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Hostility, Control and Cardiovascular Reactivity

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Thesis submitted for the degree of Doctor of
Philosophy (Psychology) on 31/08/2004.



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Abstract

Research evidence suggests that high hostile individuals are at greater risk of developing cardiovascular disease because they have a profile of cardiovascular stress arousal that is associated with greater cardiovascular disease risk. However, there is an emergence of studies that have reported null or negative findings. This dissertation addresses the presence of such discrepant findings in two ways (i) improving the measurement precision of Hostility assessment and (ii) investigates the role of control and state anger in moderating the Hostility-Cardiovascular stress arousal relationship. This is done through a new paradigm that re-defines actual control as a continuum of response-outcome contingencies rather than the presence/absence of coping behaviours. In addition, measurement precision was improved via including an attitudinal and a behavioural form of Hostility assessment in one study, confirmatory factor analysis of the Cook-Medley Hostility scale, and the development of an Implicit Cynicism assessment method. The results indicated that response-outcome contingency definition of actual control provides a better model fit for cardiovascular reactivity even when a discrepancy exists between the efficacy of coping behaviour and actual control. In addition, measurement error in Cynicism assessment can potentially result in null or negative findings. Non-interpersonal control (perceived or actual) do not moderate the Hostility-Arousal relationship. Between participant differences in non-interpersonal induced state anger moderate the Cynicism-Reactivity effect. High cynical participants did not report greater state anger reactivity due to stress. The use of visual analogue

scale partly contributes to the detection of this effect. The results indicated that state anger, but not perceived control, moderates the Cynicism-Cardiovascular stress arousal even in the absence of social conflict.

Dedication

To my parents who are ever supportive of my decision to do this.

Acknowledgements

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CHAPTER 1

MAIN INTRODUCTION

Cardiovascular disease is a major problem for most developed countries (Rayner & Petersen, 2000; American Heart Association, 2002). About 40% of all deaths in the United Kingdom are due to coronary heart disease (Petersen, Rayner, & Peto, 2003). In the same published paper it was also reported that Scotland had higher cardiovascular disease mortality than England or Wales. Cardiovascular disease has heavy economic costs as well; in the UK, coronary heart disease is estimated to cost about £7 billion a year (Liu, Maniadakis, Gray, & Rayner, 2002). Cardiovascular disease has also been the major cause of death in the United States since 1900 with the exception of 1918. The American Heart Association also published statistics that showed that cardiovascular disease accounted for about 39% of all deaths in 2000 (American Heart Association, 2002).

Trait hostility is related to cardiovascular disease (Friedman & Booth-Kewley, 1987). In 1983, the 1954 to 1959 graduates of the University of North Carolina Medical School were given a questionnaire concerning their health status. The participants had taken the Minnesota Multiphasic Personality Inventory (MMPI) as part of their medical training when they were undergraduates. Barefoot and his colleagues (Barefoot, Dahlstrom, & Williams, 1983) found that hostility as measured by the Cook-Medley Hostility Scale (Cook & Medley, 1954), derived from the

MMPI (MMPI, Hathaway & McKinley, 1951), was a significant precursor of coronary heart disease. Physicians that were more hostile as undergraduates were more likely to develop coronary heart disease during the 25-year follow-up.

Another prospective study, the Western Collaborative Group Study (WCGS), was initiated in 1960-61. Participants were interviewed and were classified into Type A or Type B individuals. Type A Behaviour Pattern is characterised by a constant sense of time urgency, competitiveness and hostility that often includes an attitude of cynical mistrust of others. Type B individuals not only have an absence of Type A Behaviours but reflect a different coping style that 'does not often lead to impatient and fast-paced type A behaviours or to inappropriate competitive and hostile/angry responses' (Rosenman, 1993). In 1986 to 1988, 1,118 elderly men from the WCGS participated in a follow-up study and the results showed that Type A individuals with low hostility scores fared better in terms of mortality than Type A individuals with high hostility scores (Carmelli & Swan, 1996). Low hostile Type A individuals had survival rates similar to Type B individuals who were non-smokers¹. Type B individuals who were smokers had similar mortality rates as hostile Type A individuals. This study showed the importance of hostility in Type A Behaviour in predicting mortality. The WCGS also found that hostility, measured by the Cook-Medley Hostility scale, was related to a higher risk of coronary heart disease over a 20 year period for a group of 1877 men

¹ Smoking status of Type A's was not reported.

(Shekelle, Gale, Ostfeld, & Paul, 1983).

In another study, cynical mistrust (a component of hostility) was shown to predict a 2-year progression of carotid atherosclerosis for a sample of 2,682 Finnish males (Julkunen, Salonen, Kaplan, Chesney, & Salonen, 1994). Williams (Williams, 1996), in suggesting ways to prevent and treat cardiovascular disease, cited several other epidemiological studies where hostility predicted significantly higher coronary heart disease risk. Hostile individuals are more likely to suffer a myocardial infarction (Everson et al., 1997), have poorer coronary arteries (Haney et al., 1996) and coronary arterial calcification at a 10-year follow-up (Iribarren et al., 2000). Matthews and her colleagues re-coded the Structured Interview taken at the Multiple Risk Factor Intervention Trial (MRFIT) to assess Hostility. High hostile individuals have an odds ratio of 1.61 for cardiovascular disease mortality after adjusting for the traditional risk factors (Matthews, Gump, Harris, Haney, & Barefoot, 2004). King (King, 1997) suggests that there is sufficient research evidence to conclude that hostility may be a crucial element in predicting health outcomes. A meta-analysis done by Miller and his colleagues (Miller, Smith, Turner, Guijarro, & Hallet, 1996) found that the two types of hostility assessment that predicted coronary heart disease are the Structured Interview Hostility assessment and the Cook-Medley Hostility Scale.

This introduction will first begin with a review of the definition and assessment of hostility since hostility is the key variable in this dissertation. One aim of this dissertation is the improvement of

measurement precision for the one of the measures of hostility - the Cook-Medley Hostility scale. Researchers have proposed pathways to explain why high hostile individuals are at a greater risk of developing cardiovascular disease. Two of such pathways that are investigated in this dissertation will be discussed - the Reactivity Hypothesis (Manuck & Krantz, 1986) and the Cardiovascular Variability Hypothesis (Sloan, Shapiro, Bagiella, Myers, & Gorman, 1999). However, there has been an increasing number of studies that have reported null or negative findings for hostility and reactivity/variability. This could be due to two reasons: (i) imprecision of the hostility measures, and (ii) the hostility-reactivity/variability relationship is moderated by other variables. Two moderators are investigated in this dissertation - state anger and control. This chapter ends with a slight digression on the justification of a male only sample for this dissertation followed by a summary.

Hostility: Definition and Measurement

There exist different methods of measuring hostility and each assessment method provides a different name to the construct it measures. The different names may be confusing and obscure the construct of hostility. A useful method is to differentiate hostility into cognitive, affective and behavioural components. The cognitive aspect of hostility (Cynicism) has been defined as a cognitive attitude of 'enduring ill will and a negative view of others' (Houston, 1986). Other similar definitions of cynicism have also been suggested (see Eckhardt,

Norlander, & Deffenbacher, 2004). Anger is the affective component while aggression is the behavioural aspect of causing or attempting to cause harm to others (Barefoot, 1992).

Hostility has been measured in two major ways: (i) vocal stylistic (verbal behaviour) assessment during a structured interview, and (ii) self-report hostility questionnaires. In this dissertation, the Cook-Medley Hostility scale is used to measure Cynicism. In Study 2, in addition to the Cook-Medley Hostility scale, the Structured Interview derived Hostile Behaviour Index (HBI) using the Interpersonal Hostility Assessment Technique is used. These two Hostility assessments are used because a meta-analysis done by Miller et al. concluded that these two Hostility measures were significantly correlated with health (Miller et al., 1996). The two Hostility assessments tap different aspect of Hostility; the Cook-Medley Hostility scale taps the attitudinal dimension of Hostility (i.e., cynical mistrust) while the HBI taps the behavioural aspect (i.e., verbal aggression).

Interpersonal Hostility Assessment Technique (IHAT)

The Interpersonal Hostility Assessment Technique, IHAT (Haney et al., 1996) is an example of hostility assessment via vocal stylistics during a structured interview. Higher IHAT scores has been associated with a greater risk of developing coronary heart disease (Haney et al., 1996; Siegman, Townsend, Civelek, & Blumenthal, 2000) and cardiovascular disease mortality (Matthews et al., 2004). The IHAT was developed from the Structured Interview for Type A Behaviour Pattern

assessment. The IHAT was a revision of the Potential for Hostility component for the Structured Interview for Type A Behaviour to make scoring easier for the assessor. It incorporates useful characteristics of both the Hostility Facet and Component scoring systems (Barefoot & Lipkus, 1994). Like the Component scoring system, the IHAT also separates the interview into smaller units for easier scoring rather than have the assessor recall and rate the entire session. Similar to the Hostility Facet scoring system, the IHAT distinguishes between hostile content and stylistics. More focus is given to vocal stylistics because hostile vocal stylistics has been found to be predictive of coronary heart disease incidence (Dembroski, MacDougall, Costa, & Grandits, 1989; Dembroski, MacDougall, Williams, Haney, & Blumenthal, 1985).

The IHAT has been shown to have construct validity with a sample of 108 African-American and Caucasian volunteers (Brummett et al., 1998). The IHAT did not correlate with self-report measures such as the Cook-Medley Hostility scale ($r = .06$), but was significantly associated with hostile facial affect (facial expressions of anger and disgust). Some self-report subscales (e.g., *Anger-Out* subscale of the STAXI) were also associated with hostile facial affect. However, unlike self-report measures, IHAT scores were not associated with happy facial affect. Since IHAT scores are only associated with hostile facial affect and have no association with the self-report hostility/anger measures, the researchers interpreted the IHAT as tapping a distinct aspect of the multidimensional construct of trait hostility. This also confirms an earlier study that reported significant associations between facial expression of

disgust and Structured Interview-derived Potential for Hostility (Chesney, Ekman, Black, & Hecker, 1990). However, the result of this study contradicts three earlier studies. One of these is a study by Rääkkönen et al. who reported a significant correlation of .38 between the Potential for Hostility and the Cook-Medley Hostility scale (Rääkkönen, Matthews, Flory, & Owens, 1999). Davidson and Hall also showed that the structured interview-derived Potential for Hostility was significantly related to the Cook-Medley Hostility scale for males ($r = .57$; Davidson & Hall, 1995). Another was Barefoot et al.'s study that showed that the IHAT correlated with the 27-item Cook-Medley Hostility scale at $r = .24$ (Barefoot, Beckham, Haney, Siegler, & Lipkus, 1993). Barefoot et al. (1993) observed that the correlation between the Structured Interview-derived Hostility and questionnaire-derived Cook-Medley Hostility tend to fluctuate from sample to sample. The inconsistent results obtained from these studies with regards to the relationship between the Structured Interview derived Hostility and the Cook-Medley Hostility scale may be due to at least two factors. Firstly, these studies differ in the gender of their samples. Brummet et al. (1998), Rääkkönen et al. (1999) and Barefoot et al. (1993) reported results based on a sample that combined both males and females while Davidson and Hall (1995) reported results by gender. There is evidence to support the contention that there are gender effects for both the Structured Interview and the Cook-Medley Hostility scale. This point is explored in greater depth in a separate section at the end of this chapter to avoid a digression here. Secondly, the fluctuation in correlations may be partly due to the measurement error

in both Hostility assessments. The measurement problems of both types of Hostility assessment will be discussed later. This dissertation focuses on improving the measurement precision of the Cook-Medley Hostility scale.

There are only two known studies that have investigated the test-retest reliability of the Structured Interview method of Hostility assessment. Blumenthal et al. administered the Structured Interview to 60 middle-aged men twice with a 4-month interval between the two interviews. The results showed that the Potential for Hostility successfully discriminated Type A's from Type B's after 4 months (Blumenthal, O'Toole, & Haney, 1984). The researchers at Duke University also reported some results for the test-retest reliability for the IHAT. Brummett et al. administered the Structured Interview to 27 male and female undergraduates and this was repeated 6 weeks later (Brummett, Maynard, Haney, Siegler, & Barefoot, 2000). Intraclass correlation coefficient for IHAT was .72. This study also found that the IHAT scores derived from the Structured Interview administered via telephone at the laboratory was correlated with the face-to-face version at .83. However, the test-retest reliability between the face-to-face administered interview and the telephone administered interview when the participant was at home was .31. This shows that the interview is less temporally stable when the mode of the interview was made changed.

This review on the various aspects of the Structured Interview method of hostility assessment reveals that not many studies have used this form of hostility assessment. This is because the Structured Interview method is laborious, requires training and more human resources than

administering a questionnaire. For the IHAT, an assessor is adequately trained if he/she obtains an inter-rater reliability of at least .80 in terms of HBI scores with one of the original formulators of this assessment technique (e.g., Dr. Thomas Haney). The interviewer also required some training in order to practise standardising the interviewing process across the participants he/she will be interviewing. In addition, to assess a participant's hostile behaviours using this technique, it would require at least 2 persons - one assessor and one interviewer. If there was a separate experimental session (as in this Study 2 of this dissertation) then it would require three persons - the interviewer, the assessor and the experimenter. This is to prevent prior exposure to the participant influencing the assessment or experimentation process. Other factors that are also important include standardising the gender between the interviewer, interviewee and assessor. Davidson et al. also reported results that indicated that female assessors tend to give lower ratings for the Potential of Hostility than male raters (Davidson et al., 1996). In this dissertation, the Structured Interview is assessed by one of the chief formulators of the IHAT - Dr. Thomas Haney.

Despite attempts to standardise and train interviewers, there remain many factors in social interaction that cannot be eliminated or controlled. In other words, Structured Interviews can never be fully standardised while questionnaires can be administered in a standardised procedure. Differences in the social interaction between the interviewer and interviewee from study to study can contribute to discrepant findings. The Western Collaborative Group Study (WCGS) is one of the often cited

evidence for Structured Interview-derived Type A predicting coronary heart disease (Rosenman et al., 1975). The Multiple Risk Factor Intervention Trial (MRFIT) was the first large scale epidemiological study to show that the Structured Interview-derived Type A/B behaviour ratings did not significantly predict coronary heart disease (Shekelle et al., 1985). Scherwitz sampled a portion of the recorded interviews from the WCGS and MRFIT and found significant differences in the interviewer behaviours between these two studies (Scherwitz, 1988). Scherwitz's analysis found that interviews in the WCGS were on average 4.9 minutes longer. Interviewers also allowed longer pauses between questions and made more polite follow-up questions during the interview. On the other hand, interviewers in the MRFIT study tended to interrupt the interviewees during the key questions more often. These differences were found despite the interviewers in both studies being personally trained by one of the chief formulators of the Type A Behaviour Structured Interview, Dr. Rosenman. Though Scherwitz's analysis is on the Structured Interview assessment of Type A Behaviour, his analysis highlights the kind of factors affecting the standardisation of the Structured Interview administration that cannot be fully anticipated.

These problems are inherent with the Structured Interview method of Hostility assessment and while a researcher can minimise the influence of such factors via training, such problems cannot be eliminated. Computerising the scoring procedure of the IHAT is also hampered by the fact that context plays a role in the identification of the

Irritation component of the IHAT². For example, an interviewee exhibiting a vocal stylistic signalling irritation during the interview in reported speech is discounted (e.g., “And he said to me: ‘Get off the road!!!”). The inclusion of this method of Hostility assessment in Study 2 with the Cook-Medley Hostility scale (discussed in the next section) is to have a more comprehensive assessment of hostility. In addition, the number of studies that have used this method of Hostility assessment is fewer than the questionnaire-format Hostility assessment. Hence, there is a need for more research into Hostility and health using this form of Hostility assessment.

Cook-Medley Hostility Scale

To date, there are at least three main self-report measures of Hostility. These are: the Cook and Medley (1954) Hostility Scale, the Buss-Durkee Hostility Index (BDHI: Buss & Durkee, 1957) and the *Angry Hostility* subscale of the revised NEO PI (NEO PI-R) (Costa, Jr. & McCrae, 1992). Since a meta-analysis done by Miller et al. (Miller et al., 1996) show that the Cook-Medley Hostility Scale significantly predicted cardiovascular disease, the current research focuses on this self-report measure of hostility. A lot of the studies cited earlier that have shown a relationship between hostility and cardiovascular disease have used this form of hostility assessment (e.g., Barefoot et al., 1983; Julkunen et al., 1994;

² The author of this dissertation is also trained in the IHAT administration and scoring procedures.

Iribarren et al., 2000). Thus, one of the focuses of this dissertation is on improving the measurement precision of this scale. By improving the measurement precision of this scale, power to detect reliable significant results for this scale increases. Specifically, two measurement aspects of the Cook-Medley Hostility scale are investigated: (i) the internal psychometric structure of this scale, and (ii) improving the measurement precision (true positives and true negatives) of this scale.

The 50-item Cook-Medley Hostility scale was derived to distinguish good and bad teachers. Walter Cook, Donald Medley and 5 other clinical psychologists derived items from the MMPI to tap Hostility and Pharisaiic-Virtue. The result is the 50-item Cook-Medley Hostility scale and 50-item Pharisaiic-Virtue scale. The Hostility scale measures the extend to which the respondent 'sees people as dishonest, unsocial, immoral, ugly, and mean, and believes they should be made to suffer for their sins' (Cook & Medley, 1954). Convergent construct validity reported for this scale agrees with this interpretation of what it measures. The Cook-Medley Hostility scale correlated with other scales measuring Ideas of Persecution, trait anger, NEO PI-R's *Angry Hostility* scale, and MMPI's Cynicism factor suggesting that the Cook-Medley Hostility measures a cynical mistrust of others (Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989; Bishop & Quah, 1998; Costa, Zonderman, McCrae, & Williams, 1986; Greenglass & Julkunen, 1989). In addition, test-retest for this scale has been reported to be around .80 while Cronbach alpha, indicating internal consistency, has also been reported to be around .80

for this scale (Bishop & Quah, 1998; Greenglass & Julkunen, 1989; Smith & Frohm, 1985).

However, the Cook-Medley Hostility scale is beset by problems as well. Though there is evidence for convergent construct validity, evidence suggests that the discriminant construct validity of this scale is poor. This is because the Cook-Medley Hostility correlates with other personality constructs not related to Hostility and health as well (Eckhardt et al., 2004). These constructs include NEO PI-R's Neuroticism, Negative Affectivity and Social Desirability (Barefoot et al., 1989; Costa et al., 1986; Greenglass & Julkunen, 1989; Jorgensen et al., 2001). Of which significant correlations with Neuroticism and Negative Affectivity present the most problem for the discriminant construct validity for the Cook-Medley Hostility scale. Both NEO PI-R's Neuroticism factor and Negative Affectivity measure a broader construct than hostility, which includes the propensity to experience anger, anxiety and depression in addition to having irrational thoughts, and emotional maladjustment (Costa, Jr. & McCrae, 1992). In other words, the Cook-Medley Hostility scale not only measures the cynical mistrust of others, and the propensity to anger and aggression but also a person's affinity towards having irrational thoughts and experiencing anxiety and depression. This is in line with two studies that have found that high Hostile participants, assessed using this scale, tend to exhibit hypochondria (e.g., report more illness symptoms) in the absence of significant actual health symptoms (Bishop & Quah, 1998; Ramsay, McDermott, & Bray, 2001).

The poor discriminant validity of this scale is confounded by a lack of empirical evidence to support a reliable internal psychometric structure for this scale. If the internal structure of the scale is stable, then a researcher is able to remove or choose items to improve the psychometric assessment precision using this scale. The current evidence suggests that this scale measures Cynicism as well as other related constructs. Exactly which questionnaire items of this scale measures what other related constructs remain empirically uncertain, though some researchers have proposed solutions. The Cook-Medley Hostility scale's problem of internal consistency is discussed next.

Since the seminal paper published by Barefoot and his colleagues reporting the ability of this scale to predict mortality (Barefoot et al., 1983), there has been renewed interest in this scale. It is clear that not all of the 50 items in the scale measure the same construct. Various studies that performed an exploratory factor analysis on this scale found that usually more than 2 factors are extracted (Costa et al., 1986; Greenglass & Julkunen, 1989; Han, Weed, Calhoun, & Butcher, 1995; Steinberg & Jorgensen, 1996)³. Therefore, it seems that the Cook and Medley Hostility scale has (i) more than one subscale and (ii) the subscales are unstable and unreliable - this is evident from these factor analytic studies showing that the items do not load onto the same factors consistently (see review in the introduction section of Chapter 2). These two points

³ At this point, the reader may realise that good internal consistency, indexed by the Cronbach alpha has been reported by some studies as reviewed earlier. However, a high Cronbach alpha does not necessarily imply good internal consistency (Shevlin, Miles, Davies, & Walker, 2000). This point is discussed in detail in the discussion section of Chapter 2.

make the measurement of Cynicism using the Cook-Medley Hostility scale imprecise because the score (obtained via the summation of the responses to all the 50 items) becomes an ambiguous interpretation of the Cynicism construct (Steinberg & Jorgensen, 1996). Two participants can have a similar score that is contributed by different scores from different subscales. This ambiguity can be reduced via selecting a particular subscale instead of using the whole 50-item scale.

Researchers frequently use subsets of the 50 items to represent this scale even when there is a lack of empirical evidence supporting the stability of using such subsets of items in the whole scale. For example, Barefoot and his colleagues found that three subscales of 27 items as defined by them were predictive of mortality for 255 physicians (Barefoot et al., 1989). In contrast, Greenglass and Julkunen used 9 items from the scale based on the items that consistently clustered together in their exploratory factor analyses (Greenglass & Julkunen, 1989). Are the researchers using comparable subsets of the scale? In other words, do the items used by these researchers measure the same construct? If all the 50 items measure the same construct then using a random selection of the 50 items will not create much problems. However, since exploratory factor analysis showed that this scale measures more than one factor, it is important to establish which subscales are reliably replicated and which items load on what subscales. This needs to be done with heterogeneous gender samples since the psychometric structure of this scale appears to be dependent on gender as well (Steinberg & Jorgensen, 1996). Hence, Study 1 in Chapter 2 aims to

provide a critical review of the known existing research on the factor structure of the Cook-Medley Hostility Scale. This review process will derive a psychometric structure of this scale that is more reliable and generalisable since it is replicated in the studies reviewed. A confirmatory factor analysis is then done on a male sample to ascertain the psychometric structure of this newly derived version of the Cook-Medley Hostility scale.

The review and confirmatory factor analysis attempts to resolve the issue of internal consistency of the scale. However, it does not address another measurement problem of this scale - the problem of false negatives and false positives. There are problems with the use of self-report questionnaires, particularly for socially undesirable traits like hostility. One problem include the possibility of response bias due to the respondent's desire to present himself or herself in a socially positive manner (Barefoot & Lipkus, 1994; Barefoot, 1992). A hostile individual may provide low ratings on a hostility questionnaire due to such reasons. These individuals are referred to as repressed hostile individuals (Helmets et al., 1995). As this section focuses on the attitudinal aspect of Hostility (Cynicism), 'Repressed Cynicism' will be used instead to avoid confusion. Repressed Cynicism represents a measurement error (false negative) for the Cook-Medley Hostility scale and is particularly problematic when assessing socially undesirable traits like Cynicism as it erodes measurement acuity. In addition, if Repressed Cynicism is considered a form of high Cynicism then it is likely that repressed cynical individuals will distort the results in a sample to produce null or negative

findings. At present, there do not exist a satisfactory method to detect and reduce the influence of repressed hostile individuals within a sample. The following paragraphs discuss the current methods and suggest a new method of assessing an implicit form of Cynicism.

One solution to this problem is to use another method of Hostility assessment that is less susceptible to impression management. The Structured Interview derived HBI is also a potential candidate to identify Repressed Hostility within a sample because it is regarded as less susceptible to social desirable responding than the Cook-Medley Hostility scale. However, there are two disadvantages of using the Structured Interview method. Firstly, it is labour-intensive, which limits its usage as an alternative form of hostility assessment. Secondly, it does not correlate with the Cook-Medley Hostility scale consistently. This is because the Structured Interview seems to measure the behavioral aspect of hostility (Aggression) rather than the cognitive aspect (Cynicism). Therefore, there is a need for an alternative form of Cynicism assessment that is (i) less labour-intensive than the structured interview, (ii) overcomes the issue of positive social presentation bias in self-report questionnaires, and (iii) measures Cynicism - the cognitive aspect of hostility.

Another solution to the problem of false negatives in Cynicism assessment would be to detect and correct for such cases using another questionnaire that measures the extent the individual is socially desirable. The current method has been to use Marlowe-Crowne Social Desirability Scale (Crowne & Marlowe, 1960) to check for such responses biases.

The main problem with this operational definition of Repressed Cynicism is that those who score low on the Cook-Medley Hostility scale and high on the Social Desirability may not be repressed cynical individuals. Construct validity for Repressed Cynicism as operationalised using the Cook-Medley/Marlowe-Crowne method confounds (i) individuals who have high trait Cynicism but are responding in a social desirable way thereby producing low Cynicism scores, and (ii) individuals that have low trait Cynicism but are also socially desirable. This distinction is important because only individuals belonging to group (i) are high cynical (but repressed) while individuals in group (ii) are low cynical and socially desirable.

Since the Cook-Medley/Marlowe-Crowne method of identifying false negatives (Repressed Cynicism) in the Cook-Medley Hostility scale is problematic, there is a need to devise another method. The other method proposed here is the development and use of an implicit measurement of Cynicism in conjunction with the Cook-Medley Hostility scale. Questionnaires are referred to as explicit measures of the construct. Since implicit measures are less susceptible to social desirability responding (Greenwald & Farnham, 2000), it can be used to help reduce the influence of Repressed Cynicism within a given sample. The Implicit Cynicism assessment is developed in this dissertation as a means to reduce/detect the influence of false positives and false negatives and not to replace the Cook-Medley Hostility scale. This is because constructs that are more susceptible to positive social presentation bias (e.g., racism, sexism), the implicit-explicit correlation

tend to be moderate (Greenwald et al., 2002; Greenwald, Nosek, & Banaji, 2003). A suitable 'replacement' of the Cook-Medley Hostility scale needs to correlate minimally at .80 with this scale. Thus, Implicit Cynicism is likely to be moderately correlated with the Cook-Medley Hostility, making the Implicit Cynicism an unsuitable candidate to replace the Cook-Medley Hostility scale. Other reasons making the Implicit Cynicism unsuitable as a replacement for the Cook-Medley Hostility scale are discussed in detail in the discussion section of Study 3 in Chapter 4. Details of the Implicit Association Test, and how it is used to reduce the influence of false negatives and false positives in a sample are discussed in the next section.

Implicit Association Test - The Cynicism Version

The Implicit Association Test (IAT) is an emerging method of assessing implicit cognitive associations (Greenwald, McGhee, & Schwartz, 1998; Nosek & Banaji, 2001). Cognitive psychologists have shown that individuals organise different concepts in terms of schematas. A schema is defined as 'a pattern of knowledge describing what is typical or frequent in a particular situation' (Reisberg, 1997). To illustrate this, Figure 1.1 shows two examples of how the schema of 'Other' (referring to other individuals) differs between a high cynical hostile individual and a low cynical hostile individual. In Figure 1.1 single headed arrows represent positive associations with thicker arrows indicating a stronger association between the two concepts. Therefore, a high cynical hostile individual (Figure 1.1a) will have stronger associations between 'Other'

and 'deceitful'. That is, they hold an implicit theory that other individuals are deceitful, which is congruent with the concept of cynical hostility.

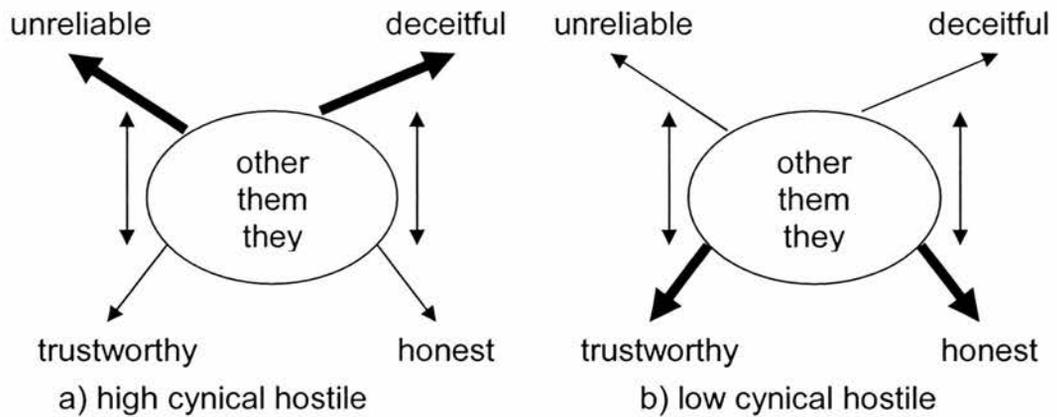


Figure 1.1 Schematic Representation of 'Others' for (a) high and (b) low Cynical Hostile Individuals (see explanation in text).

Compared with a low hostile individual (Figure 1.1.b), these individuals tend to have stronger association between 'Others' and 'Honest (i.e., they hold implicit theories about other individuals being honest). Concepts like 'deceitful' and 'honest' are semantically orthogonal. A stronger association between 'Others' and 'deceitful' will tend to inhibit the association between 'Others' and 'honest' and vice versa. This bi-directional inhibition is represented by double-headed arrows in Figure 1.1.

In order to tap these implicit cognitive associations, researchers use the psychological effect known as priming. The priming effect occurs when prior exposure enhances the processing of related concepts. For a high cynical hostile individual, paying attention to words relating to 'Other' (e.g., other, they, them, their) activates this concept and primes related

adjectives of cynical hostile trait (i.e., deceitful). By comparison, a low cynical hostile may not have such priming effects or may instead prime benign adjectives (e.g., honest). Priming is frequently assessed via reaction time to the stimuli. A concept is primed when a person's reaction time to it is faster. Using the participant's reaction to the stimuli, researchers are able to assess the strength of the association between the various concepts within an individual. In the example for cynical hostility, the reaction time to words relating to 'Other' and 'Hostile' adjectives in one trial is compared to their reaction time to words relating to 'Other' and 'Benign' in another trial for the same individual. The difference in reaction times will indicate the relative strength of the 'hostile' versus 'benign' attribute associations with regards to 'others' within an individual. High cynical hostile individuals will have faster reaction times to the 'Other' and 'Hostile' pairing compared to the 'Other' and 'Benign' pairing. This means that for high cynical hostile individuals, activating the 'Other' concept primes 'Hostile' attributes more than 'Benign' attributes. There is evidence to suggest that such associations as illustrated in Figure 1.1 exist among high and low hostile individuals. In one study, 77 male undergraduates have either a hostile or a neutral social interaction with a confederate. Individuals who scored high on the Cook-Medley Hostility scale were significantly more likely to recall hostile trait adjectives of their confederates after a hostile interaction. This shows that people who score high in cynical mistrust have stronger "hostile-other" schema (Allred & Smith, 1991).

There have been a few studies that have shown the validity of such implicit association method of assessment in other subject areas. For example, participants were faster to respond to word pairings of nouns of insects and unpleasant adjectives compared to a pairing of insects and pleasant adjectives. Participants were also more likely to respond faster to flowers and pleasant adjective pairings as well (Greenwald et al., 1998). In another study, Greenwald and Farnham also demonstrated that such implicit association tests can tap self-esteem and that implicit self-esteem assessment had a lower correlation with social desirable responding compared to the explicit self-esteem measures (Greenwald & Farnham, 2000). Implicit association has also been studied for race evaluation and a recent study showed that such implicit associations were associated with amygdala activation (a subcortical structure associated with emotional learning and evaluation) while explicit measures were not (Phelps et al., 2000). The Go/No-go Association Task (GNAT) is a variation of the implicit association task (Nosek & Banaji, 2001). Study 3 of this dissertation attempts to conceptualize and develop a GNAT version for Cynicism. The reasons for why GNAT is used instead of Greenwald's IAT version will be discussed in the introduction to Study 3 (Chapter 4). Both GNAT and IAT work on the same principles discussed here.

Such implicit association tests can help overcome problems raised earlier about Repressed Cynicism in the Cook-Medley Hostility scale because (i) it is fast and easy to apply, and (ii) the fast response required for such tests reduces the influence of positive social presentation

response bias. Using both implicit (GNAT Cynicism) and explicit Cynicism (Cook-Medley Cynicism), the different forms of Cynicism can be identified

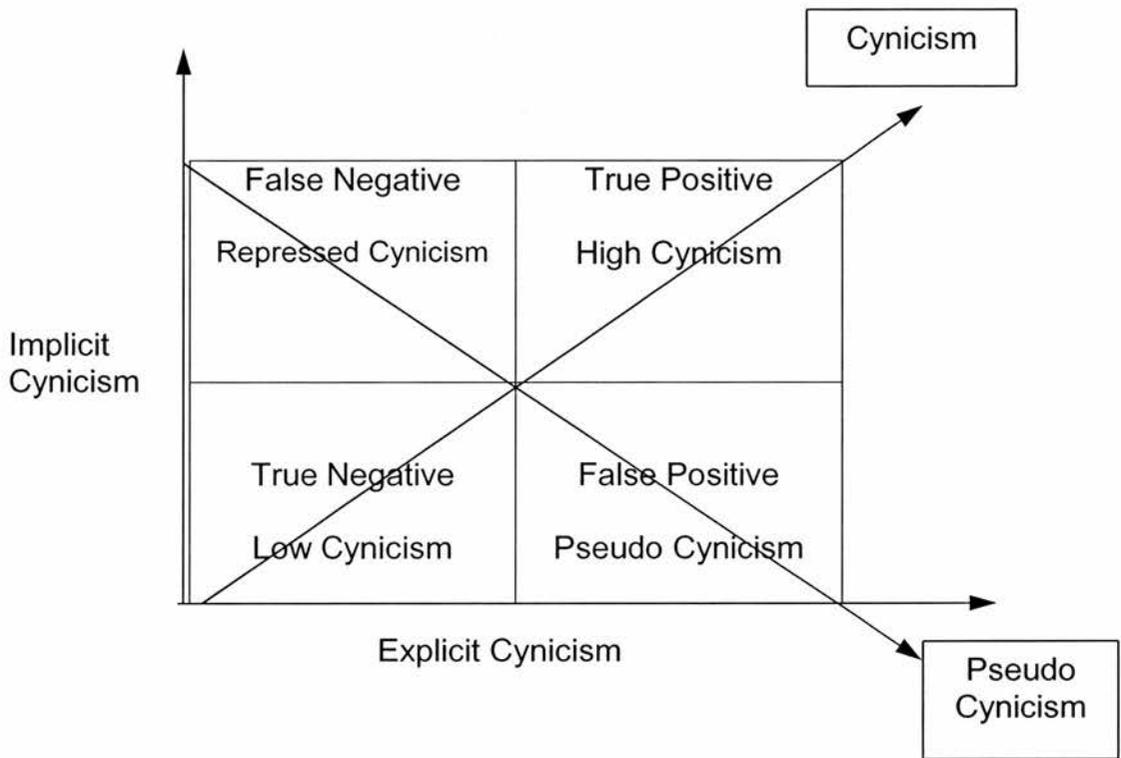


Figure 1.2 Different Forms of Cynicism identified using Explicit and Implicit Cynicism

(figure 1.2). If valid, the effects of false negatives and false positives can be reduced using factor analysis between these two forms of Cynicism assessments. Results obtained with the different forms of Cynicism assessments (explicit, implicit and factor analytic Cynicism) can provide indications of which effects are to Cynicism and which effects are due to false negative and false positives. High Cynicism scores obtained from factor analysis represents an unambiguous index of Cynicism. That is,

high Cynicism factor scores represent high Implicit and Explicit Cynicism while low Cynicism factor scores represent the reverse.

Hostility-Cardiovascular Disease Pathways

The previous sections discussed the measurement problems of Aggression and Cynicism assessment for Hostility. However, interest in assessment of these two constructs was triggered by their ability to predict cardiovascular disease risk and mortality. Two theories are proposed to mediate the Hostility-Cardiovascular Disease relationship - the Cardiovascular Reactivity Hypothesis and the Cardiovascular Variability Hypothesis. The following sections discuss the patterns of cardiovascular reactivity and variability that are associated with cardiovascular disease risk. In this dissertation such patterns of cardiovascular reactivity and variability are taken to indicate a higher risk of developing cardiovascular disease.

The Cardiovascular Reactivity Hypothesis

One of the hypothesised pathways that is used to explain why hostility is associated with an increased risk of cardiovascular disease is the Reactivity Hypothesis (Manuck & Krantz, 1986). Researchers of the Reactivity Hypothesis propose that high hostile individuals are at a greater risk of cardiovascular disease because they have greater physiological reaction to stress. There is some evidence to show that a more reactive cardiovascular response to stress is associated with an

increased risk of developing cardiovascular disease (Light, Sherwood, & Turner, 1992). The most common dependent variables used in testing the Reactivity Hypothesis have been blood pressure and heart rate.

However, mean arterial blood pressure is regulated by two main components: cardiac output (the amount of blood the heart pumps per minute) and peripheral resistance (the amount of resistance encountered by the blood to circulate through the cardiovascular system), the same blood pressure increase can have very different underlying hemodynamic profile (Shapiro et al., 1996b; Sherwood & Turner, 1992). Hence the review will also include cardiac output and peripheral resistance and their relationship with cardiovascular disease risk.

The aim of this review is to present some of the evidence for the Reactivity Hypothesis and also to present the patterns of reactivity (particularly for Cardiac Output and Total Peripheral Resistance) that are associated with cardiovascular disease. This will help in understanding the health implications of the reactivity patterns of high hostile individuals when the literature for Hostility-Cardiovascular Reactivity is reviewed as well as interpreting the clinical implications from the two experimental studies (Chapter 3 and 4) in this dissertation.

Blood Pressure Reactivity and Cardiovascular Disease. In 1988 to 1989, a sample of 51 men was taken from an initial pool of 204 that had participated in studies held from 1974 to 1978. Blood pressure and heart rate reactivity predicted resting diastolic blood pressure during follow-up independent of the standard risk factors (Light, Dolan, Davis, &

Sherwood, 1992). More recent evidence from prospective studies also points to this link between heightened blood pressure reactivity and later blood pressure status (Armario et al., 2003; Carroll, Ring, Hunt, Ford, & MacIntyre, 2003; Everson, Kaplan, Goldberg, & Salonen, 1996; Knox, Hausdorff, & Markovitz, 2002; Matthews, Salomon, Brady, & Allen, 2003). Other studies have also reported similar results between blood pressure reactivity and hypertension risk (Light et al., 1999; Steptoe & Cropley, 2000). However, null findings have also been reported (Carroll, Smith, Sheffield, Shipley, & Marmot, 1997). Generally there is evidence to indicate that heightened blood pressure reactivity is predictive of future blood pressure status.

There are fewer prospective studies that have investigated the relationship between blood pressure reactivity and coronary heart disease compared to those that have investigated blood pressure reactivity and future baseline blood pressure. Increased blood pressure reactivity has also been found to predict structural changes of the heart indicating target organ damage. A recent study found that pulse pressure responses to psychological tasks were predictive of left ventricular mass at a 10 year follow-up (Jokiniitty, Tuomisto, Majahalme, Kähönen, & Turjanmaa, 2003). The Kuopio Ischemic Heart Disease Study found that a hyper-reactive cardiovascular response is associated with increased coronary heart disease risk in a cross-sectional design for 2682 Finns (Kamarck et al., 1998).

Cardiac Output, Total Peripheral Resistance Reactivity and Cardiovascular Disease. Research has shown that peripheral resistance reactivity is frequently found among populations at risk of developing cardiovascular disease. African Americans, more likely to develop hypertension, have a more reactive sympathetic vascular stress response (Stein, Lang, Singh, He, & Wood, 2000; Llabre, Klein, Saab, McCalla, & Schneiderman, 1998). Individuals with a family history of hypertension also tend to have increased peripheral resistance reactivity during psychological stress (Lovallo & Al'Absi, 1998; Marrero, Al'Absi, Pincomb, & Lovallo, 1997). In addition, peripheral resistance reactivity has also been found to be predictive of hypertension. The Cold Pressor task is predictive of essential hypertension (Menkes et al., 1989; Kasagi, Akahoshi, & Shimaoka, 1995). Blood pressure reactivity to passive coping tasks (i.e., tasks where the participant had no way of changing its outcome) like the Cold Pressor (i.e., putting a hand/foot into ice-cold water) are found to be primarily vascular-mediated (Sherwood, Dolan, & Light, 1990b; Dishman, Jackson, & Nakamura, 2002). Girdler et.al found that the Cold Pressor was the most significant predictor of systolic blood pressure over 2 years when compared to active coping tasks (i.e., tasks where the participant could influence the outcome ;Girdler et al., 1996). Cardiac Output reactivity has also been associated with hypertension. It is proposed that enhanced cardiac output reactivity increases arterial pressure leading to a reactive increase in total peripheral resistance (Julius, 1992; Julius & Nesbitt, 1996). Cardiac output reactivity has been reported to predict systolic blood pressure reactivity in a 3-year follow-up

period for a group of 149 adolescents (Matthews, Salomon, Kenyon, & Allen, 2002). People with a family history of hypertension have also been found to have greater cardiac output reactivity than those without a family history of hypertension during a mathematical task (Schneider, Jacobs, Gevirtz, & O'Connor, 2003). However, the association between cardiac output reactivity and basal blood pressure has not been consistent (e.g., Lovallo & Al'Absi, 1998). Both cardiac output and vascular resistance appear to be elevated for hypertensives as well (Julius & Majahalme, 2000).

There is some evidence linking cardiac and peripheral resistance reactivity to coronary heart disease although all of the known studies tend to have clinical samples or are cross-sectional in nature. For instance, individuals with heart disease have been found to have increased cardiac output (Sundin, Ohman, Palm, & Strom, 1995). There is also some research evidence linking cardiac output reactivity and atherosclerosis of the cardiac and carotid arteries (Sharpley, 1998). On the other hand, the studies that have investigated the relationship between Total Peripheral Resistance reactivity and heart disease have sampled clinical populations. Some researchers found that heart disease patients have greater vascular stress reactivity (Nolan, Wieglosz, Biro, & Wielgosz, 1994) while other researches have reported attenuated vasodilation responses during exercise (Welsch, Alomari, Parish, Wood, & Kalb, 2002). Attenuated vasodilatory reactivity is also associated with a higher risk of a myocardial infarction (Castelli, 1998). Coronary heart patients on

lipid lowering therapy also resulted in more endothelium vasodilatory reactivity (Cohen et al., 2000; de Divitiis & Rubba, 1999).

To summarise, in terms of cardiac output and peripheral resistance reactivity during stress and hypertension, the research evidence is mixed for cardiac output reactivity and hypertension. There are more studies indicating that greater vascular resistance reactivity is related to hypertension risk. Some studies have shown associations between cardiac output and total peripheral resistance reactivity and heart disease. However, these studies are cross-sectional and done with mainly with clinical samples. Vascular reactivity, attenuated vasodilatory response and greater cardiac output reactivity have been found to be associated with coronary heart disease.

Heart Rate Reactivity and Cardiovascular Disease. Heart rate reactivity is related to coronary heart disease for nonhuman primates. Experimental studies have shown that heart rate reactivity is related to cardiac atherosclerosis in primates (Bassiouny et al., 2002; Kaplan, Manuck, & Clarkson, 1987; Manuck, Kaplan, & Clarkson, 1983; Manuck, Kaplan, Adams, & Clarkson, 1988; Manuck, Adams, McCaffery, & Kaplan, 1997). However, these findings have yet to be generalised to a human population though heart rate variability rather than reactivity appears to be a closer link to cardiovascular disease and mortality (e.g., Imai et al., 2003; Sharpley, 1998). The literature on heart rate variability and cardiovascular disease will also be reviewed later.

Studies have shown that blood pressure stress reactivity is related to future resting blood pressure. Vascular-mediated blood pressure reactivity and vascular (Total Peripheral Resistance) reactivity have been found to be associated with hypertension. Some studies (mainly with heart disease patients) suggest that Cardiac Output reactivity and attenuated vasodilation is associated with heart disease. For heart rate, increased heart rate and atherosclerosis have been found in experimental studies involving nonhuman primates. However, there are yet no known studies that have extended these findings to humans.

The Cardiovascular Variability Hypothesis

The cardiovascular system 'seeks' to attain homeostasis (Julius, 1992). Large variation in blood pressure increases risk of endothelium damage and hastens the process of atherosclerosis, and can increase the likelihood of atherosclerotic plaque rupture causing stroke and myocardial infarction (Lee, Grodzinsky, Frank, Kamm, & Schoen, 1991). High hostile individuals may be at greater risk of developing cardiovascular disease because they have greater variation in blood pressure during stress (Sloan et al., 1999). In this study, heart rate variability is assessed via spectral analysis. In addition, baroreflex sensitivity, a physiological mechanism involved in the regulation of blood pressure and heart rate variability, is measured by spectral analysis and the sequence method. The following paragraphs review some of the research evidence for the Cardiovascular Variability Hypothesis.

Blood Pressure/Heart Rate Variability and Cardiovascular Disease. There is research evidence to support the Cardiovascular Variability Hypothesis - cardiovascular variability is related to cardiovascular disease (Mancia & Parati, 2003). Blood pressure variability was found to be a predictor of left ventricular mass for a group of hypertensive participants over a 7-year period (Frattola, Parati, Cuspidi, Albin, & Mancia, 1993), cardiovascular morbidity over a 5-year period (Pickering & James, 1994) and atherosclerosis of the common carotid artery in a 3-year follow-up (Sander, Kukla, Klingelhofer, Winbeck, & Conrad, 2000).

A number of cross-sectional studies have reported similar findings between blood pressure variability and cardiovascular disease. Daytime systolic blood pressure variability was also found to be related to severity of target organ damage (Palatini et al., 1992), cardiovascular morbidity (Pringle et al., 2003), and the thickening of the carotid arteries among 1663 hypertensives (Mancia et al., 2001).

Heart rate variability is also related to cardiovascular mortality. However, the evidence suggest that for heart rate, lower rather than higher variability is related to cardiovascular disease and mortality. Increased heart rate variability buffers blood pressure variability (Sloan et al., 1999; Toska & Eriksen, 1993). Therefore, increased blood pressure variability but decreased heart rate variability should be related to cardiovascular disease risk and mortality. The Atherosclerosis Risk in Communities (ARIC) Study found that among 7099 normotensives, those with lower heart rate variability were significantly more likely to develop hypertension over a 9-year period (Schroeder et al., 2003). Another large

scale study, the Stockholm Female Cohort Risk Study, also found that women with reduced heart rate variability who were hospitalised after a cardiac event were at a significantly higher risk of mortality (Janszky et al., 2004). There are other studies that have also reported similar results (Aronson, Mittleman, & Burger, 2004; Huikuri et al., 1999; Imai et al., 2003; Singh et al., 1998).

These studies suggest that increased blood pressure variability and reduced heart rate variability is related to cardiovascular morbidity and mortality. In Study 2 of this dissertation, two indices of cardiovascular variability were measured: heart rate variation via spectral analysis and baroreflex sensitivity. Using spectral analysis, the two branches of the autonomic nervous system regulation of heart rate can be distinguished (Berntson et al., 1997; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). The following paragraphs list some of the research evidence illustrating increased parasympathetic cardiac activity, decreased sympathetic cardiac activity, and decreased baroreflex sensitivity are associated with an increased risk of cardiovascular disease.

Autonomic Nervous System, Baroreflex and Cardiovascular Disease. As was discussed for cardiovascular reactivity, assessing the underlying hemodynamic regulation of blood pressure (i.e., cardiac output and total peripheral resistance) enables one to understand better individual differences in the regulation of blood pressure under different conditions. Similarly for cardiovascular variability, an assessment of some of its

underlying regulatory processes will also enable a better understanding of individual differences in cardiovascular reactivity and variability. The cardiovascular system is regulated by a complex interaction of hemodynamic, humoral and electrophysiological factors and both heart rate variability and baroreceptor response have been found to be associated with disease and stress (Lanfranchi & Somers, 2002).

Basal heart rate and heart rate variability is regulated mainly by the parasympathetic system (Katona, Mclean, Dighton, & Guz, 1982; Lin et al., 2000). The role of the sympathetic nervous system and the risk of developing cardiovascular disease have been well documented. Research has shown how an increased sympathetic activation is related to the development of cardiovascular disease (Gilmour, 2001; Julius, 1998; Julius & Majahalme, 2000; Malliani & Montano, 2002). The function of the parasympathetic is often described as a conservation of energy. The effects include the slowing of heart rate and the increase of digestive functions. Studies have shown how an reduced parasympathetic activity in normal healthy individuals is associated with a higher risk of cardiovascular disease (Airaksinen, Ikaheimo, Linnaluoto, Niemela, & Takkunen, 1984; Liao et al., 1997) and sudden cardiac death (Singer et al., 1987). Individuals with hypertension have been found to have this profile of autonomic nerve activity (increased sympathetic and reduced parasympathetic cardiac activity) during psychological stress compared to normotensives (Langewitz, Rüdell, & Schächinger, 1994). A profile of high sympathetic nerve activity and low parasympathetic nerve activity is associated with a poorer health outcome (Vanoli & Schwartz, 1990).

Baroreceptors also play a role in the regulation of heart rate and blood pressure variability, which may reduce the risk of developing cardiovascular disease. Baroreceptors are mechanoreceptors located mainly at the carotid sinus and the aortic arch. They buffer changes in blood pressure by increasing vagal stimulation of the heart and inhibiting sympathetic activity to the vasculature. The effect of baroreceptor stimulation is that of reduced heart rate and vasodilation. The Baroreflex is responsible for buffering variation in blood pressure. Studies done with dogs and rats showed that if the Baroreceptors are surgically 'removed' (Sinoaortic Denervation), blood pressure rises by about 10mmHg but its variance significantly increase (Cowley, Liard, & Guyton, 1973; Cerutti, Barres, & Paultre, 1994; Cerutti, Ducher, Lantelme, Gustin, & Paultre, 1995; Persson, Ehmke, & Kirchheim Hartmut, 1989; Just et al., 1994). Stimulating the baroreflex increases parasympathetic activity to the heart. Increasing parasympathetic cardiac activity to the heart can help reduce blood pressure variability (Sloan et al., 1999; Toska & Eriksen, 1993). Though the baroreflex has often been regarded to be important in regulating short term blood pressure changes (Lanfranchi & Somers, 2002), recent evidence suggests that it may also have a potential important role in long term blood pressure regulation (Malpas, 2004; Sleight, 1997).

Though mostly cross-sectional, several studies have reported that people with cardiovascular disease or at a higher risk of developing cardiovascular disease have lower baroreceptor sensitivity. Reduced baroreceptor sensitivity predicts higher risk of mortality for chronic heart

failure patients (Mortara et al., 1997) and stroke patients (Robinson, Dawson, Eames, Panerai, & Potter, 2003). Reduced baroreflex sensitivity was significantly correlated with subclinical atherosclerosis for a group of 82 men and women without cardiovascular disease (Gianaros et al., 2002). Decreased baroreflex sensitivity has been found in individuals with stroke (Robinson et al., 2003), a family history of hypertension (Ditto & France, 1990), people with hypertension (Watkins, Grossman, & Sherwood, 1996), chronic heart failure patients (Rostagno et al., 1999) and diabetic patients (Lanfranchi et al., 1998; Parati, Di Renzo, & Mancia, 2001). These studies indicate that reduced baroreflex sensitivity is associated with cardiovascular disease and mortality.

Though the Cardiovascular Reactivity Hypothesis and Cardiovascular Variability Hypothesis are discussed separately, there is an overlap in terms of cardiovascular functions. For instance, baroreflex sensitivity is related to heart rate reactivity (Fauvel et al., 2000). 'Reactivity' refers to change from a baseline while 'variability' refers to deviations from the mean. In this dissertation, reactivity of cardiovascular variability (baroreflex sensitivity and spectral analysis of heart rate) are also analysed. In this dissertation, 'reactivity' is used to refer to blood pressure, heart rate, cardiac output and peripheral resistance changes from baseline while 'variability' refers to measures that index cardiovascular variability. Distinguish the two aspects of cardiovascular function enables the literature review to be done separately for each to avoid confusion as well as prevent confusing heart rate reactivity with spectral analysis of heart rate (variability).

Hostility, Cardiovascular Reactivity and Variability

Hostility and Cardiovascular Reactivity⁴

There are studies that have reported significant positive associations between Hostility and cardiovascular stress reactivity, which suggests that the link between hostility and cardiovascular disease may be mediated by cardiovascular reactivity to stress.

Structured Interview Hostility Assessment. Why et al. found that hostility, assessed via the IHAT, predicted increased systolic blood pressure reactivity for hostile Malay Singaporeans during an Anger Recall task (i.e., recalling an interpersonal angry event) (Why et al., 2003). Fredrickson et al. also used the IHAT to assess hostility and found that high hostile participants had greater blood pressure reactivity for the Anger Recall task (Fredrickson et al., 2000). Everson and her colleagues (Everson, McKey, & Lovallo, 1995) sampled 48 Caucasian males and had them do two sessions of a Mental Arithmetic task. Trait hostility was assessed via the Type A structured interview Potential for Hostility component scores and participants were categorised as high and low hostile based on a median split on the scores obtained from the Type A structured interview. During the execution of the second session of the Mental Arithmetic task, the participants were harassed (e.g., told to speak up, perform more

⁴ The review of the hostility-reactivity literature only includes studies that have used either the following to measure Hostility: Cook-Medley Hostility scale (attitude) and the Structured Interview Hostility assessment (behaviour) for a sample that includes males. This is to avoid any ambiguities in the interpretation of the results for the cited studies.

accurately). The results showed that harassment increased systolic blood pressure, diastolic blood pressure, heart rate, and rate-pressure product (heart rate x systolic blood pressure) more for high hostile participants than low hostile participants. Another study also reported greater systolic blood pressure reactivity among high hostile men to mental arithmetic and handgrip tasks (Engebretson & Matthews, 1992).

Due to the complexities of administering and scoring hostility obtained via the Structured Interview, there are fewer studies that have used this method of hostility assessment to investigate the hostility-reactivity hypothesis compared to the Cook-Medley Hostility scale. Hence there is a need to replicate these findings for the Structured Interview Hostility. Study 2 of this dissertation has included the IHAT as one form of hostility assessment. Overall the pattern of result obtained in these studies indicate that high hostile individuals are associated with increased blood pressure reactivity.

For cardiac output and total peripheral resistance reactivity, Why et al. is the only known study to have investigated the relationship between the Structured Interview Hostility (IHAT) and hemodynamic patterns of blood pressure reactivity. Why et al. found that high hostile Indians have greater cardiac output reactivity than low hostile Indians (Why et al., 2003). As reviewed earlier, there are studies reporting associations between greater cardiac output reactivity and cardiovascular disease.

Cook-Medley Hostility Scale. Smith, Cranford and Mann preselected 12 male and female undergraduates with hostility scores one standard

deviation above the mean (high hostile) and one standard deviation below the mean (low hostile). These participants performed an anagram task, a stationary bike task and a stationary bike task while watching videos of positive or negative content. An analysis of systolic blood pressure reactivity for these tasks revealed that high hostile participants had elevated systolic blood pressure reactivity throughout the tasks while low hostile participants systolic blood pressure reactivity habituate (i.e., decrease) over time (Smith, Cranford, & Mann, 2000). Suarez et al. also sampled 52 men based on their hostility scores (<15 and >23), and randomised them into either an anagram task without harassment or an anagram task with harassment (Suarez, Kuhn, Schanberg, Williams, Jr., & Zimmerman, 1998). The results indicated that high hostile participants who were harassed had greater blood pressure and heart rate reactivity than other participants. Lepore also found that high hostile participants had greater blood pressure reactivity regardless of whether they had a supportive confederate present or not, while low hostile participants had smaller blood pressure increases when a supportive confederate was present (Lepore, 1995). Christensen and Smith had 60 male undergraduates engage in either a task that involves self disclosure or another task that did not involve self disclosure (Christensen & Smith, 1993). High hostile participants had greater blood pressure reactivity than low hostile participants for the task involving self-disclosure. Suarez and Williams is one of the often cited study as evidence for the hostility-reactivity effect (Suarez & Williams, 1989). They preselected 53 participants based on Cook-Medley Hostility scores <14 (low hostile) or

>23 (high hostile) and found that verbal harassment from the experimenter increased blood pressure reactivity. Smith and Allred had participants engage in a debate; high hostile participants had greater systolic and diastolic blood pressure reactivity during that debate than low hostile participants (Smith & Allred, 1989). In another study, Hardy and Smith also had participants engage in a role-played interaction either with or without conflict. High hostile participants engaged in interpersonal interactions with conflict had greater diastolic blood pressure reactivity (Hardy & Smith, 1988). The results from these studies suggest that high hostility is associated with greater blood pressure reactivity. Fewer significant results for heart rate reactivity have been reported by these studies.

There are fewer studies that have reported the underlying hemodynamic processes for such blood pressure reactivity for high hostile individuals as measured by the Cook-Medley Hostility scale. The results from the available studies that have measured cardiac output and/or peripheral resistance do not indicate that hostility is consistently associated with a specific pattern of hemodynamic regulation for their blood pressure reactivity. Two studies reviewed in the previous paragraph reported increased forearm blood flow (decreased forearm vascular resistance) during stress for high hostile participants (Suarez & Williams, 1989; Suarez et al., 1998). Girdler et. al found that high hostile male individuals had greater total peripheral resistance during a speech and a paced mental arithmetic stressor. The relationship between hostility and total peripheral resistance reactivity was of borderline significance for the

speech preparation and STROOP tasks (Girdler, Jamner, & Shapiro, 1997). A study sampled 100 males and females and had them perform a mental arithmetic and a cold pressor task. No significant correlations were found between hostility and cardiac output or total peripheral resistance (Gregg, James, Matyas, & Thorsteinsson, 1999). In another study done by Lawler which categorised individuals as cardiac or vascular reactors based on their cardiac output and total peripheral resistance reactivity to arithmetic, video and anger recall tasks, found no significant differences for hostility between cardiac and vascular reactors (Lawler, 1996).

Hostility and Cardiovascular Variability

Structured Interview Hostility Assessment. A study that sampled 15 males used parasympathetic blockade (atropine) and sympathetic stimulation (isoproterenol) and found that the Potential for Hostility was related to a reduction of parasympathetic antagonism during sympathetic stimulation (Fukudo et al., 1992). Krantz et al. gave 12 males a β -adregnergic agonist (isoproterenol) and a β -adregnergic antagonist (propranolol) counterbalanced with two other placebo conditions (Krantz, Contrada, LaRiccia, & Anderson, 1987). Parallel forms of the Structured Interview were given after each treatment condition. The results revealed a borderline effect for treatment condition ($p = .06$). Compared to the placebo condition, β -adrenergic stimulation increased Potential for

Hostility ratings while β -adrenergic blockade decreased Potential for Hostility ratings. Another study by Krantz also compared 65 coronary patients having β -adrenergic blocking medication (propranolol) with 23 coronary patients without such medication. Patients with β -adrenergic blocking medication had significantly lower ratings for Potential for Hostility (Krantz et al., 1982). The results from these studies suggest that vocal stylistics during a Structured Interview, used to assess Hostility, is associated with increased β -adrenergic activity indicative of heightened sympathetic arousal. However, there has yet been any known published study that has investigated the relationship between the Structured Interview Hostility and baroreflex response during stress. Therefore, Study 2 includes this form of hostility assessment and assesses baroreflex sensitivity as well.

Cook-Medley Hostility Scale. Research has shown that hostility is related to an autonomic activity profile of high sympathetic and low parasympathetic activity that may increase the risk of developing cardiovascular disease. Research evidence suggests that a down-regulation of the β -adrenergic system due to over-stimulation (hyper-reactive sympathetic arousal) is present among high hostile individuals. A β -adrenergic agonist (isoproterenol) was administered to group of 123 males and females in order to assess β -adrenergic responsiveness. High hostile participants had reduced β -adrenergic responsiveness (i.e., higher doses of isoproterenol required to achieve a comparable increase in heart

rate) (Jain, Dimsdale, Roesch, & Mills, 2004). When given pharmacological β -adrenergic agonist (isoproterenol) and an α -adrenergic agonist (phenylephrine) were given to 149 men and women, high hostility was found to be associated with reduced cardiac β -adrenergic response (Hughes, Sherwood, Blumenthal, Suarez, & Hinderliter, 2003). The results obtained by researchers of another study was similar when done with 36 males using the same procedure as Huges et al. (2003), the result was that high hostile participants had decreased cardiac β -adrenergic response, while the results for vascular β -adrenergic response was not significant (Suarez, Sherwood, & Hinderliter, 1998). In another study, Suarez et al. sampled 30 male participants with hostility scores above 20 (high hostile) and below 14 (low hostile) (Suarez et al., 1997). The researchers found that high hostile individuals' had fewer β -adrenergic receptors (indexed by lower circulating mononuclear leukocytes). Beta-adrenergic receptors are activated by adrenaline, which is released during sympathetic activity. Demaree and Everhart found that hostility was positively related to sympathetic dominance and reduced parasympathetic activity in terms of sympathovagal balance (measured by spectral analysis of heart rate) during a resting baseline (Demaree & Everhart, 2004). Sloan et. al also found that the individuals who had higher scores on the Cook-Medley Hostility scale had reduced parasympathetic cardiac reactivity to mental arithmetic and the Stroop Colour Word task for men after statistically controlling for respiration (Sloan et al., 2001). Therefore, it appears that

research has found that hostility is related to an autonomic activity profile of low parasympathetic and high sympathetic activity that may increase the risk of developing cardiovascular disease.

Two studies have also reported associations between hostility and reduced baroreflex sensitivity. Hostility was found to be inversely related to baroreflex sensitivity for a group of 44 men (Graham, Zeichner, Peacock, & Dishman, 1996). In this study, baroreflex sensitivity was assessed via applying external pressure to the neck and tracing heart rate changes with relation to blood pressure changes (Eckberg, Cavanaugh, Mark, & Abboud, 1975). A study by Virtanen et al. reported that anxiety and hostility were related to reduced baroreflex sensitivity and increased blood pressure variability among 150 participants (Virtanen et al., 2003). However, this study used the 37-item Brief Symptoms Inventory to measure hostility, which is not a standard hostility measure. Studies that have reported associations between hostility and cardiovascular mortality or morbidity have used either the Cook-Medley Hostility scale or the Structured Interview assessing hostile paralinguistic elements (p.1-3). The results reported by Virtanen et al.'s (2003) study needs to be replicated with standard hostility measures. Therefore, one of the aims of Study 2 (Chapter 3) is to investigate whether hostility, assessed using the Cook-Medley Hostility scale and the Structured Interview-derived Hostile Behaviour Index, is associated with reduced baroreceptor sensitivity.

Hostility, Anger and Perceived Control

In contrast to the numerous studies that have reported significant associations between hostility and cardiovascular reactivity and autonomic nervous system activity, there is a growing body of research that have failed to find such significant results for the Structured Interview Hostility (Diamond et al., 1984; Schneider, Julius, & Karunas, 1989; Siegman & Anderson, 1990) as well as the Cook-Medley Hostility scale (Burns & Katkin, 1993; Carroll et al., 1997; Felsten, 1995; Felsten, 1996; Fichera & Andreassi, 1998; Sallis, Johnson, Trevorrow, Kaplan, & Hovell, 1987; Smith & Houston, 1987; Spoth, Dush, & Leonard, 1992; Weidner, Friend, Ficarrotto, & Mendell, 1989) in predicting cardiovascular reactivity. Though Fukudo et al. found that parasympathetic antagonism of sympathetic stimulation was attenuated for high hostility assessed by the Structured Interview, no significant results were reported for Cook-Medley Hostility scale (Fukudo et al., 1992). The evidence from the studies involving pharmacological blockade of the sympathetic and parasympathetic nervous system suggests that high hostile individuals (assessed by the Cook-Medley) will have blunted cardiac output reactivity and greater total peripheral resistance to stress (Sherwood, Hughes, Blumenthal, Suarez, & Hinderliter, 2004). However, as reviewed earlier, no such consistent pattern of hemodynamic blood pressure regulation was found for high hostile individuals. Of interest to note is that in a few studies, low cynical participants (measured by the Cook-Medley Hostility scale) had greater cardiovascular reactivity than high hostile participants

(Bongard, al'Absi, & Lovallo, 1998; Carroll et al., 1997; Durel, Carver, Spitzer, & Llabre, 1989; Siegman, Anderson, & Herbst, 1991).

Two explanations are investigated in this dissertation for the emergence of null and conflicting result for hostility and cardiovascular reactivity. Firstly, this could be due the non-detection of false negatives (Repressed Cynicism) in the sample. If a researcher has a large number of false negatives in his/her sample owing to sampling bias, then null or negative results can occur between Cynicism and reactivity. However, as was discussed earlier, there are problems with the current method of operationalising Repressed Cynicism. The use of both Cook-Medley Hostility scale and the Implicit Cynicism assessment is proposed in this dissertation as an alternative method. Focus is given on the Cook-Medley Hostility scale because the bulk of contradicting results have been reported when Cynicism is operationalised by this scale.

Secondly, researchers have also proposed that the Hostility-Reactivity relationship may be moderated by other factors. Some of the moderators proposed and investigated by other researchers include gender (Smith et al., 2000), ethnicity (Jain et al., 2004; Why et al., 2003), social desirability (Helmers & Krantz, 1996; Jorgensen et al., 2001; Larson & Langer, 1997; Shapiro, Goldstein, & Jamner, 1996a), state anger (Bishop & Robinson, 2000; Suarez & Williams, 1989), trait anger (Bongard et al., 1998) and interpersonal control (Smith & Brown, 1991). This chapter focuses on state anger and control as mediators for the

hostility-reactivity relationship. It is this second explanation that is discussed next⁵.

Even though state anger and control are discussed separately for clarity, these two moderators of the Hostility-Reactivity relationship are related. Appraisal theories of emotion have linked control and anger. Scherer (Scherer, 2000) reviewed the research literature and reported that appraisal patterns can successfully classify about 40% to over 50% of different emotions. Roseman et. al (Roseman, Dhawan, Rettak, Naidu, & Thapa, 1995) sampled both Indian and American undergraduates and tested whether appraisal can account for cultural differences in three emotions - sadness, fear and anger. Using Structural Equation Modelling, it was found that the appraisal of the event being caused by others was significantly related to higher anger ratings. This suggests that events caused by factors beyond one's control are related to anger. Stein and Levine proposed anger occurs when people (a) think they can accomplish a particular goal/attain a desired state, (b) find that the desired goal is lost or an aversive condition cannot be avoided, and (c) firmly believe that the desired state can be reinstated (Stein & Levine, 1990). Thus, the control over the means of goal attainment is relevant to anger. The association between control over goal attainment and anger can also be taken from another perspective; a sample of 288 community residents and students

⁵ In terms of the two measures of cardiovascular variability used in this dissertation (spectral analysis of heart rate and baroreceptor sensitivity), there is still an insufficient number of studies to deduce whether the Hostility-Variability relationship is moderated by other factors. Hence, the following discussion of the moderation of perceived control and state anger is focused on the Hostility-Reactivity relationship, as there is more evidence for this.

reported that frustration is an important cause of an angry emotional response (Averill, 1983). The frustration-aggression theorists claim that the obstruction or blocking of goal attainment, leads to anger (Dollard, Doob, Miller, Mowrer, & Sears, 1939). Again, the means of goal attainment is highlighted as an important element in an angry emotional response. How are means and control related? Skinner (Skinner, 1996) suggests that the various conceptualisations of perceived control measure relations between agent, means and ends. Hence, low perceived control can be induced through a loss of the means to goal attainment. At the same time, losing the means of goal attainment also serves as an obstacle and can induce state anger arousal. In this dissertation, state anger and low perceived control are induced through manipulating controllability of a computer task, obstructing goal attainment (i.e., task scores for monetary reward), and thereby arousing state anger and low perceived control.

Cynicism-Reactivity moderated by Anger

Suarez and Williams were one of the first researchers to suggest and present evidence that hostility is related to cardiovascular reactivity only when high hostile individuals experience anger (Suarez & Williams, 1989). These researchers used a harassment paradigm for participants with high and low hostility scores. The harassment paradigm involves a confederate making disparaging remarks at the participant while he/she is performing the task. They found a significant Cynicism-Reactivity effect. In addition, high cynical participants also reported higher levels of anger

than low hostile participants. A few other studies also found significant results for the Cynicism-Reactivity relationship were achieved via a harassment paradigm. Suarez et al. also used this harassment paradigm and found a significant Cynicism-Reactivity effect (Suarez et al., 1998). Everson and her colleagues (Everson et al., 1995) also illustrated the effects of harassment in increasing systolic blood pressure, diastolic blood pressure, heart rate, and rate pulse pressure for high cynical participants compared to low cynical participants. High cynical individuals also reported greater distress, tenseness, irritation and concentration than low cynical individuals during harassment. Smith and Allred found a significant Cynicism-Reactivity effect when a debating task was used (Smith & Allred, 1989). Hardy and Smith found a significant Cynicism-Reactivity effect using a role-played interaction with conflict (Hardy & Smith, 1988)⁶. The results of these studies led to the suggestion that Cynicism-Reactivity is significant only for situations where anger or social conflict is present (Smith & Pope, 1990).

The current harassment paradigm confounds both anger and interpersonal conflict. State anger arousal need not involve interpersonal conflict. As discussed earlier, anger can also be aroused via goal obstruction. By manipulating the controllability of a computer task, non-interpersonal anger is hypothesised to increase. Such a manipulation will help disentangle the effects of anger from the effects of interpersonal conflict. There is evidence to suggest that perhaps the Cynicism-

⁶ However two other studies using the harassment paradigm also failed to find a significant Cynicism-Reactivity effect (Felsten, 1995; Jain et al., 2004).

Reactivity relationship exist within a social context. Studies using ambulatory blood pressure and heart rate monitoring found that high cynical men have increased blood pressure reactivity during social interaction (Guyl & Contrada, 1998). Brondolo et al. also found that high cynical men had fewer social interactions, more negative social interactions, and had more negative ratings for social interactions (Brondolo et al., 2003). They are more likely to attribute ill intent to others (Epps & Kendall, 1995). This is likely to be due to the fact that hostility (assessed by the Cook-Medley Hostility scale) measures Cynicism - a cynical mistrust of others. In discussions involving conflict, high hostile husbands are also more likely to attribute blame to their spouses and rate their spouses as being responsible for the conflict (Smith, Sanders, & Alexander, 1990). High hostile individuals also have social skills that are perceived by others as less effective (Watkins & Eisler, 1988). The combination of a cynical mistrust of others and poor interpersonal skill increases the occurrence of negative social interactions and reduces their propensity to interact with others. Therefore, high hostile individuals may be more cardiovascular reactive towards social conflict situations and harassment represents a form of social conflict⁷. At present, it is not entirely clear whether state anger moderates the Cynicism-Reactivity relationship or it is just a correlate when social conflict is present. The paradigm used in this dissertation investigates whether state anger from

⁷ It is also interesting to note that the Structured Interview-derived Hostility used in this dissertation (IHAT) also reflects this interpersonal aspect of hostility as the ratings of Hostility from the IHAT is obtained through the social interaction between the interviewer and interviewee. IHAT is an acronym for the Interpersonal Hostility Assessment Technique.

a non-interpersonal origin moderates the Cynicism-Reactivity relationship. In study 2, the Anger Recall task has also been included. This task represents state anger induced through the recollection of an interpersonal event. Hence, state anger aroused by this task is influenced by interpersonal conflict. As reviewed, significant Hostility-Reactivity effects have been found for this task. This task has also found to be more cardiovascularly arousing than other experimental task, and thus may have better power in investigating the Hostility-Reactivity relationship (Jain et al., 1998; Why et al., 2003).

Moreover, there can be two versions of this 'moderator' hypothesis for state anger - a stronger and a weaker version. This dissertation distinguishes the two and tests them. The stronger version of this moderator hypothesis is when state anger is experimentally manipulated for each individual. If the moderator hypothesis is correct, increasing state anger for an individual with high cynicism will increase his cardiovascular reactivity, and decreasing his state anger will decrease his cardiovascular reactivity. In this dissertation, state anger is manipulated via adjusting the controllability of a computer task. If appraisal theories of emotion are correct, then state anger will increase, as the task becomes less controllable. In the weaker form of the 'moderator' hypothesis, the average state anger reaction to all the tasks (as well as the average cardiovascular reaction to all the tasks) is used. For the weaker version of the moderator hypothesis to be true, a high cynical individual who reports more state anger has greater cardiovascular reactivity, another high cynical individual with low state anger has lower cardiovascular reactivity,

while low cynical individuals have less cardiovascular reactivity regardless of state anger levels. The weaker form of this moderator hypothesis is the one tested in the previously quoted experimental studies (e.g., Suarez & Williams, 1989). The stronger version of the moderator hypothesis is so-called because state anger levels are manipulated within an individual. Compared to the weaker version, both hostility and state anger are not manipulated. In the weaker version, the same treatment resulted in individuals with comparable levels of hostility to report different levels of state anger. Evidence for the stronger version provides stronger empirical support for state anger moderating the Cynicism-Reactivity relationship. Significant results for the weaker version raises the possibility that high cynical participants who report high state anger may represent a subset of high cynical individuals (e.g., highly cynical individuals with high anger expression).

Cynicism-Reactivity moderated by Control

Control is related to an angry emotional response. By simultaneously decreasing control and creating goal obstacles, state anger can be aroused. At this point, 'control' refers to both actual control and perceived control. Later, the distinction between the two will be made. Cynical people are more likely to feel anger more frequently and at greater intensities (Suarez & Williams, 1989; Shapiro, Jamner, & Goldstein, 1997; Weidner et al., 1989). Therefore, if cynical individuals tend to report anger more often and control and state anger are related, then control should be an important aspect in defining their perceived

stress. The hypothesised role of control in moderating the Cynicism-Reactivity began when hostility was considered part of the Type A Behaviour Pattern.

Historically, the research about hostility and health came as a response to the increasing number of null findings that have been published with regards to Type A Behaviour (Fischman, 1987). Two cardiologists, Friedman and Rosenman proposed the role of Type A Behaviour in cardiovascular disease (Friedman & Rosenman, 1974). They had found that most of their cardiac patients had similar patterns of behaviour often characterised by a constant sense of time urgency, competitiveness and hostility. These characteristics were later used to define what is now known as Type A Behaviour Pattern. The first emerging large-scale epidemiological studies like the Western Collaborative Group study have shown that Type A Behaviour predicted heart attacks (Rosenman et al., 1975). However, the Multiple Risk Factor Intervention Trial (MRFIT) was one of the first large-scale epidemiological study to show that Type A Behaviour, assessed by the 'gold' standard the structured interview method, did not predict poorer health status (Shekelle et al., 1985). Following results published by MRFIT, several other epidemiological studies also showed similar null findings between Type A Behaviour and health outcomes (Fischman, 1987; Myrtek, 2001). In a meta-analysis done by Myrtek (Myrtek, 2001), it was found that Type A Behaviour explained for less than 1% of variance (not significant) in health outcomes. Research into Type A Behaviour has begun to wane (see, Conduit, 1991; Ray, 1991). Researchers at Duke University

published one of the first studies to show that it was hostility, a component of Type A Behaviour that predicted health outcomes (Barefoot et al., 1983). It was then suggested that hostility is the 'toxic' component of Type A Behaviour (Williams & Barefoot, 1988). Theories about Type A Behaviour could be useful in research in hostility. Theories postulated with regards to explaining the psychological processes of Type A Behaviour Pattern could be useful with regards to hostility because there has been a lack of theoretical framework to explain psychological processes relevant to hostility.

One of the earliest observation documented by Glass et al. was that individuals with Type A Behaviours tend to have greater cardiovascular reactivity when exposed to uncontrollable tasks (Glass, 1977). Type A's tend to report using active coping strategies more than Type B's (Vingerhoets & Flohr, 1984). Experiments have also showed that even when Type A's knew that their partners have better ability than themselves and could potentially improve task performance by relinquishing control, Type A's still prefer to perform the task themselves (Miller, Lack, & Asroff, 1985; Strube, Berry, & Moergan, 1985).

Researchers have suggested that at the core of Type A Behaviour and hostility is a sense of insecurity and cynical mistrust of others (Price, Friedman, Ghandour, & Fleischmann, 1995). Since what is controllable becomes predictable and reduces insecurity, individuals with Type A Behaviours try to decrease their sense of insecurity via controlling the environment (Powell, 1992) and using antagonistic social interaction styles or anger in order to gain interpersonal control (Averill, 1982).

Smith and Brown is the only known study that has investigated the role of interpersonal control in moderating the Cynicism-Reactivity relationship (Smith & Brown, 1991). Smith and Brown examined the role of Cynicism, assessed by the Cook-Medley Hostility scale, among marital couples' social interaction and its influence on cardiovascular response. Participants were either told to influence their spouse's decision (Influence condition) or to have a discussion with their spouse (Discuss condition). The results showed the Influence/Discuss manipulation interacted with hostility for systolic blood pressure reactivity for husbands but not for the wives; high hostile husbands in the Influence condition had the highest systolic blood pressure reactivity. These results were replicated in another study using another measure of hostility - the Buss-Perry Aggression Questionnaire (Smith & Gallo, 1999). Given the earlier discussion about Cynicism functioning within a social context, there are yet no known published studies that have investigated whether such results can be generalised to non-interpersonal situations. In other words, is the interaction between Cynicism and control general or interpersonal-situation specific?

At this point, it is necessary to differentiate two forms of control - perceived control and actual control (Skinner, 1996). In this dissertation actual control is refers to the response-outcome contingency for the computer task is, while perceived control refers to the participants' ratings of how controllable they perceived the computer task to be.

Actual Control (Task Control). Psychophysiological research has tackled the issue of control from another different perspective. Sherwood et al. found that tasks that were uncontrollable were associated with a passive physiological response (i.e., decreased cardiac output and increased vascular resistance) while tasks that were controllable had an active coping (i.e., increased cardiac output and decreased peripheral resistance) physiological response (Sherwood et al., 1990b). These physiological patterns have since been replicated in several other studies (e.g., Weinstein, Quigley, & Mordkoff, 2002; Tomaka, Blascovich, Kelsey, & Leitten, 1993; Hurwitz et al., 1993). Tasks were defined as passive coping if the organism could not initiate any coping responses when an aversive stimulus is present. Active coping tasks are defined as tasks where coping responses are available (Schneiderman & McCabe, 1989). Tasks like the Cold Pressor, viewing a stressful video and passive vigilance are considered passive coping tasks while mental arithmetic and memory tests are active coping tasks.

There are two problems with the current definition of active and passive coping tasks. Firstly, the definition confound the presence of coping behaviours and physical effort (Gerin, Pieper, Marchese, & Pickering, 1992). Since active coping tasks often enable the participant's to engage in active coping behaviours while passive coping do not, therefore by virtue of engaging in such behaviours, active coping tasks often require more physical effort than passive coping tasks. The current dissertation (Study 2 and 3) aims to solve these problems by manipulating the effectiveness of a computer mouse in various degrees in

order to affect task controllability. In this way, participants engage in active coping behaviours regardless of task controllability. Monetary incentive based on task performance can be offered in order to reduce the chance of participants giving up when the task has low controllability. What is manipulated is the efficacy of the coping behaviours rather than the presence/absence of such coping behaviours (e.g., Light & Obrist, 1983). Secondly, the current definition of active and passive coping tasks covers only the two extreme forms of task control; active coping tasks have task control while passive coping tasks have no task control. This appears inadequate, as it is not able to accommodate situations where task control refers to response-outcome contingencies. In Behaviourism, operant conditioning is related to response-outcome contingency (Thorndike, 1898). Response-outcome contingencies have been used in forging the concept of learned helplessness (Abramson, Seligman, & Teasdale, 1978). These constructs indicate that there are many situations where the task at hand does not fit into the current definition of active/passive coping tasks. The presence of active coping tasks like mirror tracing and STROOP colour-word tasks that elicit passive instead of active coping physiological responses also creates problems for the current definition of active/passive coping task (Hurwitz et al., 1993; Waldstein, Bachen, & Manuck, 1997).

It is proposed and tested in this dissertation that task control can be conceptualised as the contingency between a participant's response and the outcome of that response. High task control has high response-outcome contingency if a participant's response often elicits the outcome

(e.g., opening the faucet and getting water). A task's response-outcome contingency can be low as well (e.g., gambling odds) and when equipment suddenly becomes faulty (e.g., clogged water pipes decrease the response-outcome contingency of opening the faucet for water).

Using response-outcome contingencies to conceptualise actual control converts the traditional active/passive task control definition from a categorical distinction to a continuum. Using such a continuum will help eliminate the confound between coping behaviours and task control. In addition, the traditional definition of active and passive coping tasks can also be accommodated with this new continuum. Active coping and passive coping tasks represent the extreme ends of the continuum. In this dissertation, one of the aims is to test the hypothesis that even when coping behaviours are present, manipulating the response-outcome contingencies can also elicit the passive coping physiological response. In this case, the response is the movement of the computer mouse initiated by the participant while the outcome is the movement of the mouse cursor on the computer screen.

Perceived Control. In psychophysiological research, passive and active coping tasks are defined by the actual control of the task. In re-conceptualising active/passive coping tasks into a continuum of response-outcome contingencies, there is also the corresponding concept in perceived control (response-outcome expectations, Bandura, 1977).

Researchers have often cited the importance of perceived control and state that it is more important than actual control (Averill, 1973; Burger, 1989; Wallston, 2001). However, though there are studies that reported the predictive power of perceived control and future health status (e.g., Katz, Yelin, Eisner, & Blanc, 2002), few studies have actually manipulated actual control and perceived control in the same study to see their respective influence on health and cardiovascular functions. The following reviews the known studies that have done so.

Waldstein et al. found that perceived control was a better predictor of cardiovascular stress response than actual task controllability (Waldstein et al., 1997). Even for patients with a chronic illness that one has very little control over such as cancer, it is found that having higher perceptions of control were associated with less depression (Thompson, Sobolew-Shubin, Galbraith, Schwankovsky, & Cruzen, 1993). Even when situational control is present, the presence of low perceived control alone can produce helplessness (Abramson et al., 1978). Illusory control is present when an individual perceives control when actual control is non-existent or low. A person is termed to have veridical control when perceived control is congruent with actual control (Wallston, 2001). Theories on illusory control (Langer, 1975) and helplessness (Seligman, 1975) attests to the assertion that perceived control is of paramount importance in health outcomes and behaviour. These researchers suggest that when there is a discrepancy between perceived control and actual control, perceived control is more important in terms of predicting health outcomes.

On the other hand, there are also studies that show that actual control rather than perceived control is important in predicting behaviour. For example, increasing actual job control can decrease absence rates in a work environment (van Yperen & Snijders, 2000). Perceived control was also found not to be related to the actual performance on an anagram task for 80 participants (Endler, Speer, Johnson, & Flett, 2001). Moreover, if cardiovascular reactivity is related to health then the null results obtained between perceived control and cardiovascular arousal appears to limit the role perceived control in the Reactivity Hypothesis. For instance, Gerin and his colleagues had 40 students perform two sessions of mental arithmetic - one was self-paced (high task control condition) and another was externally paced (low task control condition). Blood pressure and heart rate reactivity was significantly higher during the low task control condition. However, perceived control was not significantly associated with cardiovascular reactivity (Gerin, Litt, Deich, & Pickering, 1995). Another study randomised 24 patients with lower back pain to three conditions that manipulated their perceived control via the success rate of their electromyogram biofeedback treatment (Biedermann, McGhie, Monga, & Shanks, 1987). Participants were randomised into conditions that gave feedback that either indicated high success (illusory control), linearly related to their performance (veridical control) or low success (learned helplessness). The results showed that regardless of the biofeedback given to the participants, all participants benefited equally from the biofeedback therapy. This study showed that illusory control did not improve the participants' recovery from lower back

pain. Another study by Shirom et al. also found that perceived control was not related to serum uric acid. Increased serum uric acid is associated with an increased risk of arthritis, kidney stones and cardiovascular disease (Shirom, Melamed, & Nir-Dotan, 2000). Illusory control is not always related to better health outcomes as it is also associated with high-risk sexual activity (e.g., unprotected sex) (Zuckerman, Knee, Kieffer, & Gagne, 2004) and poorer future health (Zuckerman, Knee, Kieffer, Rawsthorne, & Bruce, 1996). Mothers with high illusory control with regards to stopping infant cries tend to be susceptible to learned helplessness (Donovan, Leavitt, & Walsh, 1990).

Perceived Control versus Actual Control Debate. Perceived control and actual control need not be regarded as mutually exclusive; perceived control and actual control are not always contradictory and is frequently congruent with actual control (Wallston, 2001). For instance, Steptoe sampled 122 male and females school teachers who had high and low job control (Steptoe, 2001). He found that teachers with low job control had more instances of low perceived control and greater blood pressure and heart rate reactivity than teachers with high job control. Allowing diabetic patients to determine the time and quantity of insulin injection (after training) in line with their eating patterns was done to increase patients' actual control over their treatment (Howorka et al., 2000). This intervention also increased the patients' perceived control over diabetes. Recent evidence suggests that when actual control can be quantified, the congruence between perceived control and actual control predicts

behaviour (Sheeran, Trafimow, & Armitage, 2003) and better health behaviours (Zuckerman et al., 2004).

Furthermore, two theories that have successfully used control to predict health outcomes combines both perceived and actual control. They are Karasek and Theorell's Job Strain model (Karasek & Theorell, 1990) and Siegrist's Effort-Reward Imbalance model (Siegrist, 1996b). Since these two models are only presented here to illustrate the utility of including both actual and perceived control in predicting health outcomes, only Karasek's model will be discussed.

Karasek's model postulates that a combination of high job demand and low job control is detrimental to health (Karasek et al., 1988). Job control is assessed via a questionnaire with items asking the participant to rate the extent his/her job requires creativity, the development of new skills, control over what needs to be done and how much to be done. A review of the studies published between 1981 and 1993 that have investigated the relationship between job strain and cardiovascular disease risk or mortality found that most of these studies found positive results (Schnall, Landsbergis, & Baker, 1994). A study also found the low job control was significantly associated with higher ambulatory systolic blood pressure than high job control (Melamed, Kristal-Boneh, Harari, Fromm, & Ribak, 1998). So incorporating both actual control and perceived control clearly has its health outcome predictive utility. Therefore, there are potential research questions to be answered between actual and perceived control. For instance, does perceived control predict unique variance from actual control? Given appraisal

theories of emotion and Type A Behaviour Pattern, are high hostile individuals more sensitive to changes in actual control in a non-interpersonal situation? If so, is perceived low control a more important predictor of physiological response than task controllability?

Justification for a Male-Only Sample

The studies of this dissertation consist of male-only samples. Therefore, justifications have to be made as to why the research was restricted to this particular gender.

Firstly, the first study that attempted to validate the Interpersonal Hostility Assessment Technique has been conducted with male samples (Haney et al., 1996) though validation was subsequently extended to include female volunteers (Brummett et al., 1998; Siegman et al., 2000). However, the potential for differences between genders in terms of construct validity for hostility assessment remains a possibility as demonstrated by a study conducted by Davidson and Hall (Davidson & Hall, 1995). Davidson and Hall found that for males, the Potential for Hostility, a precursor of the IHAT, was related to the Cook-Medley Hostility scale but unrelated to negative affectivity. For females, Potential for Hostility was unrelated to the Cook-Medley Hostility scale and was related to negative affectivity. The study indicates that the concept of cynicism and overt aggression are related for males but not for females. Furthermore, the Cook-Medley Hostility Scale also appears to have a different psychometric factor structure for females when compared to males (Steinberg & Jorgensen, 1996). In addition, it could be that

cynicism as measured by the Cook-Medley Hostility scale has greater predictive validity for health in men than women. Girdler and her colleagues found that the Cook-Medley Hostility scale predicted cardiovascular reactivity in men but not in women (Girdler et al., 1997). Moreover, perceived control tend to be negatively related to negative affect in men but not in women (Hutt & Weidner, 1993). Control also appears to be more closely related to cardiovascular reactivity for men than women (Smith, Limon, Gallo, & Ngu, 1996). This suggests that earlier research evidence and theories that were discussed about anger and perceived control may be more applicable to males.

Secondly, in terms of physiological arousal, there are also gender differences for different tasks (Sloan et al., 2001; Smith et al., 2000; Carrillo et al., 2001). Men who were harassed during a mental arithmetic task had greater heart rate reactivity than women who were harassed (Delahanty et al., 2000). Stroud et. al had male and female volunteers performed a speech task about the volunteers' body image, isometric handgrip, mirror tracing and mental arithmetic tasks (Stroud, Niaura, & Stoney, 2001). Females had greater heart rate reactivity to the speech task compared to males while males had greater systolic blood pressure reactivity to the other three tasks compared to females. In terms of underlying hemodynamic regulation of blood pressure, gender differences have also been found; men also reacted to mental arithmetic, video game, and anger recall interview with greater cardiac output reactivity, and blood pressure reactivity than women (Lawler, Wilcox, & Anderson, 1995). However, in another study that used nonverbal mental arithmetic,

a mirror tracing task, the Stroop Colour-Word task, and an isometric handgrip task showed that males had greater blood pressure reactivity and total peripheral resistance reactivity while females had greater heart rate reactivity (Allen, Stoney, Owens, & Matthews, 1993). Therefore it is still unclear as to the gender differences for the hemodynamic regulation of blood pressure. But part of the differences found between genders for the hemodynamic regulation of blood pressure could be due to hormonal changes in women (Low et al., 2001).

Thirdly, gender often interacts with all the above stated variables (i.e., hostility, anger, task and reactivity) to produce complex interactions that are difficult to interpret (e.g., Saab et al., 1997). For example, high neurotic hostility in males was associated with greater diastolic blood pressure reactivity in the anger recall interview but not for females (Lawler, Harralson, Armstead, & Schmied, 1993). Using five different behavioural stressors, a task by gender interaction was found in which different genders had different levels of reactivity for different tasks (Light, Turner, Hinderliter, & Sherwood, 1993). This study found that there were no differences between men and women in terms of cardiovascular reactivity for the mathematical task but for the reaction time task, men had greater systolic blood pressure, cardiac output and peripheral resistance reactivity. Men also had greater systolic blood pressure reactivity than women for a passive-speech task. The forehead cold pressor increased systolic blood pressure and diastolic blood pressure in men while women had greater heart rate reactivity. These two studies illustrate the complexity of results that can arise when an experimental

design has gender, task and different cardiovascular reactivity indices present as variables. Since Study 2 and 3 of this dissertation are using a new method of manipulating the response-outcome contingency (i.e., task controllability) of an active coping task to predict an active and passive physiological response, the presence of such gender by task by reactivity interactions may make it difficult to interpret the main effects.

Hence for all these reasons, this dissertation limits its samples to males. That is not to say that gender is not an important variable in terms of research, but as there are already a number of variables that are investigated in this dissertation, including gender would complicate the issue and make the interpretation of the results confusing and difficult. Therefore, it is better to limit the findings of this dissertation to males alone and investigate the same psychophysiological phenomenon in females separately.

Summary

To summarise, cardiovascular disease is a major health problem in most countries and research shows that hostility is a potential risk factor. Hostility can be distinguished into attitude (Cynicism), affect (Anger) and behaviour (Aggression) components. The two main methods of hostility assessment that have been found to be significant predictors of health are the structured interview method of hostility assessment (Aggression) and the Cook-Medley Hostility scale (Cynicism). Study 2 uses these two forms of hostility assessment to investigate the role hostility, control and cardiovascular reactivity.

There are measurement problems with these two forms of Hostility assessment. Measurement imprecision decreases power and can cause null or conflicting results. This dissertation concentrates on improving the measurement acuity of the Cook-Medley Hostility scale. Specifically two measurement issues of this scale are highlighted and discussed: (i) lack of stable internal structure, and (ii) false negatives (Repressed Cynicism). The psychometric structure of the Cook-Medley Hostility scale is still unclear. Studies that have done exploratory factor analysis on this scale have found that it has more than 2 factors. Various researchers have often used different subsets of items from this scale. Without empirical evidence for its psychometric structure, it is uncertain whether these subsets of items measure the same construct. So a review and a critical selection of items are required to perform a confirmatory factor analysis, which is the aim of Study 1, in order to determine the psychometric structure of this scale. Results obtained from this study will help in the scoring of the Cook-Medley Hostility scale for Study 2 and 3.

Furthermore, the socially undesirable traits like Cynicism are prone to impression management. That is, cynical participants may give low Cynicism scores in order to present themselves in a positive light. This is referred to as 'Repressed Cynicism'. Study 3 aims to introduce an implicit association method of tapping hostility (Hostile Go/No-go Association Test - Hostile GNAT). Implicit measures have been found to be less amenable to social desirability. Using factor analysis on both the Cook-Medley Hostility scale and the Implicit Cynicism measure (GNAT Cynicism), the influence of false negatives and false positives in sample

is reduced. The effects of false positives and false negatives on the results can also be examined.

Researchers have proposed that high Hostile individuals are at greater risk of cardiovascular disease because they have a pattern of cardiovascular reactivity and variability during stress that increases this risk. This is referred to as the 'Cardiovascular Reactivity Hypothesis' and the 'Cardiovascular Variability Hypothesis' respectively. Some studies have reported evidence for the Reactivity Hypothesis. There are fewer studies investigating Hostility and cardiovascular variability but the available evidence is congruent with the Variability Hypothesis. However, there is an emerging trend of studies that have reported null results and in some cases, negative results for the Reactivity Hypothesis. This could be due to measurement error of the Cook-Medley Hostility scale and other variables moderating of the Hostility-Reactivity relationship.

Besides improving the measurement precision of the Cook-Medley Hostility scale, this dissertation also investigates the role of two moderators - control and state anger. Both control and state anger are related. According to appraisal theories of emotion, control over goal attainment is important for an angry emotional response. Via manipulating control, state anger can be induced. Current studies that have induced anger and manipulated control to investigate the Hostility-Reactivity effect confound interpersonal conflict with anger and control. There is evidence suggesting that Cynicism-Reactivity exists within a social context. Therefore, there is need to disentangle state anger inducement, control and interpersonal conflict. In this dissertation, non-

interpersonal control and anger are manipulated via adjusting the efficiency of the computer mouse. In study 2, Anger Recall task was also included to induce state anger influenced by interpersonal conflict.

Two forms of control can be distinguished - perceived and actual control. Psychophysiological research has investigated actual control via using tasks that either allow or prevent the participant from influencing the task outcome (active and passive coping tasks). This was deemed inadequate and an alternative definition of actual control using response-outcome contingency is proposed. Adjusting the efficiency of the mouse manipulated the task's response-outcome contingency.

Some researchers have argued that perceived control is more important in predicting cardiovascular stress reactivity than actual task controllability. However, perceived and actual control need not be viewed as contradictory but are often congruent. In addition, some other theories (e.g., Karasek's Job Strain model) have not discriminated either in predicating health outcomes successfully. Both perceived and actual control may be equally important.

An all-male sample is taken in this dissertation to make the sample more homogenous and thereby increasing power. Furthermore gender differences in terms of Hostility assessment, cardiovascular functioning, and their interactions may make the interpretation of the results difficult.

The following gives a general overview for the three Studies mentioned in this chapter:

Study 1 (Chapter 2): Reviews the literature on the factor structure of the Cook-Medley Hostility scale (one of the Hostility assessments used in this dissertation) and identifies the consistent psychometric structure in the scale. Confirmatory factor analysis is then performed on this structure for a sample of male participants. The results of this new structure of the Cook-Medley Hostility scale will then be applied to Study 2 and 3 in terms of scoring procedure for this scale.

Both Study 2 and 3 investigate the role of hostility, control (perceived and actual), and anger in predicting cardiovascular reactivity. Both studies analyse the underlying hemodynamic cardiovascular processes for blood pressure regulation (i.e., cardiac output and total peripheral resistance). However there are some differences in focus between the two studies when tackling these variables.

Study 2 (Chapter 3): The features of this study include:

- (i) including the two hostility assessments (Structured Interview and the Cook-Medley Hostility scale) that have been found to be significantly related to health outcomes
- (ii) analysing the regulatory mechanisms for cardiovascular variability (i.e., autonomic heart rate activity and baroreflex sensitivity using spectral analysis and sequence method),
- (iii) the first study of the three to vary the efficacy of active coping behaviours for a computer task via varying the responsiveness of the computer mouse

- (iv) comparing results from the computer task of varying control with the Anger Recall task
- (v) assessing the response-outcome contingency definition of task control

Study 3 (Chapter 4): The main focus here is the creation and testing of an implicit association test for Cynicism (Hostile GNAT). This Study aims to:

- i) describe the details of the conceptualisation and development of this form of assessment
- ii) testing its construct validity with the Cook-Medley Hostility scale and its discriminant validity with other negative affectivity and social desirability measures
- iii) investigates the use of the Hostile GNAT when used with the Cook-Medley Hostility scale to reduce the problem of false negatives (Repressed Cynicism) in the sample
- iv) Examine the consequence of non-detection of false-negatives and false positives in the sample on the overall results

CHAPTER 2: STUDY 1

INTRODUCTION

To recapitulate what was discussed in Chapter 1; the Cook-Medley scale has been frequently used by researchers to tap Cynicism. This scale consists of 50 items taken from the Minnesota Multiphasic Personality Inventory (MMPI) and responses are in a true or false format. Originally derived to differentiate good and bad teachers, it was subsequently found to be predictive of cardiovascular disease and mortality (Barefoot et al., 1983). However, the 50-item questionnaire does not seem to tap a single construct. Studies that have performed an exploratory factor analysis on this scale have found two (Costa et al., 1986), four (Han et al., 1995) and five (Steinberg & Jorgensen, 1996) factors. The aim of this introduction is to provide a review of the research literature that has investigated the psychometric structure of the Cook-Medley Scale via exploratory or confirmatory factor analysis. From this review, the stable components of this scale are then determined and a confirmatory factor analysis will be done for a male sample. The results from this Study will help to determine the scoring process for the Cook-Medley Hostility scale used for the next two studies in this dissertation as well as to increase the internal reliability of this scale for future research.

Understanding the psychometric structure of the Cook-Medley Hostility scale is important because various studies have reported that

subsets of the Cook-Medley scale predict health outcomes. This suggests that it may be a certain aspect of hostility tapped by a subset of items from the Cook-Medley Hostility scale that is more relevant to health. For example, Barefoot et al. found that 27 items of the scale significantly predicted health outcomes for 128 law students while the rest of the 23 items were not significant predictors (Barefoot et al., 1989). Other researchers have also found that a subset of 8 items of the scale that correlated at .77 with the 50-item version predict health outcome of Finns (Greenglass & Julkunen, 1991). It is necessary to assess whether the factors obtained by these researchers are consistent across studies. Doing so would enable researchers to understand which aspect of hostility is predictive of health outcomes. Since the studies of this dissertation comprised of male samples, this review will focus on results published with male samples whenever appropriate (e.g., Steinberg & Jorgensen, 1996).

Based on factor analysis or item content analysis, researchers have selected subsets from the 50 items of the Cook-Medley Hostility scale to measure specific aspects of hostility (e.g., Cynicism). Studies that have done this can be roughly divided into three main categories: (i) those using the 9 or 8-item version, (ii) those using the 27-item version, and (iii) others. As there is only one known study in the 'Others' category, a review of this category will proceed first.

Others. The one study that has investigated the psychometric structure of the Cook-Medley scale in this category is Costa et al. (Costa

et al., 1986)⁸. However, results obtained from this study have not been used by other researchers in hostility assessment. Costa et al. (Costa et al., 1986) factor analysed the Cook-Medley for two subsamples comprising of both males and females (n 's = 518, 484). Two factors were found (see Table 2.1). The first factor they labelled as Cynical Mistrust as it comprised of items that indicated a low opinion of others while the second factor was labelled Paranoid Alienation as it measures emotional distancing and feelings of persecution. These two factors were found to be significantly correlated with each other ($r = .54$) and both were also correlated with Cynicism (from their factor analysis of the MMPI) and Neuroticism factors of the MMPI. The authors concluded that these two factors measure different aspects of hostility.

9 or 8-Item Versions. The 9 or 8-item version of the Cook-Medley Scale has been derived from the research published by Greenglass and her colleagues. Greenglass and her colleagues sampled 263 undergraduates (175 females and 88 males) and performed a number of factor analyses of the 50-item Cook-Medley Hostility scale (Greenglass & Julkunen, 1989). The results revealed a three-factor structure with 9 items that consistently loaded on Factor 1, 4 items on Factor 2 and 6 items on Factor 3 (see Table 2.1). At least two studies have done a

⁸ Houston et al. is another study that has investigated the psychometric structure of this scale. However, its sample size is small ($n = 53$) making the results unreliable. Moreover, cluster analysis was done instead of factor analysis. Cluster analysis is aimed at grouping participants based on their responses on the questionnaire items rather than providing information on the psychometric structure of the scale. Hence, Houston et al.'s study will not be included in this review for the assessment of the Cook-Medley Hostility scale's psychometric structure.

confirmatory factor analysis on this set of 8 or 9 items of the Cook-Medley scale (i.e., Contrada & Jussim, 1992; Miller, Jenkins, Kaplan, & Salonen, 1995). Contrada and Jussim (1992) did a confirmatory factor analysis on these 9 items loading onto a single factor and found that it had the best fit compared to other models they tested (Bentler-Bonett normed fit index = .73). However, they also created two alternative 9-item versions of the Cook-Medley Hostility scale through random item selection and found similar good fit. Contrada and Jussim (1992) concluded that the better fit of Greenglass and Julkunen's (1989) 9-item Cook-Medley model could be due to the fact that it is easier to fit a model with only 9 items compared to other models that used 27 items or more. Another study has reported results indicating that the 9-items (or at least 8 of the 9 items) are related to a single factor; Miller et al. did a confirmatory factor analysis on the 8 item version of the Cook-Medley scale using a sample of 2682 males from the Kuopio Ischemic Heart Disease (KIHD) study (Miller et al., 1995). The sample was divided in to two subsamples (n 's = 1166, 1526). It is unclear why the researchers did a confirmatory factor analysis on 8 items when Greenglass and Contrada (1989) reported 9 items for the factor⁹. The results of this paper showed that the model with 8 items had Goodness of Fit indices of at least .97 and above. This adds to the research evidence that at least 8 of the 9 items proposed by Greenglass

⁹ Specifically, OTH436 was omitted (Table 1) by Miller et al. (1995). The item measures men's propensity to have sexual thoughts when in close proximity to women.

Table 2.1 Factor Analytic Structure (item loading) of the Cook-Medley Hostility Scale from four Studies*.

	Costa et al. (1986)	Greenglass & Julkunen (1989)	Han et al. (1995)	Steinberg & Jorgensen (1996)
Factor 1 (F1)	AGGR414 (.47) AGGR423 (.31) AGGR452 (.32) CYN50 (.40) CYN58 (.39) CYN76 (F1, .38; F2, .32) CYN81 (.40) CYN104 (.44) CYN110 (.48) CYN286 (.44) CYN306 (.43) CYN346 (.39) CYN352 (.44) CYN470 (.45) HAFT205 (.34) HAFT338 (.43) HATT124 (.44) HATT225 (.39) HATT251 (F1, .35; F2, .36) HATT259 (F1, .34; F2, .32) HATT315 (.34) HATT425 (.50) HATT445 (.55) HATT466 (.30) SOAV357 (.42)	CYN81 (.55) CYN104 (.51) CYN110 (.50) CYN241 (.58) CYN254 (.43) CYN286 (.54) CYN306 (.60) HATT124 (.44) OTH436 (.43)	AGGR414 (.36) AGGR443 (.27) CYN50 (.47) CYN58 (.50) CYN76 (.51) CYN81 (.44) CYN104 (.41) CYN110 (.47) CYN254 (.38) CYN286 (.49) CYN346 (.44) CYN352 (.44) CYN470 (.56) HATT217 (-.16) HATT358 (.47) HATT425 (.24) HATT445 (.51) OTH436 (.30) SOAV357 (.37)	AGGR414 (.46) CYN50 (.60) CYN58 (.47) CYN76 (.34) CYN254 (.38) CYN346 (.75) CYN352 (.33) CYN470 (.56) HAFT338 (.33)

*Item labels refer to item classification defined by Barefoot et al. (1989): CYN = Cynicism, HAFT = Hostile Affect, HATT = Hostile Attribution, AGGR = aggressive responding, SOAV = Social Avoidance, OTH = Others. Numbers refer to MMPI-2 booklet numbers.

Table 2.1 Factor Analytic Structure of the Cook-Medley Hostility Scale from four Studies - continued*.

	Costa et al. (1986)	Greenglass & Julkunen (1989)	Han et al. (1995)	Steinberg & Jorgensen (1996)
Factor 2 (F2)	AGGR227 (.30) AGGR248 (.34) AGGR27 (.40) CYN76 (F1, .38; F2, .32) CYN241 (.53) CYN254 (.37) HAFT136 (.31) HAFT372 (-.33) HAFT419 (.44) HATT99 (.47) HATT145 (.45) HATT251 (F1, .35; F2, .36) HATT259 (F1, .34; F2, .32) OTH398 (.46) OTH457 (.37) SOAV19 (.57) SOAV265 (.52)	CYN352 (.53) HAFT338 (.52) HATT225 (.66) HATT251 (.48)	HAFT136 (.25) HAFT205 (.40) HAFT338 (.40) HAFT372 (-.34) HATT99 (.32) HATT124 (.40) HATT145 (.42) HATT225 (.40) HATT251 (.57) HATT259 (.52) HATT315 (.28) HATT466 (.44) OTH347 (.51) OTH457 (.47) SOAV386 (.44)	CYN81 (.78) CYN104 (.32) CYN110 (.85)

*Item labels refer to item classification defined by Barefoot et al. (1989): CYN = Cynicism, HAFT = Hostile Affect, HATT = Hostile Attribution, AGGR = Aggressive Responding, SOAV = Social Avoidance, OTH = Others. Numbers refer to MMPI-2 booklet numbers.

Table 2.1 Factor Analytic Structure of the Cook-Medley Hostility Scale from four Studies - continued*.

	Costa et al. (1986)	Greenglass & Julkunen (1989)	Han et al. (1995)	Steinberg & Jorgensen (1996)
Factor 3 (F3)	AGGR414 (.44) AGGR423 (.53) CYN470 (.43) HAFT136 (.48) HATT358 (.47) HATT466 (.47)	AGGR27 (.48) AGGR227 (.42) AGGR248 (.38) AGGR406 (.45) AGGR423 (.52) AGGR452 (.35) HAFT419 (.47) OTH171 (.21) OTH393 (.41) SOAV19 (.43)	AGGR27 (.46) AGGR423 (.59) AGGR452 (.39) HAFT205 (.39) HAFT372 (F3, .36; F4, .34) HAFT419 (.65)	
Factor 4 (F4)		AGGR230 (-.24) CYN241 (.43) CYN306 (.42) OTH46 (.47) OTH398 (-.38) SOAV265 (.55)	AGGR230 (.51) CYN241 (.62) CYN286 (.39) CYN306 (.81) HAFT372 (F3, .36; F4, .34)	
Factor 5 (F5)			AGGR227 (.81) AGGR248 (.57)	

*Item labels refer to item classification defined by Barefoot et al. (1989): CYN = Cynicism, HAFT = Hostile Affect, HATT = Hostile Attribution, AGGR = Aggressive Responding, SOAV = Social Avoidance, OTH = Others. Numbers refer to MMPI-2 booklet number

and Julkunen (1989) measures a single factor.

27-item Version. The 27-item version of the Cook-Medley scale comprising of 3 subscales was first proposed by Barefoot and his colleagues (Barefoot et al., 1989). Although the model reported in Barefoot et al.'s (1989) paper was not derived from factor analysis, this is the only known paper that has systematically analysed the item content of the Cook-Medley Hostility scale. Thus, their results can help in the interpretation of the factors reported in the other studies reviewed here. Furthermore, other researchers have used this 27-item version of the scale to investigate the role of hostility in health (Barefoot et al., 1993) as well as the effects of hostility reduction on health as well (Gidron, Davidson, & Bata, 1999).

Using theories on aggression, attitudes and information processing, Barefoot and his colleagues identified 6 subscales of the Cook-Medley Hostility scale based on the content validity of the items. These 6 subscales are: (i) *Hostile Attribution* ('a tendency to interpret the behaviour of others as intended to harm the respondent', 12 items), (ii) *Cynicism* ('a generally negative view of humankind, depicting others as unworthy, deceitful, and selfish', 13 items), (iii) *Hostile Affect* ('experience of negative emotions associated with social relationships', 5 items), (iv) *Aggressive Responding* ('respondent's tendency to use anger and aggression as instrumental responses to problems or to endorse these behaviours as reasonable and justified', 9 items), and (v) *Social Avoidance* ('respondent's tendency to avoid others, refrain from social

interaction, or withdraw from interpersonal involvement', 4 items). The remaining 7 items were deemed not to fit into any of these categories and were placed in a subset labelled as '*Others*'. Fourteen clinical psychologists and psychologists-in-training were given the definitions of the 6 subscales and asked to assign each Cook-Medley Hostility item to one of the subscales. There was disagreement among the raters with regards to the assignment of 9 items and the authors retained their original assignments for these 9 items instead of removing them in the final analysis of the scale.

Two samples ($n_1 = 89$, $n_2 = 88$) consisting of almost equal numbers of men and women were used to validate the subscales of the Cook and Medley Hostility scale with the NEO Personality Inventory (NEO PI) (Costa, Jr. & McCrae, 1992). It is unclear why the researchers distinguished the two cohorts when assessing the psychometric structure of the Cook-Medley Hostility scale particularly when both sample sizes are small. The researchers intercorrelated the 7 subscales and NEO PI scales. Moderate correlations (r 's ranged from .41 to .64) were found among the 4 Cook-Medley subscales of *Cynicism*, *Hostile Attribution*, *Aggressive Responding*, and *Hostile Affect*. Compared to these 4 subscale, *Social Avoidance* and *Other* subscales did not consistently correlate significantly with these 4 subscales for the two cohorts. For example, *Social Avoidance* correlated significantly to *Hostile Affect* ($r = .25$) for the 1986-1987 cohort but not for the 1985-1985 cohort ($r = .11$). Furthermore, both *Social Avoidance* and *Others* subscales correlated to NEO PI's Hostility scale for one cohort ($r = .30$ and $.25$ respectively for

the same cohort) but not for the other cohort ($r = .09$ and $.14$ respectively). Therefore, it is questionable whether these two scales tap into the multidimensional construct of hostility that is consistent with the other four.

The results of Barefoot's analysis suggest that there are 4 subscales that measure the multidimensional construct of hostility (i.e., *Cynicism* [CYN], *Hostile Attribution* [HATT], *Aggressive Responding* [AGGR] and *Hostile Affect* [HAFT]). This can be seen in two aspects of their results: (i) the four subscales were moderately correlated with each other which suggest that they measure different aspects of a construct, and (ii) the four subscales also correlated significantly with the NEO PI's Hostility scale for both cohorts. Both the *Social Avoidance* (SOAV) and *Other* (OTH) subscales do not share these properties consistently between the two cohorts analysed. The researchers also found that three of these four subscales consisting of 27 items (*Cynicism*, *Hostile Affect* and *Aggressive Responding*) were significant predictors of later mortality (Barefoot et al., 1989). (Incidentally, it was also reported in this paper that Costa et al.'s 2-factor model also significantly predicted mortality.) Researchers have used this 27-item version of the scale to investigate the role of hostility in health (Barefoot et al., 1993) as well as the effects of hostility reduction on health as well (Gidron et al., 1999).

One of the attempts to provide research evidence for this 27-item solution of the Cook-Medley Hostility scale was done by Han et al. (Han et al., 1995). Hans et al. had a sample of 5913 men and women from colleges, marital counselling sessions, substance abusers, psychiatric

population and also other normative populations. This sample was randomly split into two smaller subsamples and a congruence coefficient was computed to show the replicability of the factors between these two subsamples. Exploratory analysis of the 50 items from the Cook-Medley Hostility reviewed 12 components with eigenvalues greater than one and a scree plot that suggested a 2 or 3 factor solution. Though they found that the congruence coefficients (i.e., replicability) was the highest for the two factor structure that was similar to that found by Costa et al.'s 2-factor model (mean congruence coefficient = .99), they presented results for Barefoot et al.'s 4 factor model because 'it allows for more detailed examination of the content dimensions represented by *Ho* scale items'. The results are presented in Table 2.1. The results showed that most of Barefoot's *Cynicism* items loaded on Factor 1, *Hostile Affect* and *Hostile Attribution* items tend to load on Factor 2 while *Aggressive Responding* items tend to load on Factor 3. This gives some support for Barefoot's 27-item model of the psychometric structure of the Cook-Medley scale though both *Hostile Affect* and *Hostile Attribution* items loaded on the same factor. However, on closer examination, the analysis suggested that Costa et al.'s 2-factor was a better solution than Barefoot et al.'s model. Two evidence suggest this; firstly, the sample was randomly separated into two halves and Costa et al.'s 2-factor solution was more reliably replicated between the two samples (congruence coefficient = .99) compared to Barefoot's 27-item solution (congruence coefficient = .96). Secondly, Costa et al.'s 2 factor model also replicated well between males and females (mean congruence coefficient = .98) compared to

Barefoot et al.'s model (mean congruence coefficient = .90). Gender differences for Barefoot et al.'s model was also reported by Steinberg and Jorgensen's study that performed an exploratory factor analysis using only Barefoot et al's (1989) 27 items from the Cook-Medley scale for 1138 males and 1462 females separately (Steinberg & Jorgensen, 1996). They found five factor solutions for both males and females. The results for males are listed in Table 2.1. According to Barefoot et al.'s (1989) classification, the 27-item Cook-Medley scale had only 3 subscales but Steinberg and Jorgensen found 5 factors. So the results obtained by Steinberg and Jorgensen contradict Barefoot et al.'s item classification. Furthermore, there were gender differences for the factor structure of the 27-item scale. For instance, while Factors 1 and 2 were correlated ($r = .64$) for males, it was less so for females ($r = .34$) though the items in these two factors were similar. This study gives credence to the view that the psychometric properties of the Cook-Medley Hostility scale change with reference to gender. Sternberg and Jorgensen suggested that gender differences of this scale could be due to the way different genders interpret the phrasing of the questionnaire items differently (Steinberg & Jorgensen, 1996). The results of this study also support the fact that the Barefoot et al's (1989) 27-item model cannot be reliably generalised across genders as found by Han et al. (Han et al., 1995).

Another study also reported contrary evidence with regards to Barefoot et al.'s (1989) model of the Cook-Medley scale (Contrada & Jussim, 1992). Contrada and Jussim sampled 470 male and female undergraduates and performed confirmatory analyses on the following

models: (i) the one factor model where all 50 items loaded onto one factor, (ii) Greenglass and Julkunen's (1989) one factor model where 9 items loaded on one factor, (iii) Costa et al.'s (1986) 2 factor model, (iv) Houston et al.'s (1989) 2 factor model, and (v) Barefoot et al.'s (1989) five factor model. For confirmatory factor analysis, fit indices show how well the model 'fit' the data obtained. A fit index greater than .90 is usually taken to indicate that the model fits the data well (Schumacker & Lomax, 1996). Contrada and Jussim (1992) found that Barefoot et al.'s model had a fit index of .51.

To summarise the various versions of the Cook-Medley Hostility scale used, there are three versions reviewed here: (i) Costa et al.'s (1986) 2-factor model, (ii) Greenglass and Julkunen's (1989) 9-item measuring 1 factor model, and (iii) Barefoot et al.'s (1989) 27-item measuring 3 subscales model. The first two models were derived from factor analytic method while the third model was obtained via analysing the item content of the Cook-Medley Hostility scale and correlating to the scales from the NEO PI.

Confirmatory Factor Analysis. Most of the studies on the psychometric structure of the scale reviewed here have used exploratory factor analysis. This method is appropriate when there is not much prior knowledge known about the psychometric structure of the scale. Confirmatory factor analysis has shown that the models reviewed here provide a poor fit of the data (Contrada & Jussim, 1992). However, research evidence suggests that *any* model provided a better fit than no

model at all (Contrada & Jussim, 1992). This means that there is some structure for this scale and the Cook-Medley Hostility scale is not simply a random selection of 50 items. However, it is still unclear what is the internal structure of the scale. Therefore this critical review of these studies attempts to pick out the stable components of the scale in order to perform a confirmatory factor analysis.

As was mentioned, there have been at least two known studies that have used confirmatory factor analysis for this scale. Miller et al.'s study has been discussed earlier because it included only 8 items in their analysis (Miller et al., 1995). The other study that used confirmatory factor analysis on this scale is Contrada and Jussim (Contrada & Jussim, 1992). Contrada and Jussim performed confirmatory analyses on all the models reviewed here. The results were disappointing; all of the models provided a goodness of fit for the data below .80 where the acceptable level is .90. Among all the tested models, Greenglass and Julkunen's (1989) 9-item version of the scale provided the best fit of the data (Bentler-Bonett normed fit index = .73). However, when a confirmatory factor analysis of two random selections of 9 items were done, both got similar goodness of fit indices as well (.63 and .68) which indicates that the better fit of Greenglass and Julkunen's (1989) model may be because their model consisted of fewer items. However, all the tested models showed significant improvement of fit compared to the null model. (The null model assumes the independence of items for each tested model. For instance, the null model for Greenglass and Julkunen's (1989) 9-item model assumes the independence of the 9 items while the model assumes that

the 9 items measure one factor.) Contrada and Jussim found that all the models tested provided a significant improvement over their corresponding null models. This means that though the models tested did not fit the data well, the items of the Cook-Medley Hostility scale has some internal psychometric structure.

A replication of what Contrada and Jussim have done is necessary because these researchers combined males and females in their sample when analysing the data. Given that there may be gender differences in the psychometric structure of this scale (Han et al., 1995; Steinberg & Jorgensen, 1996), the analyses should perhaps have separated genders. Their sample was also over-represented by females (154 males and 316 females). Therefore, the following paragraphs will review the evidence for the stable components of the Cook-Medley and suggest models to be tested using confirmatory factor analysis. The methods and results section of this chapter will describe the testing of this model on a male sample.

In determining the stability and reliability of factor components, Guadagnoli and Velicer's paper has been helpful (Guadagnoli & Velicer, 1988). The researchers used a Monte Carlo procedure that systematically manipulated sample size, number of variables, number of components and component saturation. The results demonstrated that should a component have four or more variables with loadings above .60 then that component should be regarded as stable regardless of sample size. As can be seen from Table 2.2, none of the factors reported by the four studies that have performed an exploratory factor analysis on the Cook-

Medley scale had such high component saturation levels. Guadagnoli and Velicer also suggested that if the factor has several (10 to 12) low loadings (greater than .40), then it should be interpreted as stable for sample sizes greater than 150. As can be seen from Table 2.2 all the studies that have reviewed here consisted of sample sizes above 150. Table 2.2 also shows that Factor 1 satisfies this criteria for two out of the four studies reviewed and this factor is thus likely to be stable. Factor 2 appears to satisfy this criterion for only one study (i.e., Han et al., 1995). In the same vein, Factors 3, 4 and 5 appears to have low stability as they are characterised by low component saturation levels. Guadagnoli and Velicer also suggest that if there are only a few high loadings and low loadings, the sample size should be greater than 300 for interpretation purposes. Furthermore, the researchers strongly suggested replication for such cases. In order to assess the replicability of the components within each factor, Table 2.3 re-organises the items listed in Table 2.1 and presents items that have been found to be in the same factor in at least 2 of the four studies reviewed here. For this review, only questionnaire items that have consistently loaded on the same factor for 3 out of the 4 studies reviewed here will be considered as a stable component of that factor. This criterion is taken because 4 out of 4 studies is too stringent and as Table 3 shows, none of the questionnaire items satisfy this condition. If the criterion of 2 out of 4 studies is taken, this makes the replication of the items within each factor at chance level as only results from 4 known studies are reviewed here.

Table 2.2 Methodological Information and Component Saturation in the Four Studies

	Han et. al (1995)	Greenglass & Julkunen (1989)	Costa et. al (1986)	Steinberg & Jorgensen (1996)
Sample Size	5913	263	518, 484	1138
Sample Characteristic	2900 males, 3013 females	175 females, 88 males	about 2/3 males and 1/3 females	males
Factor Derivation Method	Principal Components	Principal Components	Principal Components	Full Information Maximum Likelihood
No. of Items Analysed	50	50	50	27
Factor 1				
loadings .60 and above =	0	1	0	2
loadings .40-.59 =	12	8	14	3
loadings <.40 =	7	n.r.	10	4
Factor 2				
loadings .60 and above =	0	1	0	2
loadings .40-.59 =	11	3	8	0
loadings <.40 =	4	n.r.	7	1
Factor 3				
loadings .60 and above =	0	0		1
loadings .40-.59 =	7	6		2
loadings <.40	3	n.r.		3
Factor 4				
loadings .60 and above	0			2
loadings .40-.59	4			1
loadings <.40	2			2
Factor 5				
loadings .60 and above				1
loadings .40-.59				1
loadings <.40				0

Note. 'n.r.' = not reported.

Table 2.3 Cook-Medley Items replicated in at least 2 out of the 4 Studies*

	Costa et al. (1986)	Greenglass & Julkunen (1989)	Han et al. (1995)	Steinberg & Jorgensen (1996)
Factor 1 (F1)	CYN50 (.40)		CYN50 (.47)	CYN50 (.60)
	CYN58 (.39)		CYN58 (.50)	CYN58 (.47)
	CYN76 (F1, .38; F2, .32)		CYN76 (.51)	CYN76 (.34)
	CYN81 (.40)	CYN81 (.55)	CYN81 (.44)	
	CYN104 (.44)	CYN104 (.51)	CYN104 (.41)	
	CYN110 (.48)	CYN110 (.50)	CYN110 (.47)	
	CYN306 (.43)	CYN306 (.60)		
	CYN286 (.44)	CYN254 (.43)	CYN254 (.38)	CYN254 (.38)
	CYN346 (.39)	CYN286 (.54)	CYN286 (.49)	
	CYN352 (.44)		CYN346 (.44)	CYN346 (.75)
	CYN470 (.45)		CYN352 (.44)	CYN352 (.33)
	HATT425 (.50)		CYN470 (.56)	CYN470 (.56)
	HATT445 (.55)		HATT425 (.24)	
	HATT124 (.44)	HATT124 (.44)	HATT445 (.51)	
	HAF338 (.43)			HAF338 (.33)
AGGR414 (.47)		AGGR414 (.36)	AGGR414 (.46)	
SOAV357 (.42)		SOAV357 (.37)		
		OTH436 (.30)		
	OTH436 (.43)			

*Item labels refer to item classification defined by Barefoot et al. (1989): CYN = Cynicism, HAF3 = Hostile Affect, HATT = Hostile Attribution, AGGR = Aggressive Responding, SOAV = Social Avoidance, OTH = Others. Numbers refer to MMPI-2 booklet numbers. Items in boxes are replicated in 3 out of the 4 studies listed here.

Table 2.3 Cook-Medley Items replicated in at least 2 out of the 4 Studies - continued*

	Costa et al. (1986)	Greenglass & Julkunen (1989)	Han et al. (1995)	Steinberg & Jorgensen (1996)
Factor 2 (F2)	HAF136 (.31) HAF372 (-.33)	HAF338 (.52)	HAF136 (.25) HAF338 (.40) HAF372 (-.34)	
	HAT199 (.47) HAT145 (.45)		HAT199 (.32) HAT145 (.42) HAT225 (.40)	
	HAT251 (F1, .35; F2, .36) HAT259 (F1, .34; F2, .32) OTH457 (.37)	HAT225 (.66) HAT251 (.48)	HAT251 (.57) HAT259 (.52) OTH457 (.47)	
Factor 3 (F3)		AGGR423 (.53)	AGGR423 (.52) HAF419 (.47) AGGR27 (.48) AGGR452 (.35)	HAF419 (.65) AGGR27 (.46) AGGR452 (.39)
Factor 4 (F4)			AGGR230 (-.24) CYN306 (.42) CYN241 (.43)	AGGR230 (.51) CYN306 (.81) CYN241 (.62)

*Item labels refer to item classification defined by Barefoot et al. (1989): CYN = Cynicism, HAF = Hostile Affect, HAT = Hostile Attribution, AGGR = Aggressive Responding, SOAV = Social Avoidance, OTH = Others. Numbers refer to MMPI-2 booklet numbers. Items in boxes are replicated in 3 out of the 4 studies listed here.

As can be seen from items within boxes in Table 2.3, 11 of the 13 *Cynicism* items originally classified by Barefoot et al. (1989) have been consistently found to load on Factor 1. Thus Factor 1 can be said to be measuring cynicism. The 11 items appear to measure beliefs about others being ignorant (CYN50, CYN76), blameworthy (CYN286, CYN346, CYN352, CYN470), dishonest (CYN104), Machiavellian (CYN81, CYN110, CYN254) or deceitful (CYN58). In comparison, the two items labelled by Barefoot et al. (1989) that measure cynicism but were not included in the final 12 (i.e., CYN306 and CYN 241) include an assessment of the respondent; CYN241 measures the belief that it is better to be distrustful of others while CYN306 measures the belief that nobody cares for the respondent. These two items implicate the respondent and could be a reflection of the respondent's own assessment of his/her personality in addition to the cynical assessment of others. For example, CYN241 could also tap the distrustful nature of the respondent rather than the evaluation that others are unreliable (i.e., focuses on the evaluation of the respondent's personality rather than the evaluation of others). CYN306 could also implicate the assessment of the respondent as unworthy of concern from others instead of (or in addition to) a cynical assessment of others being unsympathetic. In addition, one *Aggressive Responding* item (AGGR414 - being rough with rude or annoying people) appears to load on this *Cynicism* subscale rather than *Aggressive Responding* subscale. Perhaps the focus of this item is the cynical appraisal of others as rude or annoying rather than on the aggressive behavioural response of the individual.

Other congruencies between these 12 items and the results from the four studies reviewed also merit discussion. For instance, 5 of the 12 items in Factor 1 (Table 3) are included in Greenglass and Julkunen's (1989) 9-item model. The 4 items from Greenglass and Julkunen's model that were excluded from the final 12 are CYN241, CYN306, HATT124 and OTH436. Both CYN 241 and CYN306 were the items that implicated the self with the cynical evaluation of others. CYN306, HATT124 and OTH436 were replicated in 2 out of the 4 studies reviewed, which made them replicable at chance level. Thus, though there was some evidence that these 3 items tend to load on Factor 1, it did not do so consistently above chance level to be included in the final 12. The 5 items from Greenglass and Julkunen's model that were included in the final 12-item model (CYN81, CYN104, CYN110, CYN254, CYN286) were also in the 7 items of Miller et al.'s confirmatory factor analysis (Miller et al., 1995). This indicates that these 5 items measure the same factor. In Miller et al.'s (1995) study, item HATT124 was excluded from the final model due to its low squared multiple correlation in the model. The conclusion of this review coincides with Miller et al.'s decision as this review shows that HATT124 only loaded on Factor 1 in one study reviewed in this chapter (i.e., Costa et al., 1986). Miller et al.'s (1995) study also provides support for items CYN241 and CYN306 loading on Factor 1 - the two items that implicate the respondent in assessing the cynical nature of others. Though this review did not find evidence for the inclusion of these two items in the final 12-item model to be above chance level in the 4 studies reviewed, the possibility of these two items being included should be

investigated further. It remains a higher possibility for these two items to be considered in the final model if subsequent studies report that these two items load on Factor 1.

The majority of items for Factor 1 reported by Steinberg and Jorgensen's (1996) study are included in the final 12 items; out of the 9 items found in Steinberg and Jorgensen's (1996) Factor 1, 8 were included in the final 12 items excluding item HAFT338 (Table 2.3). HAFT338 was found to load onto Factor 1 in two out of the 4 studies reviewed here. This item measures the belief that others frequently do not live up to the expectations of the respondent. It is quite obvious how this item can be interpreted to be an indication of a cynical evaluation of the respondent towards others - the respondent views others as unreliable. However, this item, like CYN241 and CYN306, also implicates the self and could be also a reflection of the respondent's own assessment of his/her personality in addition to the cynical assessment of others. For example, HAFT338 could also reflect the high expectations the respondent have of others, which is not necessarily a cynical evaluation of others. Hence the lack of consistency of CYN241, CYN306 and HAFT338 could be due their ambivalent nature in tapping cynicism mistrust of others and also tapping the respondent's personality.

Twelve of the 19 items reported by Han et al. (1995) that loaded on Factor 1 were also in the final 12 items. A closer inspection revealed that these 12 items tend to have two characteristics: (i) high factor loadings on Factor 1, which indicates the stability of these items on this factor, and (ii) 11 of the 12 items are *Cynicism* items. Out of these 12 items, 10 had

factor loadings of above .40 while 2 (CYN254 and AGGR414) had values close to .40 (.38 and .36 respectively). In contrast, only 8 out of the 14 items that had loadings above .40 in Costa et al.'s (1986) study were included into the final 12. The component saturation for Factor 1 between these two studies (i.e., Costa et al., 1986; Han et al., 1995) are similar (Table 2.2). Both studies also included both genders in their sample and the method of analysis for both studies was principal components factor analysis. Thus, it seems that Han et al.'s (1995) results are more generalisable because of its substantially larger sample size (about 6000) compared to Costa et al.'s (two samples of about 500 each).

To summarise, there is a total of 12 items that consistently load on Factor 1 across the 4 studies reviewed here. Content analysis of the 12 items indicated that 11 of these items measure the respondent's assessment of others as ignorant, blameworthy, dishonest, Machiavellian or deceitful. Items classified by Barefoot et al. (1989) as measuring cynicism but were not included in the final 12 items appear to also measure the respondent's personality in addition to the evaluation of others. On the other hand, an item (AGGR414) that appears to measure both the aggressive response of the respondent (had to be aggressive with others) and an assessment of others as rude or annoying, was found to load consistently on Factor 1. This shows that across the 4 studies reviewed, participants tend to interpret this item by focusing on the fact that others are rude or annoying - a cynical evaluation of others.

On the other hand, *Hostile Affect* (HAFT) and *Hostile Attribution* (HATT) items that cluster in Factor 2 do not do so consistently. From

Table 2.3, it can be seen that HAFT and HATT items are scattered between Factors 1 and 2 inconsistently across the four studies reviewed. There was only one item that was stable for Factor 2 (HATT251) but for Costa et al.'s (1986) study, this item had similar loadings for Factor 1 and Factor 2. The number of items that are found to consistently load on Factor 2 to 4 also appear to decrease.

As can be seen in Table 2.3, 12 items from Cook-Medley Hostility scale loaded on Factor 1 across the four studies reviewed here. *Hostile Affect* and *Hostile Attribution* items also tend to be found together in Factors 1 and 2, which discounts Barefoot et al.'s (1989) proposal that these are separate subscales. *Aggressive Responding* and *Hostile Affect* items tend to load on Factor 3 while Factor 4 tend to composed of one item from *Aggressive Responding* and two items from the *Cynicism* subscale. The results presented in Table 2.3 are congruent with the results from Guadagnoli and Velicer's (1988) study. Factor 1 (*Cynicism*) is most stable because of its high component saturation found in these 4 studies compared to the other factors. In terms of replicability, Factor 1 is first with 12 items that consistently loaded on this factor for 3 out of the 4 studies reviewed. One item consistently loaded on Factor 2 while none where found to be stable for Factors 3 and 4. Therefore, based on this review, it seems likely that the stable component of the Cook-Medley Hostility scale for males is a 12-item subscale of *Cynicism*. The 12 items are concluded to measure cynicism as 11 of the 12 items have been content analysed by Barefoot et. al (1989) and the investigator to be so. Hence, a model where these 12 items load onto a single factor

(*Cynicism*) is hypothesised and tested using structural equation modelling.

METHOD

Sample and Material

The sample consisted of 213 male undergraduates and postgraduates from two Scottish Universities. One hundred and thirty-two males were paid for their participation. Payment was given at a rate of £4 per hour. The average amount received per participant differs due to the participation of experiments of differing duration. Duration of each experiment is given in Chapter 3 and Chapter 4. These 132 males were from one university; ten participated in a pilot study for Study 2, 60 participated in Study 2 (Chapter 3), and 62 participated in Study 3 (Chapter 4). The remaining 81 males were from another university who volunteered for course credits in a Psychology undergraduate course. They filled in the questionnaires in groups of 35 to 41 comprising of mixed genders. However, only responses from the male participants were reported and analysed here. In addition to the Cook-Medley Hostility scale, participants also filled other measures like the DS16 scale (Denollet, 2000) and the Marlowe Crowne Social Desirability scale (Crowne & Marlowe, 1960). Details of what questionnaires the participants filled were given in Chapters 3 and 4. The order of the various scales given to the participants was randomised in each questionnaire booklet.

The 50-item Cook-Medley Hostility scale used for the analysis in this chapter was originally derived to discriminate teachers that had good rapport with students and teachers who did not have good rapport with students (Cook & Medley, 1954). Cook and Medley found that 50 items

from the Minnesota Multiphasic Personality Inventory (MMPI, Hathaway & McKinley, 1951) had such discriminatory function. Response for these 50 items were in a binary 'True/False' format. 'True' responses were coded as '1' while 'False' responses were coded as '0' with the exception of 3 reverse coded items. Unlike some other studies that have used a 4-point likert scale (e.g., Miller et al., 1995; Weidner et al., 1989), this study retained the binary response for the Cook-Medley Hostility scale as used by the MMPI.

Twenty-four items from the Cook-Medley Hostility scale was selected to test various models (see next section *Specification of Models*). Twelve items were derived from the literature review in the introduction section that measure cynicism (12-item Cynicism) while another 12 selected from the remaining 38 items were used as the alternative model (12-item Alternative). Eight participants had missing values for one or more of these 24 items. They were removed from the final sample and this reduced the total sample size to 205. Mean age (SD) of the sample analysed was 22.06 (7.48) years and the mean score (SD) for the 50-item Cook-Medley Hostility scale was 21.92 (5.42). The 12-item Cook-Medley Hostility scale was 6.22 (2.46). Intraclass correlations for the scores derived from the 50-item, Cynicism 12-item and the Alternative 12-item were listed in Table 2.4. The correlations in Table 2.4 indicated that the 50-item Cook-Medley Hostility scale measured several aspects of hostility as shown by the high correlations with 2 of the 12-item versions. The moderate correlation between the two

12-item versions indicated that these two are measuring similar but not identical constructs.

Table 2.4 Correlation and Cronbach Alpha among the 12-item Cynicism, 12-item Alternative, and 50-item versions of the Cook-Medley Hostility Scale (n = 205)

	12-item Cynicism	12-item Alternative	50-item
12-item Cynicism	--	--	--
12-item Alternative	.48*	--	--
50-item	.79*	.79*	--
Cronbach α^{**}	.75	.65	.82

* $p < .01$ using Holm's Sequential method of Bonferroni Type 1 error correction.

** (Cronbach, 1951)

Specification of Models

In addition to testing the 12-items obtained from the review in the introduction section of this chapter an alternative model was created by selecting a set of 12 items from the remaining 38 items. Specifically, the following 12 'alternative' items were selected - SOAV19, AGGR27, OTH46, HATT99, HATT124, HAFT136, SOAV265, SOAV386, AGGR406, OTH347, HAFT419, and OTH457. These 'alternative' 12 items were selected based on their item numbers in the Cook-Medley Hostility scale that were close to the original 12¹⁰. This is ensure that any tedium effects

¹⁰ Item numbers for the original 12 items in the Cook-Medley Hostility questionnaire are 4, 5, 6, 7, 9, 10, 23, 26, 30, 32, 40, 50 while alternative items are 1, 2, 3, 8, 11, 12, 25, 31, 36, 39, 41, and 48.

due to item order was comparable between the original 12 items and the alternative 12 items. In addition, items were also selected from the diverse subscales identified from the item content analysis done by Barefoot et al. (1989). These alternative 12-items were modelled to load onto one factor as well. The purpose of doing so was to demonstrate that the model fit of the original 12 items is not due to the low number of parameters that was estimated by the model nor sample size. This strategy of alternative model testing was identical to what Contrada and Jussim (1992) did to test Greenglass and Julkunen's (1989) 9-item model. Figure 2.1a shows the model for the Cynicism 12-item Model while 2.1b shows the Alternative 12-item Model. It was hypothesised that the model consisting for 12-item Cynicism (Figure 2.1a) will have a better fit than the model for the 12-item Alternative (Figure 2.1b). In order to assess the extent the 2 models tested 'fit' the data, a number of fit indices were chosen as suggested by other researchers (Curran, West, & Finch, 1996; Hoyle & Abigail, 1995; Hu & Bentler, 1995): Scaled χ^2 (Satorra & Bentler, 1988), Nonnormed Fit Index (NNFI) (Bentler & Bonett, 1980), Incremental Fit Index (IFI) (Bollen, 1989), Comparative Fit Index (CFI) (Bentler, 1990) and Root Mean-Square Error of Approximation (RMSEA) (Browne & Cudeck, 1992).

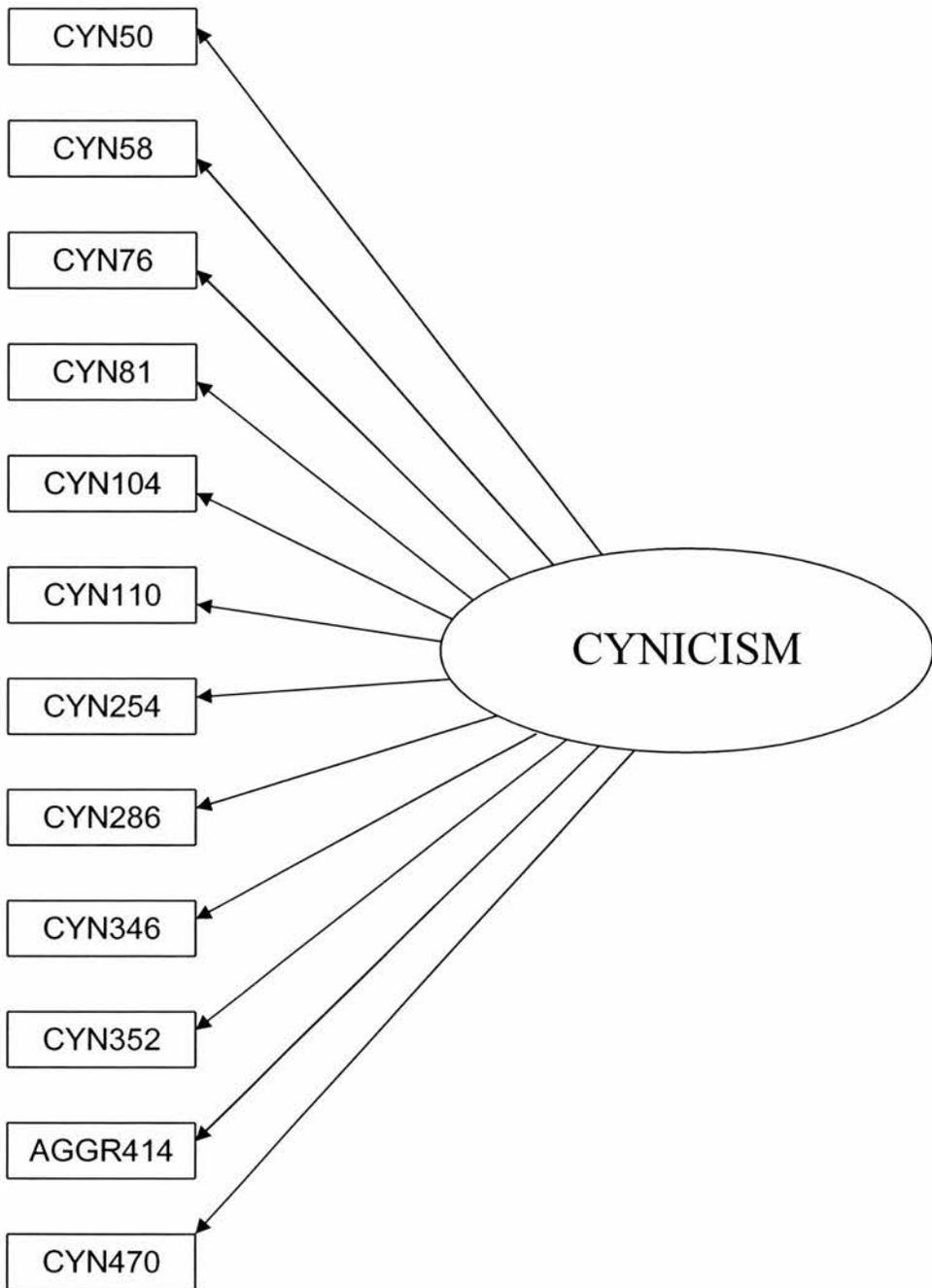


Figure 2.1a The 12-item Cynicism Model

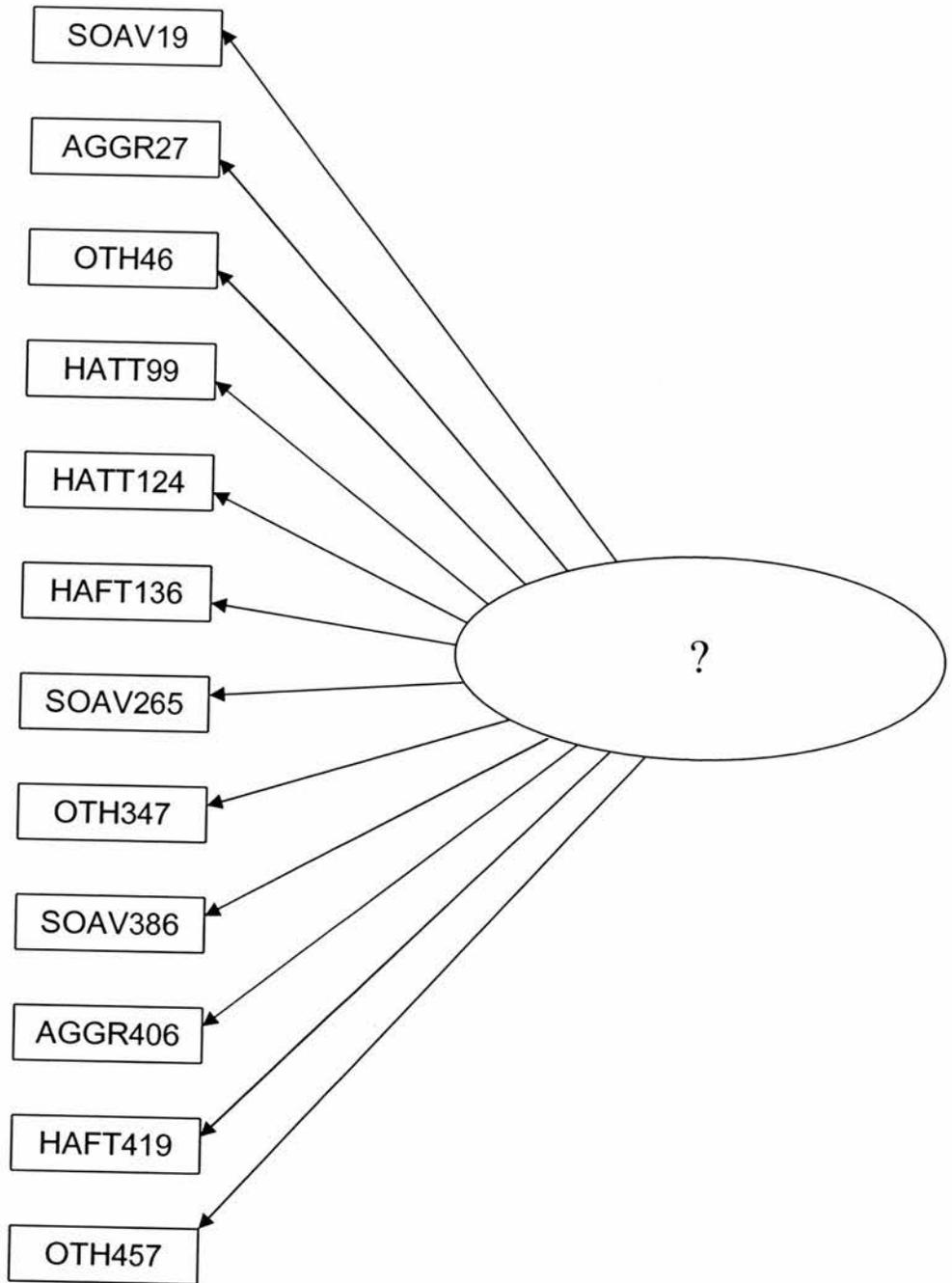


Figure 2.1b The 12-item Alternative Model

For the χ^2 statistic, an alpha level of .01 rather than .05 was taken. This is because the χ^2 statistic is affected by sample size (Hu & Bentler, 1995); it tends to be significant for sample sizes greater than 200 (Schumacker & Lomax, 1996). However the χ^2 statistic remains one of the more commonly reported fit index. Hence it was reported here. For the other fit indices (e.g., NNFI), values .90 and above have often been taken to indicate a good model fit (Bentler & Bonett, 1980; Schumacker & Lomax, 1996). However, this 'absolute' cutoff point for fit indices should be taken with caution as it is sometimes inadequate (may be too stringent or too lax) as a fit index (Hu & Bentler, 1995). The approach taken here was that not only are the fit indices interpreted in absolute terms (e.g., $\geq .90$) but also comparative - by comparing the fit indices for the main model with the alternative model. RMSEA measures the average residual when the correlations generated by the model are compared to the raw data. A value of .05 indicates that the correlations generated by the model have an average error at .05 compared to the raw data. RMSEA $< .05$ indicates a good model fit (Schumacker & Lomax, 1996).

Descriptive Data and Statistical Analysis Strategy

Responses for the 50-item Cook-Medley Hostility scale were dichotomous. Hence the data distribution was nonnormal. Structural Equation Modeling with nonnormal data leads to an inflation of the χ^2 statistic fit index as well as inaccurate parameter estimates and standard errors (West, Finch, & Curran, 1995). A method to overcome this problem

is to use the Maximum Likelihood Robust estimation procedure in EQS version 6.1 (Encino, CA: Multivariate Software, Inc.). This method of estimation derives a polychoric correlation matrix for the dichotomous data and corrects fit indices and standard errors with nonnormal distributed data (Curran et al., 1996; Hu & Bentler, 1995). Corrected fit indices, like the Scaled χ^2 (Satorra & Bentler, 1988) have been found to perform adequately when normality is violated. In addition, the Maximum Likelihood estimation method also tend to perform consistently well in small to moderate samples with nonnormal data (Hoyle & Abigail, 1995). Another advantage of using this estimation method for Structural Equation Modeling is that the Nonnormed Fit Index (NNFI) (Bentler & Bonett, 1980) derived from this method has also been found to be independent of sample size (Marsh, Balla, & MacDonald, 1988).

RESULTS

Appendix 1 and 2 presents the polychoric correlation matrix for testing the 12-item Cynicism Model and the 12-item Alternative Model. The results for the 12-item Cynicism model indicated adequate fit, SCALED χ^2 (df = 53) = 67.26, $p = .09$, NNFI = .90, IFI = .93, CFI = .92, RMSEA = .036 (CI: .000 to .060). Table 2.5 shows the unstandardised parameter estimates for this model. RMSEA was .036 indicating that this model produced correlations that had an average of .036 difference with the actual correlation matrix. It was also noted that the parameter estimate for CYN352 was the lowest of the 12 items (Table 2.5). Wald

statistic suggested that this parameter can be dropped from the model without a significant change in the χ^2 statistic, $\chi^2(df = 1) = .15, p = .70$. However, this item was still retained because the literature review in the introduction of this chapter found this item to consistently load on this factor and Barefoot et al.'s (1989) content analysis of this item indicated that it measured cynicism. On the other hand, the 12-item Alternative model (Figure 2.1b) showed poor fit, SCALED $\chi^2(df = 53) = 86.53, p = .002$, NNFI = .69, IFI = .78, CFI = .75, RMSEA = .056 (CI: .033 to .076). Table 2.6 lists the unstandardised parameter estimates, corrected standard errors and error variances. Though compared to the 12-item Cynicism model, this 12-item Alternative model had a similar number of significant parameter estimates, the overall fit of this model was markedly poorer than the 12-item Cynicism model. All fit indices were less than .90 and the SCALED χ^2 statistic was significant. This indicated that the correlations derived from this model differed significantly from the correlation matrix of the raw data. This is also supported by the fact that the RMSEA was greater than .05 and its confidence interval are higher than that obtained from the 12-item Cynicism model. This showed that the correlations deviated on average by .056 from the actual correlation matrix - an indication of poorer fit.

Table 2.5 Unstandardised Parameter Estimates (Corrected SE) and Error Variance for 12-item Cynicism Model

12-Item Cynicism Model		
	Factor Loading (corrected SE)	Error Variance (corrected SE)
F1, F1	--	.20 (.11)
1. CYN50	.48 (.29)	.95* (.05)
2. CYN58	1.03* (.38)	.79* (.08)
3. CYN76	1.06* (.35)	.77* (.09)
4. CYN81	1.85* (.52)	.31* (.11)
5. CYN104	1.19* (.39)	.71* (.11)
6. CYN110	1.84* (.53)	.32* (.11)
7. CYN254	.98* (.33)	.81* (.08)
8. CYN286	.83* (.05)	.86* (.09)
9. CYN346	.56 (.31)	.94* (.07)
10. AGGR414	.41 (.25)	.97* (.04)
11. CYN352	.10 (.39)	1.00* (.01)
12. CYN470	1.20* (.39)	.71* (.10)

* $p < .05$

Table 2.6 Unstandardised Parameter Estimates (Corrected SE) and Error Variance for 12-item Alternative Model

12-Item Alternative Model		
	Factor Loading (corrected SE)	Error Variance (corrected SE)
F1, F1	--	.20* (.09)
1. SOAV19	.78* (.30)	.88* (.08)
2. AGGR27	.75* (.27)	.89* (.07)
3. OTH46	.35 (.29)	.98* (.04)
4. HATT99	1.12* (.06)	.75* (.14)
5. HATT124	1.96* (.53)	.24 (.15)
6. HAFT136	.93* (.33)	.83* (.09)
7. SOAV265	1.29* (.35)	.67* (.11)
8. OTH347	.73* (.28)	.89* (.07)
9. SOAV386	-.29 (.24)	.98* (.03)
10. AGGR406	.75* (.30)	.89* (.08)
11. HAFT419	1.05* (.33)	.78* (.09)
12. OTH457	.60 (.31)	.93* (.06)

* $p < .05$

Overall the results showed that 12-item Cynicism model had a better fit than the 12-item Alternative model. This better fit of the Cynicism model could not be attributed to the characteristics of the model specified (e.g., number of parameters to be estimated) nor the sample size. This was because the Cynicism model had an equivalent number of parameters to be estimated as well as the same sample size compared to the Alternative model. Table 2.7 summarises the results for the two models tested.

Table 2.7 Goodness of Fit Indices for the 2 Models Tested ($n = 205$)

	χ^2 (df = 54)	NNFI	IFI	CFI	RMSEA (CI)
Cynicism Model	67.26	.90	.93	.92	.036 (.000 to .060)
Alternative Model	86.53*	.69	.78	.75	.056 (.033 to .076)

* $p < .005$

Post Hoc Analysis. Data for the Marolwe-Crowne Social Desirability scale and the DS16 Negative Affectivity scale was available for 131 and 121 participants respectively. With the Cynicism model showing better fit than the Alternative model, the 50-item, 12-item Cynicism, and 12-item alternative versions were correlated with these scales to investigate the discriminant validity of these three versions of the Cook-Medley Hostility scale.

Table 2.8 Correlations between the 3 versions of the Cook-Medley Hostility scale, social desirability and negative affectivity

Cook-Medley Hostility scale version	full 50-item	12-item Cynicism	12-item Alternative
Marlowe-Crowne Social Desirability, $n = 131$	-.47*	-.36*	-.46*
DS16 - Negative Affectivity, $n = 121$.41*	.29*	.48*

* $p < .01$ using Holm's Sequential method for Bonferroni Type 1 error correction.

Using the 12-item Cynicism version did not reduce the correlation between Cynicism and social desirability but the correlation between 12-item Cynicism and negative affectivity was lower (8% shared variance) compared to the 50-item version (17% shared variance). Unstable items, represented by the 12-item Alternative version contributed to the high correlation with negative affectivity (23% shared variance) of the 50-item version (Table 2.8). The pattern of results suggested that Cynicism was negatively related to social desirability. In addition, unstable items in the scale contributed to the poor discriminant construct validity of the scale with negative affectivity.

Study 1 Discussion

There is an increasing number of studies that have reported null associations between hostility (measured by the Cook-Medley Hostility scale) and cardiovascular reactivity (Burns & Katkin, 1993; Carroll et al., 1997; Felsten, 1995; Felsten, 1996; Fichera & Andreassi, 1998; Weidner et al., 1989)¹¹. Two reasons were proposed in Chapter 1 to explain for the existence of these null findings. Firstly, the Cook-Medley Hostility scale may have substantial measurement error, leading to low power and reliability. Hence, some studies may not have sufficient statistical power to detect the hostility-cardiovascular stress reactivity effect. The solution to this problem is to improve the scale by increasing its internal reliability and reducing its measurement error (i.e., improving its precision). Secondly, there could exist moderator variables between hostility and cardiovascular reactivity. This chapter deals with some of the measurement issues raised in Chapter 1 about the Cook-Medley Hostility scale.

Two issues regarding the measurement of cynical hostility using the Cook-Medley Hostility scale were discussed in Chapter 1. Firstly, the Cook-Medley scale did not originate as a measurement of hostility and though much research had been done to pin down the construct validity of this scale, its psychometric structure remains elusive. This creates problems for the reliability for this scale; if researchers are not able to

¹¹ For Weidner et al.'s (1989) study, the non-significant result refers to Cook-Medley Hostility scale being entered into the regression model as a continuous variable.

determine with empirical consistency the ability of the 50 items of the Cook-Medley scale to measure the proposed construct(s) then there is a limitation as to what psychological trait (if any) this scale measures that predicts health and disease. Questionnaire items should be consistent in the construct it measures. Secondly, this scale alone is not able to distinguish 'true' low cynical individuals and repressed cynical individuals (i.e., cynical individuals indicating low cynical attitudes due to positive self-presentation bias). This chapter addresses the first measurement issue of this scale while Chapter 4 addresses the second.

The 50-item Cook-Medley Hostility scale was derived to distinguish good and bad teachers. The interest in this scale increased as more studies reported significant associations between this scale and poor health outcome (e.g., Barefoot et al., 1989; Joesoef, Wetterhall, DeStefano, & Stroup, 1989; Räikkönen et al., 1999; Vahtera, Kivimaki, Uutela, & Pentti, 2000). Even though Cook and Medley carried out content analysis of the MMPI to derive the 50-item Cook-Medley Hostility scale, another study that also content analysed the 50-items using clinical psychologists concluded that the 50-items measured 5 related dimensions of hostility (Barefoot et al., 1989). Barefoot et al.'s (1989) study concluded that there were 5 subscales that measured *Cynicism*, *Hostile Affect*, *Hostile Attribution*, *Aggressive Responding* and *Social Avoidance*. The researchers could not identify the construct tapped by the remaining 7 items and these items were placed under a subscale labelled as '*Others*'. The proposition that the 50-item Cook-Medley scale measures more than one dimension of hostility is supported by at least

four studies that have shown that factor analysing the 50-item Cook-Medley Hostility scale revealed more than one factor (Costa et al., 1986; Greenglass & Julkunen, 1989; Han et al., 1995; Steinberg & Jorgensen, 1996). However, the four studies that have factor analysed the Cook-Medley Hostility scale failed to replicate all the subscales proposed by Barefoot et al. (1989). The review of these four studies done in the introduction section of this chapter showed that *Hostile Affect* and *Hostile Attribution* items as classified by Barefoot et al. (1989) tend to load onto the same factor. Some items of these subscales also tend to load together with *Cynicism* items. *Aggressive Responding* items also tend to load onto the same factor as *Hostile Affect* and *Hostile Attribution* items. Some *Aggressive Responding* items also have been reported to load on a third factor. From the review of the 4 factor analytic studies, there was only evidence to conclude that 11 of the 13 items proposed by Barefoot et al. (1989) to measure cynicism were consistent. The fact that items of the Cook-Medley Hostility scale load onto factors that are contrary to Barefoot's classification is less of a problem than the lack of consistency of the items' factor loadings.

Internal consistency of a questionnaire is of vital importance in psychometric assessment. The score for the Cook-Medley Hostility scale is derived from the summation of the its items. If all the items measure the same construct or dimensions of related constructs consistently, then the researcher is able to interpret the construct represented by the score (Steinberg & Jorgensen, 1996). Taking Barefoot et al.'s item content classification to aid in the presentation of this example, if a participant

scores 19 of which 12 points came from *Cynicism*, 4 from *Aggressive Responding* and 3 from *Hostile Attribution*, this would be different from another participant whose score of 19 came from 6 *Cynicism*, 6 *Hostile Affect*, 6 *Hostile Attribution* and 1 *Other*. If the items measure the different subscales consistently, then the researcher can attempt to focus on specific aspect of the scale (e.g., *Cynicism*) in order to reduce ambiguity in using the overall score. The review of the four studies showed that only 13 items showed consistent factor loadings occurring above chance level (i.e., occurring in 3 of the 4 studies reviewed). Of these 13 items, 12 items loaded consistently in Factor 1 and 1 item loaded consistently on Factor 2. Barefoot et al.'s (1989) item content classification suggests that 11 of the 12 items in Factor 1 are measuring cynicism. Closer inspection of the 11 items and the items that were not included revealed that these 11 items focused on the participant's evaluation of others as ignorant, blameworthy, dishonest, Machiavellian or deceitful. Barefoot et al.'s (1989) other 2 *Cynicism* items not included in the final *Cynicism* 12-item model tended to focus on or include the assessment of the respondent's personality traits. This implies that the measure of *Cynicism* as derived from this chapter taps the negative evaluation of others with little (only 1 item, AGGR414, in the *Cynicism* 12-item model) reference to the respondent. The practicality of testing Factor 2 is absent due to the fact that only 1 item was found to load onto this factor consistently. Hence, the *Cynicism* 12-item model was tested using structural equation modelling.

The purpose of employing structural equation modelling is its a priori approach in factor analysis (confirmatory factor analysis). Structural

Equation Modelling requires the researcher to define the relationships between the items and construct(s), as well as well any relationships between the different constructs. In this chapter, only confirmatory factor analysis was done - the measurement model of Structural Equation Modelling. The results showed that the Cynicism 12-item fit the data at an acceptable level. The fit indices were at the minimal level of .90 or $p > .01$ and the residuals indicated a good fit of less than .05. On the other hand, the Alternative 12-item model was a much poorer fit - all fit indices were below .90 or $p < .005$. The residuals from the Alternative 12-item model was also larger than .05 indicating that this model produced correlations that fit less well with the actual data. The poorer fit of the Alternative model indicates that the good fit of Cynicism 12-item model can neither be attributed to sample size nor the number of parameters estimated. This is because the two models tested had identical sample size and number of parameter estimates.

It was contrary to expectation that item AGGR414 of the Cynicism 12-item model did not have low loading for the Cynicism factor but item CYN352 did. This was because item AGGR414 appears to tap both the appraisal of others as annoying as well as the respondent being aggressive while the other 11 items focused primarily on the evaluation of others as negative with no direct reference to the self. The Wald statistic showed that the removal of item CYN352 would not change the χ^2 statistic significantly suggesting that this parameter is superfluous. However, removal of parameter(s) from a model (i.e., model re-specification) should be made with caution and should be supported by

theoretical and a priori empirical evidence. This is in order to prevent researchers from 'fishing' a significant model from a non-significant one. More importantly, unjustified model re-specification reduces the confirmatory nature (hence, the replicability) of the results obtained (MacCallum, 1995). The review from the introduction of this chapter shows that there is evidence supporting the consistency of CYN352 loading on this factor across the 4 studies. Content analysis of this item done by Barefoot et al. (1989) and in this chapter has also found this item to be congruent with measuring cynicism. Hence, a decision was made to retain this item in the Cynicism 12-item model.

The review and results of this chapter showed that (i) there is much inconsistency in the 50-items of the Cook-Medley scale, and (ii) only 13 of the 50 items showed reliability of which only 12 items measure cynicism, and the other factor had only 1 item. These findings caution towards using the summation of the 50-item Cook-Medley Hostility scale to tap cynicism. Using the 50-items can be misleading in a way that it is unclear what the score actually represents between participants. This could be one of the reasons why conflicting results have been obtained between the summation score based on the 50-items of the Cook-Medley Hostility scale and cardiovascular reactivity to stress. As the 50-item summation score may represent different combinations of items from the scale, its construct validity may change from sample to sample or individual to individual. This is supported by the results reported by Steinberg and Jorgensen that showed differences in item loadings on the factors obtained for males and females (Steinberg & Jorgensen, 1996).

Hence, based on the literature review and the results obtained from this chapter, it is suggested that future usage of the Cook-Medley Hostility scale tap cynicism via the 12 items in the Cynicism 12-item model.

Cronbach alphas have frequently been reported for inter-item reliability of this scale (Cronbach, 1951). Studies have reported that the 50-item Cook-Medley Hostility scale have a Cronbach alpha of about .80 (Bishop & Quah, 1998; Greenglass & Julkunen, 1989; Smith & Frohm, 1985). This study also replicated this Cronbach alpha value for inter-item reliability for the 50-item Cook-Medley Hostility scale. If the inter-item reliability for the 50-item Cook-Medley is poor, as shown by the lack of inter-item consistency in the review of factor analytic studies in this chapter, why have researchers reported such high inter-item reliabilities in the form of Cronbach alphas? Cronbach alpha is heavily influenced by systematic error and factor loadings (Shevlin et al., 2000). The presence of systematic error can inflate Cronbach alpha. Systematic errors can occur when each item in the questionnaire measure more than one latent variable. In fact, the review of the 4 factor analysis studies review that this is highly likely as many of the items load on different factors for different studies. For example, both items HAFT338 and HATT124 have been reported to load on Factor 1 as well as Factor 2 (Table 2.1). In other words, the more multidimensional the questionnaire items are (i.e., each item measures more than one construct), the higher the Cronbach alpha will be. Thus, discarding questionnaire items that loaded on different factors for different studies reduce such correlated errors and doing so will reduce Cronbach alpha inflation. Shevlin et al. (2000) concluded from

their Monte Carlo simulation that a Cronbach alpha of .80 for a scale measuring self-esteem could mean three things:

“1) The scale is measuring only self-esteem with a reliability of 0.8.

2) The scale is measuring self-esteem but the items that comprise the scale are not tau-equivalent. Therefore 0.8 is a conservative estimate of reliability.

3) The scale may be measuring self-esteem and a number of other variables. In this case, a reliability of 0.8 represents the reliability of self-esteem and all the other variables; the reliability of the self-esteem factor will be less than 0.8.” (p.236).

From the evidence reviewed in this chapter, it appears that points (2) and (3) are likely reasons to explain for the discrepancy between the different Cronbach alphas for the different versions of the Cook-Medley Hostility scale. This section discussed point (3) while the next paragraph discusses point (2). The high Cronbach alpha for the 50-item version of the Cook-Medley Hostility scale is probably due to the inclusion of a large number of questionnaire items that have correlated errors. As the number of items that have correlated errors are reduced (as in the two 12-item versions), the Cronbach alpha will be reduced. A similar reduction of Cronbach alpha as obtained in this chapter was also reported by Greenglass and Julkunen's (1989) 9-item version (50-item version $\alpha = .84$, 9-item $\alpha = .77$). However, discrepant findings were reported by Miller et al. (Miller et al., 1995). Contrary to the results reported in this chapter and Greenglass and Julkunen's (1989), Miller et al. reported high Cronbach alpha (.82) for their 8-item model (akin to Greenglass and

Jukunnen's 9-item model). This is probably due to the differences between Miller et al.'s study and Greenglass and Julkunen's paper (1989) and this chapter. Firstly, sample size was substantially larger for Miller et al.'s paper ($N_1 = 1089$ and $N_2 = 1072$) compared to Greenglass & Julkunen's ($N = 263$) and this chapter's ($N = 205$). Secondly, Miller et al. changed the response format of the scale from a dichotomy to a 4-point Likert scale. It is not known with certainty how this change in response format would affect the results in factor analysis. Thirdly, the factor loadings reported by Miller et al. were much higher than those reported elsewhere (standardised factor loadings for the two samples were about .80). These three differences could lead to a correlation matrix that is more normally distributed for the Miller et al. study, enabling a better model fit of the questionnaire items to the latent variable. However, though the 12-item versions' lower Cronbach alphas may be due to less correlated errors, this still does not mean that for the 12-item Cook-Medley versions the Cronbach alpha is a good indication of reliability. This issue will be discussed next.

There is also the discrepancy between the Cronbach alpha for the two 12-item versions and the results from the Structural Equation Modelling. Specifically, why is the Cronbach alpha low for these two 12-item versions while Structural Equation Modelling show that the Cynicism 12-item version is better than the Alternative 12-item version? The Cronbach alpha actually tests a very restricted model where all questionnaire items load onto the Cynicism factor with equal weights (tau-equivalence) and equal error. As the factor loading for the questionnaire

items fall below .80, the lower the Cronbach alpha would be (Shevlin et al., 2000). The review of the 4 factor analytic studies clearly discounts this tau-equivalent model - the majority of items had factor loadings within the regions of .59 and below (Table 2.2). The results of the Structural Equation Modelling in this chapter revealed that if the 12 questionnaire items were not restricted to have equal factor loadings and error variances, the 12-item Cynicism version will have a better fit of the data than the 12-item Alternative version. On the other hand, no amount of change in the factor loadings given to the items of the 12-item Alternative model will provide a good data fit. The corollary of this is that a summation or average of all the 12-items is actually an incorrect extension of the results from the Structural Equation Modelling. This is because a summation or average of the results assumes that each 12 item contributes equally to the measurement of the construct. The results reviewed and presented in this chapter clearly refutes this. The use of weighted item scores (Drewes, 2000) derived from the results of the Structural Equation Modelling is a more appropriate inference from the results reported here. The results from EQS assuming a 1-factor model (Bentler, 1968) reported that an inter-item reliability of .86 can be achieved using the following item weights:

$$\begin{aligned} \text{Total Cynicism Score} &= .089\text{CYN50} + .233\text{CYN58} \\ &+ .244\text{CYN76} + 1.080\text{CYN81} + .294\text{CYN104} + \\ &1.040\text{CYN110} + .215\text{CYN254} + .172\text{CYN286} + \\ &.107\text{CYN346} + .076\text{AGGR414} + .017\text{CYN352} + \\ &.301\text{CYN470} \end{aligned}$$

It can be seen from the formula that items with higher standardised weights (e.g., CYN81, CYN110) in the Structural Equation Model results contribute more to the final score than weaker items (e.g., CYN352). Coincidentally, a reliability of .84 for the 12-item Alternative can also be achieved by using item weights but it should be noted that the factor loadings obtained for this model fit the data poorly, as shown by the low goodness of fit indices. Also visible from the equation is that most of the Cynicism 12 items have low weights that are substantially different from '1' (the value assumed by Cronbach alpha). This explains for why the Cronbach alpha for this 12-item version is low. However, for the purposes of this dissertation, the summation of all the 12 items is used to measure Cynicism because the use of item weights has yet to gain wide usage. Thus, generalisation to published studies may be difficult when item-weighted total score is used.

The previous paragraphs have proposed that the high Cronbach alpha reported by previous published studies arose from the inclusion of a high number of multidimensional questionnaire items in the Cook-Medley Hostility scale, as evidenced by the 4 factor analytic studies, which inflates the Cronbach alpha. As questionnaire items become more unidimensional, Cronbach alpha will be a more accurate index of reliability. However, the Cronbach alpha also assumes equal factor loadings and equal error. The greater the violation of this assumption, the lower the Cronbach alpha will be. The 4 factor analytic studies as well as the results from this chapter clearly do not support this assumption empirically. Hence, while Cronbach alpha may be low for the Cynicism

12-item version, a less restrictive model based on differential item weights to derive a total score is a more appropriate extension of the findings reported in this chapter. However, the lack of wide usage of using item weights to derive a total score in hostility research limits its ability to compare and contrast with past studies. Hence, the summation of the 12 items without item weights is used in this dissertation.

If the 50-item version of the Cook-Medley Hostility is filled with so much inter-item inconsistencies and measurement error, then how does this be resolved with studies that have reported test-retest reliability and construct validity for this scale? For example, Barefoot et al (1989) has reported significant correlations of the 50-item Cook-Medley scale with the Hostility scale and Agreeableness factor of the NEO PI. Other studies have shown that the Cook-Medley Hostility scale is also significantly associated with trait anger expression and experience (affect component of hostility) as measured by the Spielberger's State Trait Anger Expression Inventory (Bishop & Quah, 1998; Greenglass & Julkunen, 1989). Studies have also reported that this measure tend to have a good test-retest reliability of .80 (Bishop & Quah, 1998; Greenglass & Julkunen, 1989; Smith & Frohm, 1985). The evidence for construct validity and test-retest reliability as analysed and reported in the cited studies is based on total-item reliability, while what was discussed before was inter-item reliability. Total-item reliability involve a summation of all the items in the scale (or relevant items in the scale in the case of Barefoot's (1989) 27-item version) and using the summation score for test-retest reliability and construct validation.

Construct validation using the total-item score does not necessarily imply inter-item reliability. A case to point is when 100 items have zero inter-item reliability but each item correlates with the other scale at .10, the total-item score can then correlate with the other scale perfectly at $r = 1.00$ (Guilford, 1954). Greenglass and Julkunen's (1989) paper shows this effect as well. The researchers reported that inter-item correlations of the 50-item Cook-Medley Hostility scale were low with an average of .09 (range: -.31 to .58). Yet this 50-item scale correlated significantly with ideas of persecution at .60. The independence between inter-item reliability and construct validity using total-item score is a double-edged sword. On the one hand, this independence enables the resolution of the 'contradiction' between studies that have found construct validity using the total-item score and the studies that have found little evidence for inter-item reliabilities in factor analysis. On the other hand, improving the inter-item reliability of the Cook-Medley Hostility scale may not reduce the total-item correlation with other nonhostility-related constructs (e.g., neuroticism). However, some improvement in the discriminant validity was found for the 12-item Cynicism scale. The correlations done with the 50-item, 12-item Cynicism and 12-item Alternative versions of the Cook-Medley scale revealed that using the 12-item Cynicism reduced the correlation with negative affectivity. The unstable 12-item Alternative contributed to the poor discriminant construct of the scale.

Moreover, improving inter-item reliability via the Cynicism 12-item version enables greater confidence at what is being measured more reliably than using the 50-item version. Even though the moderate

correlations between the 50-item Cook-Medley Hostility scale and other scales do suggest convergent construct validity, the scale's discriminant construct validity is questionable (Eckhardt et al., 2004). The Cook-Medley Hostility scale does not just measure the respondent's cynical mistrust of others. A significant moderate correlation between this scale and Neuroticism of the NEO PI suggests that it also measures negative affect, propensity to have irrational thoughts, and emotional maladjustment (Costa, Jr. & McCrae, 1992). Negative affect encompasses a broader range of negative emotional reactions in addition to anger; it includes anxiety (NEO PI's Neuroticism scale 1) and depression (NEO PI's Neuroticism scale 3) as well. Thus, though convergent validity for the 50-item Cook-Medley Hostility scale is present, it involves the total-item score. Construct validity using the total-item score does not imply inter-item reliability. In addition, this scale is also lacking in discriminant validity. Construct validity represents one form of analysis involving total-item score, the next section discusses inter-item reliability and test-retest reliability.

There is little point in conducting test-retest reliability if there is uncertainty in terms of inter-item reliability. If 38 items of the Cook-Medley Hostility scale have not been found to consistently measure the same hostility dimensions, then what does it mean to replicate the same total score reliably twice? The researcher is unable to distinguish whether a score of '38' obtained reliably for a participant in Time 1 and Time 2 is contributed by (i) the same items measuring the same hostility dimensions in Time 1 and Time 2, (ii) the same items measuring different

hostility dimensions in Time 1 compared to Time 2, (iii) different items measuring the same hostility dimensions in Time 1 and Time 2, and/or (iv) different items measuring different hostility dimensions in Time 1 and Time 2. The above combinations can have potential serious implications. For example as was mentioned earlier in this discussion, similar scores obtained during two time points can measure different combinations of hostility dimensions. A reduction of ambiguity by item selection can only be done if the item consistently measure the construct. Thus, only when inter-item reliability is stable will test-retest reliability make sense.

One of the potential limitations from this study may be that the results obtained from the analysis were derived from an all-male sample. This may limit the generalisation of the 12-item Cynicism model to males. However, there is reason to believe that the same model will probably be valid for females; three of the four studies reviewed in the introduction section of this chapter where the 12-item Cynicism model was derived sampled an almost equal number of males and females. Only one study reported the results of their factor analysis by gender (Steinberg & Jorgensen, 1996). Factor 1 of this study was similar across genders. Thus it is likely that the 12-item Cynicism model will be valid for females as well. However, this issue has to be resolved empirically and future studies may wish to pursue this avenue of research.

Secondly, the use of the 12-item model to tap Cynicism has problems as well. Being a relatively new model, it is unclear how this 12-item Cynicism version relates to the other versions recommended by other researchers and also how it relates to cardiovascular health as

reported by other researchers using different versions of the Cook-Medley Hostility scale. The 12-item Cynicism version recommended in this chapter does not have much in common with the other versions reviewed. It is unclear to what extent studies that have reported associations between the other versions of the Cook-Medley scale and health can be generalised to the current 12-item Cynicism version. For example, it is difficult to determine how interventions aimed at reducing hostility identified using Barefoot et al.'s (1989) 27-item version that showed reduction in blood pressure (Gidron et al., 1999) can be generalised to the current 12-item Cynicism version. However, there are suggestions that this 12-item Cynicism version should be significantly related to cardiovascular health. This is because the 12-item Cynicism version has close resemblance to Barefoot et al.'s (1989) 13-item *Cynicism* subscale. Both Barefoot's (1989) and this 12-item Cynicism version share a large overlap of 11 items. Barefoot et al. (1989) found that their *Cynicism* subscale was significantly related to mortality for 128 lawyers at a 29-year followup. Brondolo et al. also reported data for Barefoot et al.'s *Cynicism* subscale (Brondolo et al., 2003). Brondolo et al. found those individuals with high cynicism, negative ratings of their social interactions were significantly associated with ambulatory diastolic blood pressure. This was not significant for participants with low *Cynicism* scores. However, further research into such significant associations between this 12-item Cynicism version, cardiovascular reactivity and health is required. Thus, Study 2 and 3 (Chapters 3 and 4) aims to

include this 12-item Cynicism in order to test its relationship with cardiovascular stress reactivity.

Thirdly, one of the studies reviewed in the introduction section that helped derive the 12-item Cynicism version only included 27 items of the original 50 items of the Cook-Medley scale in their factor analysis (i.e., Steinberg & Jorgensen, 1996). Though this does not invalidate the findings of this chapter, it implies that the 12-item Cynicism version may be conservative. This means that there could have been more than 12-items being included in the final model had Steinberg and Jorgensen included all the 50 items in their analysis. However due to their statistical analytic approach, the reduction of items was required to ease computation and increase power. As an extension of this point, the 12-item Cynicism version was derived from the 4 studies reviewed. Future studies that published their factor analysis results of this scale may cause an alteration in the items of this version or increase the number of items in this version. However, since the generalisability and reliability of items' factor loadings helped derive the 12-item Cynicism version, major elimination of the items from the 12-item Cynicism version is not expected to occur. Item CYN352 may be such a potential candidate for future elimination given its low item loading on Factor in the Structural Equation Modelling done in this chapter.

To summarise, the main aim of this chapter is to address the inter-item measurement precision of the Cook-Medley Hostility scale. This is done so as to reduce measurement error and increase its power and internal reliability and thereby reducing the number of discrepant findings

involving this scale. A review of the four studies that have conducted factor analysis on this scale reviewed little inter-item reliabilities. What was concluded was that there were 12 items that loaded on Factor 1 reliably and 1 item that loaded on Factor 2. The practicalities of statistical analysis allowed only the investigation of Factor 1 since Factor 2 had only 1 item. Content analysis by Barefoot et al. (1989) and the current investigator found that the items measure the cynical appraisal of others as ignorant, blameworthy, dishonest, Machiavellian or deceitful. Thus, these 12-items were then hypothesised to measure the factor of Cynicism. Results of the Structural Equation Modelling revealed that this model fit the data well. A comparison with an 12-item Alternative model found that the good model fit associated with the 12-item Cynicism model could not be due to the number of parameters in the model specified nor sample size. High Cronbach alphas reported for the full 50-item scale are based on assumptions (i.e., unidimensional questionnaire items or random errors, equal factor loadings and error variances) that are unlikely to be true for this questionnaire and may inflate the Cronbach alpha. Previous studies that have reported construct validity and test-retest validity are based on total item score and this does not necessarily imply high internal (inter-item) consistency. Though the 12-item Cynicism version had little in common with most of the other versions reviewed in the introduction section of this chapter, there is some suggestion that this version may predict cardiovascular disease and mortality.

The laboratory study in the next chapter attempts to address some of the concerns raised in this chapter in the following ways: (i) determine

the correlation between the 12-item Cynicism version and a behavioural measure of hostility from the Structured Interview (i.e., the Interpersonal Hostility Assessment Technique), (ii) investigate the relationship between the two measures of hostility and cardiovascular stress reactivity/variability, and (iii) investigates the role of control as a mediator between hostility and cardiovascular stress reactivity/variability. Both laboratory studies in Chapters 3 and 4 will use the 12-item Cynicism version of the Cook-Medley Hostility scale.

CHAPTER 3: STUDY 2

INTRODUCTION

There is evidence that hostility predicts poorer cardiovascular health (Barefoot et al., 1983; Dembroski et al., 1989; Julkunen et al., 1994; Markovitz, 1998; Matthews et al., 2004). Hostility, defined as an 'enduring ill will and a negative view of others' (Houston, 1986), comprises of a cognitive (Cynicism) component, an affect (Anger) component and a behavioural (Aggression) component (Spielberger et al., 1985). This chapter assesses cynicism using the 12-item Cynicism scale from the Cook-Medley Hostility scale (Cook & Medley, 1954) and the behavioural component (Verbal Aggression) of Hostility using the Structured Interview of Hostility assessment (Haney et al., 1996). These two measurements of Hostility have been chosen because they both represent two aspects of Hostility and both Hostility assessments have been found to be significantly related to cardiovascular disease in a meta-analysis (Miller et al., 1996).

A 12-item version of the Cook-Medley Hostility scale is used in this dissertation. Based on the literature review and confirmatory factor analysis in Chapter 2, it was concluded that 12 items selected from the original 50 of this questionnaire, measures Cynicism reliably. Hence this Cynicism 12-item scale is adopted for this Chapter and the next to measure Cynicism. On the other hand, the Structured Interview-based method of hostility assessment was developed from Type A Behaviour

assessment but as later research found that it was the vocal stylistics component that predicted health outcomes (Dembroski et al., 1989), subsequent revisions focused on the assessment of this aspect of the interviewee. One such revision has been the Interpersonal Hostility Assessment Technique (IHAT; Haney et al., 1996). Scores derived from IHAT predict poorer cardiovascular health outcomes as well (Haney et al., 1996; Matthews et al., 2004; Siegman et al., 2000).

Two theories that researchers have proposed to explain for the higher cardiovascular disease risk among high hostile individuals were discussed in Chapter 1. The two theories are the Cardiovascular Reactivity Hypothesis (Manuck & Krantz, 1986) and the Cardiovascular Variability Hypothesis (Sloan et al., 1999). Researchers of the Cardiovascular Reactivity Hypothesis proposed that high hostile individuals are more likely to develop cardiovascular disease because they have greater cardiovascular stress response. Thus, greater cardiovascular reactivity of high hostile individuals during stress increases the chance of injury to the cardiovascular system, which hastens the development of cardiovascular disease. Researchers of the Cardiovascular Variability Hypothesis approached to explain for the hostility-cardiovascular disease link from another perspective; since the cardiovascular system is homeostatic, a large variation in some cardiovascular functions may indicate a dysfunction in the cardiovascular system in achieving this homeostasis. This homeostatic dysfunction of the cardiovascular system then increases the risk of developing cardiovascular disease.

As discussed in Chapter 1, the two hypotheses have similarities and differences in the way cardiovascular functioning is operationalised and analysed in this dissertation. For the Reactivity Hypothesis, blood pressure and heart rate during stress is compared to a baseline while for cardiovascular variability, spectral analysis of heart rate and an assessment of baroreflex sensitivity (known to affect blood pressure variability) during stress are done. Moreover, spectral analysis of heart rate also enables the differentiation of sympathetic and parasympathetic influences on heart rate during stress (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996).

For the Reactivity Hypothesis, studies have shown that heightened cardiovascular stress arousal is associated with cardiovascular disease risk (e.g., Light et al., 1992). For the Variability Hypothesis, a decreased baroreflex sensitivity are associated with cardiovascular disease (Frattona et al., 1993; Parati et al., 2001; Schroeder et al., 2003). For spectral analysis of heart rate, increased sympathetic cardiac activity and decreased parasympathetic cardiac activity are also associated with cardiovascular disease (Liao et al., 1996; Liao et al., 1997; Vanoli & Schwartz, 1990). Hence, such a profile of cardiovascular stress response is interpreted as having more adverse cardiovascular health consequences.

There is research evidence showing that hostility, assessed by both methods used in this chapter, is associated with greater cardiovascular stress reactivity; the Structured Interview hostility

assessment is associated with cardiovascular reactivity in the laboratory setting (Dembroski, MacDougall, Costa, & Grandits, 1979; Fredrickson et al., 2000; McCann & Matthews, 1988; Why et al., 2003) though a few other studies have reported negative or null results (Diamond et al., 1984; Schneider et al., 1989; Siegman & Anderson, 1990). However, nothing is known about this behavioural assessment of hostility and cardiovascular variability. But if hostility is related to poorer cardiovascular health, then it is reasonable to hypothesise that the Structured Interview-derived hostility should be related to a pattern of cardiovascular variability that is associated with worse health outcomes (e.g., reduced baroreflex sensitivity). Therefore, there is a need to conduct research to investigate the relationship between the Structured Interview hostility assessment and cardiovascular variability. By comparison, the research on hostility and cardiovascular reactivity/variability is over-represented by studies that have used the Cook-Medley Hostility scale (e.g., Barefoot et al., 1989; Everson et al., 1995; Houston, Smith, & Cates, 1989; Miller, Dolgoy, Friese, & Sita, 1998; Sloan et al., 2001). This is probably due to the fact that the Cook-Medley Hostility scale is less laborious to apply and does not require extensive training compared to the Structured Interview. However, there is also an increase in studies that have reported null results for the Cook-Medley Hostility scale and cardiovascular stress reactivity (Burns & Katkin, 1993; Carroll et al., 1997; Felsten, 1995; Felsten, 1996; Fichera & Andreassi, 1998; Spoth et al., 1992; Weidner et al., 1989).

Chapter 1 discusses two explanations for such conflicting findings, which are recapitulated here in brief. Firstly, this could be due to measurement error of both methods of hostility assessment. Since the Cook-Medley Hostility scale is the most frequently used method of hostility assessment, an attempt was made in Chapter 2 to reduce its measurement error. This was done via testing the internal consistency of a Cynicism 12-item version obtained from a review of four known factor analytic studies. This Cynicism 12-item version of the Cook-Medley scale will be used here to test its discriminant construct validity.

Secondly, conflicting results for the hostility-reactivity association may also be due to the existence of moderators. This chapter focuses on state anger and control as moderators for the hostility-reactivity relationship. Studies have reported that hostility is related to cardiovascular reactivity only when self-reported anger among high hostile individuals is elicited. These studies have used the harassment paradigm to elicit anger among high hostile individuals in the experimental laboratory to test the mediation of state anger (Everson et al., 1995; Miller et al., 1998; Suarez & Williams, 1989)¹². The harassment paradigm involves a confederate making deprecating remarks to the participant while the participant is executing the experimental task¹³. Smith and Brown also showed that interpersonal control moderates the

¹² A few other studies have used the Anger Recall, but this task is discussed later.

¹³ At least two studies have reported null results using this harassment paradigm as well. One study used the Structured Interview Hostility assessment (Diamond et al., 1984) while the other used the Cook-Medley Hostility scale (Felsten, 1995).

hostility-reactivity relationship (Smith & Brown, 1991). Forty-five couples were randomly assigned into two conditions - the Discuss and Influence conditions. In the Discuss condition, the couples were to discuss about a solution to a problem presented to them. Participants in this condition were told that their chances to win a lottery were determined randomly. In the Influence condition, each spouse was told to convince the other partner to take a specified solution in order to increase his/her chances of winning a lottery. For husbands, Cook-Medley Hostility scores were positively and significantly related to systolic blood pressure reactivity during the Influence condition but not for the Discuss condition.

The methodology used in investigating the role of state anger and interpersonal control as mediators for the hostility-reactivity relationship make generalisations to non-interpersonal settings difficult. The use of the harassment paradigm in investigating the role of anger as a mediator confounds both social conflict and state anger inducement. State anger can be induced in a non-interpersonal (e.g., goal obstruction) and interpersonal manner (e.g., harassment). Some evidence suggests that the hostility related reactivity may exist in an interpersonal context. High hostile individuals have greater ambulatory blood pressure during social interaction (Brondolo et al., 2003; Guyll & Contrada, 1998) and this is partly due to the fact that their interpersonal skills are perceived as less effective than low hostile individuals (Watkins & Eisler, 1988). If this is true then harassment may mediate the hostility-reactivity not because it induces anger but because harassment is also a form of social conflict. Thus, it is important to test the function of state anger and control without

implicating social conflict. In this chapter, the role of non-interpersonal control is investigated via manipulating the controllability of an experimental task. Reduced controllability of the task hinders the participants from performing well on the task. Monetary compensation based on task performance increases the participants' motivation to perform well. According to appraisal theories of emotion, obstruction to goal attainment (in this case, via manipulating the controllability of the task) will increase state anger (Berkowitz, 2000). Two known studies that have investigated the role of state anger (Why et al., 2003) and non-interpersonal control (Diamond et al., 1984) as moderators for the Hostility-Reactivity relationship have found non-significant results. These studies used the Structured Interview method of hostility assessment. Therefore, this chapter aims to extend the research to include both the Structured Interview as well as the Cook-Medley Hostility scale (using the Cynicism 12-item version).

Furthermore, there has been some debate as to whether perceived control or actual control is more important. Cardiovascular psychophysiological studies have reported evidence for task control and cardiovascular reactivity. A number of studies have reported that controllable tasks are associated with an active coping physiological response (i.e., increased cardiac output and decreased total peripheral resistance) while uncontrollable tasks are associated with a passive coping physiological response (i.e., decreased cardiac output and increased total peripheral resistance) (e.g., Weinstein et al., 2002; Tomaka et al., 1993; Hurwitz et al., 1993). Other researchers have also

reported results that showed the importance of perceived control over actual control (Averill, 1973; Burger, 1989; Waldstein et al., 1997). However, actual and perceived control are often congruent (Steptoe, 2001; Wallston, 2001) and some successful theories that predict health outcomes have measured both actual and perceived control as indices of overall control (i.e., Karasek and Theorell's demand-control theory and Siegrist Effort-Reward Imbalance theory) (Karasek & Theorell, 1990; Siegrist, 1996a). Hence, to help determine the congruence of perceived and actual control, it would be useful to investigate whether actual and perceived control tap the same or difference variance in cardiovascular reactivity. In this dissertation, actual control is manipulated by changing the efficacy of a mouse for a computer task.

This chapter also includes Anger Recall as one of the experimental tasks. Its inclusion serves a few purposes; firstly, Anger Recall involves recalling an interpersonal anger provoking event and this task elevates self-reported state anger (Boltwood, Taylor, Burke, Grogin, & Giacomini, 1993; Why et al., 2003). Hence, if state anger is a mediator of the hostility-reactivity or hostility-variability relationship, then Anger Recall represents another method to induce state anger among the participants. With reference to the earlier discussion about the harassment paradigm confounding anger inducement with interpersonal conflict, state anger induced by Anger Recall is also influenced by interpersonal conflict since it is the recollection of such an event. On the other hand, state anger induced by manipulating the controllability of the computer task is non-interpersonal. Thus, these two types of tasks cover the two forms of state

anger - of interpersonal origin and of non-interpersonal origin. Secondly, Anger Recall has also been found to elicit greater cardiovascular reactivity than most of the other laboratory-used stressors (Jain et al., 2001; Why et al., 2003). This increases the power of detecting statistical significant relationships between hostility and its mediators in predicting cardiovascular reactivity. Studies have also reported that this task has a profile of cardiac activity that is associated with poorer cardiac health (Ironson et al., 1992; Jain et al., 1998). Thus, the cardiovascular effects of this task have clinical significance as well.

To summarise, in this chapter, hostility is assessed via the Cynicism 12-item version of the Cook-Medley Hostility scale and the Structured Interview. The former represents the cognitive aspects of hostility (Cynicism) while the latter represents the behavioural aspect (i.e., verbal aggression). Two mediators of the hostility-reactivity/variability relationship are tested in this chapter: (i) state anger, (ii) non-interpersonal control (actual and perceived). State anger is induced via using an Anger Recall task and control is manipulated via adjusting the effectiveness of a computer mouse for a computer task (SYNWORK; Elsmore, 1994). According to appraisal theories of emotion, goal obstruction via such a manipulation of task controllability will increase state anger as well (Berkowitz, 2000). State anger induced by the Anger Recall is influenced by interpersonal conflict while state anger induced by the computer task is non-interpersonal.

Hence, the following are the hypotheses for Study 2:

Hypothesis 1: Cynicism and Cardiovascular reactivity/variability

1a) Cynicism 12-item scores are positively related to cardiovascular stress reactivity.

1b) Cynicism 12-item scores are negatively related to baroreflex sensitivity (BRS_{seq}). High Cynicism scores are associated with reduced baroreflex sensitivity.

1b) Cynicism 12-item scores are negatively related to parasympathetic cardiac activity (Normalised HF HR).

Hypothesis 2: Verbal Aggression (HBI) and cardiovascular reactivity and variability.

2a) Structured Interview-derived IHAT scores are positively related cardiovascular reactivity.

2b) Structured Interview-derived IHAT scores are negatively related to baroreflex sensitivity (BRS_{seq}). Participants who higher levels of verbal aggression during the structured interview have reduced baroreflex sensitivity during stress.

2c) Structured Interview-derived IHAT scores are negatively related to parasympathetic cardiac activity (Normalised HF HR). Participants who higher levels of verbal aggression during the structured interview have lower parasympathetic cardiac activity during stress.

Hypothesis 3: Hostility and cardiovascular reactivity/variability

Since both Cynicism and HBI are two aspects of Hostility, it is hypothesised that participants scoring high in both will have significantly

greater cardiovascular reactivity/variability. A Cynicism X HBI interaction will be significantly in predicting cardiovascular reactivity/variability.

3a) The pattern of the interaction would be that participants scoring high in both Cynicism and HBI would have greater reactivity than participants scoring low in both.

3b) Participants scoring high in both Cynicism and HBI would have greater reduction in Baroreflex Sensitivity (BRS) than participants scoring low in both.

3c) Participants scoring high in both Cynicism and HBI would have greater reduction in Parasympathetic Heart Rate activity (Normalised HF HR) than participants scoring low in both.

Hypothesis 4: Task differences for Cardiovascular Reactivity and Variability

4a) Task with Normal Control is characterised by an active coping physiological response (increased CO and decreased TPR) while the Tasks with Variable Pauses and More Variable Pauses are characterised by passive coping physiological response (decreased CO and increased TPR). Task Control main effect is significant for both CO and TPR.

Hypothesis 5: Task Control (Actual Control) and Perceived Control

5a) Task Control and Perceived Control predict common/unique variance for the dependent variables. The literature review in Chapter 1 revealed that there is research evidence supporting the views that both forms of control predict common and unique variance. However, it is

important to investigate this issue as whether Task Control (Actual Control) and Perceived Control predict unique or common will affect how Hostility X Control interaction is analysed (the next set of hypotheses). In order to investigate this hypothesis, Task Control and its interactions are entered into the regression model in a counterbalanced order with Perceived Control and its interactions. The changes in effect sizes when the order of Task Control (and its interactions) and Perceived Control (and its interactions) from these analyses will enable one to determine whether both forms of Control predict unique or common variance in cardiovascular reactivity/variability.

Hypothesis 6: Hostility and Cardiovascular reactivity/variability moderated by control (perceived or actual)

6a) There is a significant interaction between Cynicism and Control (Actual or Perceived) for cardiovascular reactivity. Specifically, participants scoring high in Cynicism are more reactive as the Task becomes less controllable. Participants scoring low in Cynicism would have similar cardiovascular reactivity as the Task becomes less controllable. The effect of Control (perceived/actual) is not significant among low Cynicism participants.

6b) Cynicism is associated with decreased parasympathetic cardiac activity (Normalised HF HR) and decreased BRS (BRS_{seq}) as the Task becomes less controllable.

6c) There is a significant HBI X Task Control interaction for cardiovascular reactivity. Specifically, participants scoring high in HBI have greater cardiovascular reactivity as the Task becomes less controllable.

6d) There is a significant HBI X Task Control interaction for cardiovascular variability. Specifically, participants scoring high in HBI have reduced parasympathetic cardiac activity (reduced Normalised HF HR) and reduced BRS as the Task becomes less controllable.

6e) There is a significant HBI X Cynicism X Task Control interaction. Specifically, participants scoring high in both HBI and Cynicism have greater cardiovascular reactivity as the Task becomes less controllable.

6f) There is a significant HBI X Cynicism X Task Control interaction. Specifically, participants scoring high in HBI and Cynicism have reduced parasympathetic cardiac activity (reduced Normalised HF HR) and reduced BRS as the Computer Task becomes less controllable.

Hypothesis 7: Hostility, Perceived Control and Cardiovascular reactivity/variability

7a) Hostility-Cardiovascular reactivity/variability relationship is moderated by Perceived Control. It is hypothesised that participants high in 12-item Cynicism or HBI will have greater cardiovascular reactivity, decreased parasympathetic cardiac activity (Normalised HF HR) and decreased BRS when Perceived Control is low. This is a Hostility x Perceived Control interaction.

7b) Participants scoring high in both 12-item Cynicism and HBI will have greater cardiovascular reactivity, reduced parasympathetic cardiac activity (Normalised HF HR) and reduced BRS. A 12-item Cynicism x HBI x Perceived Control interaction is hypothesised to be significant.

Hypothesis 8: Hostility, State Anger and Cardiovascular reactivity/variability.

8a) Hostility is positively associated with State Anger. Cynicism is positively associated with State Anger (significant Cynicism main effect), HBI is also positively associated with State Anger (significant HBI main effect), and participants scoring high in both Cynicism and HBI also tend to reported higher levels of State Anger (a significant HBI X Cynicism interaction).

8b) There is a significant Task Control main effect when State Anger is the Dependent Variable. Following appraisal theories of emotion, Synwork with less control is associated with higher ratings of State Anger.

8c) Hostility and cardiovascular reactivity/variability is moderated by State Anger. This hypothesis refers to significant State Anger X HBI, State Anger X Cynicism, and State Anger X Cynicism X HBI interactions. Specifically, participants scoring (i) high in both State Anger and HBI, (ii) high in State Anger and Cynicism, or (iii) high in State Anger, Cynicism and HBI have greater cardiovascular reactivity, greater reduction in parasympathetic cardiac activity (Normalised HF HR) and reduced BRS (BRS_{seq}).

Method

Sample

Table 3.1 Sample Characteristics (n = 59)

	Mean (SD)
Age, years	21.27 (2.99)
Gender	Male
Height, cm	179.28 (6.06)
Weight, kg	72.55 (8.90)
BMI, kg/m ²	22.57 (2.55)
BSA, m ²	1.91 (.13)
Baseline Systolic Blood Pressure, mmHg	106.35 (14.95)
Baseline Diastolic Blood Pressure, mmHg	71.80 (10.22)
Baseline Mean Arterial Pressure, mmHg	83.52 (10.65)
Baseline Heart Rate, bpm	70.48 (10.66)
Cook-Medley Scale	19.81 (7.83)
Cook-Medley Cynicism Subscale	6.22 (2.94)
Cook-Medley Alternative Subscale	3.87 (2.26)
Hostile Behaviour Index (HBI)	.10 (.08)
Transformed HBI	.78 (.16)

Table 3.1 lists the sample characteristics for this study.

Participants were recruited by word of mouth and advertisements posted at various venues within the University campus (e.g., University Student

Halls of Residence). Sixty-one males volunteered for this experiment. One participant only attended the psychological assessment session and withdrew participation from the experimental session. Thus physiological data was not obtained from this participant. Another participant's Structured Interview was not recorded due to a technical fault with the recording equipment. These two participants were removed from the sample and this reduced the sample analysed to 59. In this study (and the next in Chapter 4), a target sample of 60 was set because other studies reported significant result between hostility (derived from the Structured Interview) and cardiovascular stress reactivity during the Anger Recall task for 66 participants (Fredrickson et al., 2000) and 39 participants (Why et al., 2003).

Alpha level was set at .01 since a number of hypotheses were tested. All analyses were done using SPSS version 10 and MS Excel 97.

Psychological Measures

The cognitive and behavioural components of hostility were measured. Based on the literature review and results in Chapter 2, the 12-item Cynicism subscale from the Cook-Medley Hostility Scale (Cook & Medley, 1954) was used to tap the cognition component of Cynicism while the Structured Interview measured the behavioural aspect of hostility (i.e., verbal aggression).

The interviews were taped and were assessed using the Interpersonal Hostility Assessment Technique (IHAT, Haney et al., 1996) which measures the behavioural aspect of hostility. The interview

consisted of questions from the Type A structured interview, which was adapted for an undergraduate sample. For example, 'Are you satisfied with your job?' was changed to a question about their satisfaction with their studies. All interviewers were female undergraduate volunteers who underwent some IHAT interview training. Interviewer N. interviewed 30 males (50.8%), both interviewers K.S.₁ and K.S.₂ interviewed 10 males (16.9%) each, while interviewer P. interviewed 9 males (15.3%). Dr. Haney, one of the formulators of the IHAT, scored the interviews. For each participant, component scores for Irritation, Hostile Evade/Withhold, Indirect Challenge, Direct Challenge were combined and divided by the total number of questions asked during the interview to yield a Hostile Behaviour Index (HBI). Skewness and kurtosis indices for HBI were significant: skewness = 1.17 (SE = .31) and kurtosis = 1.20 (SE = .61). The tendency for HBI scores to be positively skewed has been reported before and researchers either dichotomised the scores (Matthews et al., 2004) or used transformed HBI scores (Fredrickson et al., 2000; Why et al., 2003). Given the undesirable effects of dichotomising continuous scores (MacCallum, Zhang, Preacher, & Rucker, 2002), transformations of the HBI is done instead. Using first $1/(HBI + 1)$ and then HBI^3 , the distribution for HBI was transformed to normal. Indices for skewness and kurtosis were no longer significant: skewness = -.47 (SE = .31) and kurtosis = -.33 (SE = .61). The means and SD for the transformed HBI is listed in Table 3.1. Cynicism 12-items scores were normally distributed, skewness = -.08 (SE = .31), kurtosis = -.92 (SE = .61). Statistical analysis used the centred HBI (i.e., $HBI - \text{Mean}_{HBI}$) and centred Cynicism scores.

This was to reduce multicollinearity between these two between-subject main effects and their interaction term (Aiken & West, 1991).

Desirability of control scale (Burger and Cooper, 1979) was used to measure trait desirability of control. This was to determine if there were any potential confounds between hostility and desire for control. If hostility was positively related to cardiovascular reactivity/variability during low task control, this may not be due to low control but because of a positive correlation between hostility and desirability of control. That is, because high hostile individuals tend to also desire for control, low task controllability elicits greater cardiovascular reactivity for them than low hostile individuals. Thus a confound between these two traits can also explain the Hostility x Task Control interaction. Any confound between the two Hostility measures and Desirability of Control would be statistically controlled for in subsequent analyses. Desirability of Control scale was also included to test the discriminant construct validity of the Cynicism 12-item scale. The correlation between the Cynicism 12-item scale, DS16 and Marlowe-Crowne Social Desirability scale has been analysed in Chapter 2 and will not be repeated here. Only inter-correlations between the 12-item Cynicism scale, HBI and Desirability of Control are reported here.

State mood ratings and situational appraisal were also assessed. State mood items were taken from the UNWIST mood adjective checklist (Matthews, Jones, & Chamberlain, 1990). Table 3.2 lists the 15 words used from this Checklist. The words were rated on a 4-point Likert scale: '1' for 'Definitely Not', '2' for 'Slightly not', '3' for 'Slightly' and '4' for

‘Definitely’. Appraisal questions are included in Appendix 3. Self reported Anger was the mean of all the ratings given for the 5 Anger items. Only Anger items were used in this dissertation because it was the moderator of interest. Participants would be primed towards state anger if they were

Table 3.2 The 15 Mood Adjective items derived from Matthews et. al (Matthews et al., 1990)

Satisfied	}	Hedonic tone items	}	Hedonic tone (Factor 1)
Happy				
Cheerful				
Sad				
Impatient	}	Anger items	}	Hedonic tone (Factor 1)
Annoyed				
Angry				
Irritated				
Grouchy				
Anxious	}	Tense arousal (Factor 2)	}	Hedonic tone (Factor 1)
Relaxed				
Calm				
Alert	}	Energetic arousal (Factor 3)	}	Hedonic tone (Factor 1)
Energetic				
Tired				

to respond to only Anger items repeatedly. To reduce this from happening, other items from the other factors of the UNWIST mood adjective checklist were also included. Participants were asked to fill in the mood adjective checklist before and after every baseline and experimental task. The order of items in the mood adjective checklist and items in the appraisal questionnaire were randomised.

Item 2 listed in Appendix 3 (‘To what extend were you able to influence the outcome of the task?’) was used to indicate the participants’ Perceived Control. Perceived control after the three sessions of the

computer task (Normal, Variable Pauses and More Variable Pauses) were all normally distributed.

Physiological Measures

For cardiovascular reactivity, there were 6 relevant dependent variables. They were: Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Mean Arterial Pressure (MAP), Heart Rate (HR), Cardiac Output (CO) and Total Peripheral Resistance (TPR). For cardiovascular reactivity, there were two covariates - the respective cardiovascular baseline and BSA. The Finapres and Beatscope were used to derive these dependent variables. The Ohmeda 2300 Finapres and Beatscope (TNO Biomedical Instrumentation, Amsterdam) were used to provide continuous cardiovascular readings of the participants. Blood pressure and Heart Rate were obtained using the Finapres, which measured finger arterial pressure wave. Before a Finapres reading was recorded, the Servo Self-Adjust was allowed to operate until it occurred once every 30 blood pressure cycles. The Servo Self-Adjust was then disabled during data acquisition. A review on the Finapres indicated that once the Servo Self-Adjust has occurred at least once every 30 cycles, the readings were more likely to be accurate (Imholz, Wieling, van Montfrans, & Wesseling, 1998). The Beatscope software (Modelflow) was used to estimate cardiac output and total peripheral resistance from the obtained finger pressure wave (Wesseling, Jansen, Settels, & Schreuder, 1993). Finger arterial pressure was significantly correlated with intra-brachial pressure (Jellema, Imholz, van Goudoever, Wesseling, & van Lieshout, 1996) and

the Finapres track blood pressure and heart rate changes accurately (Imholz et al., 1998). Modelflow's estimation of stroke volume (cardiac output = stroke volume x heart rate) has been found to be significantly correlated with clinical assessments of stroke volume using Doppler echocardiography and thermodilution (Gratz et al., 1992; Sugawara et al., 2003).

Three dependent variables assessed cardiovascular variability: Normalised High Frequency Heart Rate, Baroreflex Sensitivity using spectral analysis, and Baroreflex Sensitivity using the Sequence Method. The following paragraphs detail the equipment used to collect and process these dependent variables. For cardiovascular variability, there were three covariates - the respective cardiovascular baseline, BSA and respiration. Spectral analysis of Electrocardiogram (ECG) was used to derive the Normalised High Frequency Heart Rate and Normalised High Frequency Respiration. ECG was also used with the continuous SBP data obtained from the Finapres to assess Baroreflex Sensitivity via spectral analysis and the sequence method. Electrocardiogram was taken via three Ag/Cl electrodes (Unilect model 1010; MSB, Wiltshire, UK) using a conventional lead placement (figure 3.1).

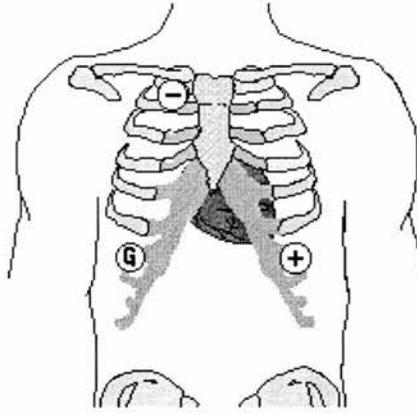


Figure 3.1 Schematic diagram showing conventional lead placement for electrocardiogram.

Respiration was measured via putting a Pneumotrace respiration transducer model 1130 (UFI, CA, USA) across the abdominal of the participant where the greatest motion was felt during respiration. It was a solid-state strain gauge transducer used to measure respiratory changes in the abdominal circumference. It was a resistive device that changes its resistance linearly as the band is stretched. Resistance decreased as the participant inhaled and increased as he exhaled. Respiration was measured in Arbitrary Units (AU). Respiration was an arithmetic mean of ± 15 samples data points from the R-wave. Since respiration was sampled at 100Hz, this meant that respiration values obtained ± 150 milliseconds of an R-wave were averaged. Averaging respiration data this way removes minor fluctuations (Mulder, van Roon, & Schweizer, 1995).

Electrocardiogram (ECG) and respiration were obtained via a polygraph D.C. driver amplifier (Model 7DAF; Grass Medical Instruments, MA, USA). Electrocardiogram, respiration and finger arterial blood pressure were then converted from analogue to digital format (1401 Plus; CED, Cambridge, UK). This was then recorded by specialised software

(SPIKE 2 Version 3.14, CED, Cambridge, UK) in real time and stored in an IBM desktop computer. Electrocardiogram was sampled at 500hz, respiration at 100hz and blood pressure at 100hz according to published guidelines (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996; Berntson et al., 1997). However, due to a technical oversight, respiration data for the first 7 participants were sampled at 10Hz. One participant's respiration data during Synwork Variable Pauses was corrupted due to equipment failure. Respiration data was thus treated as missing for these eight participants. CARSPAN (ie ProGAMMA, Groningen) was used to perform spectral analysis on the dependent variables. Pharmacological blockade studies showed that High Frequency band of heart rate is due to parasympathetic activity (Berntson et al., 1997; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). Low Frequency (LF) HR spectral band was defined as between .04 to .15Hz, (changes in heart rate between 2.4 to 9 beats per minute) while High Frequency (HF) HR was defined as between .15 to .40Hz (changes in heart rate between 9 to 24 beats per minute). Normalised versions of the High Frequency HR was used here. Normalisation for High Frequency HR spectral band was done via the following formula: $\text{High Frequency HR} / (\text{HF HR} + \text{LF HR}) \times 100$. Therefore, normalised HF HR had the property of 'normalised HF HR + normalised LF HR = 1'. Normalisation reflects the balance between the two branches of the autonomic system (sympathovagal cardiac activity) as well as controlling for the overall changes in total power (Task Force of

the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). Since 'Normalised HF Heart Rate + Normalised LF HR = 1', analysis was only done for Normalised HF HR. Results from this variable could deduce the corresponding results for normalised LF HR. The measurement unit for Normalised LF and HF HR is 'nu' (normalised units).

Baroreflex sensitivity was calculated in two ways. Firstly, using CARSPAN to calculate coherence and modulus between SBP and HR (see Data Processing section) and using the Sequence Method (Parati et al., 1988).

In order to enable CARSPAN to process information from the SPIKE files, the times of R-waves onset were obtained from ECG. Systolic Blood Pressure was obtained from the Finapres were matched according to the R-wave of the same cardiac cycle as Baroreceptor Sensitivity is usually assessed using both heart rate and SBP. CARSPAN can also calculate the coherence, modulus and phase between each R-wave and SBP (Mulder et al., 1995)¹⁴. Coherence is akin to a correlation between the two data streams. The modulus indicates how much SBP would change according to one unit change in R-wave. Phase provides information about the time latency between the two data streams (i.e., how long the changes in one data stream is preceded by the other). To assess Baroreflex Sensitivity, the coherence between the SBP and HR spectral data should be $> .50$ to ensure statistical reliability (Persson,

¹⁴ For the interested reader, the formulae for coherence, modulus and phase are provided in the cited work.

1997). Baroreflex Sensitivity is measured by the modulus, once coherence is $>.50$; the higher the modulus, the more sensitivity the baroreflex response. A recent study demonstrated that the results from CARSPAN reliably and accurately detect baroreflex changes when compared to standardised bolus angiotensine II method of baroreflex sensitivity assessment (Schachinger et al., 1996). This method of Baroreflex Sensitivity assessment was found to be significantly correlated with the phenylephrine method of baroreceptor stimulation (Pitzalis et al., 1998; Watkins et al., 1996).

In addition, Baroreflex Sensitivity was also assessed using the Sequence Method (Parati et al., 1988). Sequences of 3 to 5 pairs of Inter Beat Interval (IBI) and SBP were found that fulfil the following criteria: (i) there was a change of at least 1ms for IBI, (ii) there was a change of at least 1mmHg for SBP, (iii) both IBI and SBP changes were in the similar direction (i.e., both IBI and SBP were increasing or decreasing), and (iv) the correlation between IBI and SBP was greater than $.80$ to ensure linear trends. The average of the regression slopes for these sequences provided an index of the Baroreflex Sensitivity. Good agreement between the Sequence Method and more standard invasive methods (e.g., phenylephrine method) of baroreflex sensitivity assessment has been reported (Watkins, Fainman, Dimsdale, & Ziegler, 1995; Parlow, Viale, Annat, Hughson, & Quintin, 1995; Pitzalis et al., 1998).

In order to assess autonomic cardiac activity and baroreflex sensitivity reliably via spectral analysis, respiration needed to be statistically controlled for (Althaus, Mulder, Mulder, Van Roon, &

Minderaa, 1998; Grossman, Karemaker, & Wieling, 1991; Novak et al., 1993).

Task

A computerised task, Synwork, was used. Synwork was a multitasking task that divided the monitor display into four equal quadrants (see Figure 3.2; Elsmore, 1994). Each quadrant on the computer monitor had a distinct task.

The top left hand corner was a memory search task. Participants had to remember an alphabet list and determine whether a presented alphabet was contained within the list. Participants had about 5 seconds to view the list and respond by clicking on the 'Yes' or 'No' buttons. After 5 seconds the list would be covered by the words 'Retrieve List'. Clicking on these words would reveal the list again for another short period. Scoring for this task was +10 points for a correct response and -10 for an incorrect response. Once the 'Yes' or 'No' button was clicked, the items in the list changed. Participants could review the list as many times as they wished without being penalised for points. There was no penalty should the participant choose not to respond to this task. The lower left quadrant was a visual monitoring task. A cursor moved randomly (i.e., left or right) along an axis and the participant had to keep the cursor away from the two ends of the axis. The cursor was set to move at the speed of 1 pixel per 80 millisecond and the scale was at 201 pixels. Points were gained should the participant click on the button marked 'Reset' to move the cursor to the centre of the scale. The further away the cursor was from

the centre, the more points would be gained when the 'Reset' button was clicked. A maximum of 10 points was awarded when the cursor was at

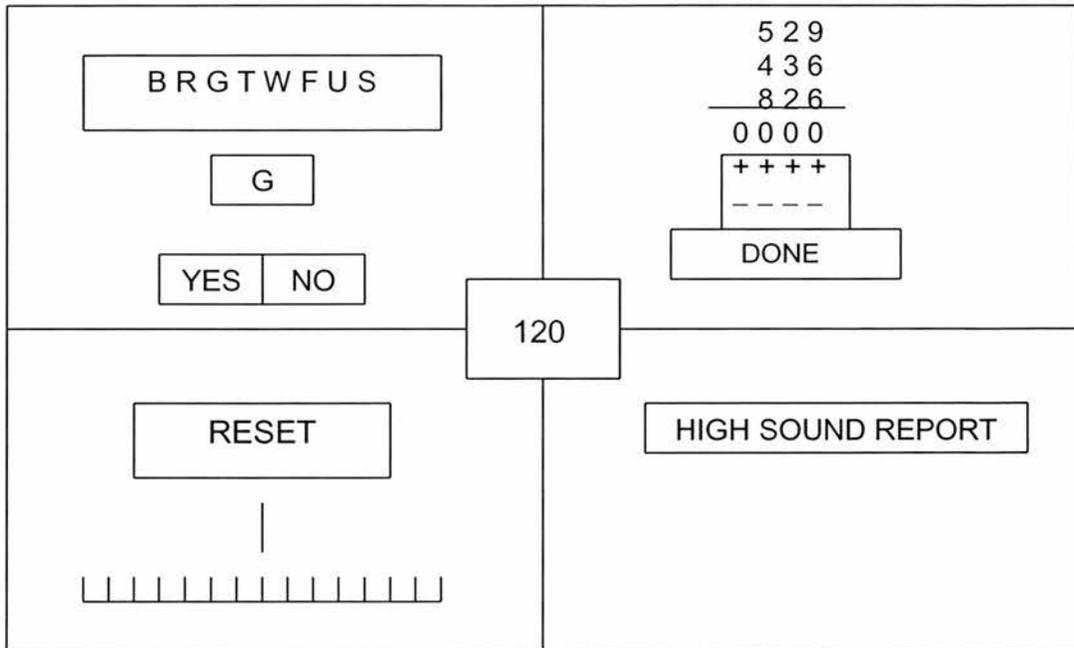


Figure 3.2 Presentation Layout of Synwork

either end of the scale when the 'Reset' button was clicked. However, if the cursor was allowed to stay at either end of the scale for longer than a second, 10 points were deducted per second. The top right hand quadrant was a mathematical task. Participants were to compute the sum of three 3-digit figures via clicking on the '+' and '-' buttons at the corresponding column. The 'Done' button was clicked once the participants thought that they have entered the correct answer. Ten points were awarded for a correct answer and 10 points were deducted for an incorrect answer. The lower right hand quadrant was an auditory monitoring task. The computer played high and low pitched tones at constant time intervals. Whenever a high-pitched tone was heard, the

participant was to click on the button labelled 'High Sound Report' in that quadrant as a response. High-pitched tones were played at 1500 Hertz while low pitched tones were at 5500 Hz. Probability of the high pitched tones being played was .3. No points were deducted when the participant did not make a response when there was a high-pitched tone. However, 10 points were deducted if the participant clicked the 'High Sound Report' button after a low-pitched tone. Conversely, 10 points were awarded when the participant clicked on the 'High Sound Report' after a high-pitched sound was played. Each participant wore headphones when executing this task using a trackball mouse. A trackball mouse was used to minimise hand and arm movements. The score, based on the summation or subtraction of the points from the four tasks, was displayed in the centre of the monitor.

The Task Control (Actual Control) manipulation

Each participant performed the Synwork task four times of 5 minutes each. A practise session came first followed by a Normal (i.e., the mouse worked normally) Synwork session. This was followed by the Variable Pauses and then the More Variable Pauses condition. The order of Task Control was fixed for three reasons. Firstly, fixing Task Control from Normal to Variable Pauses allowed participant to experience and develop coping strategies during the Variable Pauses Task Control condition in order to improve the efficacy of their coping behaviour for the More Variable Pauses Task Control condition. Task Control condition was defined as the response-outcome contingency between the movement of

the trackball mouse and the mouse cursor on the computer screen. So while participants may improve their coping behaviour for the Variable and More Variable Pauses Task Control, More Variable Pauses Task Control had lower response-outcome contingency. Thus, a discrepancy between efficacy of coping behaviour and response-outcome contingency was created. Secondly, counterbalancing the various tasks in this study entailed a 2 (Anger Recall - Synwork order) x 6 (3 Synwork Task Control conditions order) presentation orders. With sample size of 60, this meant that each counterbalancing condition would have only $n = 5$. Such a small sample size increases the likelihood of differences in Hostility scores between the counterbalancing groups. Thirdly, a pilot study counterbalancing 2 Synwork difficulty levels (Normal and Variable Pauses) showed that the counterbalancing affected the participants' appraisal of control for the next Synwork session. Specifically, those participants in the pilot study who experienced the Variable Pauses Task Control first, reported lower perceived control of their next Synwork session (Normal). The pattern of results in the pilot study suggested that if participants were to experience a Variable Pauses (or a More Variable Pauses) condition first, this will lower their Perceived Control ratings of subsequent Task Control conditions even when Task Control may be Normal. By fixing the order of the Task Control conditions (in the order of Normal Control, Variable Pauses and More Variable Pauses) in this study, the carryover effects of Task Control condition on Perceived Control between participants was also fixed. This allowed an investigation of the interaction between the two between-subject hostility measures

(Cynicism and IHAT-derived HBI scores) and Perceived Control while standardising Task Control order effects on Perceived Control between subjects. The disadvantage of fixing the order of Task Control was that results obtained from the study have limited generalisability (i.e., results may be true only for the fixed Task Control order used in this study). Task Control was counterbalanced in Study 3.

In the Variable Pauses and More Variable Pauses condition, the mouse malfunctioned. The malfunction of the computer mouse was accomplished via having a random pause generator (i.e., the 'Faulty Mouse') connected between the mouse and the computer (see Appendix 4 for a technical description of this device and its schematic diagram). The Faulty Mouse device was used to create low response-outcome contingency for Synwork during the Variable and More Variable Pauses conditions.

During the normal sessions of the Synwork task (i.e., practise and Normal condition), this was switched off. During the Variable Pauses condition, the Faulty Mouse device generated pauses between 25 to 300ms. During the More Variable Pauses condition, the device created random pauses between 25 to 500ms. Participants did not know that the Synwork task would be less controllable as the experimental session progresses. If participants complained about the uncontrollability of the task, the experimenter would explain that it was due to a software incompatibility between the Synwork task and the computer's Operating Software. The participant would then be told that the computer would be rebooted and their scores for Synwork corrected accordingly. In actuality,

nothing was done. Participants were encouraged to try their best even when Synwork was less controllable. At the end of the experiment, participants were debriefed that the purpose of this deception was to enable them to maintain their motivation to do well in the task even when it became less controllable.

Procedure

Data collection was separated into two sessions. The first session lasted for about 30 minutes and was held between 9am to 11am on weekdays. In the first session, participants were briefed about the general procedures of the experiment and informed consent was obtained. They were then interviewed for the IHAT and filled in the personality questionnaires (i.e., Cook and Medley Hostility Scale, Marlowe Crowne Social Desirability Scale, Desirability of Control Scale, and DS16 scale). The order of the personality questionnaires was randomised for each participant. In addition, half the participants filled in questionnaires first while the other half had the interview first. The second session (Session 2) was held one day to one week after session one and lasted for about 2 hours. Session 2 was also held on weekdays either from 9am to 11am or from 11am to 1pm. The second session consisted of the experiment. When the participant arrived, 10 to 15 minutes was spent briefing the participant about the general procedures of this session, taking their weight and height, fixing the electrodes, respiration belt, and the Finapres onto the participant as well as making sure that the physiological equipment was functioning well. Participants sat in front of the computer

that would present Synwork with the electrodes, respiration belt and Finapres placed on them. The Finapres was placed on their left hand index finger and an adjustable armrest kept their left arm and hand at the participants' heart level. This was to minimise the Finapres blood pressure assessment from being affected by the finger-to-heart height for each participant. Once in operation, it was switched off once every 30 minutes for 1 minute. After 1 minute, the Finapres was switched on again and once the Servo Self-Adjust occurs once every 30 cardiac cycles, cardiovascular physiological data collection resumed. The Servo Self-Adjust was switched off when data was recorded and stored onto the desktop computer. This was to enable an uninterrupted stream of physiological data readings to be recorded. Extended use of the Finapres has been found to be associated with an upward drift of blood pressure (Ristuccia, Grossman, Watkins, & Lown, 1997). The 1-minute rest of the Finapres was to reduce such effects. A five-minute baseline was placed at the beginning of the whole experiment. Another 5-minute recovery baseline was placed at the end of the whole experiment. Duration of the resting periods between trials was 3 minutes each. Participants were randomised into two conditions: (i) Anger Recall-Synwork condition, or (ii) Synwork-Anger Recall condition. The first condition had the Anger Recall task first while in the second condition, the Synwork sessions came first. Before and after every task session, as well as at the end of the experimental study, participants were told to answer questions relating to their appraisal of the task, expected performance of the next Synwork session (participants were told they had to do four testing sessions of

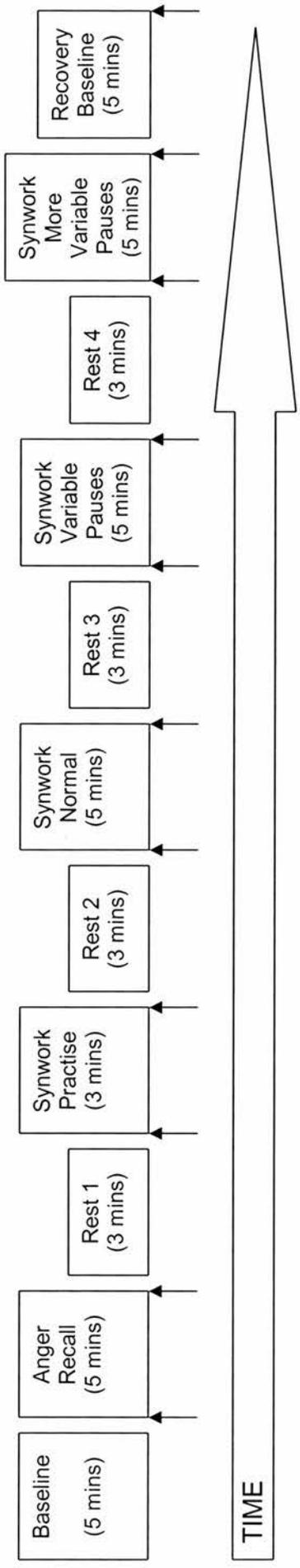
Synwork instead of three; Appendix 3) as well as their mood. Participants were told that the amount of money they could get for their participation was partly based on their performance on the Synwork task. This was to ensure engagement and motivation in the task of the participants even when the mouse became 'faulty'. However, at the end of the experiment, all participants were given compensation at the rate of £4 per hour dependent on the total time they had taken to participate in the two sessions combined. This deception was also explained to the participants at the end of the session during the debriefing period. The average time for the two sessions combined was 2.5 hrs.

For the Anger Recall task, the participant was told to recall an event that had occurred in the last six months when someone made them really angry. Specifically, an incident that still made them angry when they recalled it. They were told to imagine the situation, the people involved, their reaction, thoughts, feelings and reaction for one minute. All participants had no problem recalling such an incident. During this phase, no measurements were taken. After one minute, the participant was asked to talk about the incident. Each participant was requested to describe this incident for five minutes. If a participant stopped before the end of five minutes, a male experimenter gave standardised prompt questions. Standard prompts included: "Describe in detail what you were feeling at that time.", "What were your thoughts then?", "What did you do when that event occurred?", "How did the other person react?", and "What was it about the event that made you angry?". Their disclosures were not recorded. At the end of the Anger Recall task, participants

responded to the mood questionnaire. Their appraisal was not assessed because the appraisal of the angry event narrated during Anger Recall was retrospective and the time lapse between the event and the experiment (within 6 months before the experiment) reduced the accuracy of their appraisal of the event. Hence appraisal of the anger-provoking event was not assessed.

At the end of the experiment, participants were debriefed about the deceptions, the general purpose of the experiment and were paid. They were also asked whether they thought the amount of cash they were getting to be dependent on their performance or dependent on the amount of time they had taken to attend both sessions for this study. This was to check whether the deception to motivate them to perform well for Synwork was successful. Figure 3.3 shows the sequence of events for Session 2 (the experimental session) of this study.

Task Order 1: Anger Recall Task 1st



Task Order 2: Synwork Task 1st

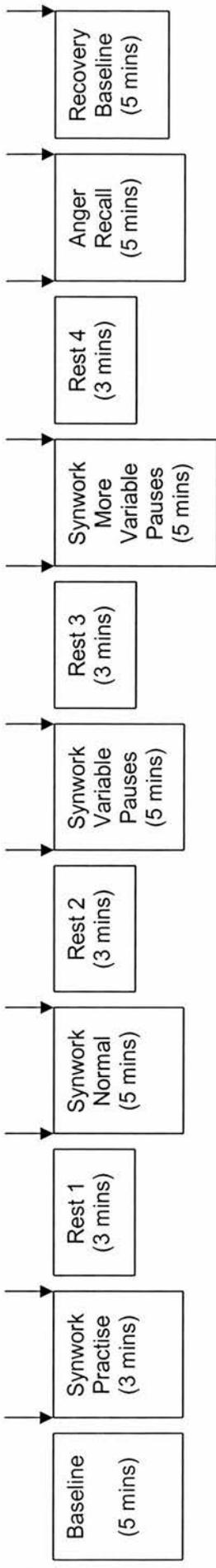


Figure 3.3 Schematic Diagram showing the Sequence of Events in Session 2 (Experimental Session) of Study*

*Black arrows indicate when participants' mood and/or appraisal are assessed. Participants are randomised into Task Order 1 or 2 (see text).

Data Analyses Strategy

ANOVA can be regarded as a case of regression. A number of researchers have shown that regression can duplicate results from ANOVA and ANCOVA (Cohen, 1968; Edwards, 1985; Pedhazur, 1982). Both GLM and Regression are used here instead of ANOVA because they allowed the use of continuous between subject independent variables (e.g., Cook-Medley Cynicism). However for analysis involving State Anger reactivity and Perceived Control, regression was used instead of GLM. This was because compared to GLM procedure, SPSS has yet to allow the inclusion of a continuous within subject variable (e.g., state anger reactivity) into its analysis.

It would be beyond the scope of this dissertation to provide a detailed exposition of how GLM, Regression and ANOVA are related as well as the how to use regression to derive results in the ANOVA format. A brief explanation is given here and the interested reader may wish to refer to Pedhazur (Pedhazur, 1982) on how this is done. Two studies have been published using this method (Bishop & Robinson, 2000; Why et al., 2003). The following paragraphs describe how the various Independent Variables were entered into the model and the different models tested in line with the hypotheses.

All continuous variables (i.e., Perceived Control, HBI and Cook-Medley Cynicism) were centred ($x - \text{MEAN}$) to reduce multicollinearity among the predictors (Aiken & West, 1991). Interaction terms were the multiplication of the respective main effects. That is, a HBI X Cook-Medley Cynicism interaction term for the regression model was created

via multiplying HBI with Cook-Medley Cynicism in order to produce a predictor for this interaction effect. In order to derive an error term for the between-subjects model, a vector using criterion scaling was created (Pedhazur, 1977). Criterion scaling involved using the mean of all the conditions for each participant. For this study, Task Control had 3 levels (Normal, Variable Pauses and More Variable Pauses) and each participant had three values for each task control condition. So if a participant had a heart rate of 70, 75 and 80 for these three task control conditions respectively, the criterion scaling vector value for this participant for heart rate as the dependent variable would be $(70+75+80)/3 = 75$. However, though the Sums of Squares predicted by this criterion scaling method was correct, the degrees of freedom for this vector was wrong as SPSS output prints its df as '1'. The correct df for the error term of this criterion scaling vector should be $N - 1 - \text{Sum of all the df's for the between subject model predictors}$.

For categorical within-subject variables like Task Control, effect coding was used. Two vectors were created to do this; vector T1 and T2. Table 3.4 shows the codes given for T1 and T2. These two vectors were entered into the regression model in a single block, thus created the predictor for Task Control with 2 degrees of freedom. The interaction involving Task Control was also created by multiplying these two vectors with the other variable of concern. All components of the interactions were entered into the regression model first. That is, for the HBI X Cook-Medley Cynicism interaction, the corresponding main effects for HBI and Cook-Medley Cynicism were entered first and retained in the regression

model even if non-significant, followed by the interaction term. Both multivariate and epsilon corrected univariate repeated measures revealed similar patterns of significant results, epsilon corrected results from univariate repeated measure are reported here.

Table 3.3 Effect Coding for Task Control

Vectors	T1	T2
Task Control		
Normal	-1	-1
Variable Pauses	1	0
More Variable Pauses	0	1

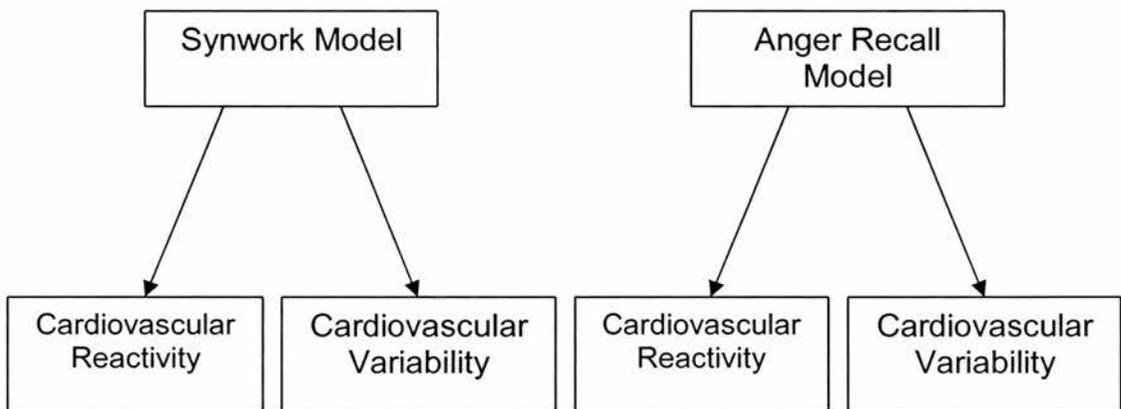


Figure 3.4 Diagram of Model and Data Analytic Structure

The three versions of the computer task (Synwork with Normal, Variable Pauses and More Variable Pauses) were tested under 'Task Control' while Anger Recall was tested separately. This was because Anger Recall was not a task that control was manipulated (i.e., not

another level of Task Control). Hence it was not analysed together with Synwork. As can be seen from Figure 3.3, participants were randomly assigned to two conditions that counterbalanced the order between Anger Recall and the three versions of Synwork. There were two types of cardiovascular stress responses - cardiovascular reactivity and cardiovascular variability. Figure 3.4 shows the data analytic structure of this chapter.

The following paragraphs illustrates the model tested for Synwork and Anger Recall.

Synwork Model. For Synwork, the following general model was tested. Some changes to this model were made when testing specific hypotheses and will be specifically mentioned in text. Predictors were entered as listed:

Between Subjects Model

1. Covariate
2. Hostile Behaviour Index (HBI)
3. Cook-Medley Cynicism
4. HBI X Cook-Medley Cynicism
5. Within Subject Error (Criterion scaling)

Within Subjects Model

1. Task Control (Synwork Normal, Variable & More Variable Pauses)
2. Task X HBI
3. Task X Cook-Medley Cynicism
4. Task X HBI X Cook-Medley Cynicism

For cardiovascular reactivity, there were two covariates - the cardiovascular physiological Baseline and BSA. For cardiovascular

variability the two covariates were the respective cardiovascular baseline and respiration.

Anger Recall Model. For Anger Recall model, it was a between-subjects model since there was only one task condition:

Between Subjects Model

1. Covariate
2. Hostile Behaviour Index (HBI)
3. Cook-Medley Cynicism
4. HBI X Cook-Medley Cynicism

For the Anger Recall model, the covariates for cardiovascular reactivity were the same for the Synwork Model - Baseline and BSA. For cardiovascular variability, the two covariates were Baseline and Respiration.

Operationalisation of Reactivity. There are several ways to operationalise cardiovascular reactivity. These include, using the arithmetic difference between baseline and task conditions (gain scores), using the residuals based on regression analysis, incorporating the baseline as a covariate (ANCOVA) and incorporating the baseline as another level of a repeated measure. Other considerations include the definition of what a baseline is. This study used the first Baseline (Figure 3.3) as the resting baseline. The justification for the operationalisation of cardiovascular baseline and reactivity in this dissertation is complex. This was discussed in detail in the discussion section of this chapter. At this point, it is sufficient to state that none of the current methods of operationalising baseline and

reactivity are perfect and each method is plagued by particular problems. The use of ANCOVA in operationalising reactivity in this study served to maximise statistical power and reliability in the analyses. Hence, this approach was taken here.

Results

Manipulation Check

Out of 61 participants, 52 were asked whether they thought monetary compensation was dependent on Task performance or time. (Nine participants were not asked due to human error.) Forty-four (84.62%) thought monetary compensation was dependent on task performance, five (9.62%) thought monetary compensation was fixed for every participant or time-dependent while three (5.77%) gave other reasons (e.g., did not think about it). Thus, linking performance with monetary compensation was largely successful.

Table 3.3 Intercorrelations between the Psychological Measures (n = 59)

	Hostile Behaviour Index	Burger-Cooper Desirability of Control
1) Hostile Behaviour Index	--	.17
2) 50-item Cook-Medley Hostility Scale	-.17	-.09
3) Cynicism 12-item	-.07	-.04

Correlations among the Psychological Variables

The Structured Interview derived Hostility assessment, HBI, was correlated with three versions of the Cook-Medley Hostility scale: the full 50-item version, and the Cynicism 12-item version (from Chapter 2). Table 3.3 shows the correlations among the various psychological measures. None of the correlations were significant at $p < .01$ after controlling for Type 1 error. However, compared to the Cynicism 12-item version, the 50-item version of the Cook-Medley Hostility scale tended to have higher negative correlations with HBI.

Testing the 'Suitability' of Covariates

Baseline. The Baseline operationalised as the average of all resting periods can provide a more reliable estimate. Table 3.5 shows the means and standard deviations for the first baseline as well as the between task rest periods and Recovery Baseline for cardiovascular reactivity. Baselines should represent tonic levels of cardiovascular functioning and should not be influenced by the experimental tasks. Significant differences between the baselines indicate that an average of all the rest periods cannot be operationalised as a 'Baseline'. The Baseline, 4 Rest Periods and Recovery Baseline were entered as single repeated measures with 6 levels termed 'Baselines'. Significant 'Baselines' main effect was found for SBP, $F(3.15, 166.99) = 9.90, p = .03 \times 10^{-4}, \eta^2 = .16$, DBP, $F(3, 159.01) = 8.47, p = .03 \times 10^{-3}, \eta^2 = .14$, MAP, $F(3.07, 162.64) =$

7.56, $p = .08 \times 10^{-3}$, $\eta^2 = .13$, CO, $F(2.66, 141.19) = 7.12$, $p = .03 \times 10^{-2}$, $\eta^2 = .12$, and TPR, $F(9.96, 141.19) = 7.12$, $p = .07 \times 10^{-4}$, $\eta^2 = .16$. 'Baselines' main effect was not significant for Heart Rate, $F(2.70, 143.04) = 2.92$, $p = .04$, $\eta^2 = .05$. The linear components of 'Baselines' for SBP, DBP, MAP, CO and TPR were significant. As shown in Table 3.5, there was a gradual increase in blood pressure. The hemodynamic profile showed that this increase in blood pressure was due to a decrease in CO and increase in

Table 3.5 Mean (SD) for the Baselines and Rest Periods

	Baseline	Rest Period 1	Rest Period 2	Rest Period 3	Rest Period 4	Recovery Baseline
SBP, mm Hg	106.28 (15.07)	113.41 (14.84)	115.17 (14.25)	115.32 (15.94)	113.53 (17.25)	115.97 (18.08)
DBP, mm Hg	71.82 (10.25)	75.36 (9.81)	75.75 (10.09)	77.42 (9.76)	76.45 (11.15)	79.21 (11.87)
MAP, mm Hg	83.52 (10.74)	87.24 (10.19)	87.64 (10.85)	88.95 (10.85)	87.70 (12.14)	90.50 (12.85)
HR, bpm	70.48 (10.66)	71.85 (10.14)	71.87 (9.80)	71.21 (10.33)	70.15 (9.42)	69.31 (9.31)
CO, l/min	4.28 (.91)	4.35 (1.02)	4.37 (1.06)	4.12 (1.06)	4.01 (1.06)	3.90 (1.05)
TPR, dyne- sec*cm ⁻⁵	1642.13 (483.68)	1731.43 (538.84)	1752.25 (520.42)	1919.94 (604.19)	1928.44 (605.23)	2066.61 (693.39)

TPR. Most of the cardiovascular indices increased over time indicating that the Finapres had an upward bias in estimating blood pressure and hemodynamic processes over time (Ristuccia et al., 1997). Therefore, the rest periods and Recovery Baseline should not be taken as resting

baselines as they were higher than the Baseline. Hence, in this chapter, the first Baseline of the whole experiment was taken as the baseline.

Correlation Between Covariates and Independent Variables. In this study, there were two between-subjects independent variables that measured hostility - HBI and Cook-Medley Cynicism. Using BSA and Baseline as the covariates helped reduce the error term for the between subjects model. A lack of significant correlation between the covariates and the between-subject independent variables (i.e., HBI, Cook-Medley Cynicism, and HBI X Cook-Medley Cynicism) enabled a less ambiguous interpretation of the between subject variables' effects since there was no confound between the covariate and the between subjects variables (Maxwell & Delaney, 2004).

Synwork Model. For cardiovascular reactivity, BSA and Baselines of SBP, DBP, MAP, HR, CO, TPR and State Anger were covariates. For cardiovascular reactivity, the Between-Subjects model was as follows:

Between Subjects Model

1. Hostile Behaviour Index (HBI)
2. Cook-Medley Cynicism
3. HBI X Cook-Medley Cynicism

The covariates were used as dependent variables. Non significant results indicated the independence between the covariates and the Hostility measures.

The results showed that there were no significant results when the two covariates were used as dependent variables, p 's ranged from .08 to 1.00. Therefore, hostility was not significantly related to cardiovascular baselines, State Anger baseline or BSA.

For cardiovascular variability, Normalised HF HR Baseline, Baroreceptor Sensitivity assessed via spectral analysis (BRS_{spectral}) Baseline, Baroreceptor Sensitivity assessed via the Sequence method (BRS_{seq}) Baseline, and Normalised HF Respiration were covariates. Analysis of BRS_{spectral} was also not feasible because the coherence of a large number of participants fell below the requirement of .50 during Anger Recall. Mean coherence (SD) between SBP and Inter Beat Interval during baseline was .65 (.18) and during Anger Recall was .41 (.19). During Baseline, 10 participants had coherence values below .50 but during the Anger Recall task, this increased to 42. Thus, Baroreflex Sensitivity will be assessed only via the Sequence Method (BRS_{seq}). Table 3.6 shows the number and percentage of cardiac cycles used in the assessment of Baroreflex Sensitivity via the Sequence Method. Percentage of sequences detected during the baseline was similar to a spontaneous breathing baseline reported by other researchers (28.39%, Pitzalis et al., 1998). Table 3.7 shows the Mean and SD of the normalised High Frequency IBI, normalised High Frequency Respiration and

Table 3.6 Number and percentages (SD) of cardiac cycles and sequences used in the calculation of Baroreflex Sensitivity (n = 58)

Dependent Variable	Baseline	Anger Recall	Task Control		
			Normal	Variable Pauses	More Variable Pauses
Total Number of Cardiac Cycles Paired with SBP	345.98 (54.65)	377.62 (54.06)	373.14 (54.82)	366.44 (49.24)	363.71 (49.13)
Number of Pairs Analysed	116.02 (79.94)	142.29 (48.11)	75.34 (42.23)	79.00 (40.37)	69.93 (38.41)
Percentage of Pairs Analysed	32.02 (18.98)	37.50 (10.81)	19.67 (9.71)	21.22 (10.23)	18.87 (9.51)
No. of Sequences Analysed	33.60 (20.56)	40.81 (12.73)	22.40 (11.61)	23.41 (11.44)	20.88 (10.93)

Baroreflex Sensitivity data by Task. Baroreflex Sensitivity for the baseline was also close to that reported elsewhere using the Phenylephrine method (19.6ms/mm Hg, Watkins et al., 1996). There was a substantial drop in the percentage of cardiac cycles analysed during the computer

task, this could reduce the reliability of this method of baroreflex assessment for this task.

There were no significant results with BRS_{seq} Baseline as the dependent variable, p 's .46 to .80. No significant results were also found when Normalised HF HR Baseline was the dependent variable, p 's .09 to .96. With Normalised HF Respiration Baseline, there were no significant results, p 's .28 to .69. Therefore, Hostility was not related to differences in BRS_{seq} Baseline, Normalised HF HR Baseline or Normalised HF Respiration Baseline.

Respiration was not only taken during Baseline but also during the three versions of Synwork. The following model was used to test for Respiration obtained during the three conditions of Task Control as the dependent variable:

Between Subjects Model

1. Hostile Behaviour Index (HBI)
2. Cook-Medley Cynicism
3. HBI X Cook-Medley Cynicism

Within Subjects Model

1. Task Control (Normal, Variable & More Variable Pauses)
2. Task X HBI
3. Task X Cook-Medley Cynicism
4. Task X HBI X Cook-Medley Cynicism

The only significant result was for Task Control, $F(1.80, 84.41) = 11.88$, $p = .06 \times 10^{-3}$, $\eta^2 = .20$. Table 3.7 shows the means and standard deviations of Normalised HF Respiration by Task Control condition.

Simple effects analysis showed that HF Respiration significantly differed

between Normal and Variable Pauses and Normal and More Variable Pauses. There was no significant difference between Variable and More Variable Pauses. Hence Respiration during Synwork was also assessed as a potential covariate to control for such differences among the three levels of Task Control.

Table 3.7 Means (SD) [Sample Size] of Normalised High Frequency Heart Rate, Normalised High Frequency Respiration and Baroreceptor Sensitivity.

Dependent Variable	Baseline	Anger Recall	Task Control		
			Normal	Variable Pauses	More Variable Pauses
High Frequency HR, nu	34.10 (18.99) [59]	22.72** (12.91) [59]	33.39 (17.86) [59]	32.83 (18.29) [58]	32.21 (16.98) [59]
High Frequency Respiration, au	84.07 (15.47) [52]	70.62 (11.87) [52]	90.50** _a (7.48) [52]	85.18 _b (10.74) [51]	86.30 _b (8.14) [52]
Baroreflex Sensitivity, ms/mm Hg	18.17 (12.00) [59]	13.77** (6.46) [59]	15.58 (7.32) [58]	16.64 (10.28) [59]	15.57 (7.95) [59]

_{a, b, c} Means with different subscripts are significantly different at $p < .01$ using Holm's Sequential Method of Bonferroni Type 1 error correction, ** Significantly different ($p < .01$) from Baseline using paired t-test and Holm's Sequential Method of Bonferroni Type 1 error correction.

To summarise the results for the Synwork model, Hostility was not related to Baselines of SBP, DBP, MAP, HR, CO, TPI, Normalised HF

HR, and Normalised HF Respiration. There were significant differences for Normalised HF Respiration by Task Control. Therefore, Normalised HF HR and BRS_{seq} needed to be corrected for changes in respiration among the different versions of Synwork. These analyses were to determine whether there were baseline differences for the between subject variables in the Synwork model. The absence of such associations reduced the ambiguity of interpreting the effects of the between subject variables on dependent variables obtained during the various experimental tasks.

Anger Recall. For cardiovascular reactivity and variability, BSA and the Baselines of SBP, DBP, MAP, HR, CO, TPR, Normalised HF HR, Normalised HF Respiration and BRS_{seq} were not related to the two measures of hostility (Cook-Medley Cynicism and HBI). Therefore, there is no need to repeat the same analyses here.

For the Synwork model and Anger Recall model, almost all of the covariates were not significantly associated with the Independent Variables. The only exception to this was for Normalised HF Respiration where differences associated with Task Control were found. Thus, the function of this covariate was to 'adjust' for Task Control differences in Normalised HF Respiration.

Homogeneity of Regression Coefficients. An important assumption when using covariates was that it should not have any significant interactions with the other between-subject independent variables. This was

particularly important if the covariate was used to adjust for between group differences in the covariate. However, as the earlier analyses showed a lack of between group differences in the covariates, the use of the covariates here was to reduce error variance. Hence the test of the homogeneity of regression coefficient was to maximise error reduction in order to increase power.

Synwork model. For cardiovascular reactivity, BSA and the Baselines of SBP, DBP, MAP, HR, CO and TPR were centred and entered into the following model as a covariate separately.

Between Subjects Model

1. Baseline (covariate)
2. Hostile Behaviour Index (HBI)
3. Cook-Medley Cynicism
4. HBI X Cook-Medley Cynicism
5. HBI X Baseline
6. Cook-Medley Cynicism X Baseline
7. HBI X Cook-Medley Cynicism X Baseline

To test for the assumption of the homogeneity of regression coefficients for BSA, the same model as that listed above was also tested but all effects with 'Baseline' were replaced with BSA. Only the relevant results for the covariates and their interactions for the between-subjects model are reported here. Any significant results involving the covariates were retained into the model.

For SBP, the main effect for SBP Baseline was significant, $F(1, 51) = 55.70$, $p = .01 \times 10^{-7}$, $\eta^2 = .52$. For DBP, there were two significant effects, DBP Baseline main effect was significant, $F(1, 51) = 32.98$, $p =$

$.05 \times 10^{-5}$, $\eta^2 = .39$ and a DBP Baseline x HBI was of borderline significance, $F(1, 51) = 5.09$, $p = .03$, $\eta^2 = .09$. Figure 3.5 shows this interaction graphically using the method described by Aiken and West (Aiken & West, 1991). The interaction was that for participants with -1SD DBP Baseline, HBI scores was positively associated with DBP reactivity and participants with +1SD DBP Baseline, the relationship between HBI and DBP reactivity was reversed.

The same pattern of results were found for MAP as well, MAP Baseline main effect was significant, $F(1, 51) = 51.35$, $p = .03 \times 10^{-7}$, $\eta^2 = .50$, while the Baseline x HBI interaction was of borderline significance,

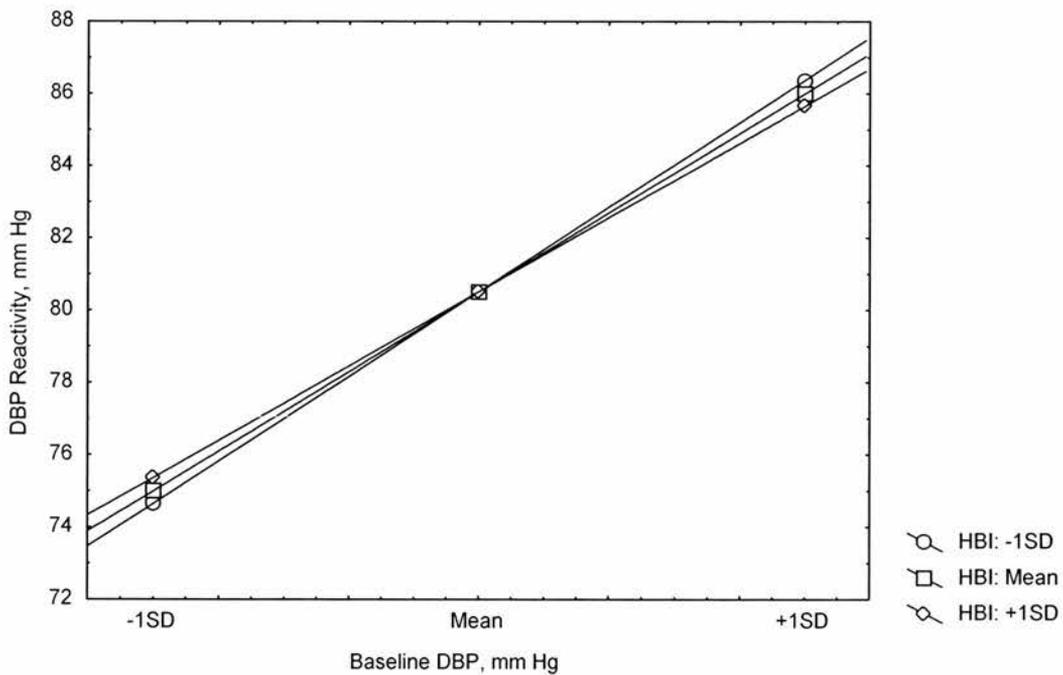


Figure 3.5 DBP Baseline x HBI interaction for DBP Reactivity

$F(1, 51) = 6.16$, $p = .02$, $\eta^2 = .11$. The nature of the Baseline x HBI interaction for MAP reactivity is the same for DBP (Figure 3.6). For Heart

Rate, a HR Baseline main effect was significant, $F(1, 51) = 66.58, p = .08 \times 10^{-9}, \eta^2 = .54$. A Cardiac Output Baseline main effect was also found, $F(1, 51) = 13.97, p = .05 \times 10^{-2}, \eta^2 = .22$. A significant TPR Baseline main effect was also found, $F(1, 51) = 9.41, p = .001, \eta^2 = .16$. Therefore, the Baseline main effect was entered as a covariate for all the dependent variables. The borderline significant interactions were not entered.

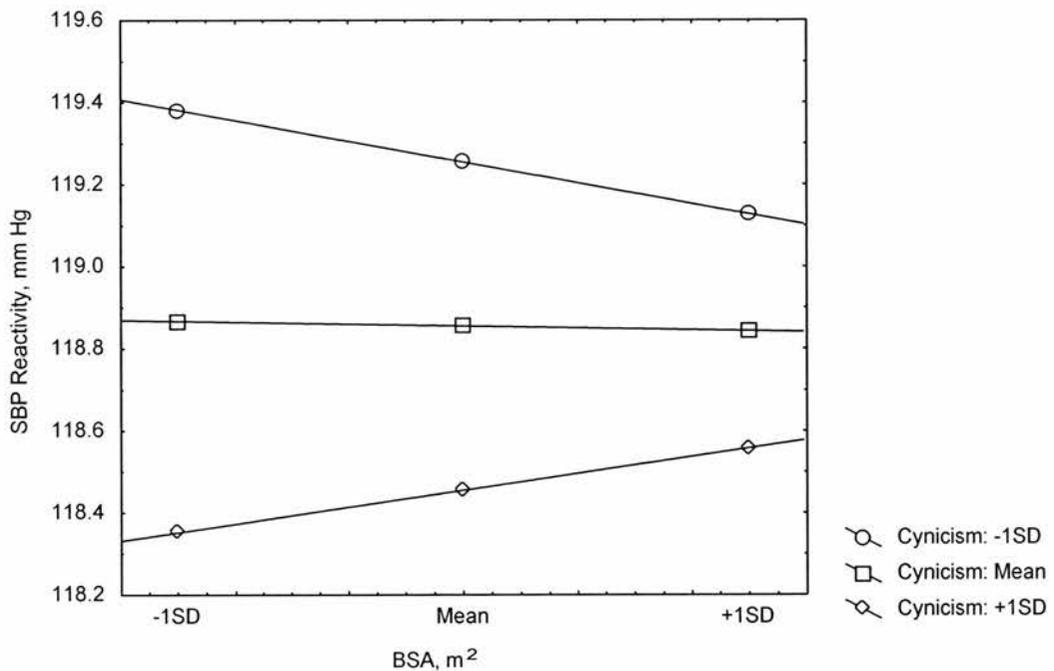


Figure 3.6 SBP Baseline x Cook-Medley Cynicism for SBP Reactivity

For SBP, BSA main effect was non-significant, $F(1, 51) = 1.85, p = .18, \eta^2 = .04$. There was a borderline significant BSA X Cook-Medley Cynicism interaction, $F(1, 51) = 6.06, p = .02, \eta^2 = .11$. Figure 3.5 shows the pattern of this interaction. For participants with -1SD BSA, low Cook-Medley Cynicism scorers had greater SBP reactivity than high Cook-

Medley Cynicism scorers. The difference in SBP reactivity between low and high Cynicism scorers was small (about 1mm Hg). This SBP reactivity difference between low and high Cynicism scorers narrowed with increasing BSA.

For DBP, BSA main effect was also non significant, $F(1, 51) = 1.63$, $p = .21$, $\eta^2 = .03$, and a borderline significant BSA x Cook-Medley Cynicism interaction, $F(1, 51) = 5.43$, $p = .02$, $\eta^2 = .10$. The pattern of this interaction was the same as for SBP as shown in Figure 3.5. BSA main effect was also not significant for MAP, $F(1, 51) = 1.18$, $p = .28$, $\eta^2 = .02$, and a BSA x Cook-Medley Cynicism interaction was of borderline significance, $F(1, 51) = 5.95$, $p = .02$, $\eta^2 = .10$. The pattern of the BSA x Cynicism interaction was similar to SBP. For HR, BSA main effect was non significant, $F(1, 51) = .72$, $p = .40$, $\eta^2 = .01$. BSA main effect was also not significant for CO, $F(1, 51) = .84$, $p = .36$, $\eta^2 = .02$, and TPR, $F(1, 51) = 2.73$, $p = .10$, $\eta^2 = .05$.

BSA was analysed here because CO and TPR are frequently divided by BSA to control for BSA differences (Sherwood et al., 1990a; Steptoe, Willemsen, Kunz-Ebrecht, & Owen, 2003). The lack of significant BSA main effects and the presence of borderline significant interactions for BSA show that this covariate would not substantially reduce error. In addition, there were no significant associations between the two Hostility measures (Cynicism and HBI) and BSA. Cardiovascular baselines could explain for as much as .50 (SBP) variance while for BSA, the highest effect size was .11. A number of studies have also reported a lack of BSA

or BMI main effect in predicting cardiovascular reactivity (Al'Absi, Everson, & Lovallo, 1995; Kamarck et al., 2000; Why et al., 2003). Hence only cardiovascular Baselines were entered as a covariate for cardiovascular reactivity.

For cardiovascular variability, Baselines of Normalised HF HR, BRS_{seq} , and Normalised HF Respiration were covariates for their respective dependent variables. Covariates were entered into the following model with Normalised HF HR reactivity and BRS_{seq} during Synwork as dependent variables:

Between Subjects Model

1. Covariate
2. Hostile Behaviour Index (HBI)
3. Cook-Medley Cynicism
4. HBI X Cook-Medley Cynicism
5. HBI X Covariate
6. Cook-Medley Cynicism X Covariate
7. HBI X Cook-Medley Cynicism X Covariate

Significant results were found for Normalised HF HR Baseline main effect, $F(1, 50) = 79.51, p = .07 \times 10^{-10}, \eta^2 = .61$, and none of the interactions involving Normalised HF HR Baseline were significant, p 's .07 to .84. A significant BRS_{seq} Baseline main effect was also found, $F(1, 53) = 36.56, p = .02 \times 10^{-5}, \eta^2 = .41$. None of the interactions involving BRS_{seq} were significant, p 's .04 to .37. When Normalised HF HR during Synwork was used as the Dependent Variable, Normalised HF Respiration Baseline main effect was not significant, $F(1, 43) = .54, p = .47, \eta^2 = .01$ and so were any interactions involving Normalised HF Respiration Baseline, p 's .32 to .76. When BRS_{seq} was used as the

dependent variable, Normalised HF Respiration Baseline main effect was also not significant, $F(1, 43) = .04$, $p = .84$, $\eta^2 = .001$ and none of the interactions involving Normalised HF Respiration Baseline were significant, p 's .23 to .90. The results showed that only Baselines of Normalised HF HR and BRS_{seq} significantly reduced error for the Between Subjects model. Hence only these two Baselines and not Normalised HF Respiration Baseline were included as a between subject covariate for their respective dependent variables.

As was mentioned earlier, respiration was also measured during the three versions of Synwork. Two versions of this Respiration data were created; one was created by taking the mean of the Normalised HF Respiration obtained during the three versions of Synwork. This new variable ($Resp_{btw}$) represent Normalised HF Respiration during Synwork averaged over the three Synwork conditions and was entered into the between subjects model. Another version ($Resp_{wtn}$) matched the Normalised HF Respiration to the conditions of Task Control and was entered into the within subjects model.

The following model was used to test Normalised HF $Resp_{btw}$ and $Resp_{wtn}$ as a covariate using Normalised HF HR during the three Synwork Control conditions as dependent variables:

Between Subjects Model

1. Normalised HR $Resp_{btw}$
2. Hostile Behaviour Index (HBI)
3. Cook-Medley Cynicism
4. HBI X Normalised HR $Resp_{btw}$
5. Cynicism X Normalised HR $Resp_{btw}$

6. HBI X Cook-Medley Cynicism
7. HBI X Cook-Medley Cynicism X Normalised HR Resp_{btw}

Within Subjects Model

1. Normalised HF Resp_{wtn}
2. Task Control (Synwork Normal, Variable & More Variable Pauses)
3. Task Control X Normalised HF Resp_{wtn}
4. Task Control X HBI
5. Task Control X Cook-Medley Cynicism
6. Task Control X HBI X Normalised HF Resp_{wtn}
7. Task Control X Cook-Medley Cynicism X Normalised HF Resp_{wtn}
8. Task Control X HBI X Cook-Medley Cynicism
9. Task Control X HBI X Cook-Medley Cynicism X Normalised HF Resp_{wtn}

The main effects for Normalised HF Resp_{btw} and HF Resp_{wtn} were not significant, $F(1, 39.90) = .15, p = .70, \eta^2 = .003$, and $F(1, 79.81) = 2.27, p = .14, \eta^2 = .02$ respectively. All interactions involving either Normalised HF Resp_{btw} or Resp_{wtn} were not significant, p 's .20 to .95. Hence, Normalised HF Resp_{wtn} was not used as a covariate for normalised HF HR since it did not predict significant variance in the model. To summarise the results for cardiovascular variability, only the Baselines of Normalised HF HR and BRS_{seq} were found to significantly reduce error variance. Hence, for cardiovascular variability only Normalised HF HR Baseline and BRS_{seq} Baseline were used as covariates.

For the Synwork Model, the covariates were not significantly associated with any of the between-subject independent variables. This showed that Hostility was not significantly related to differences in Baselines of cardiovascular reactivity/variability and BSA. No covariates interacted significantly with the independent variables as well. The

Baselines of the various dependent variables were found to significantly reduce error variance in the model. BSA and Respiration did not have any significant effect. Hence only Baseline was used as a covariate in the Synwork model.

Anger Recall Model. The series of analyses done for the Synwork model was repeated here for the Anger Recall model. This was the model tested for the Anger Recall model:

Between Subjects Model

1. Covariate
2. Hostile Behaviour Index (HBI)
3. Cook-Medley Cynicism
4. HBI X Cook-Medley Cynicism
5. HBI X Covariate
6. Cook-Medley Cynicism X Covariate
7. HBI X Cook-Medley Cynicism X Covariate

For cardiovascular reactivity, the Baseline of the respective dependent variables and BSA were covariates. For SBP, SBP Baseline main effect was significant, $F(1, 51) = 51.03, p = .03 \times 10^{-7}, \eta^2 = .50$ and none of the interactions involving SBP Baseline were significant, p 's .35 to .91. For DBP, a DBP Baseline main effect was found, $F(1, 51) = 42.76, p = .03 \times 10^{-6}, \eta^2 = .46$, and a DBP Baseline x HBI was also significant, $F(1, 51) = 10.18, p = .001, \eta^2 = .17$. Figure 3.7 shows this interaction. The pattern of the interaction was the same as that for the Synwork model except that the effect size was larger for the Anger Recall model. Low HBI scores with low DBP Baseline was associated with the lowest DBP reactivity during Anger Recall but Low HBI scores with high DBP Baseline

were associated with the highest DBP reactivity. For MAP, MAP Baseline main effect was significant, $F(1, 51) = 62.40, p = .02 \times 10^{-8}, \eta^2 = .55$, and a MAP Baseline x HBI interaction was significant, $F(1, 51) = 8.21, p = .006$,

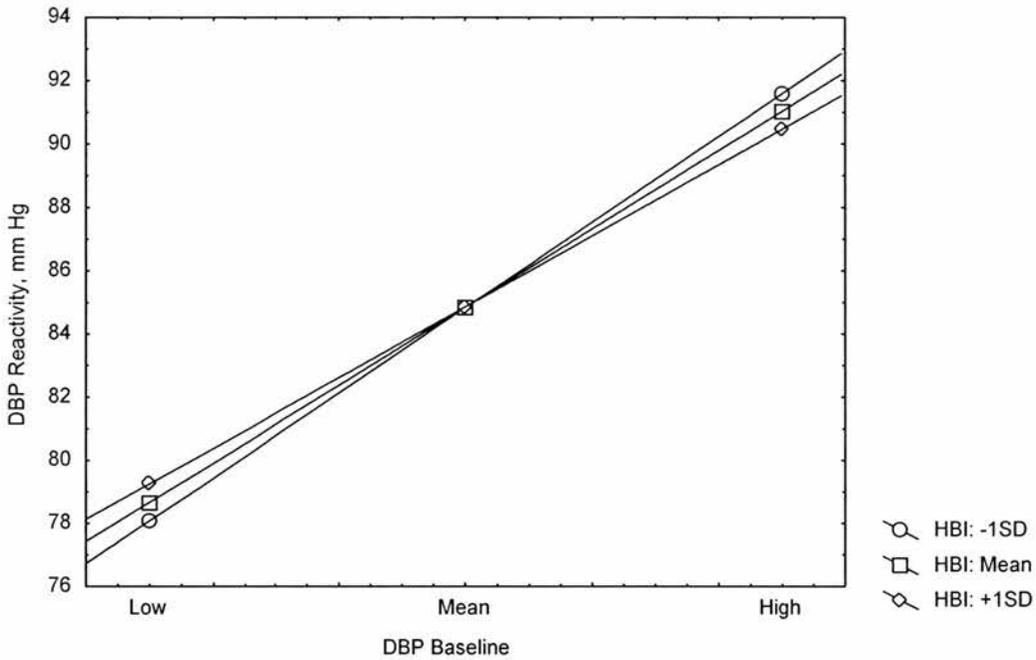


Figure 3.7 DBP Baseline x HBI interaction for Anger Recall

$\eta^2 = .14$. The pattern of the MAP Baseline x HBI interaction was similar to the one found for DBP. For HR, only HR Baseline main effect was significant, $F(1, 51) = 96.29, p = .02 \times 10^{-11}, \eta^2 = .65$. No other interactions involving HR Baseline was significant, p 's .06 to .88. For CO, CO Baseline was of borderline significance, $F(1, 51) = 6.28, p = .02, \eta^2 = .11$, no other significant results involving CO Baseline were found, p 's .12 to .93. For TPR, TPR Baseline main effect was of borderline significance,

$F(1, 51) = 5.95, p = .02, \eta^2 = .10$. A TPR Baseline x Cynicism interaction was also of borderline significance, $F(1, 51) = 6.35, p = .02, \eta^2 = .11$.

To summarise the cardiovascular reactivity results for the Anger Recall model, the baselines of SBP, DBP, MAP and HR were significant predictors of cardiovascular reactivity during Anger Recall. CO and TPR Baselines main effects were of borderline significance. For DBP and MAP, significant Baseline x HBI interactions were found. Therefore, for these two dependent variables, this interaction term was included into the Anger Recall model. For CO and TPR, though their respective baseline main effects were of borderline significance, they were also entered into the model because all the other dependent variables in the Synwork model as well as the Anger Recall model had the Baseline entered as a covariate.

For cardiovascular variability, Baselines of Normalised HF HR and BRS_{seq} were covariates. Respiration collected during Anger Recall model was used as a covariate for this model as well.

For Normalised HF HR, Normalised HF HR Baseline main effect was significant, $F(1, 51) = 51.16, p = .03 \times 10^{-11}, \eta^2 = .50$. There was also a borderline significant Normalised HF HR Baseline x Cynicism x HBI interaction, $F(1, 51) = 6.77, p = .01, \eta^2 = .12$. For BRS_{seq} , BRS_{seq} Baseline main effect was significant, $F(1, 51) = 37.82, p = .01 \times 10^{-9}, \eta^2 = .43$. No interactions involving BRS_{seq} were significant, p 's .17 to .82. When Normalised HF HR was used as the dependent variable, Respiration Reactivity main effect was not significant, $F(1, 44) = 1.23, p =$

.27, $\eta^2 = .03$. No significant interactions involving respiration were significant, p 's .54 to .95.

For cardiovascular variability of the Anger Recall model, Baselines of Normalised HF HR and BRS_{seq} were entered as covariates while respiration was not used as a covariate.

Control Variables

Task control was defined as the duration of random pauses generated by the Faulty Mouse device. There were three levels of task control - 'Normal', 'Variable Pauses' and 'More Variable Pauses'. An one-way repeated measures ANOVA showed that there was a significant main effect for Task scores, $F(1.79, 103.64) = 86.84, p = .02 \times 10^{-13}, \eta^2 = .60$. Simple effects analysis showed that task scores obtained during all the three Task Control conditions were significantly different from one another (see Table 3.8). An one-way repeated measures ANOVA also revealed a significant main effect for Perceived Control, $F(1.93, 111.82) = 119.55, p = .03 \times 10^{-17}, \eta^2 = .67$. The linear and quadratic components of this main effect were both significant, $F(1, 58) = 134.24, p = .01 \times 10^{-14}, \eta^2 = .70$, and $F(1, 58) = 103.11, p = .02 \times 10^{-12}, \eta^2 = .64$ respectively. As expected, participants' perceived control of the computer task with Variable Pauses was significantly lower than the Normal Control condition. However, their perceptions of control for the computer task with More Variable Pauses was not the lowest but was between the Normal and the Variable Pauses condition. The actual difference of Perceived

Control ratings between the Variable and More Variable Pauses Control conditions is much smaller ($3.58 - 3.15 = .43$). Perceived Control

Table 3.8 Means (SD) of Synwork Scores, and Perceived Control by Task Control (n = 59)

	Task Control		
	Normal	Variable Pauses	More Variable Pauses
Synwork Scores	585.8 _a (131.5)	183.8 _b (270.5)	386.0 _c (195.6)
Perceived Control, cm	5.55 _a (.87)	3.15 _b (1.20)	3.58 _c (1.18)

^{a, b, c} Means with different subscripts are significantly different at $p < .05$ using Bonferroni correction for Type 1 error. All Synwork Scores are significantly different '0', $p < .01$.

differences were about 5 times greater between Normal and Variable Pauses, and Normal and More Variable Pauses. Simple effects analysis showed the same patterns as that reported for Task scores (Table 3.8).

Main Analyses for Physiological Reactivity

Table 3.9 shows the means and standard deviations for the six dependent variables during the first baseline, Anger Recall, active and passive versions of the Synwork Task. Hypotheses testing for Anger Recall are reported first followed by Synwork. As the hypotheses testing in this chapter used the Baselines as a covariate and ANCOVA to operationalised reactivity, the results would not indicate whether

cardiovascular functions changed significantly from baseline. Table 3.7 showed that Normalised HF HR and BRS_{seq} decreased significantly from Baseline during Anger Recall. During Synwork Normal Control, Normalised HF Respiration increased significantly from Baseline though later analysis found that Respiration did not predict significant variance in Normalised HF HR.

Table 3.9 Means (SD) for the Physiological Data (n = 59)

	Baseline	Anger Recall	Synwork Task Control		
			Normal	Variable Pauses	More Variable Pauses
SBP, mm Hg	106.28 (15.07)	121.43* (19.17)	120.68* (16.34)	118.40* (18.54)	116.29* (18.75)
DBP, mm Hg	71.82 (10.31)	84.67* (14.67)	81.27* (11.81)	80.33* (11.95)	79.82* (11.81)
MAP, mm Hg	83.52 (10.74)	96.57* (15.77)	93.59* (13.06)	92.15* (13.39)	91.23* (13.27)
HR, bpm	70.48 (10.66)	77.18* (9.71)	75.77* (10.71)	74.63* (9.20)	73.38* (9.75)
CO, l/min	4.28 (.91)	4.30 (1.10)	4.69 (1.16)	4.33 (1.12)	4.19 (1.11)
TPR, dyne-sec*cm ⁻⁵	1642.13 (483.68)	1950.85 (760.64)	1693.76 (510.79)	1818.59 (578.18)	1860.34 (561.60)

*Significantly different from Baseline ($p < .01$) using paired samples t-test with Holm's Sequential method of Bonferroni Type 1 error correction.

As Table 3.9 shows, blood pressure and heart rate measures were significantly different from Baseline. However, there were no significant changes in the underlying hemodynamic processes (CO and TPR) from

Baseline. This was because $MAP = \log CO + \log TPR$ (Gregg, Matyas, & James, 2002). Therefore large effects found in blood pressure could be 'translated' into smaller effects in both CO and TPR.

The same analysis was done for the UNWIST Hedonic Tone mood ratings. Table 3.10 shows that Variable or More Variable Pauses conditions were associated with a decrease in Hedonic mood item ratings and an increase in Anger item ratings. Anger Recall was also associated

Table 3.10 Mean (SD) of UNWIST Hedonic Tone Mood Ratings

	Baseline	Anger Recall	Task Control		
			Normal	Variable Pauses	More Variable Pauses
Hedonic Tone					
Hedonic items	3.19 (.44)	2.36* (.71)	3.08 (.42)	2.60* (.59)	2.68* (.58)
Anger items	1.27 (.40)	2.35* (.81)	1.57* (.57)	2.35* (.84)	2.27* (.81)

*Significantly different from Baseline ($p < .01$) using paired samples t-test with Holm's Sequential method of Bonferroni Type 1 error correction.

with a significant decrease in Hedonic item ratings and a significant increase in Anger mood item ratings.

The aim of manipulating Task Control was to increase State Anger via decreasing Perceived and Task Control. Pearson correlations between Perceived Control and the Hedonic Tone mood items used showed that only in the More Variable Pauses condition was Perceived Control significantly associated with higher levels of Anger and lower

levels of Happiness (Table 3.11). For State Anger, the negative correlation between Perceived Control and State Anger increases in magnitude as Task Control decreases.

Table 3.11 Correlations between Perceived Control and State Anger by Task Control condition (n=59)

	Task Control		
	Normal	Variable Pauses	More Variable Pauses
Hedonic Tone - Hedonic items			
Perceived Control	.24	.21	.49*
Hedonic Tone - Anger items			
Perceived Control	-.13	-.27	-.43*

*Significant at $p < .05$ using Holm's Sequential method of Bonferroni Type 1 error correction

Hypotheses Testing

To test Hypotheses in (1) and (2), the following between subjects model was tested¹⁵:

Between Subjects Model

1. Baseline (covariate)
2. Hostile Behaviour Index (HBI)
3. Cook-Medley Cynicism
4. HBI X Cook-Medley Cynicism

¹⁵ Though the Between Subject and Within Subject models are reported separately for hypotheses testing, they are analysed simultaneously.

Two effect sizes were reported for the between subject model: η^2 and partial η^2 . Partial η^2 was calculated using a denominator that excluded the sums of squares predicted by Baseline or any of its related interactions. This effect size allowed comparison with other studies that eliminated the effect of the Baseline via other methods (e.g., gain scores). However, the within subject model does not have covariates, so the η^2 allows comparison of the effect sizes between the between subject model and within subject model.

Hypothesis 1: Cynicism and Cardiovascular reactivity/variability

1a) Cynicism 12-item scores are positively related to cardiovascular stress reactivity.

Synwork model. For cardiovascular reactivity, Cynicism main effect was not significant. Specifically, SBP, Cynicism main effect was $F(1, 54) = .68, p = .41, \eta^2 = .006, \text{partial } \eta^2 = .01$, DBP, $F(1, 54) = 2.27, p = .14, \eta^2 = .03, \text{partial } \eta^2 = .04$, MAP, $F(1, 54) = 2.09, p = .15, \eta^2 = .02, \text{partial } \eta^2 = .04$, HR, $F(1, 54) = 5.01, p = .03, \eta^2 = .04, \text{partial } \eta^2 = .08$. The trend of the Cynicism main effect for HR was negative instead of positive. CO, $F(1, 54) = .07, p = .80, \eta^2 = .001, \text{partial } \eta^2 = .001$, and TPR, $F(1, 54) = .69, p = .41, \eta^2 = .01, \text{partial } \eta^2 = .01$.

Five of the 6 Dependent Variables showed non significant results for Cynicism in predicting cardiovascular reactivity. The trend for HR was in the opposite direction to that hypothesised.

Anger Recall model. The main effect of Cynicism was not significant for cardiovascular reactivity¹⁶. SBP, $F(1, 54) = .16, p = .90, \eta^2 = .02 \times 10^{-2}$, partial $\eta^2 = .03 \times 10^{-2}$, DBP, $F(1, 53) = .48, p = .49, \eta^2 = .004$, partial $\eta^2 = .009$, MAP, $F(1, 53) = .34, p = .56, \eta^2 = .003$, partial $\eta^2 = .006$, HR, $F(1, 54) = 2.14, p = .15, \eta^2 = .01$, partial $\eta^2 = .04$, CO, $F(1, 54) = .40, p = .53, \eta^2 = .007$, partial $\eta^2 = .007$, and TPR, $F(1, 54) = 1.58, p = .21, \eta^2 = .03$, partial $\eta^2 = .03$. The main effect of Cynicism was also found not to be significant for the Anger Recall model.

Therefore, Hypothesis 1a was not supported - Cynicism was not positively associated with cardiovascular reactivity.

1b) Cynicism 12-item scores are negatively related to baroreflex sensitivity (BRS_{seq}). High Cynicism scores are associated with reduced baroreflex sensitivity.

Synwork Model. The main effect of Cynicism for BRS_{seq} was not significant, $F(1, 54) = 4.50, p = .04, \eta^2 = .05$, partial $\eta^2 = .08$. There was a trend for BRS_{seq} . However, the pattern was in the opposite direction as hypothesised - participants with high Cynicism had greater BRS_{seq} reactivity during Synwork.

Anger Recall Model. The main effect of Cynicism for BRS_{seq} , $F(1, 54) = .92, p = .34, \eta^2 = .01$, partial $\eta^2 = .02$.

¹⁶ For the Anger Recall model, a Baseline x HBI has been entered for DBP and MAP as dependent variables. Hence the change in degrees of freedom (see section 'Testing the Suitability of Covariates': 'Homogeneity of Regression Coefficients').

Therefore Hypothesis 1b was not supported. Cynicism was not positively related to decreased BRS reactivity during psychological stress.

1c) Cynicism 12-item scores are negatively related to parasympathetic cardiac activity (Normalised HF HR).

Synwork Model. For cardiovascular variability, the main effect of Cynicism for Normalised HF HR was $F(1, 54) = .16, p = .70, \eta^2 = .001$, partial $\eta^2 = .003$.

Anger Recall Model. The main effect of Cynicism for Normalised HF HR was $F(1, 54) = .06, p = .81, \eta^2 = .001$, partial $\eta^2 = .001$.

Hypothesis 1c was not supported. Cynicism was not related to decreased parasympathetic cardiac responses (Normalised HF HR) during stress.

Hypothesis 2: *Verbal Aggression (HBI) and cardiovascular reactivity and variability.*

2a) Structured Interview-derived HBI scores are positively related cardiovascular reactivity.

Synwork model. Main effect of HBI was not significant. Specifically, for SBP, $F(1, 54) = .48, p = .49, \eta^2 = .004$, partial $\eta^2 = .009$, DBP, $F(1, 54) = .17, p = .69, \eta^2 = .002$, partial $\eta^2 = .003$, MAP, $F(1, 54) = .38, p = .54, \eta^2 = .004$, partial $\eta^2 = .007$, HR, $F(1, 54) = .29, p = .59, \eta^2 = .002$, partial $\eta^2 =$

.005, CO, $F(1, 54) = .07$, $p = .79$, $\eta^2 = .03 \times 10^{-3}$, partial $\eta^2 = .03 \times 10^{-3}$, and TPR, $F(1, 54) = .28$, $p = .60$, $\eta^2 = .004$, partial $\eta^2 = .005$.

Anger Recall model. Main effect of Cynicism was not significant for cardiovascular reactivity. SBP, $F(1, 54) = .17$, $p = .68$, $\eta^2 = .002$, partial $\eta^2 = .003$, DBP, $F(1, 53) = 1.67$, $p = .20$, $\eta^2 = .02$, partial $\eta^2 = .03$, MAP, $F(1, 53) = 1.27$, $p = .26$, $\eta^2 = .01$, partial $\eta^2 = .02$, HR, $F(1, 54) = 1.92$, $p = .17$, $\eta^2 = .01$, partial $\eta^2 = .03$, CO, $F(1, 54) = .25$, $p = .62$, $\eta^2 = .004$, partial $\eta^2 = .005$, and TPR, $F(1, 54) = 1.51$, $p = .22$, $\eta^2 = .02$, partial $\eta^2 = .03$.

The results for the Synwork and Anger Recall model show that HBI was not positively related to cardiovascular reactivity. Hence Hypothesis 2a was not supported.

2b) Structured Interview-derived IHAT scores are negatively related to baroreflex sensitivity (BRS_{seq}). Participants who higher levels of verbal aggression during the structured interview have reduced baroreflex sensitivity during stress.

Synwork model. Main effect of HBI for BRS_{seq} was $F(1, 54) = .004$, $p = .95$, $\eta^2 = .04 \times 10^{-3}$, partial $\eta^2 = .06 \times 10^{-3}$.

Anger Recall model. Main effect of HBI for BRS_{seq} , $F(1, 54) = .068 \times 10^{-4}$, $p = 1.00$, $\eta^2 = .01 \times 10^{-5}$, partial $\eta^2 = .01 \times 10^{-5}$.

Hypothesis 2b was not supported - HBI was not negatively related to BRS_{seq} .

2c) Structured Interview-derived HBI scores are negatively related to parasympathetic cardiac activity (Normalised HF HR). Participants who higher levels of verbal aggression during the structured interview have lower parasympathetic cardiac activity during stress.

Synwork model. Main effect of HBI for Normalised HF HR is $F(1, 54) = .01 \times 10^{-5}$, $p = 1.00$, $\eta^2 = .08 \times 10^{-8}$, partial $\eta^2 = .02 \times 10^{-7}$.

Anger Recall model. Main effect of HBI for Normalised HF HR was $F(1, 54) = .005$, $p = .94$, $\eta^2 = .05 \times 10^{-3}$, partial $\eta^2 = .01 \times 10^{-2}$.

Hypothesis 2c was not supported. HBI was not negatively associated with a reduction of parasympathetic cardiac activity during Synwork and Anger Recall.

Hypothesis 3: Hostility and cardiovascular reactivity/variability

Since both Cynicism and HBI are two aspects of Hostility, it was hypothesised that participants scoring high in both would have significantly greater cardiovascular reactivity/variability. A Cynicism x HBI interaction would be significant in predicting cardiovascular reactivity/variability.

3a) The pattern of the interaction would be that participants scoring high in both Cynicism and HBI would have greater reactivity than participants scoring low in both.

Synwork model. HBI x Cynicism interaction was not significant.

Specifically, SBP, HBI x Cynicism interaction was $F(1, 54) = .04, p = .85, \eta^2 = .03 \times 10^{-2}$, partial $\eta^2 = .07 \times 10^{-2}$, DBP, $F(1, 54) = 1.64, p = .21, \eta^2 = .003$, partial $\eta^2 = .005$, MAP, $F(1, 54) = .17, p = .68, \eta^2 = .002$, partial $\eta^2 = .003$, HR, $F(1, 54) = 2.59, p = .11, \eta^2 = .02$, partial $\eta^2 = .04$, CO, $F(1, 54) = .49, p = .49, \eta^2 = .007$, partial $\eta^2 = .009$, and TPR, $F(1, 54) = .03, p = .86, \eta^2 = .001$, partial $\eta^2 = .001$.

Anger Recall model. HBI x Cynicism interaction was not significant. Specifically, SBP, HBI x Cynicism interaction was $F(1, 54) = .13, p = .72, \eta^2 = .001$, partial $\eta^2 = .002$, DBP, $F(1, 53) = .37, p = .55, \eta^2 = .003$, partial $\eta^2 = .007$, MAP, $F(1, 53) = .50, p = .48, \eta^2 = .004$, partial $\eta^2 = .009$, HR, $F(1, 54) = .35, p = .56, \eta^2 = .002$, partial $\eta^2 = .006$, CO, $F(1, 54) = .008, p = .93, \eta^2 = .01 \times 10^{-2}$, partial $\eta^2 = .02 \times 10^{-2}$, and TPR, $F(1, 54) = .34, p = .56, \eta^2 = .005$, partial $\eta^2 = .006$.

Hypothesis 3a was not supported. Cynicism x HBI interaction was not significant.

3b) Participants scoring high in both Cynicism and HBI would have greater reduction in Baroreflex Sensitivity (BRS_{seq}) than participants scoring low in both.

Synwork model. Cynicism x HBI interaction for BRS_{seq} is $F(1, 54) = .50, p = .48, \eta^2 = .005$, partial $\eta^2 = .008$.

Anger Recall model. Cynicism x HBI interaction for BRS_{seq} , $F(1, 54) = 2.23, p = .14, \eta^2 = .02, \text{partial } \eta^2 = .04.$

Hypothesis 3b was not supported - Cynicism x HBI interaction was not significant for BRS_{seq} reactivity.

3c) Participants scoring high in both Cynicism and HBI would have greater reduction in Parasympathetic Heart Rate activity (Normalised HF HR) than participants scoring low in both.

Synwork model. Cynicism x HBI interaction for Normalised HF HR was $F(1, 54) = .65, p = .42, \eta^2 = .005, \text{partial } \eta^2 = .01.$

Anger Recall model. Cynicism x HBI interaction for Normalised HF HR was $F(1, 54) = 1.13, p = .29, \eta^2 = .01, \text{partial } \eta^2 = .02.$

Hypothesis 3c was not supported. Cynicism X HBI interaction was not significant for Normalised HF HR.

Hypothesis 4: Task Control differences for Cardiovascular Reactivity and Variability

This hypothesis focuses on the hemodynamic processes (CO and TPR) for cardiovascular reactivity in the Synwork model. However, an analysis of blood pressure is important in order to interpret the underlying hemodynamic profile. The following are the blood pressure results for Task Control main effect:

Synwork model. Task Control main effect was not significant for SBP, $F(1.90, 107.39) = 3.43, p = .07, \eta^2 = .06$, DBP, $F(1.74, 102.58) = .92, p = .34, \eta^2 = .01$, and MAP, $F(1.73, 102.39) = 2.13, p = .15, \eta^2 = .03$. The results showed that there were no differences in SBP, DBP and MAP for the 3 versions of Synwork (Normal, Variable and More Variable Pauses).

4a) Synwork with Normal Control is characterised by an active coping physiological response (increased CO and decreased TPR) while Synwork with Variable and More Variable Pauses is characterised by passive coping physiological response (decreased CO and increased TPR). Task Control main effect is significant for both CO and TPR.

Synwork model. Task Control main effect was significant for CO, $F(1.96, 109.12) = 23.47, p = .04 \times 10^{-4}, \eta^2 = .29$. For TPR, Task Control was borderline significant, $F(1.66, 99.98) = 5.78, p = .02, \eta^2 = .08$. Table 3.12 shows the means (SD) for CO and TPR by Task Control condition. Simple effects analysis was done for CO since Task Control main effect was significant for this dependent variable. As Table 3.12 shows, blood pressure during Synwork Normal Control was mainly due to increase in CO as TPR values were close to baseline. When Synwork had Variable or More Variable Pauses, CO fell while there was a gradual increase in TPR. Blood pressure during these conditions was maintained by TPR. Hence Hypothesis 4a was supported - Synwork during Normal Control was associated with an active coping physiological response (increased

Table 3.12 Simple Effects Analysis for CO by Task Control condition*

	Baseline	Task Control		
		Normal	Variable Pauses	More Variable Pauses
MAP, mm Hg	83.52 (10.74)	93.59 (13.06)	92.15 (13.39)	91.23 (13.27)
CO, l/min	4.28 (.91)	4.69 _a (1.16)	4.34 _b (1.12)	4.19 _b (1.11)
TPR, dyne-sec*cm ⁻⁵	1642.13 (483.68)	1693.76 (510.79)	1818.59 (578.18)	1860.34 (561.60)

^{a, b, c} MAP, TPR and Baseline data are included in this table to aid interpretation. Numbers with different subscripts are significantly different using Holm's Sequential Bonferroni method for Type 1 error correction.

CO and decreased TPR) while Synwork during Variable and More Variable Pauses was associated with a passive physiological response (decreased CO and increased TPR).

Hypothesis 5: Task Control (Actual Control) and Perceived Control

5a) Task Control and Perceived Control predict common/unique variance for the dependent variables.

Synwork model. This was tested for Synwork model using CO since a significant Task Control main effect was found for this dependent variable. Therefore, this dependent variable will be best suited to investigate whether Perceived Control and Task Control predict common or unique variance. This model was tested for Task Control:

Between Subjects Model

1. Co Baseline
2. Hostile Behaviour Index (HBI)
3. Cook-Medley Cynicism
4. HBI X Cook-Medley Cynicism

Within Subjects Model (Actual Control)

1. Task Control (Synwork Normal, Variable & More Variable Pauses)
2. Task X HBI
3. Task X Cook-Medley Cynicism
4. Task X HBI X Cook-Medley Cynicism

Another model was tested where the Within Subject model is replaced with the following:

Within Subjects Model (Perceived Control)

1. Perceived Control
2. Perceived Control X HBI
3. Perceived Control X Cook-Medley Cynicism
4. Perceived Control X HBI X Cook-Medley Cynicism

Then both Within Subjects Model for Actual and Perceived Control were entered into the model. Order in which the two Within Subject models were entered into the regression model was counterbalanced. Doing so would show whether Perceived and Actual Control predict unique or common variance and also which form of Control assessment predicts more variance.

When Perceived Control and its interactions replaced Task Control, Perceived Control main effect was significant, $F(1, 112.90) = 16.98$, $p = .07 \times 10^{-3}$, $\eta^2 = .13$. The slope showed that Perceived Control was positively associated with CO, $b(SE) = .099 (.02)$. This pattern was similar to that of Task Control. Table 3.13 shows the changes in effect

Table 3.13 Effect sizes from the Two Within Subject Models involving Task and Perceived Control

Main Effect	Task & Perceived Control Entered Separated	Task Control Entered 1st	Perceived Control Entered 1st
Task Control	$\eta^2 = .291$	$\eta^2 = .291$	$\eta^2 = .166$
Perceived Control	$\eta^2 = .128$	$\eta^2 = .04 \times 10^{-2}$	$\eta^2 = .128$

sizes (η^2) for the various models analysed. The analyses showed that Perceived Control and Task Control did not predict unique variance for CO reactivity in the Synwork model. Task Control predicted more variance than Perceived Control. In addition, Perceived Control predicted about 44% of what Task Control predicted for CO reactivity. Task Control offered a better model fit than Perceived Control.

Since Perceived Control did not predict unique variance from Task Control and Task Control predicted more variance than Perceived Control, subsequent hypotheses testing involving Hostility x Control interactions will only report results from Task Control.

Hypothesis 6: The Hostility and Cardiovascular reactivity/variability relationship is moderated by Task Control. The hypotheses tested involve only the Synwork model.

6a) There is a significant interaction between Cynicism and Task Control for cardiovascular reactivity. Specifically, participants scoring high

in Cynicism have greater cardiovascular reactivity, as Synwork becomes less controllable.

For cardiovascular reactivity, Cynicism x Task Control interaction was not significant. For SBP, $F(1.90, 107.39) = .56, p = .45, \eta^2 = .009$, DBP, $F(1.74, 102.58) = .57, p = .45, \eta^2 = .009$, MAP, $F(1.73, 102.39) = .62, p = .43, \eta^2 = .009$, HR, $F(1.74, 102.58) = .35, p = .56, \eta^2 = .005$, CO, $F(1.96, 109.12) = .08, p = .78, \eta^2 = .09 \times 10^{-2}$, and TPR, $F(1.66, 99.98) = 1.40, p = .24, \eta^2 = .02$. Hypothesis 6a was not supported, Cynicism was not positively related to cardiovascular reactivity as Synwork task becomes less controllable.

6b) Cynicism is associated with decreased parasympathetic cardiac activity (Normalised HF HR) and decreased BRS (BRS_{seq}) as Synwork becomes less controllable.

For cardiovascular variability, Cynicism x Task Control interaction was also not significant. For Normalised HF HR, $F(1.86, 105.29) = .03, p = .85, \eta^2 = .06 \times 10^{-2}$, and BRS_{seq} , $F(1.73, 101.65) = .25, p = .62, \eta^2 = .004$. Therefore Hypothesis 6b was not supported, Cynicism was not related to decreased parasympathetic cardiac activity and decreased BRS as Synwork becomes less controllable.

6c) There is a significant HBI x Task Control interaction for cardiovascular reactivity. Specifically, participants scoring high in HBI

have greater cardiovascular reactivity as the Computer Task becomes less controllable.

For cardiovascular reactivity, Cynicism x Task Control interaction was not significant. For SBP, $F(1.90, 107.39) = .68, p = .41, \eta^2 = .01$, DBP, $F(1.74, 102.58) = .36, p = .55, \eta^2 = .006$, MAP, $F(1.73, 102.39) = .68, p = .41, \eta^2 = .01$, HR, $F(1.74, 102.58) = .16, p = .69, \eta^2 = .002$, CO, $F(1.96, 109.12) = .62, p = .43, \eta^2 = .008$, and TPR, $F(1.66, 99.98) = .002, p = .96, \eta^2 = .03 \times 10^{-3}$. Hypothesis 6c was not supported, HBI was not positively related to cardiovascular reactivity as Synwork task became less controllable.

6d) There is a significant HBI x Task Control interaction for cardiovascular variability. Specifically, participants scoring high in HBI have reduced parasympathetic cardiac activity (reduced Normalised HF HR) and reduced BRS as the Computer Task becomes less controllable.

For cardiovascular variability, HBI x Task Control interaction was also not significant. For Normalised HF HR, $F(1.86, 105.29) = .78, p = .38, \eta^2 = .01$, and BRS_{seq} , $F(1.73, 101.65) = .85, p = .36, \eta^2 = .01$. Therefore Hypothesis 6c was not supported, HBI was not related to decreased parasympathetic cardiac activity and decreased BRS as Synwork became less controllable.

6e) There is a significant HBI x Cynicism x Task Control interaction for cardiovascular reactivity. Specifically, participants scoring high in both HBI and Cynicism have greater cardiovascular reactivity as the Synwork Task becomes less controllable.

HBI x Cynicism x Task Control interaction was not significant. For SBP, $F(1.90, 107.39) = .86, p = .36, \eta^2 = .01$, DBP, $F(1.74, 102.58) = .70, p = .41, \eta^2 = .01$, MAP, $F(1.73, 102.39) = .60, p = .44, \eta^2 = .01$, HR, $F(1.74, 102.58) = .17, p = .68, \eta^2 = .003$, CO, $F(1.96, 109.12) = .81, p = .37, \eta^2 = .01$, and TPR, $F(1.66, 99.98) = 1.20, p = .28, \eta^2 = .02$.

Hypothesis 6c was not supported, participants scoring high in both HBI and Cynicism did not have greater cardiovascular reactivity as Synwork task became less controllable.

6f) There is a significant HBI x Cynicism x Task Control interaction for cardiovascular variability. Specifically, participants scoring high in HBI and Cynicism have reduced parasympathetic cardiac activity (reduced Normalised HF HR) and reduced BRS as the Computer Task becomes less controllable.

For cardiovascular variability, HBI X Cynicism X Task Control interaction was also not significant. For Normalised HF HR, $F(1.86, 105.29) = 1.32, p = .25, \eta^2 = .02$, and BRS_{seq} , $F(1.73, 101.65) = .32, p = .57, \eta^2 = .005$. Therefore Hypothesis 6c was not supported, participants

scoring high for HBI and Cynicism did have decreased parasympathetic cardiac activity and decreased BRS as Synwork became less controllable.

Hypothesis 7: Hostility, Perceived Control_{btw} and Cardiovascular reactivity/variability

Perceived Control was given by the participants after each Synwork session. In testing Hypothesis 6, it was shown that Perceived Control did not explain unique variance from Task Control and that Task Control accounted for the variance explained by Perceived Control and more. However, Perceived Control could also be averaged for each participant to create a between subject variable - Perceived Control_{btw}. Perceived Control_{btw} was tested as a between subject variable in the between subject model.

7a) Hostility-Cardiovascular reactivity/variability relationship is moderated by Perceived Control_{btw}. It is hypothesised that participants high in 12-item Cynicism or HBI will have greater cardiovascular reactivity, decreased parasympathetic cardiac activity (Normalised HF HR) and decreased BRS when Perceived Control_{btw} is low. This is a Hostility x Perceived Control_{btw} interaction. This hypothesis is only applicable to the Synwork model.

Synwork model. The following between subject model was tested:

Between Subject

- 1) Covariate
- 2) HBI
- 3) 12-item Cynicism
- 4) Perceived Control_{btw}
- 5) HBI X Cynicism
- 6) HBI x Perceived Control_{btw}
- 7) 12-item Cynicism x Perceived Control_{btw}
- 8) HBI x 12-item Cynicism x Perceived Control_{btw}

The Cynicism x Perceived Control_{btw} interaction was not significant, SBP, $F(1, 50) = 1.56, p = .22, \eta^2 = .01, \text{partial } \eta^2 = .03$, DBP, $F(1, 50) = 2.80, p = .10, \eta^2 = .004, \text{partial } \eta^2 = .007$, MAP, $F(1, 50) = .35, p = .56, \eta^2 = .003, \text{partial } \eta^2 = .006$, HR, $F(1, 50) = .11, p = .74, \eta^2 = .001, \text{partial } \eta^2 = .002$, CO, $F(1, 50) = .61, p = .44, \eta^2 = .008, \text{partial } \eta^2 = .01$, TPR, $F(1, 50) = .21, p = .65, \eta^2 = .003, \text{partial } \eta^2 = .04$, Normalised HF HR, $F(1, 49) = .009, p = .92, \eta^2 = .07 \times 10^{-7}, \text{partial } \eta^2 = .02 \times 10^{-2}$, and BRS, $F(1, 49) = .18, p = .68, \eta^2 = .002, \text{partial } \eta^2 = .003$.

HBI x Perceived Control_{btw} interaction was also not significant: SBP, $F(1, 50) = 3.34, p = .07, \eta^2 = .03, \text{partial } \eta^2 = .06$, DBP, $F(1, 50) = .80, p = .38, \eta^2 = .009, \text{partial } \eta^2 = .01$, MAP, $F(1, 50) = 1.89, p = .17, \eta^2 = .02, \text{partial } \eta^2 = .03$, HR, $F(1, 50) = .45, p = .50, \eta^2 = .004, \text{partial } \eta^2 = .008$, CO, $F(1, 50) = 4.44, p = .04, \eta^2 = .06, \text{partial } \eta^2 = .08$, TPR, $F(1, 50) = 1.14, p = .29, \eta^2 = .02, \text{partial } \eta^2 = .02$, Normalised HF HR, $F(1, 49) = 3.76, p = .06, \eta^2 = .03, \text{partial } \eta^2 = .07$, and BRS, $F(1, 49) = .001, p = .97, \eta^2 = .01 \times 10^{-7}, \text{partial } \eta^2 = .02 \times 10^{-7}$.

Hypothesis 7a was not supported - Hostility-cardiovascular reactivity/variability relationship was not moderated by Perceived Control_{btw}.

7b) Participants scoring high in both 12-item Cynicism and HBI will have greater cardiovascular reactivity, reduced parasympathetic cardiac activity and reduced BRS_{seq}. A 12-item Cynicism x HBI x Perceived Control_{btw} interaction is hypothesised to be significant.

The Cynicism x HBI x Perceived Control_{btw} interaction was not significant: SBP, $F(1, 50) = .52, p = .47, \eta^2 = .004, \text{partial } \eta^2 = .09$, DBP, $F(1, 50) = .004, p = .95, \eta^2 = .05 \times 10^{-7}, \text{partial } \eta^2 = .08 \times 10^{-7}$, MAP, $F(1, 50) = .35, p = .56, \eta^2 = .003, \text{partial } \eta^2 = .006$, HR, $F(1, 50) = 1.03, p = .31, \eta^2 = .008, \text{partial } \eta^2 = .02$, CO, $F(1, 50) = 2.88, p = .10, \eta^2 = .04, \text{partial } \eta^2 = .05$, TPR, $F(1, 50) = 1.30, p = .26, \eta^2 = .02, \text{partial } \eta^2 = .02$, Normalised HF HR, $F(1, 49) = .64, p = .43, \eta^2 = .005, \text{partial } \eta^2 = .01$, and BRS_{seq}, $F(1, 49) = .97, p = .33, \eta^2 = .01, \text{partial } \eta^2 = .02$.

Main effect of Perceived Control_{btw} was not significant: SBP, $F(1, 50) = .01, p = .90, \eta^2 = .01 \times 10^{-2}, \text{partial } \eta^2 = .03 \times 10^{-2}$, DBP, $F(1, 50) = 2.29, p = .14, \eta^2 = .005, \text{partial } \eta^2 = .008$, MAP, $F(1, 50) = .71, p = .40, \eta^2 = .007, \text{partial } \eta^2 = .01$, HR, $F(1, 50) = .03, p = .86, \eta^2 = .02 \times 10^{-2}, \text{partial } \eta^2 = .05 \times 10^{-2}$, CO, $F(1, 50) = .25, p = .62, \eta^2 = .003, \text{partial } \eta^2 = .004$, TPR, $F(1, 50) = .43, p = .52, \eta^2 = .007, \text{partial } \eta^2 = .008$, Normalised HF

HR, $F(1, 49) = .99, p = .32, \eta^2 = .007$, partial $\eta^2 = .02$, and $BRS_{seq}, F(1, 49) = .80, p = .38, \eta^2 = .009$, partial $\eta^2 = .01$.

Hypothesis 8: *Hostility, State Anger and Cardiovascular reactivity/variability.*

8a) Hostility is positively associated with State Anger. Cynicism is positively associated with State Anger (significant Cynicism main effect), HBI is also positively associated with State Anger (significant HBI main effect), and participants scoring high in both Cynicism and HBI also tend to reported higher levels of State Anger (a significant HBI X Cynicism interaction).

Synwork model. The following model was tested using State Anger¹⁷ as the Dependent Variable and State Anger Baseline as a covariate:

Between Subject Model

- 1) State Anger Baseline
- 2) HBI
- 3) Cynicism
- 4) HBI X Cynicism

Within Subject Design

- 1) Task Control
- 3) Task Control X HBI
- 4) Task Control X Cynicism
- 7) Task Control X HBI X Cynicism

¹⁷ State Anger refers to the ratings of the Anger mood items after the corresponding Synwork session. State Anger Baseline refers to the ratings of the Anger mood items after the Baseline.

Main effect of Cynicism was not significant, $F(1, 53) = .04, p = .84, \eta^2 = .001$. HBI main effect was not significant, $F(1, 53) = .08, p = .78, \eta^2 = .002$, and HBI x Cynicism interaction was also not significant, $F(1, 53) = .03, p = .87, \eta^2 = .05 \times 10^{-2}$. There was a borderline significant State Anger Baseline main effect, $F(1, 53) = 6.24, p = .01, \eta^2 = .11$.

Anger Recall model. The between subject model analysed in the Synwork model was used here. Main effect of Cynicism was not significant, $F(1, 53) = .04 \times 10^{-7}, p = 1.00, \eta^2 = .07 \times 10^{-5}$. HBI main effect was not significant, $F(1, 53) = .07, p = .80, \eta^2 = .001$, and HBI x Cynicism interaction was also not significant, $F(1, 53) = .15, p = .71, \eta^2 = .003$. State Anger Baseline main effect was $F(1, 53) = 2.94, p = .09, \eta^2 = .05$.

Hypothesis 7a was not supported - Hostility was not related to higher ratings of State Anger after Synwork and Anger Recall.

8b) There is a significant Task Control main effect when State Anger is the Dependent Variable. Following appraisal theories of emotion, Synwork with less control is associated with higher ratings of State Anger.

The results from the model tested for Hypothesis 7a revealed a significant Task Control main effect for State Anger, $F(1.61, 86.94) = 57.13, p = .04 \times 10^{-9}, \eta^2 = .50$, and State Hedonic tone, $F(1.97, 114.26) = 24.76, p = .01 \times 10^{-7}, \eta^2 = .30$. Table 3.14 shows the means (SD) of UNIWST Hedonic Tone Mood ratings by Task Control condition. Both

Table 3.14 Means (SD) of UNWIST Hedonic Tone Mood Ratings by Task Control*

	Task Control		
	Normal	Variable Pauses	More Variable Pauses
Hedonic Tone			
Hedonic items, cm	3.08 _a (.42)	2.60 _b (.59)	2.68 _b (.58)
Anger items, cm	1.57 _a (.57)	2.35 _b (.84)	2.27 _b (.81)

*Means with different subscripts within rows are significantly different at $p < .01$ using Holm's Sequential Bonferroni Type 1 error correction, **Differences are of borderline significance.

linear and quadratic terms for the State Anger main effect were significant, $F(1, 53) = 55.13, p = .09 \times 10^{-7}, \eta^2 = .49$, $F(1, 53) = 57.84, p = .05 \times 10^{-8}, \eta^2 = .52$. As can be seen from Table 3.14, items from Hedonic Tone differentiated the Normal Task Control from the Variable and More Variable Pauses. Hypothesis 7b was supported, Task Variable and More Variable Pauses was associated with increased State Anger.

8c) Hostility and cardiovascular reactivity/variability is moderated by State Anger. Hypothesis 7c refers to significant State Anger x HBI, State Anger x Cynicism, and State Anger x Cynicism x HBI interactions. Specifically, participants scoring high in both State Anger and HBI, high in State Anger and Cynicism, or high in State Anger, Cynicism and HBI have greater cardiovascular reactivity. For cardiovascular variability, participants scoring high in (i) State Anger and HBI, (ii) State Anger and

Cynicism, or (iii) State Anger, Cynicism and HBI, have greater reduction in parasympathetic cardiac activity (Normalised HF HR) and reduced BRS (BRS_{seq}).

As for Perceived Control, two forms of State Anger can be distinguished - State Anger after each Synwork session and the average of State Anger for each participant. The former was termed as 'State Anger_{wtn}' while the latter 'State Anger_{btw}'.

Synwork model. The following regression model was tested:

Between Subject Model

- 1) Cardiovascular Baseline
- 2) HBI
- 3) Cynicism
- 4) State Anger_{btw}
- 5) HBI X Cynicism
- 6) HBI x State Anger_{btw}
- 7) Cynicism x State Anger_{btw}
- 8) HBI x Cynicism x State Anger_{btw}

Within Subject Model

- 1) State Anger_{wtn}
- 2) State Anger_{wtn} X HBI
- 3) State Anger_{wtn} X Cynicism
- 4) State Anger_{wtn} X Cynicism X HBI

State Anger_{btw} x Cynicism interaction was not significant: SBP, $F(1, 50) = 1.74, p = .19, \eta^2 = .01, \text{partial } \eta^2 = .03$, DBP, $F(1, 50) = 1.61, p = .21, \eta^2 = .02, \text{partial } \eta^2 = .03$, MAP, $F(1, 50) = 1.27, p = .26, \eta^2 = .01, \text{partial } \eta^2 = .02$, HR, $F(1, 50) = .72, p = .40, \eta^2 = .006, \text{partial } \eta^2 = .01$, CO, $F(1, 50) = .11, p = .74, \eta^2 = .002, \text{partial } \eta^2 = .002$, TPR, $F(1, 50) =$

1.70, $p = .20$, $\eta^2 = .02$, partial $\eta^2 = .03$, Normalised HF HR, $F(1, 49) = 3.70$, $p = .06$, $\eta^2 = .03$, partial $\eta^2 = .07$, and BRS_{seq} , $F(1, 49) = 1.20$, $p = .28$, $\eta^2 = .01$, partial $\eta^2 = .02$.

State Anger_{btw} x HBI interaction was also not significant: SBP, $F(1, 50) = 3.01$, $p = .09$, $\eta^2 = .03$, partial $\eta^2 = .05$, DBP, $F(1, 50) = 4.34$, $p = .04$, $\eta^2 = .05$, partial $\eta^2 = .07$, MAP, $F(1, 50) = 3.91$, $p = .05$, $\eta^2 = .04$, partial $\eta^2 = .07$, HR, $F(1, 50) = .45$, $p = .51$, $\eta^2 = .04 \times 10^{-2}$, partial $\eta^2 = .08 \times 10^{-2}$, CO, $F(1, 50) = 2.08$, $p = .16$, $\eta^2 = .03$, partial $\eta^2 = .04$, TPR, $F(1, 50) = 4.06$, $p = .05$, $\eta^2 = .06$, partial $\eta^2 = .07$, Normalised HF HR, $F(1, 49) = .43$, $p = .51$, $\eta^2 = .003$, partial $\eta^2 = .008$, and BRS_{seq} , $F(1, 49) = .02$, $p = .88$, $\eta^2 = .03 \times 10^{-2}$, partial $\eta^2 = .04 \times 10^{-2}$.

State Anger_{btw} main effect was also not significant: SBP, $F(1, 50) = .12$, $p = .73$, $\eta^2 = .001$, partial $\eta^2 = .002$, DBP, $F(1, 50) = 1.64$, $p = .21$, $\eta^2 = .003$, partial $\eta^2 = .005$, MAP, $F(1, 50) = .26$, $p = .61$, $\eta^2 = .002$, partial $\eta^2 = .004$, HR, $F(1, 50) = .25$, $p = .62$, $\eta^2 = .002$, partial $\eta^2 = .004$, CO, $F(1, 50) = 1.06$, $p = .31$, $\eta^2 = .02$, partial $\eta^2 = .02$, TPR, $F(1, 50) = 1.93$, $p = .17$, $\eta^2 = .03$, partial $\eta^2 = .03$, Normalised HF HR, $F(1, 49) = .60$, $p = .44$, $\eta^2 = .004$, partial $\eta^2 = .01$, and BRS_{seq} , $F(1, 49) = .57$, $p = .45$, $\eta^2 = .006$, partial $\eta^2 = .01$.

For cardiovascular reactivity, there were no significant State Anger_{wtw} x HBI interactions. For SBP, $F(1, 108.19) = .41$, $p = .52$, $\eta^2 = .004$, DBP, $F(1, 98.95) = 1.42$, $p = .24$, $\eta^2 = .01$, MAP, $F(1, 98.61) = 2.11$, $p = .15$, $\eta^2 = .02$, HR, $F(1, 98.95) = .46$, $p = .50$, $\eta^2 = .004$, CO, $F(1,$

111.95) = .02, $p = .89$, $\eta^2 = .02 \times 10^{-2}$, and TPR, $F(1, 94.51) = .61$, $p = .44$, $\eta^2 = .006$. State Anger_{wtn} x Cynicism interactions were also not significant, SBP, $F(1, 108.19) = .13$, $p = .72$, $\eta^2 = .001$, DBP, $F(1, 98.95) = .16$, $p = .69$, $\eta^2 = .002$, MAP, $F(1, 98.61) = .14$, $p = .71$, $\eta^2 = .001$, HR, $F(1, 98.95) = .42$, $p = .52$, $\eta^2 = .004$, CO, $F(1, 111.95) = .67$, $p = .42$, $\eta^2 = .006$, and TPR, $F(1, 94.51) = 1.63$, $p = .21$, $\eta^2 = .02$. State Anger_{wtn} x HBI x Cynicism interaction was also not significant; SBP, $F(1, 108.19) = .16$, $p = .69$, $\eta^2 = .001$, DBP, $F(1, 98.95) = 1.10$, $p = .30$, $\eta^2 = .01$, MAP, $F(1, 98.61) = .74$, $p = .39$, $\eta^2 = .007$, HR, $F(1, 98.95) = 1.17$, $p = .28$, $\eta^2 = .01$, CO, $F(1, 111.95) = .72$, $p = .40$, $\eta^2 = .006$, and TPR, $F(1, 94.51) = 1.96$, $p = .16$, $\eta^2 = .02$.

For cardiovascular variability, State Anger_{wtn} x HBI interactions were not significant either. For Normalised HF HR, $F(1, 104.86) = 1.07$, $p = .30$, $\eta^2 = .01$, and BRS_{seq}, $F(1, 97.97) = .64$, $p = .43$, $\eta^2 = .006$. State Anger_{wtn} x Cynicism interactions were also not significant, Normalised HF HR, $F(1, 104.86) = .01$, $p = .11$, $\eta^2 = .02$, and BRS_{seq}, $F(1, 97.97) = .32$, $p = .57$, $\eta^2 = .003$. State Anger_{wtn} x HBI x Cynicism interactions were also not significant, Normalised HF HR, $F(1, 104.86) = 2.63$, $p = .11$, $\eta^2 = .02$, and BRS_{seq}, $F(1, 97.97) = .76$, $p = .39$, $\eta^2 = .008$.

For both cardiovascular reactivity and variability, State Anger_{wtn} main effects were not significant, p 's .04 to .89.

Anger Recall model. As the Anger Recall model was a between subject analysis, reactivity scores for State Anger were calculated. State

Anger Baseline was subtracted from State Anger ratings after Anger Recall. This new variable is called 'State Anger Reactivity' and the following regression model was tested:

Between Subject Model

- 1) Cardiovascular Baseline
- 2) HBI
- 3) Cynicism
- 4) State Anger Reactivity
- [4a) Baseline X HBI - for DBP and MAP only]
- 5) HBI X Cynicism
- 6) State Anger Reactivity X HBI
- 7) State Anger Reactivity X Cynicism
- 8) State Anger Reactivity X HBI X Cynicism

Hypothesis 8c postulated that State Anger Reactivity x HBI, State Anger Reactivity x Cynicism, and State Anger Reactivity x HBI x Cynicism interactions would be significant.

For cardiovascular reactivity, State Anger Reactivity x HBI interaction was not significant. SBP, $F(1, 49) = .002$, $p = .96$, $\eta^2 = .02 \times 10^{-3}$, partial $\eta^2 = .04 \times 10^{-3}$, DBP, $F(1, 49) = .12$, $p = .73$, $\eta^2 = .001$, partial $\eta^2 = .002$, MAP, $F(1, 48) = .03$, $p = .85$, $\eta^2 = .03 \times 10^{-2}$, partial $\eta^2 = .07 \times 10^{-2}$, HR, $F(1, 49) = .004$, $p = .95$, $\eta^2 = .03 \times 10^{-3}$, partial $\eta^2 = .08 \times 10^{-3}$, CO, $F(1, 49) = 1.56$, $p = .22$, $\eta^2 = .02$, partial $\eta^2 = .03$, and TPR, $F(1, 49) = 3.48$, $p = .07$, $\eta^2 = .05$, partial $\eta^2 = .06$.

State Anger Reactivity x Cynicism interactions were also not significant, SBP, $F(1, 49) = .21$, $p = .65$, $\eta^2 = .002$, partial $\eta^2 = .004$, DBP, $F(1, 48) = .03$, $p = .86$, $\eta^2 = .03 \times 10^{-2}$, partial $\eta^2 = .06 \times 10^{-2}$, MAP, $F(1, 48) = .08$, $p = .79$, $\eta^2 = .06 \times 10^{-2}$, partial $\eta^2 = .001$, and, HR, $F(1, 49) = 1.70$, $p = .20$, $\eta^2 = .01$, partial $\eta^2 = .03$. For CO and TPR, State Anger Reactivity x

Cynicism interaction was of borderline significance, CO, $F(1, 49) = 5.66, p = .02, \eta^2 = .09, \text{partial } \eta^2 = .10$, and TPR, $F(1, 49) = 4.58, p = .04, \eta^2 = .07, \text{partial } \eta^2 = .07$. Figure 3.8 & 3.9 show the pattern of interaction for CO and TPR using the method proposed by Aiken and West (1991). State Anger Reactivity x HBI x Cynicism interactions were not significant, SBP, $F(1, 49) = .08, p = .77, \eta^2 = .08 \times 10^{-2}, \text{partial } \eta^2 = .002$, DBP, $F(1, 48) = .40, p = .53, \eta^2 = .004, \text{partial } \eta^2 = .008$, MAP, $F(1, 48) = .18, p = .67, \eta^2 = .002, \text{partial } \eta^2 = .003$, HR, $F(1, 49) = .02, p = .88, \eta^2 = .02 \times 10^{-2}, \text{partial } \eta^2 = .04 \times 10^{-2}$, CO, $F(1, 49) = .36, p = .55, \eta^2 = .006, \text{partial } \eta^2 = .006$, and TPR, $F(1, 49) = .06, p = .81, \eta^2 = .001, \text{partial } \eta^2 = .001$.

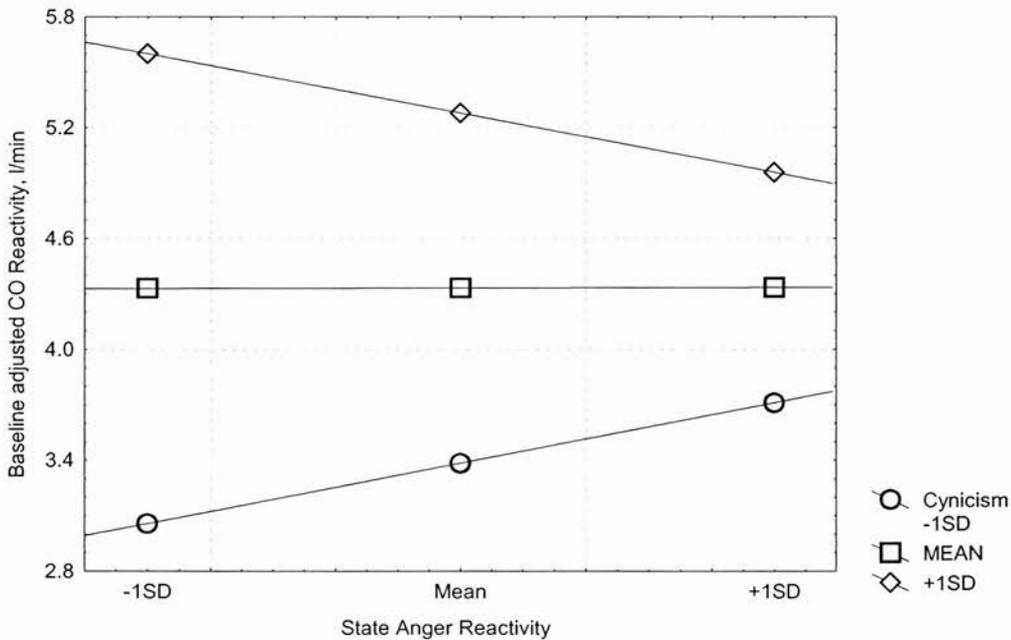


Figure 3.8 State Anger Reactivity x Cynicism interaction for CO

For cardiovascular variability, State Anger Reactivity x HBI interactions were not significant either. For Normalised HF HR, $F(1, 49) =$

.35, $p = .56$, $\eta^2 = .004$, partial $\eta^2 = .006$, and BRS_{seq} , $F(1, 49) = 3.42$, $p = .07$, $\eta^2 = .04$, partial $\eta^2 = .06$. State Anger Reactivity x Cynicism interactions were also not significant, Normalised HF HR, $F(1, 49) = 2.16$, $p = .15$, $\eta^2 = .02$, partial $\eta^2 = .04$, and BRS_{seq} , $F(1, 49) = 2.20$, $p = .14$, $\eta^2 = .02$, partial $\eta^2 = .04$. State Anger x HBI x Cynicism interactions were also not significant, Normalised HF HR, $F(1, 49) = .33$, $p = .57$, $\eta^2 = .004$, partial $\eta^2 = .006$, and BRS_{seq} , $F(1, 49) = .13$, $p = .72$, $\eta^2 = .001$, partial $\eta^2 = .002$.

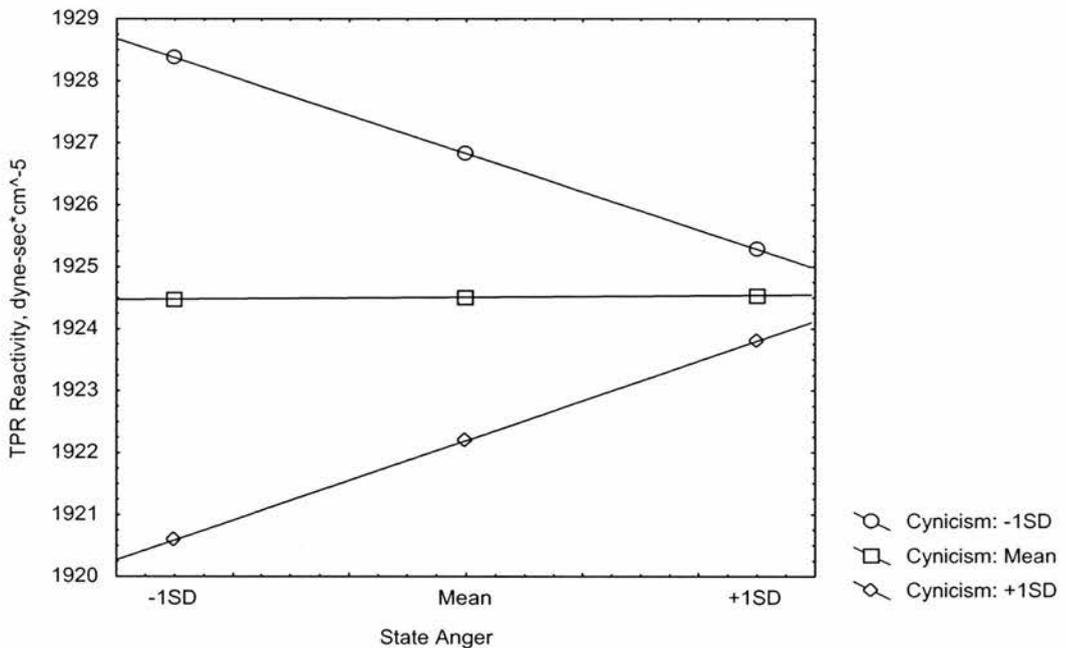


Figure 3.9 State Anger Reactivity x Cynicism interaction for TPR

State Anger main effects were not significant for cardiovascular reactivity and variability, p 's .07 to .72.

The results from the Synwork and Anger Recall model showed that Hostility-Cardiovascular reactivity/variability relationship was not moderated by State Anger. There was a trend for State Anger as a moderator for Cynicism with CO and TPR reactivity for Anger Recall. In

terms of these two hemodynamic processes of blood pressure, the greatest difference was found at low ratings of State Anger during Anger Recall. Specifically, at low ratings of State Anger, participants with high Cynicism had active coping physiological response (increased CO and decreased TPR) while participants with low Cynicism had passive coping physiological response (decreased CO and TPR). This pattern of reactivity between people with high and low Cynicism was attenuated with increasing State Anger. The results for CO and TPR were the reverse of predictions for hypothesis 8c, which predicted the greatest cardiovascular reactivity for people with high Cynicism and high State Anger.

Synwork Scores

Perceived Control and Synwork Task scores were lowest in the Variable Pauses condition and average for the More Variable Pauses Task Control condition. This raised concerns about the validity of the More Variable Pauses Task Control condition. Perceived Control and Task Control were analysed and found that Task Control offered a better model fit than Perceived Control. This meant that defining Task Control via response-outcome contingencies offered a better explanation of the data than Perceived Control. This section extends this analysis to Task Scores. As significant Task Control main effect was found for CO, this dependent variable was analysed here. The aim of this analysis was to determine whether Task Scores offer a better model fit than Task Control.

If so, this gives credence to the concern about the validity of the More Variable Pauses condition.

Like Perceived Control and State Anger, a between subject and within subject version of Task Scores were created ($Score_{btw}$, $Score_{wtn}$).

The following regression model was tested:

Between Subject Model

- 1) CO Baseline
- 2) $Score_{btw}$
- 3) HBI
- 4) Cynicism
- 5) HBI x Cynicism

Within Subject Model

- 1) $Score_{wtn}$

$Score_{btw}$ main effect was not significant, $F(1, 54) = 1.41, p = .24, \eta^2 = .02$, partial $\eta^2 = .03$. $Score_{wtn}$ main effect was significant, $F(1, 112.93) = 15.02, p = .02 \times 10^{-2}, \eta^2 = .12$, partial $\eta^2 = .12$. The effect size for $Score_{wtn}$ was similar to Perceived Control_{wtn} ($\eta^2 = .13$). Compared to $Score_{wtn}$ and Perceived Control_{wtn}, Task Control provided a better model fit of the data ($\eta^2 = .29$). Therefore, the response-outcome contingency was a better description of CO reactivity patterns than Task Scores or Perceived Control. The patterns of Task Scores and Perceived Control by Task Control condition did not threaten the validity of the More Variable Pauses Control condition.

Study 2 Discussion

There is an increasing number of studies that have reported null or negative findings between hostility and cardiovascular reactivity (Burns & Katkin, 1993; Carroll et al., 1997; Felsten, 1995; Felsten, 1996; Fichera & Andreassi, 1998; Spoth et al., 1992; Weidner et al., 1989). Two main possible explanations for the emergence of conflicting results were discussed in Chapter 1. Firstly, it could be due to measurement error in Hostility assessment. Specifically, this dissertation addresses the measurement error in the Cook-Medley Hostility scale since this is the most frequently used form of hostility assessment. Chapter 2 addresses the internal consistency of the Cook-Medley Hostility scale and found that there was evidence for a 12-item Cynicism subscale. Secondly, some researchers have also showed that the Hostility-Reactivity relationship is moderated by other variables. Therefore, it could be that hostility is positively related to cardiovascular reactivity only under certain conditions. This chapter investigates the role of state anger and control as moderators.

Methodology Issues

Most studies have used harassment to induce state anger (Everson et al., 1995; Miller et al., 1998; Suarez & Williams, 1989) and interpersonal control during social conflict (Smith & Brown, 1991) to demonstrate the role of state anger and control as moderators of the hostility-reactivity relationship. Such methodology does not distinguish

interpersonal-induced anger from non interpersonal-induced anger. There is evidence to suggest that hostility functions within an interpersonal context (Brondolo et al., 2003; Guyll & Contrada, 1998; Watkins & Eisler, 1988).

This chapter utilises two forms of experiment tasks to induce anger (i.e., Synwork and Anger Recall). The controllability of a computer task, Synwork, was manipulated. According to appraisal theories of emotion, control or goal obstruction leads to increase feelings of anger (Berkowitz, 2000). This was supported by the results reported in this chapter; though Synwork with Normal Control elicited significant higher ratings of State Anger from Baseline, the ratings for State Anger given after Synwork Variable and More Variable Pauses were significantly higher than Synwork Normal Control. This shows that goal obstruction can lead to increased levels of State Anger. Manipulating Task Control for Synwork was to elicit non-interpersonal State Anger and this was shown to be successful. Another task used in this chapter, Anger Recall, was to elicit State Anger from an interpersonal origin. The mood ratings showed that though Anger Recall elicited significant increases in Anger from Baseline. Synwork and Anger Recall also elicited comparative blood pressure compared to another study that used a white male sample of similar age (Everson et al., 1995). Everson et al. sampled 48 males (mean age = 24.9) and used a mental arithmetic task with and without harassment. Heart rate reactivity for the Mental Arithmetic with Harassment was 15.5bpm, SBP reactivity was 17.5mm Hg, DBP reactivity was 11.5mm Hg. In this study, Anger Recall elicited a Heart Rate reactivity of 6.7bpm,

SBP reactivity of 15.15mm Hg, and DBP reactivity of 12.85mm Hg. The blood pressure reactivity values obtained in this study were close to those elicited by Everson et al's (1995) task with harassment. There were differences in Heart Rate reactivity is probably due to the fact that Everson et al.'s sample probably consisted of participants with low basal heart rate (64.5bpm)¹⁸.

In addition to blood pressure and heart rate, the underlying hemodynamic processes (CO and TPR) were also derived using the Beatscope software. Both CO and TPR are usually divided by Body Surface Area to control for such differences (Sherwood et al., 1990a; Steptoe et al., 2003). The results of this chapter adds onto a growing body of published research that have reported null effects for BSA or BMI as a covariate for cardiovascular reactivity (Al'Absi et al., 1995; Kamarck et al., 2000; Why et al., 2003). The reason for how BSA or BMI affects blood pressure reactivity is not entirely clear. An increase in bodyfat increases the number of blood vessels, which in turn raises resting blood pressure (Marieb, 1998). Hence it could be possible that bodyfat (rather than proxy measures of it in terms of BMI and BSA) is closely associated with TPR reactivity.

Three indices of cardiovascular variability were also processed - BRS_{spectral} , BRS_{seq} , and Normalised High Frequency Heart Rate. The analysis of BRS_{spectral} presented some problems as the mean coherence fell below .50 during the various experimental task. The number of

¹⁸ Basal blood pressure is not compared because the Finapres is not accurate in basal blood pressure assessment (Imholz et al., 1998).

cardiac cycles included for the assessment of BRS_{seq} during Synwork also decreased. This could imply that the reliability of these measures of BRS could be affected. However, the values obtained for BRS_{seq} replicated the phenylephrine method (Watkins et al., 1996). BRS analysed via spectral analysis has often been reported for resting baseline with spontaneous or paced breathing (Fauvel et al., 2000; Virtanen et al., 2003) whereas BRS assessed via the Sequence Method has been used to studies involving psychological stress (Stephoe, Evans, & Fieldman, 1997; Steptoe & Sawada, 1989). Attempt at measuring $BRS_{spectral}$ during psychological stress has yielded null findings (AlKubati, Fiser, & Siegelova, 1997). The pattern of findings from published studies suggest that BRS assessed via spectral analysis may be less reliable during psychological stress (i.e., coherence $<.50$) and hence it is seldom done or reported. In this chapter, the number of cardiac cycles included for the assessment of BRS for the Anger Recall task was similar to Baseline but the coherence value fell below $.50$ for BRS assessed via spectral analysis during Anger Recall. BRS assessed via the Sequence Method is less affected in terms of reliability during psychological stress than BRS assessed via spectral analysis.

The ANCOVA analysis also showed that Normalised HF Respiration was not a significant predictor of Normalised HF HR changes in the two experimental task (i.e., Anger Recall and Synwork). The null findings for Normalised HF Respiration is in no way contradictory to past studies that have shown the importance of controlling for changes in respiration when analysing HF Heart Rate (Althaus et al., 1998;

Grossman et al., 1991; Novak et al., 1993). Normalised HF Respiration was found to increase significantly from Baseline only during Synwork Normal Task Control (Table 3.7). Though Normalised HF respiration changed significantly from Baseline during Synwork Normal Control, there were no significant changes in Normalised HF Heart Rate. Therefore, there were no significant effects in the Dependent Variable (Normalised HF Heart Rate) to 'correct' for by respiration. Secondly, changes in Normalised HF Heart Rate for Anger Recall was significant from Baseline but this was not followed by any significant change in respiration. Therefore, in this study, the task effects of Anger Recall and Synwork on Normalised HF Heart Rate are not confounded with the changes in Normalised HF Respiration. In the event when it is, then it would be necessary to statistically control for respiratory changes.

The only significant change in cardiovascular variability dependent variables was for Normalised HF Heart Rate for Anger Recall. This replicates previous work (Jain et al., 2001; McCraty, Atkinson, Tiller, Rein, & Watkins, 1995) that reported anger is associated with reduced HF Heart Rate and correspondingly increases LF Heart Rate. However, both Jain et al. and McCraty et al. did not measure Baroreflex Sensitivity and respiration. Respiratory Sinus Arrhythmia occurs in the high frequency range and is also vagally-mediated. A downward shift of respiration frequency into the low frequency range (i.e., increase respiration rate) can also induce a decrease in HF HR. The results of this study suggest that the significant decrease of parasympathetic cardiac activity, increase in sympathetic cardiac activity associated with the Anger Recall task is

not related to respiration. Secondly, this study also extends the work of Jain et al. and McCraty et al. by assessing Baroreflex Sensitivity. It was found that Baroreflex Sensitivity was significantly reduced during Anger Recall as well. This can explain for the greater blood pressure variability (indexed by greater SD) during Anger Recall compared to the Baseline (table 3.9). As reviewed in Chapter 1, reduced BRS has been found to be associated with cardiovascular disease (Gianaros et al., 2002). Anger Recall used in this study and Jain et al.'s involved speech. It is unlikely that speech could have contributed to this effect. McCraty et al.'s study devised a form of Anger Recall without speech that produced identical results with HF HR.

In this chapter, reactivity is operationalised in an ANCOVA analysis. There have been different methods to operationalise reactivity. In this chapter, a single baseline was used instead of an average of all rest periods and baselines. In addition, reactivity was also analysed in an ANCOVA instead of calculating reactivity scores. Other methods include using the residuals as reactivity. These various methods of operationalising reactivity are discussed next. The aim is to show that no single method is without problems and the approach taken in this chapter represents some attempt to balance the pros and cons of the various methods.

A Baseline can be conceptualised as an average of all rest periods and baselines to derive a more reliable estimate of the resting baseline. However, there are two assumptions to be met before an 'averaged' Baseline is taken. Firstly, Baselines should not be affected by the

treatment. If it is, then the treatment's effect size will be attenuated since the baseline is also removing variance associated with the treatment (Fredrickson et al., 2000). Secondly, all the rest periods constituting the Baseline should not differ significant from one another since baselines indicate tonic levels of cardiovascular function. The analysis of the cardiovascular functions during the baselines and rest periods of this study showed that there was significant upward bias in blood pressure over time. Hence, the average of all baselines cannot be assumed to reflect tonic levels of cardiovascular functions. This upward drift of blood pressure due to increased vascular resistance has been reported (Ristuccia et al., 1997). Ristuccia et al. showed that removing the finger cuff from the participants' fingers and initiating a brief hand exercise reduced this upward drift. However, though much care were taken by the researchers to replace the cuff back in its original position, this method of removing and attaching the finger cuff introduces the problem of cuff placement accuracy during the experimental procedure. This upward drift also presents some problems in the interpretation of some of the Task Control main effects for the Synwork model. Task Control for Synwork replicated previous findings with regards to active and passive physiological response. The order of Task Control was not counterbalanced and since there is an upward drift of vascular resistance and Task Control was from Normal to More Variable Pauses - the effects of increased vascular resistance with decreasing control could be contributed by the upward drift of vascular resistance in the Finapres. Hence, there is a need to counterbalance the order of Task Control in the

next study (chapter 4). This problem affects the Anger Recall task much less because it is counterbalanced with Synwork. For half of the participants, Anger Recall occurred right after Baseline and the results showed that Anger Recall elicited the greatest vascular response while CO remained similar to Baseline. Since half of the participants had the Anger Recall after Baseline, the upward bias of vascular resistance affected these participants less.

A vanilla baseline can also be used to calculate reactivity (Jennings, Kamack, Stewart, Eddy, & Johnson, 1992). Jennings et al. used a colour detection task as a baseline. They showed that using the cardiovascular reactivity during this task produced more reliable cardiovascular values than a resting baseline. Since this task is minimally demanding on the participants, the researchers proposed the use of this task as a vanilla baseline. However, though increasing reliability of any measurement is a good thing, the reliability of a Baseline does not necessarily equate to reliability of cardiovascular reactivity scores (or sometimes called gain scores). The reliability of reactivity scores (the difference between baseline values and values during psychological stress) is dependent on the reliabilities of both the baseline and the values obtained during the various treatments as well as the correlation between the two (Thorndike & Hagen, 1977). This point will be discussed later. So increasing the reliability of the Baseline while keeping the reliability of the value obtained during stress constant does little to change the reliability of reactivity scores (Gerin, Pieper, & Pickering, 1993). In addition, reliability is not a defining characteristic of a Baseline.

Researchers would wish to have reliable estimates of cardiovascular reactivity during stress as well. One of the common understanding of what a baseline is that is it a resting baseline that is relatively treatment-free. Therefore, even though participants' cardiovascular reaction to a colour detection task is reliable, it introduces ambiguity into the concept of a 'Baseline'. Since a vanilla baseline already introduces a minimally stressful task, can it still be used as a 'stress-free' baseline?

Reactivity can also be operationalised in a few ways. One of the ways is to calculate gain scores via subtracting the value obtained during the baseline from the value during psychological stress. It is generally reported that gain scores have lower reliability than the raw scores from which it is computed (Cronbach & Furby, 1970). Some researchers have found that as the correlation between the two scores is more similar to the average reliability of each of the score, gain scores have a reliability of '0' (Thorndike & Hagen, 1977). For example, if baseline and stress value of a dependent variable each has a reliability of .80, and they are both correlated at .80, then the reactivity score will have a reliability of '0'. If in this hypothetical case, the correlation between the two scores is .60 (as found in this and other studies), then the reliability of gain score is .50. Though in some circumstances, gain scores can achieve a reliability of .80 (Llabre, Spitzer, Saab, & Ironson, 1991), a meta-analysis shows that this seldom occurs. Swain and Suls did a meta-analysis of over 20 studies and found that the average test-retest reliability of SBP reactivity was .55, for DBP reactivity, it was .41 while for HR reactivity it was .35 (Swain & Suls, 1996). Therefore, gain scores are generally less reliable

than the raw scores. But gain scores are conceptually appealing method of operationalising reactivity. Hence reactivity is often operationalised as gain scores. Another method of operationalising reactivity is to analyse residuals. By using regression, values obtained during stress can be regressed onto baseline values and the residuals derived. Thus the residuals represent “baseline-free” reactivity (Manuck, Kasprovicz, Monroe, Larkin, & Kaplan, 1989). Such residuals have been found to have similar reliability compared to gain scores (Llabre et al., 1991). However, it has been found that the analysis of residuals can produce a test statistic that is not normally distributed and an analysis using residuals should be avoided (Maxwell, Delaney, & Manheimer, 1985). Reactivity can also be operationalised as another level of a repeated measure. For example, in this chapter, the Anger Recall model can be conceptualised of as a repeated measure with 2 levels - Baseline and Anger Recall. Therefore, the hypothesis of a Hostility main effect would now be conceptualised as Hostility by Task interaction since hostility is predicting values during Anger Recall and not Baseline. Huck and McLean found that this method of analysing reactivity obtains the same F ratio as gain scores (Huck & McLean, 1975). Hence the low reliability that affects gain scores is also applicable when reactivity is operationalised as another level of a repeated measure. Furthermore, in certain instances, this may not be a probable solution. For this study, Perceived Control was one of the independent variables, but there is no baseline for this variable. There will be no corresponding Perceived Control rating if the

cardiovascular baseline is included as another level of a repeated measure.

Finally, reactivity can also be operationalised as a covariate as in ANCOVA as done in this chapter. Some researchers have compared reactivity operationalised as gain scores, as another level of a repeated measure and as a covariate in ANCOVA. The general conclusion is that the ANCOVA method of operationalising reactivity tend to have better power (Huck & McLean, 1975; Jennings, 1988; Maris, 1998; Maxwell & Delaney, 2004)¹⁹. However, too many covariates may make the ANCOVA analysis unwieldy. This is because for every covariate the assumption for the homogeneity of regression coefficients has to be ascertained. If there any significant interactions found between the independent variables and the covariate then the covariate cannot 'adjust' for pretest (baseline) differences adequately. This would entail the inclusion of the interaction term in order to obtained an appropriate error term. If there are several covariates or several independent variables, there could potentially be more than one significant interaction, and this could make the whole statistical model large and unwieldy. In a regression model, this can exacerbate the problem of collinearity and produce unreliable results with large standard errors. Therefore, though ANCOVA method of operationalising reactivity is taken here, it is has assumptions that may not be met at times. In those instances, gains scores or reactivity as a repeated measure may be the next alternative.

¹⁹ (cf., Jamieson, 2004)

The hypotheses tested in Study 2 follow three major themes. They are:

- 1) Hostility and cardiovascular reactivity/variability.
- 2) Hostility and cardiovascular reactivity/variability moderated by Control
- 3) Hostility and Cardiovascular Reactivity/Variability moderated by State Anger

The discussion of the results of Study 2 will follow these themes in the order listed.

Hostility and Cardiovascular Reactivity/Variability

In this study, Hostility was measured by the two ways - the Structured Interview and 12-item Cynicism scale from the Cook-Medley Hostility scale. The Structured Interview represents the gold standard of hostility assessment while the Cook-Medley questionnaire is the most common and convenient form of hostility assessment. While the Structured Interview taps the behavioural aspect of hostility (verbal aggression), the 12-item Cynicism scale taps the cognitive aspect (cynical mistrust). The results showed that regardless of its method of assessment, Cynicism was not related to cardiovascular reactivity/variability. In addition, using both the Structured Interview and 12-item Cynicism scale (HBI X Cynicism interaction) did not predict reactivity/variability. There was a lack of significant Hostility main effect for both the Anger Recall and Synwork tasks. The null findings for Hostility in predicting cardiovascular reactivity and variability in this study

joins with the other studies cited earlier that have reported similar non significant results. Suls and Wan's meta-analysis, using 28 studies and based on 16 effect sizes, reported that the mean effect size estimate for the Cook-Medley Hostility scale for SBP, DBP and HR all had a non significant average r^2 of less than .01 (Suls & Wan, 1993). The effect size for the Anger Recall for SBP, DBP and HR reported in this chapter were all less than .01. The average effect size for the Potential for Hostility Style reported by Suls and Wan was $r^2 < .03$. The effect sizes obtained in this study were on average less than .05. The results from this chapter are congruent with Sul and Wan's finding that the effect size for both Hostility measures tend to be less than .05.

The pattern of null results (and some negative trends) for these two Hostility assessments suggests that it would be fruitful to investigate the influence of Repressed Cynicism in the 12-item Cynicism scale. Improving the internal consistency and discriminant construct validity by using the 12-item version of the Cook-Medley Hostility scale does not rectify the problem of false negatives. In the next chapter, the developing and testing of a implicit Cynicism assessment (GNAT Cynicism) is used in conjunction with the 12-item Cynicism scale to reduce the influence of false negatives in the sample. Secondly, some studies that have reported significant results for the Cynicism-Reactivity effect have selected groups with extreme scores on this scale (Smith et al., 2000; Suarez et al., 1998; Suarez & Williams, 1989). This suggests that the Cynicism-Reactivity relationship could be cubic. The effect is only present at extreme score values while Cynicism scores near the mean are not related to reactivity.

The next chapter explores such nonlinear relationship between Cynicism and reactivity.

Another explanation discussed in Chapter 1 is that non significant results for Hostility may be due to the presence of moderators. Two moderators were investigated in this dissertation - Control and State Anger.

Hostility and cardiovascular reactivity/variability moderated by Control

Smith and Brown reported that Cynicism was related to cardiovascular reactivity when interpersonal control was low (Smith & Brown, 1991). Hostility is a component of Type A Behaviour Pattern. Earlier investigations on Type A Behaviour focused on the importance of control for Type A Behaviour (Glass, 1977; Miller et al., 1985; Powell, 1992). Hence it is also hypothesised that Hostility might also be moderated by control for cardiovascular reactivity and variability.

In this study non interpersonal control was manipulated via adjusting the efficiency of the computer mouse. The efficiency of the mouse was manipulated so participants can actively cope with the task to gain scores. In other words, the task was not made impossible to gain scores. As was discussed in Chapter 1, it is found that a controllable task (active coping task) elicits increased CO and decreased TPR reactivity while an uncontrollable task (passive coping task) elicits decreased CO and TPR reactivity (Hurwitz et al., 1993; Sherwood et al., 1990b; Tomaka et al., 1993; Weinstein et al., 2002). In this dissertation, three levels of task control were defined by the duration of the random pauses

generated by the Faulty Mouse device. In the Normal Control condition, this device was switched off so the mouse could function normally. In the Variable Pauses condition, the duration of the random pauses were from 25 to 300ms and during the More Variable Pauses condition, the duration of the random pauses were from 25 to 500ms. The efficiency of the computer mouse represents actual task control.

Currently, the presence or absence of coping behaviours defines active and passive coping tasks. This definition of active and passive coping task is inadequate as it does not cover other situations where coping behaviours are present but task control is low (i.e., low response-outcome contingency). In this chapter, it is shown that manipulating the response-outcome contingency, the corresponding active and passive coping physiological response can be elicited. The results of this chapter provide evidence that response-outcome contingency, rather than presence/absence of coping behaviours, can define Task Control.

Chapter 4 rectifies some of the problems encountered in this chapter with regards to Task Control. Firstly, the downward drift of cardiac output and upward drift of total peripheral resistance during the rest periods and recovery baseline might also have contributed to the active and passive physiological response since Task Control was fixed from Normal to More Variable Pauses. Therefore, the experimental study in the next chapter counterbalances the order of the three versions of Synwork. Secondly, participants' ratings of their perceptions of task control after each session of Synwork showed that the Task Control manipulation significantly decreased their perceptions of control.

However, perceived control and Task scores were slightly but significantly higher in the More Variable Pauses condition compared to the Variable Pauses condition. The same pattern was found for Synwork scores. This lead to doubts as to whether More Variable Pauses condition has the lowest control. The pattern for Perceived Control and Synwork scores suggest that More Variable Pauses Task Control condition is actually *more* controllable than the Variable Pauses Task Control condition. This is unlikely to be true because the hemodynamic processes during More Variable Pauses did not reverse significantly from Variable Pauses - there was no reversal from a passive coping physiological response during the Variable Pauses to a more active coping physiological response during the More Variable Pauses. The equivalent level of cardiac output and total peripheral resistance reactivity between Variable Pauses and More Variable Pauses suggest that both conditions are equally uncontrollable. In addition, using Perceived Control and Synwork Scores as alternatives in measuring active/passive coping physiological response for cardiac output produced a poorer model fit. This shows that response-outcome contingency (represented by Task Control) predicts the active/passive physiological coping responses better than Perceived Control and Synwork Task Scores. However, there was not a dose-dependent effect between Task Control and active/passive coping physiological response. Passive physiological response during the Variable Pauses condition was not between Normal and More Variable Pauses. This could be because of habituation to the loss of control since Task Control proceeded from

Normal to More Variable Pauses. Counterbalancing would control for habituation effects.

Despite discrepancies between actual task control (defined by the duration of the random pauses) and perceived control, perceived control predicted the same variance as actual control. Actual control predicted about 60% more variance than perceived control. This is congruent with the view that actual and perceived are often congruent even though minor discrepancies may exist (Wallston, 2001). This result also has implications for studies that may have to use perceived control as a proxy for actual control. This can occur in naturalistic studies where participants fill in diaries to give their ratings of perceived control. In such situations, it is difficult to have an assessment of actual situational control the participants have. The results from this study show that if perceived control is used as a proxy for actual control, 60% less variance is predicted for CO reactivity. Thus, statistical power has to be increased (e.g., more measurements) in order to detect the significant effects when perceived control instead of actual control is assessed.

There were no significant interactions between Hostility and control in predicting cardiovascular stress reactivity or variability. Individuals scoring high in Hostility did not have greater cardiovascular reactivity or variability when the Synwork got less controllable. Chapter 4 investigates the possibility that the presence of such null findings could be due to the presence of false positives and false negatives when the 12-item Cynicism scale is used to assess Hostility. This point will be discussed later in this chapter.

Self Reported Anger and Cardiovascular Reactivity

Researchers have reported that harassment increased self-reported irritation and anger among high hostile participants more than low hostile participants (Everson et al., 1995; Suarez & Williams, 1989). This increased feelings of anger and irritation among high hostile predicted greater cardiovascular reactivity during the harassment task (cf., Diamond et al., 1984; Felsten, 1995; Felsten, 1996). The use of harassment to induce anger confounds interpersonal conflict with anger inducement. Smith and Brown showed that interpersonal conflict increases SBP reactivity among high hostile males (Smith & Brown, 1991). High hostile individuals have more negative social interactions and also greater cardiovascular reactivity during such interpersonal interactions (Brondolo et al., 2003; Gyll & Contrada, 1998). Therefore, the harassment paradigm of anger inducement may work depending on the extent high hostile participants appraise the situation as one of interpersonal conflict.

One way to investigate this proposition is to differentiate anger induced from interpersonal origins and that induced from non-interpersonal origins. Anger Recall represents a method of interpersonal anger inducement and it has been reported that participants scoring high in Hostility (assessed by the HBI) have greater cardiovascular reactivity during Anger Recall than participant scoring low in HBI (Fredrickson et al., 2000). However, the results for Cynicism in Fredrickson et al.'s study were not reported. Contrary to the results reported by Fredrickson et al., the results of this chapter showed that neither Cynicism nor HBI were

significantly related to cardiovascular reactivity during Anger Recall. Similar to Fredrickson et al.'s study, HBI was not related to self-reported anger. This chapter also extended the analysis from Fredrickson et al.'s study to include Cynicism into the analysis and found that Cynicism was not related to anger, or moderated by anger in predicting cardiovascular reactivity/variability. A closer look at Fredrickson et al.'s study revealed that DBP and HR reactivity obtained during Anger Recall were smaller than that obtained in this chapter. They reported the following reactivity for Anger Recall: HR, 1.18 bpm, SBP, 16.29mm Hg, and DBP, 7.06mm Hg. The reactivity obtained for Anger Recall in this study were: HR, 6.7bpm, SBP, 15.15mm Hg, DBP, 12.85mm Hg. The participants in Fredrickson et al.'s study were also significantly older (mean age = 62). Studies that reported null findings for Structured Interview derived Hostility measures moderated by anger in predicting cardiovascular reactivity consisted of participants who were much younger (mean age = 21.27 for this Study, male undergraduates [age not reported] for Diamond et al., 1984, and mean age = 27.3 for Why et al., 2003). Therefore, the Structured Interview-derived Hostility assessment may be related to cardiovascular reactivity during Anger Recall for older participants.

Anger Recall was used here to elicit anger of an interpersonal origin. Three versions of Synwork were used to elicit anger of a non-interpersonal origin. Synwork Variable Pauses, More Variable Pauses and Anger Recall elicited similar increase in anger while Synwork Normal Control elicited significant less anger compared to these tasks. Diamond et al. was one of the first study to investigate the relationship between

Hostility, Type A Behaviour and cardiovascular reactivity (Diamond et al., 1984). Participants competed with a confederate in a video game in three treatment conditions: a control condition, with distraction (to reduce control) and with harassment. Diamond et al. did not report the results of mood changes associated with each treatment condition but they found that hostility (assessed by the Structured Interview derived Potential for Hostility) did not predict cardiovascular reactivity in any of the treatments. Like Diamond et al.'s study, the results in this chapter also showed that Structured Interview derived HBI was not associated with cardiovascular reactivity in Synwork with different levels of Task Control. No significant interactions between Cynicism and Task Control were found as well. Synwork Variable and More Variable Pauses Task Control significantly increased anger compared to Normal Control. This lends support to appraisal theories of emotion that low perceived control is related to anger arousal. However, Hostility (both Cynicism and HBI) was not associated with anger. Like the Anger Recall task used in this study, Hostility and cardiovascular reactivity/variability relationship was also not moderated by anger for Synwork with different Task Control.

From their meta-analysis, Suls and Wan suggest that anger moderates the Hostility-Cardiovascular Reactivity relationship only when the anger inducement procedure elicits moderate amount of anger. If the anger inducement is great, then both high and low hostile participants will report equivalent amounts of anger (Suls & Wan, 1993). If this is true, then it limits the ability for researchers to test this experimentally. This is because at present, it is not known what procedures can reliably elicit

higher anger among high hostile individuals. As reviewed, harassment and Task Control manipulations do not do so consistently.

State anger tends not to predict cardiovascular reactivity. Feldman et al.'s meta-analysis found that anger's relation to cardiovascular reactivity for speech task, star-tracing task and the handgrip task had effect sizes ranging from .64% to .12% (Feldman et al., 1999). It has been suggested by other researchers that the experimental tasks reviewed by Feldman et al. do not elicit strong emotions (Gerin et al., 1999). The results of Feldman et al.'s meta-analysis are also congruent with other studies that have used harassment or Anger Recall - tasks with the specific aim to elicit anger. In these instances, there are also several studies (including the results reported in this chapter) that have reported null findings for anger in predicting cardiovascular reactivity even when Hostility did predict cardiovascular reactivity (e.g., Everson et al., 1995; Fredrickson et al., 2000; Why et al., 2003). Schwartz suggests that there are possibly two explanations for this weak association between mood and cardiovascular stress reactivity (Schwartz, 1999). One of which is the low reliability of gain scores that frequently used to compute changes in mood. The low reliability of gain scores decreases the power of detecting an effect of mood on cardiovascular reactivity. In this chapter changes in self reported anger were not operationalised as gain scores. Therefore, this reason is not applicable. However, the issue of measurement error is an important one and deserves close scrutiny. One source of measurement error for mood could be the use of Likert scales. Two studies have shown that the reliability of Likert scales is not improved

significantly from 5 to 7 categories (Cicchetti, Showalter, & Tyrer, 1985; Likert, 1932). However it was also found that measurement error on a visual analogue scale is not significantly greater than a 5-point Likert scale (Russell & Bobko, 1992). Researchers have also highlighted the fact that reducing a continuous variable to categories decreases power and increases Type 1 error (Cohen, 1983; MacCallum et al., 2002; Maxwell & Delaney, 1993). Therefore, the absence of Hostility by Anger interaction in predicting cardiovascular reactivity could be due to the frequent use of Likert scale in mood assessment. Chapter 4 changes the 4-point Likert scale used in mood and Perceived Control assessment to a visual analogue scale in order to investigate its impact on detecting a significant Hostility by Anger interaction for cardiovascular reactivity.

Measurement Error in the Cook-Medley Hostility Scale Revisited

One of the explanations given for the presence of null findings is that the Cook-Medley Hostility has a considerable amount of measurement error. One source of measurement error of this scale is attributed to its lack of a stable psychometric structure. As Chapter 2 shows, factor analyses on the Cook-Medley Hostility scale have produced largely inconsistent findings with regards to its internal psychometric structure. This was partly reduced by using a 12-item Cynicism in this chapter based on the review and analysis of Chapter 2. However, this does not rectify another source of measurement error of the Cook-Medley Hostility - the presence of false positives and false negatives.

The presence of false negatives has been the focus of research. False negatives are individuals who give low scores on the Cook-Medley Hostility scale but are hostile. Currently researchers use the Cook-Medley Hostility and the Marlowe-Crowne Social Desirability scale to detect repressed hostile individuals (Helmers et al., 1995; Helmers & Krantz, 1996; Jorgensen et al., 2001; Larson & Langer, 1997; Shapiro et al., 1996a; Shapiro, Goldstein, & Jamner, 1995). Using this method, repressed hostile individuals are defined as participants scoring low in Cook-Medley Hostility scale and high in Marlowe-Crowne Social Desirability. However, the use of the Cook-Medley and Marlowe-Crowne scales to detect false negatives is problematic and this was discussed in detail in Chapter 1. Briefly, this method confounds two different constructs - Repressed Cynicism and low Cynicism-high socially desirability. Repressed Cynicism refers to high cynical individuals but low Cynicism-high social desirability refers to low cynical individuals. The alternative to the Cook-Medley and Marlowe-Crowne method of detecting false negatives is to develop a new assessment of Cynicism that is less affected by social desirability (i.e., GNAT Cynicism).

Chapter 4 will introduce and test a new method for Cynicism assessment that is also less affected by Social Desirability than the Cook-Medley Hostility scale - the Go/No Go Association Task (Nosek & Banaji, 2001). One of the aims of Chapter 4 is to develop and offer the use of the Implicit measure of Cynicism using the Go/No Go Association (GNAT) with the Cook-Medley 12-item Cynicism scale as an operationally more

feasible alternative to tap Repressed Hostility than the Cook-Medley/Marlowe-Crowne method.

CHAPTER 4: STUDY 3

INTRODUCTION

Measurement error in psychometric assessment decreases power and subsequently reduces the ability to detect significant results.

Measurement error in psychometric assessment can also produce unreliable and conflicting results. Measurement error in the Cook-Medley Hostility scale could explain for the presence of null findings with this scale. Though Chapter 2 tries to reduce measurement error of this scale by improving its internal consistency, it does not rectify another source of measurement error. This other source of measurement error for the Cook-Medley Hostility scale (or its derivatives) is the presence of false negatives - repressed cynical individuals.

The current approach taken by researchers is to use both Marlowe-Crowne Social Desirability scale and the Cook-Medley Hostility scale to identify Repressed Cynicism. Using the Cook-Medley/Marlowe-Crowne method, Repressed Cynicism is defined as individuals scoring low in Cook-Medley and high in Marlowe-Crowne. If Repressed Cynicism are not low cynical individuals, then their cardiovascular stress reactivity may not be as low as true low hostile individuals. If a researcher does not identify repressed cynical individuals within his/her sample, conflicting results can appear. For instance, if a researcher unknowingly has a disproportionate number of repressed cynical individuals in the sample, then null findings for Cynicism main effect on cardiovascular reactivity may happen because repressed cynical individuals with low Cynicism

scores may have average or greater reactivity than true low cynical individuals. This chapter investigates the effect of false negatives and false positives on the Cynicism-Reactivity results.

The Cook-Medley/Marlowe-Crowne method of detecting repressed cynical individuals is problematic because it confounds two constructs: Repressed Cynicism and low hostility-social desirability. One construct refers to high cynical individuals (Repressed Cynicism) while the other refers to low cynical individuals (low hostility-social desirability). To date, there is no logical or empirical evidence to prove that individuals scoring high low in Cook-Medley and high in Marlowe-Crowne are hostile. In fact, the results of some analysis with the samples obtained in Study 2 and Study 3 suggest that individuals scoring low in Cook-Medley and high in Marlowe-Crowne are not hostile (Appendix 5).

An alternative method of detecting Repressed Cynicism is to devise another measure of Cynicism that is less affected by Social Desirability. This new method of Cynicism assessment should have moderate correlation (.30 to .70) with the Cook-Medley Hostility scale for convergent construct validity but should have close to '0' correlation with Social Desirability. Recently, the development of Implicit Association Tests (IAT) have been shown to possess such characteristics for the assessment self esteem, racial stereotypes and ageism (Greenwald et al., 1998; Greenwald et al., 2003; Nosek & Banaji, 2001). If the development of the IAT form of Cynicism assessment is successful, then a researcher could define repressed cynical individuals as those scoring low in the Cook-Medley Hostility scale and high in Implicit Cynicism. This

new method of identifying Repressed Cynicism makes better conceptual sense than the Cook-Medley/Marlowe-Crowne method because both IAT Cynicism and Cook-Medley Hostility scale are assessing Cynicism.

The introduction of this chapter details the development of the IAT for Cynicism assessment. The laboratory study reported in this chapter investigates the construct validity of this IAT Cynicism and together with the Cook-Medley Hostility scale, explores the effect of reducing measurement error on the results for the Cynicism-Reactivity relationship. In this chapter, a variation of the IAT, the Go/No Go Association Test (GNAT) is used (Nosek & Banaji, 2001). The reasons for this will be made clear later in this introduction. Both the IAT and GNAT function on the same principles of schemas and priming. This was discussed in Chapter 1 and will only be recapitulated briefly here. Implicit attitudes can be conceptualised as a schema consisting of the evaluation of a certain object. For instance, a person who has high cynical mistrust may have a schema of others being 'dishonest', 'untrustworthy' and 'deceitful'. Researchers can tap the strength of such implicit associations between the evaluative terms and the object via priming. Priming occurs when the prior exposure to a construct increases the processing speed of a related construct. Priming is usually operationalised as a change in reaction time to the stimuli; a stimulus is primed when a participant's reaction to it decreases. When applied to implicit association tests, this means that exposing the participant to the object or increasing its salience (e.g., via repeated exposure) will prime their evaluations related to the object. Their processing speed to these evaluations will then be faster. In the example

for Cynicism, repeated exposure to words pertaining to 'others' (e.g., 'they', 'them', 'their') for a cynical person will prime the adjectives 'dishonest', 'untrustworthy', and 'deceitful'. On the other hand, a person who is not cynical will not have such priming but 'benign' adjectives may be primed instead (e.g., 'honest', 'reliable').

Figure 4.1 Sequence of Trial Blocks in a Racial Stereotype Implicit Association Test

Block	No. of Trials	Function	Items assigned to left key response	Items assigned to right key response
1	20	Practice	Black Names	White Names
2	20	Practice	Pleasant words	Unpleasant words
3	20	Practice	Black Names + Pleasant words	White Names + Unpleasant words
4	40	Test	Black Names + Pleasant words	White Names + Unpleasant words
5	20	Practice	White Names	Black Names
6	20	Practice	Pleasant words	Unpleasant Words
7	40	Test	White Names + Pleasant words	Black Names + Unpleasant words

An example of an Implicit Association Test for Racial Stereotype is shown in Figure 4.1. Results of this form of IAT for racial stereotype has been done and reported by Greenwald and his colleagues (Greenwald et al., 1998; Greenwald et al., 2003). Students rated adjectives as pleasant or unpleasant (Bellezza, Greenwald, & Banaji, 1986) and whether first names as more likely to belong to a Black or White person (Greenwald et

al., 1998). In the first Block of the assessment, the participants practised identifying Black and White Names by pressing the left (for Black names) or right (for White names) key on the computer keyboard whenever a name is displayed on the computer monitor. In the next practise block (Figure 4.1, Block 2), participants had to distinguish the Pleasant and Unpleasant words.

The actual IAT tests are in Blocks 4 and 7. In Block 4, participants' reaction times are an indication of their associations for 'Black-Pleasant' and 'White-Unpleasant'. In Block 7, the participants are response times are tapping into their associations for 'Black-Unpleasant' and 'White-Pleasant'. The order of Block 4 (and its related practise Blocks) and Block 7 (and its related practise Blocks) are counterbalanced. Results showed that White subjects had faster reaction times for Block 7 than Block 4. This means that White subjects had stronger associations between White-Pleasant words than Black-Pleasant words (Greenwald et al., 1998). This is congruent with the general finding that a person tends to evaluate his/her in-group in more positive terms than the out-group (Brewer, 1979).

However the IAT cannot be directly adopted for Cynicism assessment. A closer look at the design of this form of IAT shown in Figure 4.1 shows that at Block 4, subjects responded to both 'Black Names-Pleasant words' and 'White Names-Unpleasant words' associations at the same block. This is congruent with the concept of racial stereotype where the evaluation is comparative (i.e., between two racial groups). However, the concept of Cynicism is as measured by the

Cook-Medley Cynicism scale (from Chapter 2) is not comparative. The content analysis for the 12-item Cynicism scale from the Cook-Medley Hostility scale in Chapter 2 revealed that 11 of the 12 items measure the respondent's evaluation of others without reference to the respondent. Items that included references about the respondent's personality were found not to load onto the same factor as the Cynicism 12 items consistently. Thus, the concept of Cynicism as measured by the Cook-Medley 12-item Cynicism scale is not comparative like attitudes of racial stereotype. This means that a person who is cynical is distrustful of others (Implicit Cynicism), but this does not necessarily imply that they evaluate themselves as more benign (Implicit Trait Hostility). Thus, a cynical person can have a negative evaluation of others as well as a negative evaluation of himself/herself. Therefore, the IAT is not an appropriate design to assess Cynicism. Nosek and Banaji also mentions other instances when the in-group versus out-group comparative nature of the IAT design may not be appropriate (Nosek & Banaji, 2001).

The Go/No Go Association Task (GNAT) is a variation of the IAT and its design removes the in-group versus out-group comparative nature of the IAT. The GNAT is designed so that participants responds to only one class of stimuli in any particular Block ('Go' or target stimuli) via tapping a key and ignores the distractors ('No Go' or distracter stimuli). Nosek and Banaji showed that the results produced using the IAT could be replicated using the GNAT (Nosek & Banaji, 2001). Figure 4.2 shows an abbreviated version of the GNAT for Cynicism (the practice Blocks are not included).

Figure 4.2 Abbreviated example of the Cynicism Go/No-Go**Association Test (GNAT Cynicism)**

Block	No. of Trials	Function	Target ('Go') Stimuli	Distracter ('No Go') Stimuli*
1	40	Test	Self Nouns + Benign	Positive + Negative adjectives
2	40	Test	Self Nouns + Hostile adjectives	Positive + Negative adjectives
3	40	Test	Other Pronouns + Benign adjectives	Positive + Negative adjectives
4	40	Test	Other Pronouns + Hostile words	Positive + Negative adjectives

*The stimuli in this category refers to adjectives rated by participants that are not related to Hostile or Benign adjectives (e.g., 'energetic').

As shown in Figure 4.2, participants respond to the 4 different combinations of 'Self-Benign', 'Self-Hostile', 'Other-Benign' and 'Other-Hostile' in separate Blocks. Blocks 3 and 4 are used to derive the implicit Cynicism while Blocks 1 and 2 are used to derive implicit Trait Hostility. A person who has high implicit Cynicism will respond faster to 'Other-Hostile' (Block 2) than 'Other-Benign (Block 1). An individual with high Trait Hostility will respond faster to 'Self-Hostile' (Block 2) than 'Self-Benign' (Block 1). The participants have to focus their attention and respond to the appropriate target stimuli due to the presence of distracter items. The distracter items are the same for all Blocks and Blocks 1 to 4 (and their respective practice Blocks) will be randomised.

The aims of Study 3 are:

1) To investigate the convergent and discriminant construct validity of the Implicit Cynicism.

1b) Implicit Cynicism's convergent construct validity is indicated by moderate correlation (r .30 to .70) with the Cook-Medley Hostility scale.

1c) Implicit Cynicism's discriminant construct validity is indicated by close to '0' or non-significant correlation with non-Cynicism related constructs. Non-Implicit Cynicism related constructs in this chapter are indexed by: Marlowe-Crowne Social Desirability scale (Crowne & Marlowe, 1960), STAXI's Anger Reaction (Spielberger, 1988), DS16 Negative Affectivity subscale (Denollet, 2000).

2) To investigate the relationship between Implicit Cynicism and cardiovascular stress reactivity. Is Implicit Cynicism positively related to cardiovascular stress reactivity?

3) To investigate the effects of measurement error reduction in Cynicism assessment on the Cynicism-Reactivity relationship. Repressed Hostility is defined as individuals scoring low in the Cook-Medley Hostility scale and high in Implicit Cynicism.

The following are the hypotheses of Study 3 involving the Cook-Medley 12-item Cynicism scale, Control (actual and perceived), Anger and cardiovascular stress reactivity.

Hypothesis 1: Cynicism and cardiovascular Reactivity. Cynicism is positively associated with cardiovascular reactivity - a significant Cynicism main effect. Nonlinear trends between Cynicism and cardiovascular reactivity are also explored.

Hypothesis 2: Task Control differences for Cardiovascular Reactivity

Normal Task Control is associated with increased CO reactivity and decreased TPR reactivity (active coping physiological response) while Variable and More Variable Pauses Task Control are associated with decreased CO and increased TPR (passive coping physiological response).

Hypothesis 3: Task Control (Actual Control) and Perceived Control.

Task Control and Perceived Control predict common/unique variance for the dependent variables. As there is evidence for Task Control and Perceived Control predicting common and unique variance. This is an exploratory analysis rather than a hypothesis. As in Study 2, the results of these analyses helps to determine the analysis for Hypothesis 4.

Hypothesis 4: Cynicism and Cardiovascular Reactivity moderated by Control. It is hypothesised that participants scoring high in Cynicism have greater cardiovascular reactivity in situations of low control (actual or perceived) - a significant Cynicism x Control interaction.

Hypothesis 5: *Cynicism and Cardiovascular Reactivity is moderated by State Anger.* A Cynicism x State Anger interaction is hypothesised to be significant for cardiovascular reactivity. Participants with high Cynicism scores have greater reactivity when self-reported state anger is high.

5a) Cynicism x State Anger_{btw} and Cynicism x State Anger_{wth} are significant for cardiovascular reactivity. Participants with high Cynicism scores have greater reactivity when State Anger is high.

Method

Sample

Table 4.1 Sample Characteristics (n = 59)

	Mean (SD)
Physiological	
Age, years	23.53 (5.32)
Gender	Male
Height, cm	180.43 (7.09)
Weight, kg	77.76 (14.03)
BMI, kg/m ²	23.91 (4.00)
BSA, m ²	1.97 (.19)
Baseline Systolic Blood Pressure, mmHg	110.27 (13.42)
Baseline Diastolic Blood Pressure, mmHg	73.64 (10.53)
Baseline Mean Arterial Pressure, mmHg	86.01 (10.56)
Baseline Heart Rate, bpm	70.23 (8.72)
Psychological	
Cook-Medley Scale	21.46 (6.46)
Cook-Medley 12-item Cynicism Scale	5.78 (2.18)
Marlowe-Crowne Social Desirability	14.76 (5.48)
GNAT Hostility, ms	-.06 (.39)
GNAT Cynicism, ms	-.07 (.47)
STAXI - Trait Anger Reaction	10.25 (2.78)
DS16, Negative Affectivity Subscale	10.85 (6.09)

Table 4.1 shows the sample characteristics of Study 3. Like Study 2 (Chapter 3) participants were recruited by word of mouth and advertisements posted at various venues within the University campus. Sixty-one participants volunteered for this study. All participants were paid at the rate of £4 per hour. One participant withdrew from participation and another participant had Baseline Total Peripheral Resistance value greater than +4.7SD from the mean. These two cases were dropped from the cardiovascular reactivity analysis, reducing the sample size to 59. However, for the assessing the correlation among the various psychological scales, the last participant was included.

Independent t-tests were conducted to examine any differences in sample characteristics between Study 2 and this study. Alpha for this Study was set at .05. When Levene's test for the equality of variances was significant, the correct adjustment was applied. For the physiological variables, BMI, $t(98.12) = -2.25, p = .03$, BSA, $t(100.65) = -2.15, p = .03$, and Age, $t(91.73) = -2.81, p = .006$, were found to be significantly different between the two samples. The sample of this study was slightly older (mean age = 23.53) than the sample in Study 2 (mean age = 21.27). The sample for this chapter also had higher BMI (mean = 23.91) and BSA (mean = 1.97) than Study 2's participants (BMI mean = 22.52, BSA mean = 1.91). Baselines for SBP, DBP, MAP and HR were not significantly different between the two samples, p 's range from .13 to .89. For the psychological measures, there were also no significant differences for the 50-item Cook-Medley scale, Cook-Medley 12-item Cynicism scale, Marlowe-Crowne Social Desirability scale, and DS16

Negative Affectivity subscale, p 's range from .14 to .92. Therefore, besides age, BMI and BSA, the two samples had similar psychological and cardiovascular profiles.

Psychological Measures

Participants were asked to fill in the following measures: Cook and Medley Hostility scale, Marlowe-Crowne Social Desirability scale, DS16 scale, and State Trait Anger Expression Inventory (Spielberger, 1988) after the computer assessment for the Implicit measures of Cynicism and Trait Hostility. The order of these scales in the questionnaire booklet was randomised.

Implicit Measures of Cynicism and Trait Hostility. A pool of 40 words were selected to be rated in order to obtain the stimuli for the Implicit Cynicism and Hostility assessment. Ten of the 40 words were chosen to describe hostile attributes (e.g., 'deceitful'), another 10 were chosen to describe benign attributes (e.g., 'sincere'), another 10 words were chosen to describe positive non-hostile attributes (e.g., 'sensible'), and another 10 words were chosen to represent negative non-hostile attributes (e.g., 'illogical'). Out of these 40 words and based on the ratings from 10 participants, 5 words were to be selected from each group to represent 'Hostile', 'Benign', 'Positive', and 'Negative' adjectives. 'Hostile' and 'Benign' adjectives were used as target stimuli while 'Positive' and 'Negative' adjectives were used as distracter stimuli in the GNAT.

Ten male participants who did not participate in this study (mean age [SD] = 25.30 [3.40]) were recruited by word of mouth to rate these 40 words. Each participant rated the 40 words on two dimensions: the 'Hostile-Benign' dimension and the 'Positive-Negative' dimension. Five participants rated the 'Hostile-Benign' dimension first while the other 5 participants rated the 'Positive-Negative' dimension first. The order of the words presented in the questionnaire booklets was also randomised within each dimension rated. Each participant rated each dimension on a 7-point Likert like scale. The ends of the scale were counterbalanced for each participant. For example, some participants rated the words using the 7-point Likert scale where '7' is for Hostile and '1' is for Benign. Some other participants rated the words using the 7-point Likert scale where '7' is for Benign, and '1' is for Hostile. The two dimensions in the same questionnaire booklet have opposite polarities in the Likert scales used. This meant that if one participant rated the words using a Likert scale where '1' for Hostile and '7' is for Benign, then the Likert scale used for the 'Positive-Negative' dimension will be '1' for 'Positive' and '7' for 'Negative'. This complex balancing process ensured that the participant paid attention to the scales used to rate the words. If the polarities of the Likert scales used for the two dimensions were the same for each questionnaire booklet, the participant might simply give identical ratings without paying attention to the scale used.

Based on the participants' ratings, words selected as 'Hostile' adjectives were rated more likely as hostile and negative. Words selected as 'Benign' were rated more likely as benign and positive. Positive

distracter items have neutral ratings (near '4') for the 'Hostility-Benign' dimension and rated as being positive. Negative distracter items have also neutral ratings ('4') for the 'Hostility-Benign' dimension and rated as being more negative. Five 'Hostile' adjectives, 5 'Benign' adjectives, 5 'Positive' adjectives, and 5 'Negative' adjectives were selected from the 40 to maximise these differences in the ratings on the two dimensions as

Table 4.2 Mean (SD) ratings of the 10 Hostile adjectives used to derive the stimuli for Cynicism GNAT (n = 10)*

	Hostile-Benign dimension	Negative-Positive dimension
Hostile adjectives		
1) aggressive	1.60 (.97)	2.20 (.92)
2) annoying	2.50 (.85)	1.80 (.79)
3) antagonistic	1.70 (.95)	1.80 (1.03)
4) conniving	2.20 (.92)	1.60 (.84)
5) deceitful	1.90 (.99)	1.50 (.97)
6) dishonest	2.10 (.99)	1.60 (.97)
7) offensive	2.30 (.82)	2.40 (1.35)
8) peeved	3.50 (1.18)	2.80 (.79)
9) scheming	2.50 (.97)	2.40 (1.35)
10) touchy	3.50 (1.18)	2.60 (.52)

*Bold words and figures are selected for use as stimuli for the Cynicism GNAT.

well as have comparative syllable length. Tables 4.2 to 4.5 show the mean (SD) ratings for these 40 words. Words selected are in bold.

Ratings for the two dimensions were recoded so that higher ratings on the 'Hostile-Benign' dimension indicated more Benign ratings and higher ratings on the 'Negative-Positive' dimension indicated more Positive ratings.

Table 4.3 Mean (SD) ratings of the 10 Benign adjectives used to derive the stimuli for Cynicism GNAT (n = 10)*

	Hostile-Benign dimension	Negative-Positive dimension
Hostile adjectives		
1) amiable	6.00 (.67)	5.40 (.84)
2) friendly	6.10 (1.10)	6.30 (.82)
3) genuine	5.60 (1.17)	6.40 (.70)
4) helpful	6.10 (.88)	6.50 (.71)
5) peaceful	6.50 (.97)	6.30 (.82)
6) reliable	5.80 (1.23)	6.10 (1.10)
7) sincere	5.50 (1.08)	6.30 (.67)
8) supportive	5.90 (.88)	6.00 (1.05)
9) truthful	5.90 (.99)	6.50 (.71)
10) trustworthy	5.80 (1.03)	6.30 (.95)

*Bold words and figures are selected for use as stimuli for the Cynicism GNAT.

Table 4.4 Mean (SD) ratings of the 10 Positive adjectives used to derive the stimuli for Cynicism GNAT (n = 10)*

	Hostile-Benign dimension	Negative-Positive dimension
Hostile adjectives		
1) active	4.70 (.95)	5.40 (1.35)
2) busy	4.40 (.97)	4.70 (.82)
3) diligent	5.30 (.67)	6.00 (1.05)
4) industrious	4.90 (1.10)	5.50 (.71)
5) practical	5.10 (1.10)	5.60 (.84)
6) prudent	4.90 (.88)	5.70 (.82)
7) rational	5.20 (.79)	5.40 (.97)
8) sensible	5.40 (1.07)	5.80 (.92)
9) quick	4.40 (1.07)	4.80 (1.32)
10) vigilant	4.30 (1.25)	5.30 (1.34)

*Bold words and figures are selected for use as stimuli for the Cynicism GNAT.

Table 4.6 shows the mean ratings by GNAT Stimuli group and rated dimensions. The ratings shown in Table 4.6 were obtained via averaging the 5 word stimuli for each GNAT stimuli group. For example, Hostile GNAT stimuli ratings for the Hostile-Benign dimension was the average Hostile-Benign dimension ratings for the items 'annoying', 'deceitful', 'dishonest', 'offensive' and 'scheming'. Paired *t*-tests using Bonferroni Type 1 error correction showed that the four groups of GNAT stimuli differentiated well in terms of the Hostile-Benign dimension. Table

4.5 Mean (SD) ratings of the 10 Negative adjectives used to derive the stimuli for Cynicism GNAT (n = 10)*

	Hostile-Benign dimension	Negative-Positive dimension
Hostile adjectives		
1) foolish	3.00 (.94)	2.20 (1.03)
2) idle	3.90 (1.66)	2.60 (.97)
3) illogical	3.50 (.97)	2.00 (1.05)
4) lazy	3.50 (1.65)	2.10 (1.29)
5) listless	3.90 (1.20)	3.00 (1.05)
6) sloppy	3.20 (1.23)	2.00 (.47)
7) sluggish	3.90 (1.29)	2.30 (.95)
8) submissive	4.80 (1.48)	2.80 (1.03)
9) unrealistic	3.50 (.97)	2.70 (.95)
10) unwise	3.30 (.82)	2.30 (.82)

*Bold words and figures are selected for use as stimuli for the Cynicism GNAT.

Table 4.6 Mean Ratings (SD) by Stimuli Group and Dimension (n = 10)*

Dimensions	GNAT Stimuli			
	Hostile	Benign	Negative	Positive
Hostile-Benign	2.26 _a (.71)	5.92 _b (.84)	3.86 _c (1.22)	4.62 _c (.90)
Negative-Positive	1.94 _a (.83)	6.02 _b (.70)	2.36 _a (.79)	5.22 _c (.64)

_{a, b, c} Means with different subscripts are significantly different ($p < .05$) within rows.

4.6 shows that Hostile and Benign GNAT stimuli were significantly different from each other and with Positive and Negative GNAT stimuli as well. On the other hand, Positive and Negative GNAT items were not significantly different from each other in terms of Hostile-Benign ratings. The ratings showed that Hostile GNAT stimuli tended to have Hostile ratings, Benign GNAT stimuli tended to have Benign ratings while Positive and Negative GNAT stimuli tended to have scores near '4' on the Hostile-Benign dimension (i.e., neither rated Hostile nor Benign). The Hostile-Benign dimension discriminated the four groups of GNAT stimuli in a desired pattern. Figure 4.3 and 4.4 show the results for the Hostile-Benign dimension and Negative-Positive dimension ratings for the four groups of GNAT stimuli respectively.

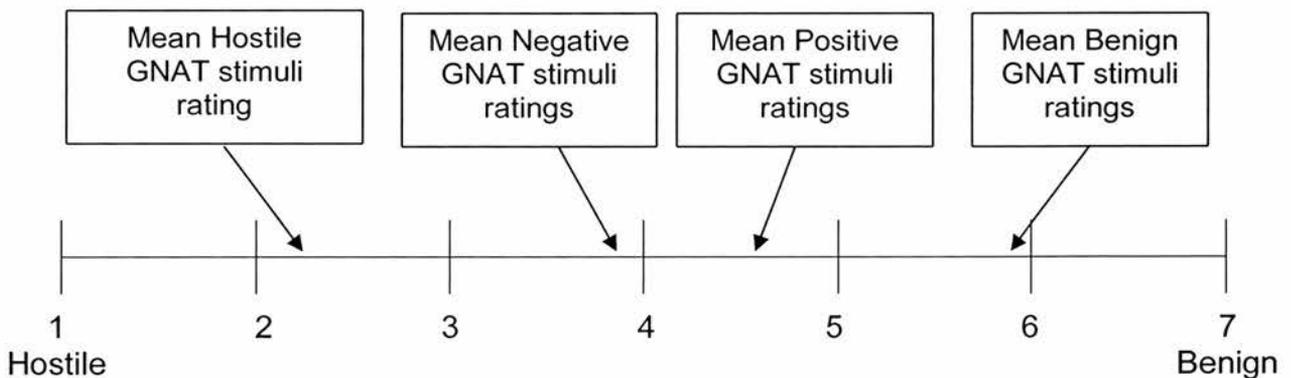


Figure 4.3 Graphic Representation of the mean Hostile-Benign ratings for the 4 GNAT stimuli groups

The Positive-Negative ratings for the four GNAT stimuli groups were slightly less ideal. Both Hostile GNAT stimuli and Negative GNAT stimuli had similar negative ratings for the Negative-Positive dimension. However, Benign GNAT stimuli had significantly higher positive ratings

than the Positive GNAT stimuli. But Positive GNAT stimuli still had significantly higher positive ratings than Hostile GNAT and Negative

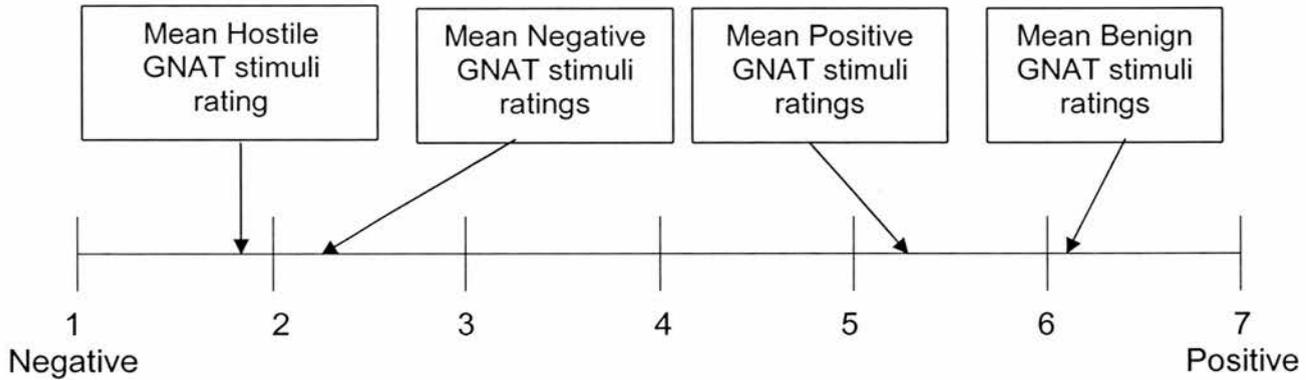


Figure 4.4 Graphic Representation of the mean Negative-Positive ratings for the 4 GNAT stimuli groups

GNAT stimuli. Overall, the ratings for the Hostile-Benign dimension and the Negative-Positive dimension discriminated the four GNAT stimuli quite well in the desired directions.

Besides the four group of GNAT adjectives, another two groups of GNAT stimuli were needed. These two groups consisted of proper nouns referring to the self and pronouns referring to others. Self Nouns consisted of the participant's city, country of origin, gender, ethnicity, and first name. These were used to refer to the participant ('Self Nouns'). Greenwald and Farnham found that the use of such ideographic Self

Figure 4.5 GNAT Cynicism and Trait Hostility

Block	No. of Trials	Function	Target ('Go') Stimuli	Distracter ('No Go') Stimuli
1a	40	Practice	Self Nouns + Benign	Positive + Negative adjectives
1b	40	Test	Self Nouns + Benign	Positive + Negative adjectives
2a	40	Practice	Self Nouns + Hostile adjectives	Positive + Negative adjectives
2b	40	Test	Self Nouns + Hostile adjectives	Positive + Negative adjectives
3a	40	Practice	Other Pronouns + Benign adjectives	Positive + Negative adjectives
3b	40	Test	Other Pronouns + Benign adjectives	Positive + Negative adjectives
4a	40	Practice	Other Pronouns + Hostile words	Positive + Negative adjectives
4b	40	Test	Other Pronouns + Hostile words	Positive + Negative adjectives

Nouns (rather than 'I' or 'me') increased the implicit to explicit correlation (Greenwald & Farnham, 2000). Pronouns used to indicate 'others' were 'they', 'them', 'their', 'it', and 'other' as used by Greenwald and Farnham in their study.

These stimuli were used in the different combinations as shown in Figure 4.5. Figure 4.5 shows the structure of the GNAT used in this chapter. It includes the practice trials. These blocks were presented via a desktop computer using Inquisit software (2003). At the start of the Implicit Cynicism/Hostility assessment, instructions were displayed on screen. The program then prompted the participants to answer five

questions in order to derive the GNAT stimuli for Self Nouns (i.e., the participant's city, country of origin, gender, ethnicity, and first name). All Nouns entered by the participants were in lowercase letters. This was because all the stimuli used by the GNAT that were not provided by the participant were in lowercase letters. If the participant entered a Noun with uppercase letters, this could enable the easy identification of these Nouns. The experimenter also stayed at the first block of the Implicit Cynicism/Hostility assessment to observe whether the participant understood the instructions. (The first block at the beginning of the Implicit Cynicism/Hostility assessment was always a practice block.) Participants were told that speed ('respond to target stimuli as fast as possible') and accuracy ('do not press the spacebar for every single stimuli', 'use the feedback from the computer to improve your accuracy') were important in this assessment. Feedback was given in the form of a green 'O' appearing on the screen for a correct response lasting 250ms. Feedback for an incorrect response was a red 'X' displayed on the monitor for 450ms.

There was a practise trial preceding each actual test trial. The presentation order of the 4 blocks (Block 1a+1b, Block 2a+2b, Block 3a+3b, Block 4a+4b in Figure 4.5) was randomised. In each block there were 20 GNAT stimuli and each one was presented twice. For instance, in blocks 2a and 2b, the 20 items were 5 Self Nouns, 5 Hostile adjectives, 5 Positive adjectives, and 5 Negative adjectives. In each block, the Inquisit software randomly selected from the pool of 20 stimuli twice without replacement. Thus, each stimulus was presented to the

participant twice in each block. During each block, the items were presented for a period of 1 second. If the participant did not respond for the 1 second duration, the stimulus will disappear from the computer screen. This duration was chosen so as to enable the response of the participant to be fast (in order for this assessment of Cynicism to be implicit) and not commit too many errors (Nosek & Banaji, 2001). Before and after each block, there was a pause and the participant can initiate the next block when he was ready. All responses to the GNAT stimuli were made via tapping (or not tapping) the 'spacebar' on the computer keyboard. The participant's response time for each stimuli was recorded onto the computer by the Inquisit software.

Implicit Cynicism was calculate by subtracting the mean response time obtained during Block 4b ('Other-Hostile') from the mean response time obtained during Block 3b ('Other-Benign') and divided by the SD of the response times in Blocks 4b and 3b combined. This was to control for individual variation in response times (Greenwald et al., 2003). Thus, positive scores represent higher levels of Implicit Cynicism. Implicit Trait Hostility was calculated by subtracting the mean response time during Block 2b ('Self-Hostile') from Block 1b ('Self-Benign') and divided by the SD of all the response times in Blocks 2b and 1b combined. Positive scores represent higher levels of Implicit Hostility.

While Greenwald et al. found that recoding errors increased the implicit-explicit correlation, this was not found in this study (Greenwald et al., 2003). In fact, when errors were recorded (mean + 2SD) as recommended by Greenwald et al., the implicit-explicit correlation

decreased to below .30 for Cynicism. Hence, errors were excluded from the computation of Implicit Cynicism and Trait Hostility. The number of correct responses obtained under each condition was shown in Table 4.7. If a participant made 20 Hits and 20 Correct Rejections, this meant that for that particular block, there were no mistakes made since there were 40 trials in each Block. A 2 x 2 x 2 repeated measures analysis was conducted using the data presented in Table 4.7 to investigate differences in the number of correct responses across the various conditions. The model tested had three repeated measures: Self-Other (2 levels), Hostile-Benign (2 levels), Hit-Reject (2 levels). Two significant main effects were found, a Self-Other main effect, $F(1, 58) = 7.66, p = .008, \eta^2 = .12$, and a Hit-Reject main effect, $F(1, 58) = 38.77, p = .06 \times 10^{-10}, \eta^2 = .40$. Participants were more accurate when responding to Blocks using Self Nouns (18.23) compared to 'Other' pronouns (17.80). They also had more Hits (18.64) than Correct Rejections (17.40). However the differences were small. Overall, the participants had a good level of accuracy for the GNAT task.

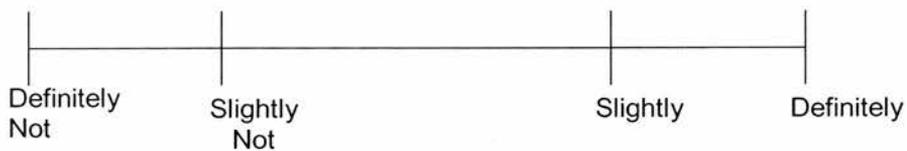
Table 4.7 Number of Hits and Correct Rejections by Block (n=59)

	Self-Benign	Self-Hostile	Other-Benign	Other-Hostile
Hit (Min = 0, Max = 20)	18.68 (1.44)	18.78 (1.38)	18.72 (1.16)	18.37 (1.96)
Correct Rejection (Min = 0, Max = 20)	17.68 (2.21)	17.77 (2.38)	16.98 (3.40)	17.15 (2.36)

Table 4.8 The 15 Mood Adjective items derived from Matthews et al. (Matthews et al., 1990)

Satisfied	}	Hedonic tone items	}	Hedonic tone (Factor 1)
Happy				
Cheerful				
Sad				
Impatient	}	Anger items	}	
Annoyed				
Angry				
Irritated				
Grouchy				
Anxious	}	Tense arousal (Factor 2)	}	
Relaxed				
Calm				
Alert	}	Energetic arousal (Factor 3)	}	
Energetic				
Tired				

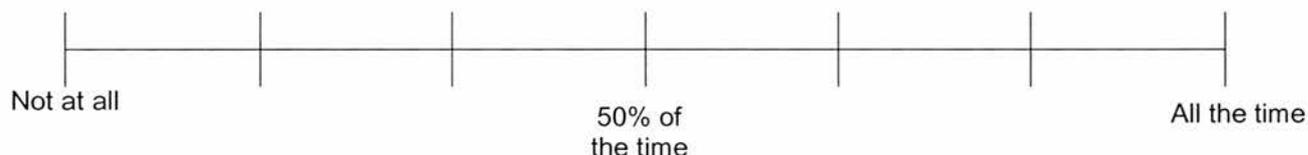
During the experiment, the participants also filled in the UNIWIST mood adjective checklist consisting of 15 items used in Study 2 (Table 4.8). However, the rating scale was not a 4-point Likert scale as in Study 2 but a visual analogue scale consisting of a 10cm line. The following continuous scale was used:



Participants were shown examples and instructed to draw an 'X' on the line and within the scale to indicate their mood. '0'cm represents 'Definitely Not', 2.5cm represents 'Slightly Not', 7.5cm represents 'Slightly' and 10cm represents 'Definitely'. Labels used are the same as in

Study 2. Perceived control of Synwork was assessed using the following 15cm visual analogue scale as well:

1. To what extent were you able to influence the outcome of the task?



For Perceived Control, '0' represents 'Not at all', 7.5cm represents '50% of the time' and 15cm represents 'All the time'.

Physiological Measures

The dependent variables for this study were Systolic Blood Pressure (SBP) Diastolic Blood Pressure (DBP) Mean Arterial Pressure (MAP) Heart Rate (HR) Cardiac Output (CO) and Total Peripheral Resistance (TPR). SBP, DBP, MAP and HR were assessed using the Finapres while CO and TPR were estimated from the finger arterial pressure obtained from the Finapres by Modelflow software. This method was identical to that used in the previous chapter. Finger arterial pressure is significantly correlated with intra-brachial pressure (Jellema et al., 1996) and the Finapres can track blood pressure and heart rate changes accurately (Imholz et al., 1998). Modelflow's estimation of stroke volume significantly correlates with clinical assessments of stroke volume using Doppler echocardiography and thermodilution (Gratz et al., 1992; Sugawara et al., 2003).

Task

The task used here was the same as Study 2 - Synwork (Elsmore, 1994). To describe this task briefly, it was a multi-task computer program. Participants performed four tasks simultaneously to gain points. The four tasks were: a memory task, a mental arithmetic task, a visual monitoring task and an auditory task. Participants wore headphones to detect the auditory signals for the auditory task.

The Task Control (Actual Control) manipulation

Each participant performed four sessions of Synwork of 3minutes duration each. One session was a practice session. The other three Synwork sessions had Normal, Variable Pauses or More Variable Pauses. The practice session of Synwork was given first. The three treatment conditions for Synwork (Normal, Variable and More Variable Pauses) were counterbalanced and each participant was randomly assigned to one of the 6 Synwork treatment counterbalancing orders after the Synwork practice session.

Like Study 2, Synwork's controllability was also manipulated via the Faulty Mouse Device (Appendix 4). During the normal sessions of the Synwork task (i.e., practise and Normal condition), this was switched off. During the Variable Pauses condition, the Faulty Mouse device generated pauses between 25 to 300ms. During the More Variable Pauses condition, the device created random pauses between 25 to 500ms. Participants did not know that the Synwork task would be less controllable as the experimental session progresses. These settings of

the Faulty Mouse were the same as in Study 2. If participants complained about the uncontrollability of the task, the experimenter would explain that it was due to a software incompatibility between the Synwork task and the computer's Operating Software. The participant would then be told that the computer would be rebooted and their scores for Synwork corrected accordingly. In actuality, nothing was done. Participants were encouraged to try their best even when Synwork was less controllable. At the end of the experiment, participants were debriefed that the purpose of this deception was to enable them to maintain their motivation to do well in the task even when it became less controllable.

Procedure

Study 3 was conducted on weekdays from 9am to 6pm. Each session lasted from 2 to 2.5 hours depending on the duration it took for participants to complete the psychological assessments. When the participant arrived, he was debriefed about the aims and general procedures of the experiment. After obtaining informed consent, weight and height of the participant was taken and the participant was seated in front of a computer to perform the GNAT for Implicit Cynicism and Trait Hostility assessment. This was followed by the explicit questionnaires (e.g., Cook-Medley Hostility scale).

After filling in the questionnaires, the Finapres was placed on the left hand index finger and the left arm rested on an adjustable armrest that kept the finger at the participant's heart level as finger arterial pressure is affected by distance from the heart. After ensuring that the

physiological data collected was stable, the experimenter informed the participant about the commencement of the first Baseline and the experimental procedures.

Participants were told that the Synwork sessions after the practice session would determined part of the monetary compensation that they would receive. In actuality, this was a deception to ensure their motivation to perform well even when Synwork had Variable and More Variable Pauses Task Control. At the end of the experiment, the Finapres was removed and the participant was debriefed about the aims of the study as well the purpose of the study and the appropriate amount of monetary compensation was given.

Data Analyses Strategy

The same data analytic method used in Study 2 was also used here; SPSS version 10 GLM and Regression modules and MS Excel 97 were used to perform ANCOVA. Continuous variables were centred and the same effect coding used in Study 2 to represent the Task Control levels were also used (Table 4.10).

Table 4.9 Effect Coding for Task Control

Vectors	T1	T2
Task Control		
Normal	-1	-1
Variable Pauses	1	0
More Variable Pauses	0	1

Multivariate repeated measures revealed the same pattern of significant results as epsilon-corrected univariate repeated measures. The results for epsilon-corrected repeated measures are reported here.

GNAT Cynicism was a new measure of Cynicism and it was significantly correlated with its corresponding explicit measure (12-item Cynicism scale). Therefore, there was a problem of collinearity when both were analysed together. This chapter analysed GNAT Cynicism and 12-item Cynicism separately. This was because the analyses for GNAT Cynicism was exploratory while the analyses for the 12-item Cynicism was to replicate some of the results obtained in Study 2. Doing separate analysis enables comparison with between the two measures of Cynicism on cardiovascular stress reactivity as well as with Study 2. However separating the analysis between Implicit and Explicit Cynicism did not address the collinearity issue.

One way the collinearity issue can be addressed was via using exploratory factor analysis to reduce the correlated variables into uncorrelated ones (Stevens, 1996). Factor analysis via principal components was done here to eliminate the collinearity between GNAT Cynicism and 12-item Cynicism. However, this approach was taken only if both measures load onto one factor at .80. That is, the factor obtained was correlated with both measures of Cynicism at .80. Only when this criterion was fulfilled can the factor claim to measure Cynicism. The remaining factor(s) would represent measurement error.

Principal components factor analysis revealed that a 2-factor

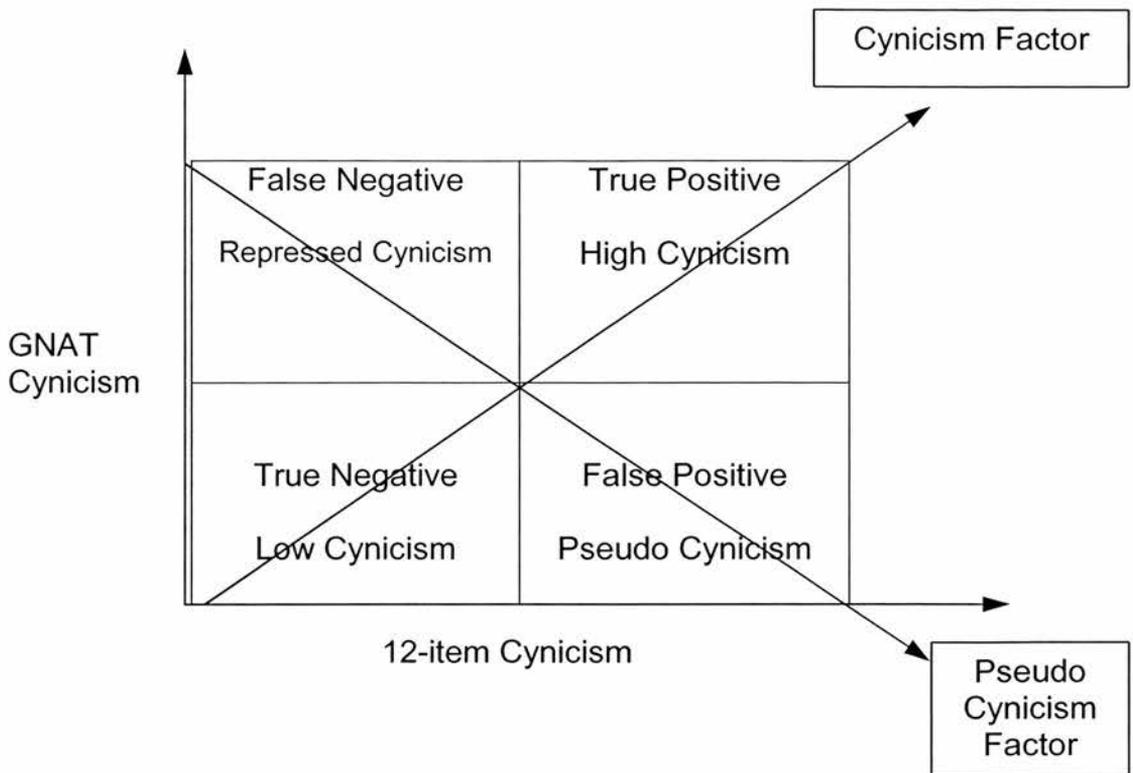


Figure 4.6 Graphic representation of the Conceptual Meaning of the Cynicism Factor and the Pseudo Cynicism Factor

solution accounted for 100% of the variance for GNAT Cynicism and 12-item Cynicism. The first factor accounted for 65.13% variance

(eigenvalue = 1.30) while the second factor accounted for the remaining 34.87% variance (eigenvalue = .70). Both GNAT Cynicism and 12-item Cynicism loaded onto the first factor with the same loading (.81).

Therefore, this factor was labelled as the Cynicism Factor. For the second factor, GNAT Cynicism factor loading was negative (-.59) while 12-item Cynicism was positive (.59). This factor represented measurement error from the Cynicism Factor and was labelled as Pseudo Cynicism Factor. Based on the factor loadings of the two Cynicism measures on the Pseudo Cynicism Factor, a high score on this factor indicate a high score on the 12-item Cynicism and a low score on the

GNAT Cynicism. Since the Pseudo Cynicism factor was orthogonal to the Cynicism Factor, this would be a false positive. Low scores on the Pseudo Cynicism factor would indicate a false negative (repressed cynical individuals). Figure 4.6 graphically shows the conceptual interpretation of these two factors. Since the results of the principal components factor analysis is conceptually sound, these two factors were used in investigating the relationship of these different types of Cynicism on cardiovascular stress reactivity. Scores for these two factors were obtained via regression. These variables had a mean of '0' and SD = '1'. The factor analysis removed about 35% of measurement error (Pseudo Cynicism Factor) from both 12-item Cynicism and GNAT Cynicism. Both Cynicism Factor and Pseudo Cynicism were uncorrelated, and can be included to investigate the relationship between Cynicism, Pseudo Cynicism and cardiovascular stress reactivity without the collinearity problem. Current problem with Cynicism assessment was its failure to discriminate Cynicism from Negative Affectivity. The correlation between the Cynicism Factor and DS16 Negative Affectivity scale is $r(60) = .19$, while between Pseudo Cynicism Factor and DS16 Negative Affectivity scale is $r(60) = .29$. As reported in Table 4.9, the 12-item Cynicism scale correlates significantly with negative affectivity at $r(60) = .33$. Thus, factor analysis reduced the correlation between Cynicism and negative affectivity.

In Study 2 (Chapter 4) there were two models tested - the Anger Recall and Synwork model. In this chapter, there were three: the GNAT Cynicism model, the 12-item Cynicism model, and the Cynicism Factor

model (including the Pseudo Cynicism Factor). Nonlinear terms (quadratic, cubic) for Cynicism were also assessed.

Results

Alpha level was set at .05 since part of the analyses in this chapter concerning the Implicit Cynicism measure and curvilinear trends were exploratory. Secondly, as in Study 2, two effect sizes were reported - η^2 and partial η^2 . The denominator for the partial η^2 was computed via subtracting the sums of squares due to the covariates (i.e. dependent variables' Baselines and its interactions) from the total sums of squares. This was to enable comparisons with other studies that have removed the effects of the Baseline from the error term (e.g., gain scores or residuals analysis). However the within subject model does not have covariates and since certain variables were present in both the within and between subject models (e.g., Perceived Control), η^2 would enable a comparison between the within and between subject models.

Correlations among the Psychological Variables. Table 4.9 shows that the 12-item Cynicism had poor discriminant construct validity. It correlated significantly with social desirability, negative affectivity and trait anger reaction. On the other hand, GNAT Cynicism (the implicit measure of Cynicism) had convergent construct validity as it is correlated significantly with the 12-item Cynicism and good discriminant construct validity. Specifically, GNAT Cynicism was not significantly correlated with

social desirability, negative affectivity or trait anger reaction. The moderate correlation (.30) between the two forms of Cynicism (i.e., 12-item Cynicism scale of the Cook-Medley Hostility scale and GNAT Cynicism) indicated that these two measure similar but not identical constructs. There was also a lack of correlation between GNAT Trait Hostility and GNAT Cynicism. This was consistent with the assertion that a cynical view of others (GNAT Cynicism) does not necessarily imply a benign view of the self (as assessed by GNAT Trait Hostility). GNAT measures were the implicit measures of Cynicism and Trait Hostility.

Table 4.10 Intercorrelations Between the Psychological Measures (n = 60)

	GNAT Trait Hostility	GNAT Cynicism	12-Item Cynicism
1) GNAT Trait Hostility	--	.04	--
2) 12-item Cynicism	.16	.30*	--
3) 50-item Cook-Medley Hostility scale	.10	.33*	--
4) Marlowe-Crowne Social Desirability	-.16	-.11	-.40*
5) DS16 - Negative Affectivity	.18	-.02	.33*
6) Trait Anger Reaction	-.02	.13	.36*

*Significant at $p < .05$ using Holm's Sequential Method of Bonferroni Type 1 error correction.

Testing the 'Suitability' of Covariates

Correlation between Covariates and Independent Variables. As discussed in Chapter 3, the interpretation of the results would be less ambiguous in ANCOVA if there were no significant associations between the independent variables and the covariate. The covariates used for the three models were the same: BSA and Baseline of the respective cardiovascular dependent variable. The 6 cardiovascular Dependent Variables were: Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Mean Arterial Pressure (MAP), Heart Rate (HR), Cardiac Output and Total Peripheral Resistance. BSA was a covariate because CO and TPR were frequently divided by it to form Cardiac Output Index and Total Peripheral Index to control for inter-individual differences (Sherwood et al., 1990a; Steptoe et al., 2003). However, there was no need to do so if (i) there was no confound between BSA and the independent variable, and (ii) BSA did not predict cardiovascular reactivity. The following tests the relationship between the independent variables and the covariates.

12-item Cynicism. Regression was done using the following predictors for BSA and each of the baseline physiological dependent variables:

- 1) Cynicism
- 2) Cynicism²
- 3) Cynicism³

There were no significant effects predicted by any of the predictors in the 12-item Cynicism model. 12-item Cynicism linear term was not significant for all the covariates, SBP Baseline, $F(1, 55) = .54, p = .47, \eta^2 = .01$, DBP Baseline, $F(1, 55) = .70, p = .41, \eta^2 = .01$, MAP Baseline, $F(1, 55) = .92, p = .34, \eta^2 = .02$, HR Baseline, $F(1, 55) = .004, p = .95, \eta^2 = .07 \times 10^{-3}$, CO Baseline, $F(1, 55) = .85, p = .36, \eta^2 = .02$, TPR Baseline, $F(1, 55) = .18, p = .68, \eta^2 = .003$, and BSA, $F(1, 55) = 1.68, p = .20, \eta^2 = .03$. The quadratic and cubic terms for Cynicism were mostly not significant, p 's range from .20 to 1.00. The exception is BSA, the quadratic term for Cynicism was of borderline significance, $F(1, 55) = 3.82, p = .06, \eta^2 = .07$. Cynicism score greater than ± 4 (1.85SD) tended to have higher BSA (Figure 4.7).

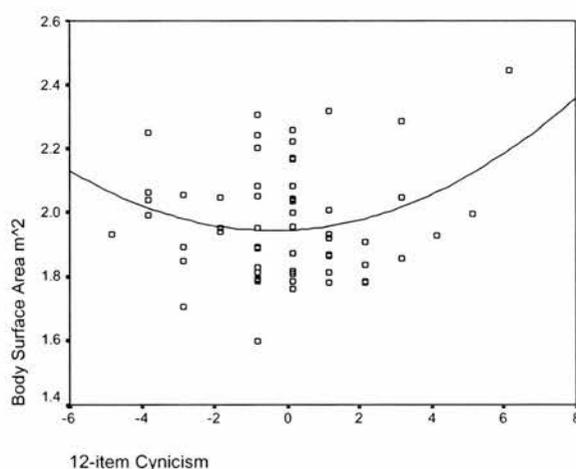


Figure 4.7 Body Surface Area as a function of 12-item Cynicism.

GNAT Cynicism. The model used for 12-item Cynicism was also used here. The linear term for GNAT Cynicism was not significant, SBP

Baseline, $F(1, 55) = .003, p = .96, \eta^2 = .05 \times 10^{-7}$, DBP Baseline, $F(1, 55) = .03, p = .87, \eta^2 = .001$, MAP Baseline, $F(1, 55) = .004, p = .95, \eta^2 = .06 \times 10^{-7}$, HR Baseline, $F(1, 55) = .002, p = .96, \eta^2 = .04 \times 10^{-7}$, CO Baseline, $F(1, 55) = 2.53, p = .12, \eta^2 = .04$, TPR Baseline, $F(1, 55) = .71, p = .40, \eta^2 = .01$, and BSA, $F(1, 55) = 2.71, p = .11, \eta^2 = .05$. The quadratic and cubic terms of GNAT Cynicism were not significant for all the covariates, p 's .22 to .99.

Cynicism and Pseudo Cynicism Factors. These two were analysed together in the following model:

- 1) Cynicism Factor
- 2) Pseudo Cynicism Factor
- 3) Cynicism Factor²
- 4) Pseudo Cynicism Factor²
- 5) Cynicism Factor³
- 6) Pseudo Cynicism Factor³

The interaction between Cynicism and Pseudo Cynicism was not entered into the model because it did not make conceptual sense; both Cynicism and Pseudo Cynicism cover the 4 aspects of Cynicism assessment, their interaction would be conceptually meaningless.

The linear term for Cynicism Factor was not significant, SBP Baseline, $F(1, 55) = .29, p = .59, \eta^2 = .006$, DBP Baseline, $F(1, 55) = .07, p = .80, \eta^2 = .001$, MAP Baseline, $F(1, 55) = .22, p = .64, \eta^2 = .004$, HR Baseline, $F(1, 55) = .02, p = .88, \eta^2 = .05 \times 10^{-2}$, CO Baseline, $F(1, 55) = 2.82, p = .10, \eta^2 = .05$, TPR Baseline, $F(1, 55) = 1.00, p = .32, \eta^2 = .02$,

and BSA, $F(1, 55) = 2.40$, $p = .13$, $\eta^2 = .04$. The quadratic and cubic terms for Cynicism Factor were not significant, p 's range from .18 to .96.

The linear term for Pseudo Cynicism was also not significant, SBP Baseline, $F(1, 55) = .67$, $p = .42$, $\eta^2 = .01$, DBP Baseline, $F(1, 55) = .38$, $p = .54$, $\eta^2 = .007$, MAP Baseline, $F(1, 55) = .28$, $p = .60$, $\eta^2 = .005$, HR Baseline, $F(1, 55) = .08$, $p = .78$, $\eta^2 = .002$, CO Baseline, $F(1, 55) = .16$, $p = .69$, $\eta^2 = .003$, TPR Baseline, $F(1, 55) = .002$, $p = .97$, $\eta^2 = .03 \times 10^{-5}$, and BSA, $F(1, 55) = .66$, $p = .42$, $\eta^2 = .01$. The quadratic term for Pseudo Cynicism was mostly not significant, p 's range from .07 to .96. The exception was the quadratic term for BSA, $F(1, 55) = 3.90$, $p = .05$, $\eta^2 = .07$ (Figure 4.8). Pseudo Cynicism (high scores on the Pseudo Cynicism Factor) and Repressed Hostility (low scores on the Pseudo Cynicism Factor) were associated with higher BSA. The cubic term for CO was also borderline significant, CO, $F(1, 55) = 3.31$, $p = .06$, $\eta^2 = .07$ (Figure 4.9). The pattern showed that participants who have high ($> +2SD$) and low ($-1.5SD$) scores on Pseudo Cynicism Factor tend to have high Baseline CO.

To summarise, Baselines of SBP, DBP, MAP, HR, CO and TPR were not related to 12-item Cynicism, GNAT Cynicism or Cynicism Factor. However, the quadratic term was significant for Pseudo Cynicism Factor on BSA. The cubic term for the Pseudo Cynicism Factor on CO was also significant. Thus the covariates were not related to any of the Cynicism variables but some significant nonlinear effects were found for the Pseudo Cynicism Factor.

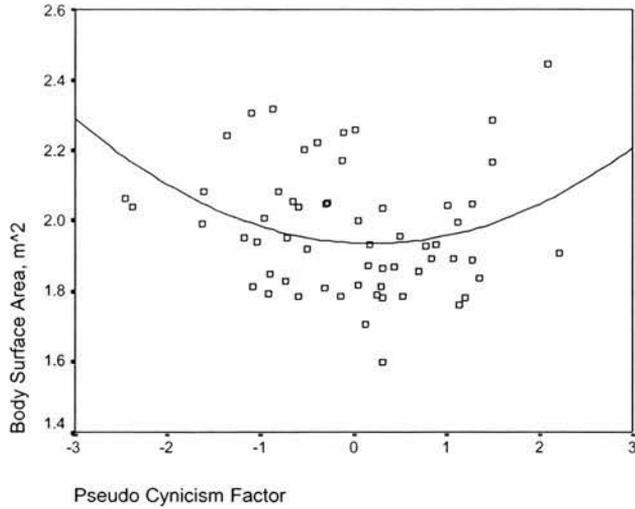


Figure 4.8 Quadratic effect of Pseudo Cynicism Factor on Body Surface Area

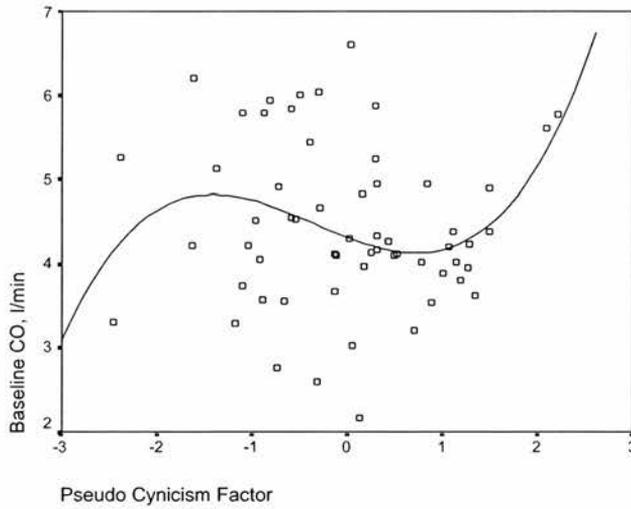


Figure 4.9 Cubic term for Pseudo Cynicism Factor on Baseline Cardiac Output (CO)

Operationalisation of Reactivity. This has been discussed in Chapter 3. ANCOVA was used to operationalised reactivity in this chapter as well to enable comparisons of the results between Study 2 and 3.

Homogeneity of Regression Coefficients. In this section, the assumption for the homogeneity of regression coefficients for ANCOVA was tested. The following model was used to test for the homogeneity of regression coefficients using the respective cardiovascular values obtained in the Task Control conditions as dependent variables:

Between subject model

- 1) Covariate
- 2) Cynicism
- 3) Cynicism²
- 4) Cynicism³
- 5) Cynicism x Covariate
- 6) Cynicism² x Covariate
- 7) Cynicism³ x Covariate

Any significant interactions involving the covariate were retained in the model. This was to derive an 'covariate interaction-free' error term for the computation of the F ratio (Rogosa, 1980). Likewise, any non-significant interactions involving the covariate were removed. The exception to this rule to when a higher order interaction was significant (e.g., Cynicism² x Covariate), then the lower order effects would be retained (i.e., Covariate, Cynicism, Cynicism², Cynicism x Covariate).

12-item Cynicism. The Baselines of the six cardiovascular dependent variables were analysed first followed by BSA.

For SBP, DBP, MAP, HR, CO and TPR, Baseline main effect was found to be significant and was retained in the final regression model²⁰:

SBP Baseline main effect, $F(1, 56) = 27.70$, $p = .02 \times 10^{-8}$, $\eta^2 = .33$, DBP

Baseline main effect, $F(1, 56) = 29.49$, $p = .01 \times 10^{-8}$, $\eta^2 = .35$, MAP

Baseline main effect, $F(1, 56) = 36.17$, $p = .01 \times 10^{-9}$, $\eta^2 = .39$, HR

Baseline main effect, $F(1, 55) = 84.13$, $p = .01 \times 10^{-14}$, $\eta^2 = .58$, CO

Baseline main effect, $F(1, 52) = 58.62$, $p = .04 \times 10^{-12}$, $\eta^2 = .47$, and TPR

Baseline main effect, $F(1, 56) = 27.38$, $p = .03 \times 10^{-8}$, $\eta^2 = .33$. For CO, a

significant Cynicism² x CO Baseline interaction was found, $F(1, 52) =$

5.33, $p = .02$, $\eta^2 = .04$ (Figure 4.10).

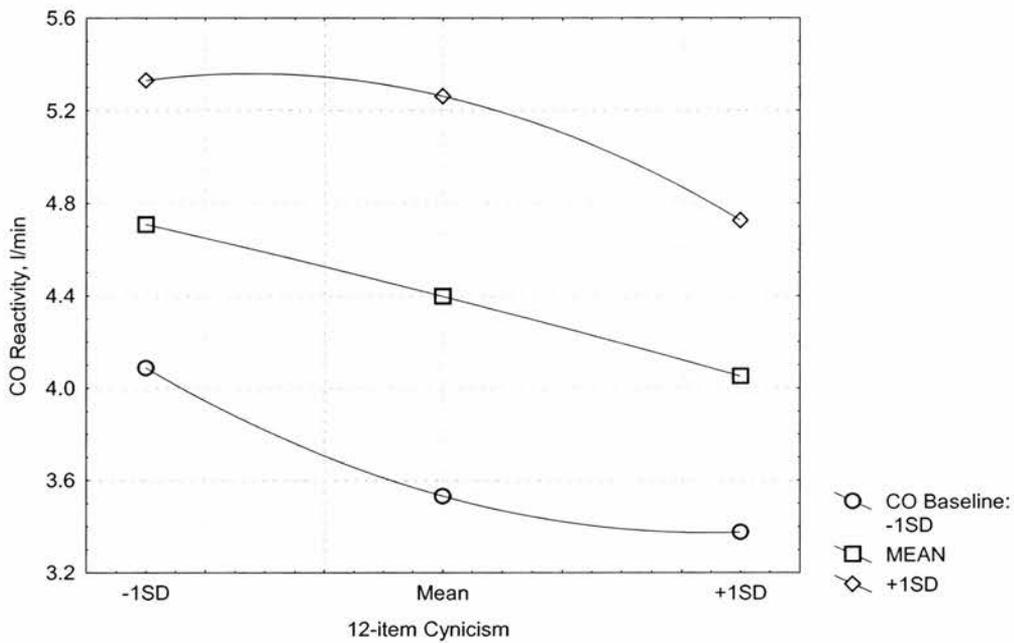


Figure 4.10 Cardiac Output (CO) Baseline as a quadratic function of 12-item Cynicism

Overall, the Baseline main effects were significant for all of the dependent variables as well. The next paragraph details the testing of BSA as a covariate.

²⁰ Discrepancies in the degrees of freedom among the different dependent variables were due to the inclusion of significant linear, quadratic and/or cubic terms of Cynicism.

For SBP, none of the effects involving BSA were significant, p 's .37 to .71. The absence of significant effects involving BSA was found for DBP as well, p 's .31 to .83. BSA and its interactions were also not significant for MAP, p 's .44 to .79. There was also a lack of significant results involving BSA for HR, p 's .15 to .85. For CO, all the interactions involving BSA were not significant and excluded from the regression model, p 's .18 to .69. BSA main effect was significant, $F(1, 51) = 7.11$, $p = .01$, $\eta^2 = .11$. For CO, the following between subjects model was tested for the two covariates (CO Baseline and BSA) to predict CO reactivity during the three Task Control conditions:

Between Subject model for CO

- 1) CO Baseline
- 2) BSA
- 3) Cynicism
- 4) Cynicism²
- 5) Cynicism³

When CO Baseline was entered into the regression model, BSA main effect become non-significant, $F(1, 53) = 1.61$, $p = .21$, $\eta^2 = .01$ while CO Baseline remained significant, $F(1, 53) = 54.68$, $p = .01 \times 10^{-11}$, $\eta^2 = .42$. This indicated that the effect of BSA on CO reactivity during Synwork was largely related to CO Baseline differences. The results suggested that with CO Baseline as a covariate, BSA became superfluous. Therefore, BSA was not entered as a covariate for CO reactivity.

For TPR, BSA main effect was significant, $F(1, 54) = 4.45$, $p = .04$, $\eta^2 = .07$. As for CO, the following model was tested with TPR Baseline and BSA as covariates for TPR reactivity:

Between Subject model

- 1) TPR Baseline
- 2) BSA
- 3) Cynicism
- 4) Cynicism²
- 5) Cynicism³

When TPR Baseline was entered into the regression model, BSA was no longer significant, $F(1, 53) = 1.16, p = .29, \eta^2 = .02$, while TPR Baseline remained significant, $F(1, 53) = 28.10, p = .02 \times 10^{-8}, \eta^2 = .32$. The pattern of results indicated that BSA's prediction of TPR reactivity was largely associated with between subject TPR Baseline differences. With the inclusion of TPR Baseline as a covariate, BSA became redundant as a covariate for TPR reactivity. Hence BSA was not entered for TPR reactivity as well.

GNAT Cynicism. The Baseline main effects of all the dependent variables were found to be significant, and will not be reported here, as the results would be identical. Only the results for the GNAT Cynicism (linear, quadratic and cubic terms) x Baseline interactions will be reported. For SBP Baseline, all interactions involving Cynicism and the SBP Baseline were not significant, p 's .37 to .94. For DBP, the Cynicism x DBP Baseline interactions were not significant, p 's .17 to .99. For MAP, Cynicism x MAP Baseline interactions were also not significant, p 's .19 to .93. For HR, none of the interactions involving HR Baseline were significant, p 's .14 to .71. For CO, interactions involving Cynicism and CO

Baseline were also not significant, p 's .40 to .87. For TPR, Cynicism x TPR Baseline interactions were not significant, p 's .18 to .89.

As BSA was tested as a covariate for the 12-item Cynicism model and found not to be a significant covariate when the Baseline main effects were entered into the regression models first, BSA was not considered as a covariate here.

Cynicism and Pseudo Cynicism Factors. These two orthogonal factors obtained from the factor analysis were analysed together in the following model:

Between subject model

- 1) Covariate
- 2) Cynicism
- 3) Pseudo Cynicism
- 4) Cynicism²
- 5) Pseudo Cynicism²
- 6) Cynicism³
- 7) Pseudo Cynicism³
- 8) Cynicism x Covariate
- 9) Pseudo Cynicism x Covariate
- 10) Cynicism² x Covariate
- 11) Pseudo Cynicism² x Covariate
- 12) Cynicism³ x Covariate
- 13) Pseudo Cynicism³ x Covariate

Baseline main effects were identical to that reported for the 12-item Cynicism scale and will not be reported here. For SBP, Pseudo Cynicism³ x SBP Baseline was significant, $F(1, 50) = 6.84$, $p = .01$, $\eta^2 = .07$. For Pseudo Cynicism scores below -2SD from the mean, high SBP

Baseline was associated with higher SBP reactivity during Synwork while low SBP Baseline was associated with low SBP reactivity. This pattern

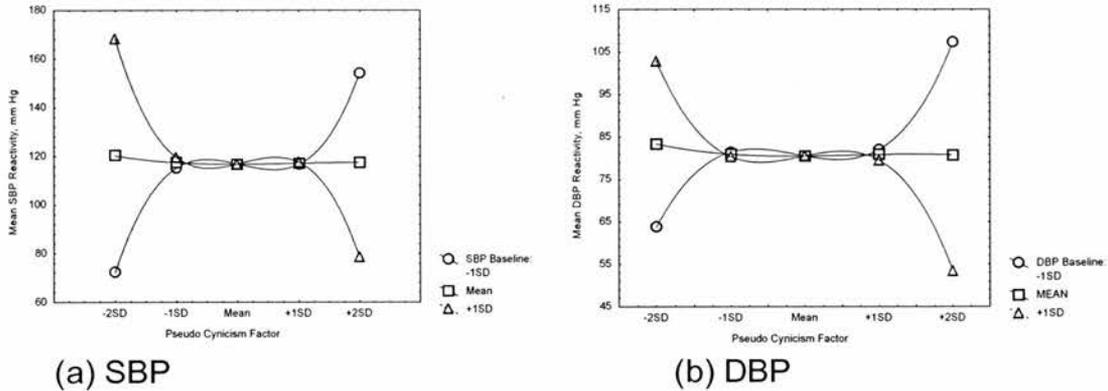


Figure 4.11 Systolic Blood Pressure (SBP) and Diastolic Blood Pressure (DBP) reactivity as a function of Pseudo Cynicism³ and Baseline blood pressure

was reversed for Pseudo Cynicism scores above +2SD (figure 4.11a).

For DBP, a Pseudo Cynicism³ x DBP Baseline interaction was of borderline significance and retained in the model, $F(1, 50) = 3.69, p = .06, \eta^2 = .04$. The pattern for this interaction was similar to that found for SBP (figure 4.11b). For MAP, the Pseudo Cynicism³ x MAP Baseline was not significant, $F(1, 45) = 3.50, p = .07, \eta^2 = .04$. None of the other interactions involving MAP Baseline were significant, p 's .29 to .87.

For HR, all interactions involving HR Baseline were not significant, p 's .09 to .92, and were removed from the regression model. For CO and TPR, all interactions involving the Baseline were also not significant and removed, CO, p 's .23 to .99, and TPR, p 's .10 to .56.

BSA as a covariate was tested in the 12-item Cynicism model and will not be repeated here. Appendix 1 lists the final regression models used after removing the terms that were not significant.

Control Variables

Like Study 2, two forms of control were analysed in this chapter - task control and perceived control. Task Control had three levels (Normal, Variable and More Variable Pauses) that were counterbalanced. A one-way repeated measures ANOVA found Task score differences by Task Control condition, $F(1.94, 112.32) = 42.44, p = .04 \times 10^{-16}, \eta^2 = .42$. Perceived Control was also significantly different by Task Control, $F(1.96, 113.82) = 71.46, p = .01 \times 10^{-15}, \eta^2 = .55$. Simple effects analysis (Table 4.11) showed that Task Scores and Perceived Control during Variable and More Variable Pauses Task Control conditions were significantly different from Normal Task Control. But Task Scores and Perceived Control during Variable and More Variable Pauses Task Control conditions were not significantly different from each other.

Table 4.11 Means (SD) of Synwork Scores, and Perceived Control by Task Control (n = 60)*

	Task Control		
	Normal	Variable Pauses	More Variable Pauses
Synwork Scores	330.75 _a (91.80)	53.62 _b (282.44)	40.45 _b (275.45)
Perceived Control, cm	11.33 _a (2.17)	6.89 _b (3.38)	6.73 _b (3.54)

a, b, c Means with different subscripts are significantly different at $p < .05$ using Bonferroni correction for Type 1 error. Synwork Scores for Variable and More Variable Pauses conditions are not significantly different from '0', $p > .05$.

Main Analyses for Physiological Reactivity

Table 4.12 shows the cardiovascular reactivity for the Baseline and during the three Task Control conditions. As ANCOVA analysis would not indicate whether cardiovascular responses during Synwork was significantly different from Baseline, paired *t*-tests were done. The analyses showed that blood pressure and heart rate increased significantly from Baseline during the three Task Control conditions. The source of this rise in blood pressure in vascular-mediated as seen in the significant increase in TPR (Table 4.12). The same analyses were done for the UNWIST mood checklist as well (Table 4.13). Synwork during the three Task Control conditions elicited significant increases in Anger.

Table 4.12 Means (SD) for the Physiological Data (n = 60)

	Baseline	Normal	Task Control Variable Pauses	More Variable Pauses
SBP, mm Hg	110.46 (13.39)	119.57* (18.05)	121.22* (17.53)	123.31* (16.89)
DBP, mm Hg	74.09 (11.01)	82.34* (13.21)	84.12* (14.49)	85.23* (14.49)
MAP, mm Hg	86.40 (10.90)	94.86* (14.58)	96.74* (14.64)	97.97* (15.17)
HR, bpm	70.17 (8.66)	74.97* (11.19)	74.55* (9.23)	74.39* (8.71)
CO, l/min	4.41 (1.01)	4.49 (1.23)	4.41 (1.21)	4.42 (1.11)
TPR, dyne-sec*cm ⁻⁵	1274.90 (474.00)	1390.16* (599.43)	1453.38* (618.35)	1474.70* (661.25)

*Significantly different from Baseline ($p < .05$) using paired samples *t*-test with Holm's Sequential method of Bonferroni Type 1 error correction.

Table 4.13 Mean (SD) of UNWIST Hedonic Tone Mood Ratings

	Baseline	Task Control		
		Normal	Variable Pauses	More Variable Pauses
Hedonic Tone				
Hedonic items, cm	8.20 (1.88)	8.53 (2.00)	6.91* (2.00)	6.92* (2.00)
Anger items, cm	1.58 (1.65)	2.21* (2.01)	4.08* (2.62)	4.19* (2.55)

*Significantly different from Baseline ($p < .05$) using paired samples t-test with Holm's Sequential method of Bonferroni Type 1 error correction.

The aim of manipulating the controllability of Synwork was to increase non-interpersonal State Anger. Appraisal theories of emotions point out that uncontrollable events obstructing goal attainment can increase State Anger (Berkowitz, 2000). This was investigated via correlating Perceived Control with the various mood ratings during the three Task Control conditions. Perceived Control was significantly negatively associated with State Anger when Task Control was Variable or More Variable Pauses but not when Task Control was Normal. During Normal Task Control, Perceived Control was significantly above the midpoint of the scale (7.5cm, Table 4.11) indicating that Perceived Control was above average. When Perceived Control fell below the midpoint of the scale during the Variable and More Variable Pauses Task Control conditions, it became significantly associated with State Anger. This lends support for the appraisal theory of emotion that low perceived control is associated with increased State Anger. Perceived Control and

the Hedonic items reveal the reverse pattern to that obtained for the Anger items (Table 4.14).

Table 4.14 Correlations between Perceived Control and State Anger by Task Control condition (n = 59)

	Task Control		
	Normal	Variable Pauses	More Variable Pauses
Hedonic Tone - Hedonic items			
Perceived Control, cm	.13	.31*	.26
Hedonic Tone - Anger items			
Perceived Control, cm	-.10	-.30*	-.31*

*Significant at $p < .05$ using Holm's Sequential method of Bonferroni Type 1 error correction

Hypotheses Testing

Hypothesis 1: Cynicism and cardiovascular Reactivity. Cynicism is positively associated with cardiovascular reactivity - a significant Cynicism main effect. Nonlinear trends between Cynicism and cardiovascular reactivity are also explored. Only significant results for these nonlinear trends will be reported.

12-item Cynicism. For cardiovascular reactivity, the linear term for Cynicism was not significant, SBP, $F(1, 54) = .39, p = .54, \eta^2 = .004$, partial $\eta^2 = .007$, DBP, $F(1, 56) = .32, p = .58, \eta^2 = .003$, partial $\eta^2 = .006$,

MAP, $F(1, 54) = .43, p = .51, \eta^2 = .004, \text{partial } \eta^2 = .007$, HR, $F(1, 56) = .23, p = .63, \eta^2 = .002, \text{partial } \eta^2 = .004$, CO, $F(1, 55) = 1.77, p = .19, \eta^2 = .01, \text{partial } \eta^2 = .03$, and TPR, $F(1, 54) = .07, p = .80, \eta^2 = .03 \times 10^{-2}, \text{partial } \eta^2 = .001$. A Cynicism quadratic term for HR was found to be significant, $F(1, 55) = 5.51, p = .02, \eta^2 = .04, \text{partial } \eta^2 = .09$. Figure 4.12 shows that HR reactivity is higher among participants with 12-item Cynicism scores at ± 4 ($\pm 1.80\text{SD}$) or greater from the mean. The cubic term for Cynicism was also significant for CO, $F(1, 52) = 4.34, p = .04, \eta^2 = .04, \text{partial } \eta^2 = .07$ (Figure 4.13). For participants scoring within 1SD for the 12-item Cynicism scale, there was little association between Cynicism and CO reactivity. Participants scoring ± 4 ($\pm 1.80\text{SD}$) or greater than the mean had the most CO and TPR reactivity. Extremely low Cynicism was associated with decreased CO while extremely high Cynicism was associated with an increased CO reactivity.

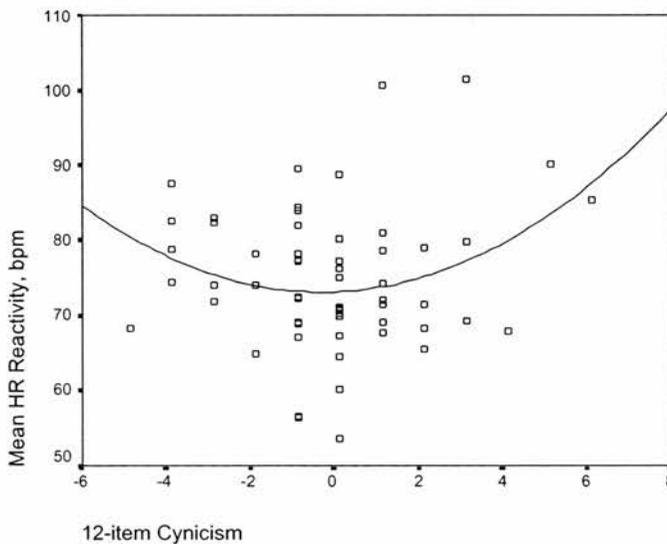


Figure 4.12 Quadratic trend between 12-item Cynicism and Heart Rate Reactivity.

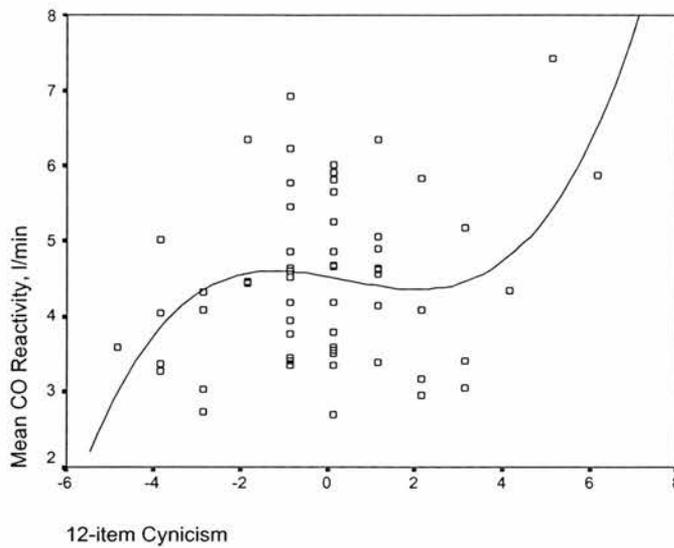


Figure 4.13 Cubic trend between 12-item Cynicism and Cardiac Output (CO) reactivity

Overall, the results for the linear term for the 12-item Cynicism scale replicated that obtained in Study 2 (Chapter 3). There were no significant results for the linear term of Cynicism for cardiovascular reactivity. Like Study 2, effect sizes were typically less than .01. Though there were no significant results for Cynicism, the underlying hemodynamic process regulating blood pressure for CO were different for extremely low ($-1.8SD$) and extremely high ($+1.8SD$) scores on the 12-item Cynicism scale. For individuals scoring within $\pm 1.5SD$ from the mean, there was little association between Cynicism and hemodynamic cardiovascular stress reactivity.

GNAT Cynicism. The same hypothesis was explored when Cynicism was operationalised as GNAT Cynicism. Linear Cynicism main

effects were not significant; SBP, $F(1, 57) = 1.10, p = .30, \eta^2 = .01$, partial $\eta^2 = .03$, DBP, $F(1, 56) = .31, p = .58, \eta^2 = .004$, partial $\eta^2 = .006$, MAP, $F(1, 56) = .13, p = .72, \eta^2 = .001$, partial $\eta^2 = .002$, HR, $F(1, 56) = .01, p = .92, \eta^2 = .08 \times 10^{-3}$, partial $\eta^2 = .02 \times 10^{-2}$, CO, $F(1, 56) = .15, p = .70, \eta^2 = .001$,

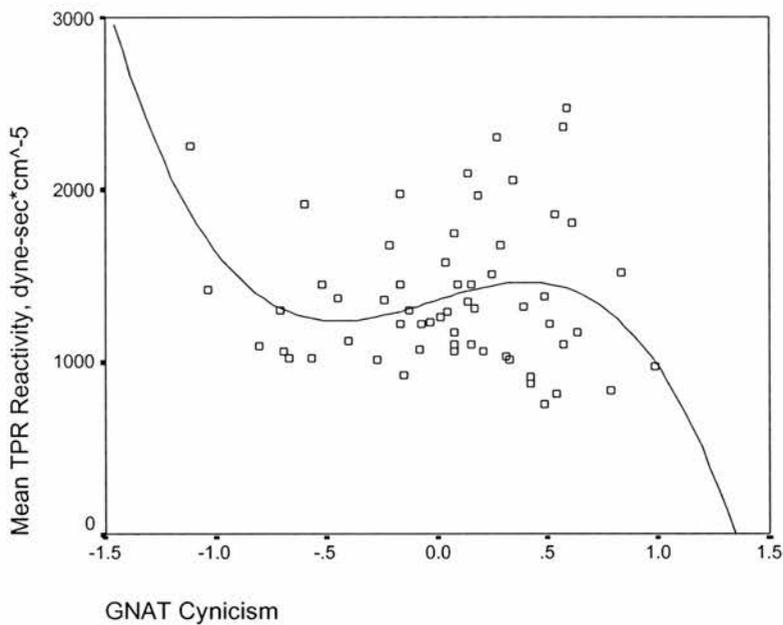


Figure 4.14 GNAT Cynicism³ main effect for average Total Peripheral Resistance (TPR) reactivity.

partial $\eta^2 = .003$, and TPR, $F(1, 53) = .20, p = .66, \eta^2 = .002$, partial $\eta^2 = .003$. A Cynicism³ main effect was found to be significant for TPR, $F(1, 54) = 5.14, p = .03, \eta^2 = .06$, partial $\eta^2 = .08$. GNAT Cynicism scores less than -2SD (-.92) tended to have greater TPR reactivity and Cynicism scores greater than +2SD tended to have slightly less TPR reactivity (figure 4.14).

Cynicism and Pseudo Cynicism Factors. Linear term for the Cynicism Factor was not significant: SBP, $F(1, 50) = .04, p = .83, \eta^2 = .04 \times 10^{-2}$, partial $\eta^2 = .001$, DBP, $F(1, 50) = .04, p = .84, \eta^2 = .04 \times 10^{-2}$, partial $\eta^2 = .001$, MAP, $F(1, 54) = .01, p = .92, \eta^2 = .01 \times 10^{-2}$, partial $\eta^2 = .001$, HR, $F(1, 52) = .24, p = .63, \eta^2 = .002$, partial $\eta^2 = .004$, CO, $F(1, 53) = .59, p = .45, \eta^2 = .005$, partial $\eta^2 = .01$, and TPR, $F(1, 51) = .13, p =$

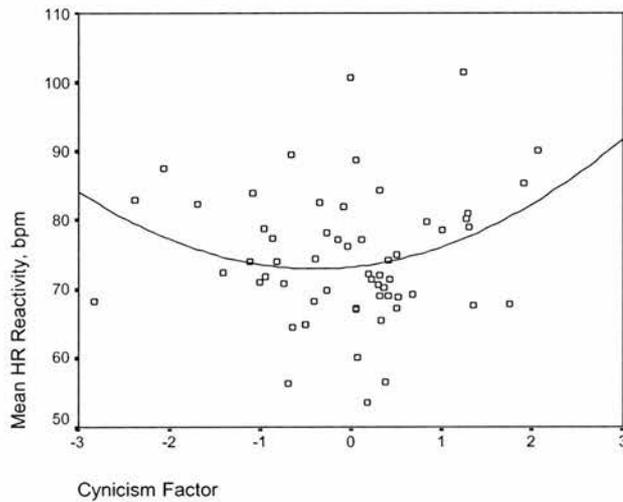
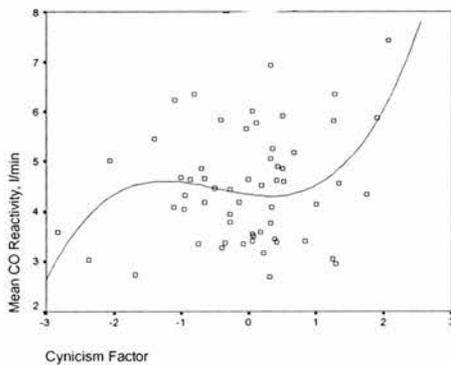
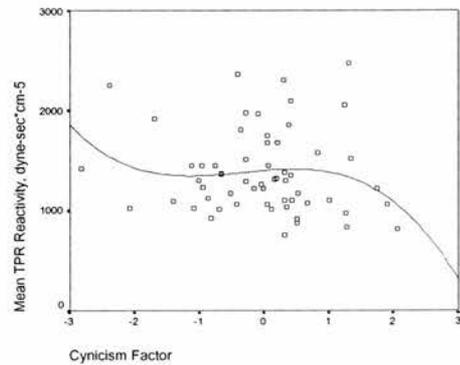


Figure 4.15 Cynicism² Factor main effect on Heart Rate (HR) reactivity



(a) CO



(b) TPR

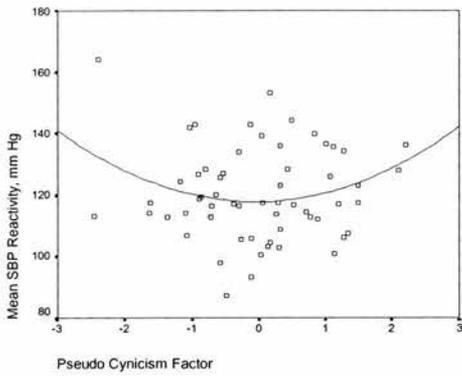
Figure 4.16 Cynicism³ Factor main effect on Cardiac Output (CO) and Total Peripheral Resistance (TPR) reactivity

.72, $\eta^2 = .001$, partial $\eta^2 = .002$.

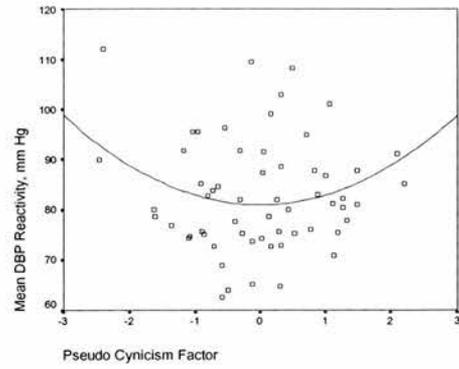
Some significant effects were found for the nonlinear terms of the Cynicism Factor. A Cynicism² was of borderline significance for HR, $F(1, 52) = 3.85$, $p = .06$, $\eta^2 = .03$, partial $\eta^2 = .06$. Cynicism Factor scores greater than $\pm 2SD$ ($\pm .92$) tended to have greater HR reactivity (figure 4.15). Extremely low ($> -2SD$) Cynicism scores were associated with a passive coping physiological response (reduced CO reactivity and increased TPR reactivity) while extremely high Cynicism scores ($> +1.5SD$) were associated with an active coping physiological response (increased CO and decreased TPR reactivity; figure 4.16).

Next, the results for the Pseudo Cynicism Factor are reported. Linear Pseudo Cynicism Factor main effect was not significant; SBP, $F(1, 50) = .51$, $p = .48$, $\eta^2 = .005$, partial $\eta^2 = .01$, DBP, $F(1, 50) = .53$, $p = .47$, $\eta^2 = .005$, partial $\eta^2 = .01$, MAP, $F(1, 54) = .23$, $p = .63$, $\eta^2 = .002$, partial $\eta^2 = .004$, HR, $F(1, 52) = .23$, $p = .63$, $\eta^2 = .002$, partial $\eta^2 = .004$, CO, $F(1, 53) = 2.85$, $p = .10$, $\eta^2 = .02$, partial $\eta^2 = .04$, and TPR, $F(1, 51) = 1.50$, $p = .23$, $\eta^2 = .02$, partial $\eta^2 = .02$. The Pseudo Cynicism² main effect was significant for SBP, $F(1, 50) = 7.69$, $p = .008$, $\eta^2 = .08$, partial $\eta^2 = .13$, DBP, $F(1, 50) = 6.42$, $p = .01$, $\eta^2 = .07$, partial $\eta^2 = .11$, and MAP, $F(1, 54) = 5.41$, $p = .02$, $\eta^2 = .06$, partial $\eta^2 = .09$. The pattern of results for SBP, DBP and MAP was similar - participants who have Pseudo Cynicism scores less than $-1SD$ (Repressed Cynicism) had greater blood pressure reactivity. Participants who have Pseudo Cynicism scores

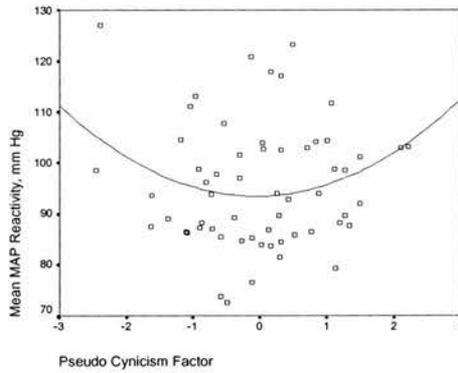
greater than +1SD (Pseudo Cynicism) also tend to have greater blood pressure reactivity (Figure 4.17).



(a) SBP



(b) DBP



(c) MAP

Figure 4.17 Pseudo Cynicism² main effect on Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), and Mean Arterial Blood Pressure (MAP) reactivity

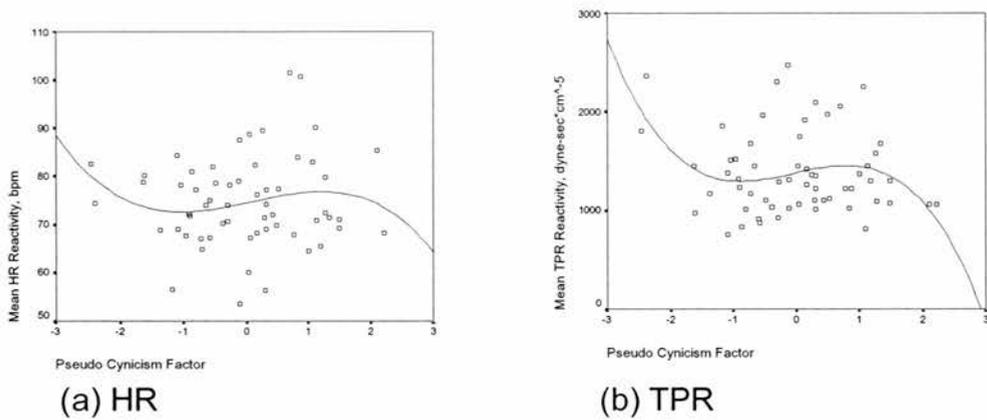


Figure 4.18 Pseudo Cynicism³ main effect on Heart Rate (HR) and Total Peripheral Resistance (TPR) reactivity

Pseudo Cynicism³ main effect was also significant for HR, $F(1, 52) = 4.33$, $p = .04$, $\eta^2 = .03$, partial $\eta^2 = .07$, and TPR, $F(1, 51) = 5.19$, $p = .03$, $\eta^2 = .06$, partial $\eta^2 = .08$. Pseudo Cynicism scores $>-2SD$ from the mean (Repressed Hostile) had greater HR and TPR reactivity (figure 4.18).

Overall, Hypothesis 1 was not supported regardless of how Cynicism was operationalised for the linear term of Cynicism - high Cynicism was not associated with greater cardiovascular reactivity. However, a number of nonlinear Cynicism main effects were found. The effects was present for Cynicism scores greater than $\pm 1SD$ from the mean. For the 12-item Cynicism and Cynicism Factor, scores greater than $\pm 1SD$ from the mean had greater HR reactivity. Participants with 12-item Cynicism and Cynicism Factor scores $>+1SD$ had greater CO reactivity while scores for these two Cynicism assessments below $-1SD$ had less CO reactivity. Both GNAT Cynicism³ and Pseudo Cynicism³

predicted similar main effects for TPR reactivity. Those scoring less than -1SD from mean had greater TPR reactivity while scores greater than +1SD had less TPR reactivity. It is noted that no matter how Cynicism was operationalised, the underlying hemodynamic processes of blood pressure reactivity (CO and TPR) patterns predicted by nonlinear Cynicism terms were complimentary. All scores less than -1SD had passive physiological responses (reduced CO and increased TPR) and scores greater than +1SD had active coping physiological responses (increased CO and decreased TPR). The results obtained for reactivity for the Cynicism and Pseudo Cynicism Factors were more coherent than for the other two method of Cynicism assessment. For Cynicism and Pseudo Cynicism Factors, the hemodynamic processes reflected the pattern of results obtained for blood pressure reactivity. In contrast, for 12-item Cynicism, a significant CO effect and a non-significant TPR effect, suggesting hemodynamic dysregulation of blood pressure reactivity, were not accompanied by significant blood pressure effects. For GNAT Cynicism, significant TPR effect was also not accompanied by significant blood pressure effects. On the other hand for Cynicism Factor, a significant CO and TPR changes regulated blood pressure sufficiently so that no significant results for blood pressure reactivity were present. For Pseudo Cynicism Factor, blood pressure effects were TPR-mediated.

Hypothesis 2: Task Control differences for Cardiovascular Reactivity

An examination of the blood pressure and HR reactivity during the three levels of Task Control would help in the interpretation of CO and TPR

reactivity. Results obtained using the GNAT Cynicism, Cynicism Factor and Pseudo Cynicism Factor had identical effect sizes for Task Control²¹. Task Control main effect was significant for SBP, $F(1.83, 104.02) = 4.02$, $p = .048$, $\eta^2 = .07$, DBP, $F(1.83, 104.42) = 5.79$, $p = .02$, $\eta^2 = .09$, and MAP, $F(1.88, 107.39) = 5.30$, $p = .02$, $\eta^2 = .09$. Task Control main effect was not significant for HR, $F(1.43, 81.28) = .51$, $p = .48$, $\eta^2 = .009$. Table 4.15 shows that the less controllable Synwork is, the higher the blood pressure reaction to the task.

Table 4.15 Mean (SD) of SBP, DBP, MAP, CO and TPR by Task Control condition (n = 59)*

	Task Control		
	Normal	Variable Pauses	More Variable Pauses
SBP, mm Hg	119.57 _a (18.05)	121.22 _{a,b} (17.53)	123.31 _b (16.89)
DBP, mm Hg	82.34 _a (13.21)	84.12 _{a,b} (14.49)	85.23 _b (14.49)
MAP, mm Hg	94.86 _a (14.58)	96.74 _b (14.64)	97.97 _b (15.17)
CO, l/min	4.49 (1.23)	4.41 (1.21)	4.42 (1.11)
TPR, dyne-sec*cm ⁻⁵	1390.16 _a (599.43)	1453.38 _b (618.35)	1474.70 _b (661.25)

a, b, c Means with different subscripts are significantly different ($p < .05$) within rows using paired samples t-test with Holm's Sequential method of Bonferroni Type 1 error correction.

²¹ Task Control and its interactions belong to the within subject model and the within subject model does not have covariates. Thus, partial η^2 (as presented for the between subject model) will not be calculated for the within subjects model.

2a) Normal Task Control is associated with increased CO reactivity and decreased TPR reactivity (active coping physiological response) while Variable and More Variable Pauses Task Control is associated with decreased CO and increased TPR (passive coping physiological response).

Task Control main effect was significant for TPR, $F(1.86, 105.91) = 5.16, p = .03, \eta^2 = .08$ but not for CO, $F(1.80, 102.83) = .93, p = .34, \eta^2 = .02$. Table 4.15 shows that the rise in blood pressure with decreasing Task Control was associated with a rise in TPR. TPR reactivity for Task Control main effect for this study has the same pattern and effect size as for study 2. In contrast, CO reactivity was significant in Study 2 and

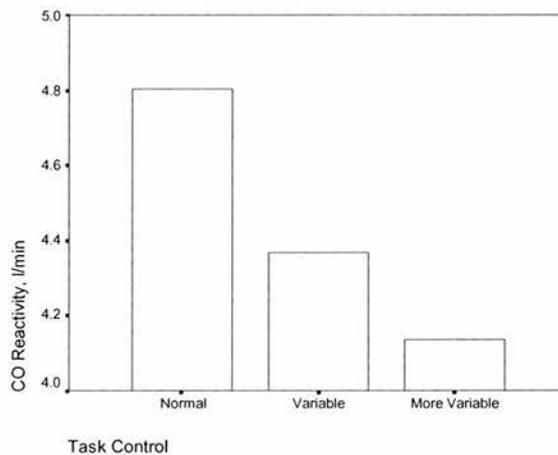


Figure 4.19 Cardiac Output (CO) reactivity for the Normal-Variable-More Variable Pauses condition

not significant in this study. An examination of the Task Control condition in this study that was the same as study 2 (Normal-Variable-More Variable Pauses), revealed that the patterns of CO reactivity was

significant for Task Control main effect, $F(1.84, 35.02) = 4.81, p = .02, \eta^2 = .20$. The pattern of CO reactivity obtained for the Normal-Variable-More Variable Pauses condition in this study was similar to that obtained in Study 2 (figure 4.19). Hence, the significant Task Control main effect for CO in Study 2 was due to the fixed order for Task Control conditions, which was also replicated in this study.

Hence Hypothesis 2a was partly supported for Task Control - Task Control was associated with an active coping physiological response (decreased TPR reactivity) while low Task Control was associated with a passive coping physiological response (increased TPR reactivity). No significant changes in CO reactivity were found.

Perceived Control was given by the participants after every Synwork session. This rating was matched to the corresponding dependent variable (e.g., SBP reactivity). For instance, Perceived Control given after a Synwork with Variable Pauses was matched to the corresponding SBP reactivity during Synwork Variable Pauses by participant. This form of Perceived Control was entered as a continuous within subject variable (Perceived Control_{wtw}). The average of Perceived Control for each participant over the three Task Control condition was also taken and this represents the between subjects Perceived Control (Perceived Control_{btw}). Only a borderline significant effect was found for Perceived Control_{btw} for CO reactivity, $F(1, 50) = 4.07, p = .05, \eta^2 = .03, \text{partial } \eta^2 = .06$. The slope for this effect indicated a positive association between Perceived Control_{btw} and CO reactivity, $b(SE) = .077$

(.025). Hypothesis 2a is partially supported for Perceived Control_{btw} for CO reactivity (albeit a weak effect) but not for TPR reactivity.

Hypothesis 3: Task Control (Actual Control) and Perceived Control

Task Control and Perceived Control predict common/unique variance for the dependent variables. The results reported in the next paragraph and in Table 4.16 used Perceived Control_{wtm}.

SBP, DBP, MAP and TPR were used to test this hypothesis since Task Control differences were found for these dependent variables. Both between and within subject versions of Perceived Control were centred. Compared to Study 2 (Table 3.13), Perceived Control_{wtm} and Task Control did not predict common variance. The amount of variance explained by Perceived Control_{wtm} is much smaller than Study 2. When Task Control was entered into the regression model first, the effect size for Perceived Control was attenuated somewhat but not completely eliminated as in Study 2. Since Perceived Control assessed using a visual analogue scale does not explain common variance with Task Control, its interaction with Cynicism was analysed separately.

Hypothesis 4: Cynicism and Cardiovascular Reactivity moderated by Control.

It was hypothesised that participants scoring high in Cynicism have greater cardiovascular reactivity in situations of low control (actual or perceived) - a significant Cynicism x Control interaction. Perceived Control is analysed first followed by Task Control.

Table 4.16 Effect sizes from the Two Within Subject Models involving Task and Perceived Control_{wtn}

Main Effect	Task Control Entered 1st	Perceived Control Entered 1st
	SBP, mm Hg	
Task Control	$\eta^2 = .072$	$\eta^2 = .005$
Perceived Control	$\eta^2 = .005$	$\eta^2 = .072$
	DBP, mm Hg	
Task Control	$\eta^2 = .095$	$\eta^2 = .010$
Perceived Control	$\eta^2 = .002$	$\eta^2 = .087$
	MAP, mm Hg	
Task Control	$\eta^2 = .090$	$\eta^2 = .006$
Perceived Control	$\eta^2 = .010$	$\eta^2 = .093$
	TPR, dyne-sec*cm ⁻⁵	
Task Control	$\eta^2 = .070$	$\eta^2 = .008$
Perceived Control	$\eta^2 = .005$	$\eta^2 = .067$

Perceived Control & 12-item Cynicism. In order to interpret the effects of Perceived Control and its potential interaction with the 12-item Cynicism scale, Perceived Control was entered as a three level repeated dependent variable corresponding to each Task Control condition. The linear, quadratic and cubic terms for the 12-item Cynicism scale were entered into the regression model as well. This was to investigate whether there are significant associations between Cynicism and

Perceived Control. A lack of significant associations would enable a less ambiguous interpretation of Cynicism x Perceived Control interactions. The results showed that none of the effects involving 12-item Cynicism were significant in predicting Perceived Control, p 's .44 to .78. Cynicism is not significant associated with Perceived Control.

The results for 12-item Cynicism scale revealed that Cynicism was not moderated by Perceived Control (both Perceived Control_{btw} and Perceived Control_{wtn}) for cardiovascular reactivity. For Cynicism x Perceived Control_{btw} interaction: SBP, $F(1, 54) = 1.00, p = .32, \eta^2 = .01$, partial $\eta^2 = .02$, DBP, $F(1, 54) = .35, p = .56, \eta^2 = .01$, partial $\eta^2 = .02$, MAP, $F(1, 52) = .88, p = .35, \eta^2 = .01$, partial $\eta^2 = .02$, HR, $F(1, 54) = 1.48, p = .23, \eta^2 = .01$, partial $\eta^2 = .03$, CO, $F(1, 50) = .92, p = .34, \eta^2 = .007$, partial $\eta^2 = .01$, and TPR, $F(1, 54) = .62, p = .43, \eta^2 = .007$, partial $\eta^2 = .01$. For Cynicism x Perceived Control_{wtn} interaction: SBP, $F(1, 106.02) = .39, p = .53, \eta^2 = .003$, DBP, $F(1, 106.26) = .04, p = .84, \eta^2 = .04 \times 10^{-2}$, MAP, $F(1, 109.27) = .18, p = .67, \eta^2 = .002$, HR, $F(1, 82.71) = .16, p = .69, \eta^2 = .002$, CO, $F(1, 104.63) = .85, p = .36, \eta^2 = .008$, and TPR, $F(1, 107.76) = .43, p = .51, \eta^2 = .004$.

Task Control & 12-item Cynicism. Cynicism x Task Control interaction was not significant for SBP, $F(1.83, 104.20) = .32, p = .57, \eta^2 = .005$, DBP, $F(1.83, 104.42) = .53, p = .47, \eta^2 = .008$, MAP, $F(1.88, 107.39) = .61, p = .44, \eta^2 = .01$, HR, $F(1.43, 81.28) = 1.09, p = .30, \eta^2 = .02$, and CO, $F(1.80, 102.83) = 1.80, p = .18, \eta^2 = .03$.

The Cynicism x Task Control interaction was found to be significant for TPR, $F(1.86, 105.91) = 4.42, p = .04, \eta^2 = .07$. Figure 4.20 shows that participants with low Cynicism had greater TPR reactivity compared to participants with high Cynicism when Task Control was very low. This pattern was opposite of Hypothesis 4, which hypothesised that high (not low) Cynicism was associated with greater cardiovascular reactivity when control was low.

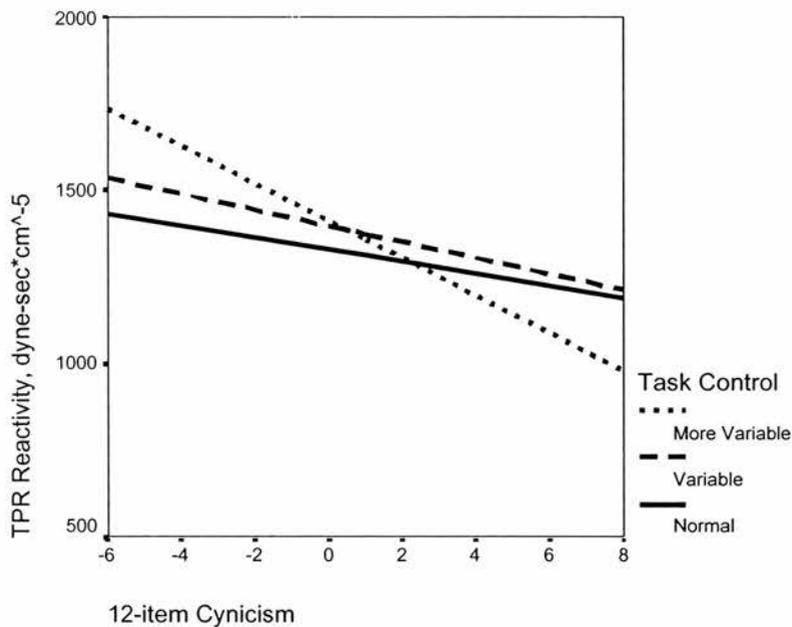


Figure 4.20 12-item Cynicism x Task Control interaction for Total Peripheral Resistance (TPR) reactivity

For 12-item Cynicism, Perceived Control and Task Control did not significantly moderate the Cynicism-Reactivity relationship. A significant Cynicism x Task Control interaction was found but the pattern of this

interaction was the reverse of Hypothesis 4. Therefore, Hypothesis 4 for 12-item Cynicism was not supported.

Perceived Control & GNAT Cynicism. As was done for the 12-item Cynicism, Perceived Control was entered as a 3-level repeated measure dependent variable in order to investigate the associations between GNAT Cynicism and Perceived Control. No significant effects for GNAT Cynicism were found, p 's .53 to .84. GNAT Cynicism was not significantly associated with Perceived Control.

There were no significant Cynicism x Perceived Control_{btw} interactions: for SBP, $F(1, 54) = .77, p = .39, \eta^2 = .009, \text{partial } \eta^2 = .01$, DBP, $F(1, 54) = 1.12, p = .29, \eta^2 = .01, \text{partial } \eta^2 = .02$, MAP, $F(1, 54) = .96, p = .34, \eta^2 = .01, \text{partial } \eta^2 = .02$, HR, $F(1, 54) = .04, p = .85, \eta^2 = .03 \times 10^{-2}, \text{partial } \eta^2 = .07 \times 10^{-2}$, CO, $F(1, 54) = .005, p = .94, \eta^2 = .05 \times 10^{-7}, \text{partial } \eta^2 = .09 \times 10^{-7}$, and TPR, $F(1, 52) = .13, p = .72, \eta^2 = .001, \text{partial } \eta^2 = .002$. Cynicism x Perceived Control_{wtn} interactions were also non-significant: SBP, $F(1, 106.02) = .09, p = .77, \eta^2 = .08 \times 10^{-2}$, DBP, $F(1, 106.26) = .05, p = .82, \eta^2 = .005 \times 10^{-2}$, MAP, $F(1, 109.27) = .42, p = .52, \eta^2 = .004$, HR, $F(1, 82.71) = .03, p = .87, \eta^2 = .03 \times 10^{-2}$, CO, $F(1, 104.63) = .46, p = .50, \eta^2 = .004$, and TPR, $F(1, 107.76) = 1.56, p = .21, \eta^2 = .01$.

Task Control & GNAT Cynicism. There were no significant Cynicism x Task Control interactions: SBP, $F(1.82, 105.44) = 3.42, p = .07, \eta^2 = .05$, DBP, $F(1.91, 108.64) = 2.44, p = .12, \eta^2 = .04$, MAP,

$F(1.83, 104.08) = 2.63, p = .11, \eta^2 = .04$, HR, $F(1.49, 84.70) = .24, p = .63, \eta^2 = .004$, CO, $F(1.81, 102.94) = .28, p = .60, \eta^2 = .005$, and TPR, $F(1.74, 100.69) = 1.04, p = .31, \eta^2 = .02$. Hypothesis 4 was not supported, GNAT Cynicism and cardiovascular reactivity was not moderated by Task Control.

Perceived Control, Cynicism & Pseudo Cynicism Factors.

Cynicism Factor x Perceived Control_{btw} was not significant for cardiovascular reactivity: SBP, $F(1, 47) = 2.74, p = .10, \eta^2 = .03$, partial $\eta^2 = .05$, DBP, $F(1, 47) = 2.11, p = .15, \eta^2 = .02$, partial $\eta^2 = .04$, MAP, $F(1, 51) = .03, p = .86, \eta^2 = .02$, partial $\eta^2 = .03$, HR, $F(1, 48) = .66, p = .42, \eta^2 = .004$, partial $\eta^2 = .01$, CO, $F(1, 46) = .04, p = .84, \eta^2 = .02 \times 10^{-2}$, partial $\eta^2 = .05 \times 10^{-2}$, and TPR, $F(1, 46) = .002, p = .96, \eta^2 = .01 \times 10^{-2}$, partial $\eta^2 = .01 \times 10^{-2}$. Cynicism x Perceived Control_{wth} interaction for cardiovascular reactivity was also not significant: SBP, $F(1, 105.11) = .06 \times 10^{-2}, p = .98, \eta^2 = .08 \times 10^{-7}$, DBP, $F(1, 105.34) = .08 \times 10^{-2}, p = .98, \eta^2 = .08 \times 10^{-7}$, MAP, $F(1, 108.33) = .01 \times 10^{-2}, p = .99, \eta^2 = .01 \times 10^{-2}$, HR, $F(1, 82.00) = .04 \times 10^{-2}, p = .98, \eta^2 = .04 \times 10^{-2}$, CO, $F(1, 103.73) = .05 \times 10^{-2}, p = .98, \eta^2 = .05 \times 10^{-2}$, and TPR, $F(1, 106.84) = .01, p = .90, \eta^2 = .01$.

Pseudo Cynicism Factor x Perceived Control_{btw} interaction was also not significant: SBP, $F(1, 47) = .16, p = .69, \eta^2 = .002$, partial $\eta^2 = .003$, DBP, $F(1, 47) = .04, p = .84, \eta^2 = .04 \times 10^{-2}$, partial $\eta^2 = .07 \times 10^{-2}$, MAP, $F(1, 51) = .22, p = .65, \eta^2 = .002$, partial $\eta^2 = .004$, HR, $F(1, 48) = 2.08, p = .16, \eta^2 = .01$, partial $\eta^2 = .03$, CO, $F(1, 46) = .34, p = .56, \eta^2 =$

.002, partial $\eta^2 = .004$, and TPR, $F(1, 46) = .11, p = .90, \eta^2 = .001$, partial $\eta^2 = .002$. Interactions involving the nonlinear term of Pseudo Cynicism Factor were found for CO and TPR reactivity. For CO and TPR, Pseudo Cynicism³ x Perceived Control_{btw} interaction was significant, $F(1, 46) = 10.07, p = .003, \eta^2 = .07$, partial $\eta^2 = .13$, and TPR, $F(1, 46) = 7.20, p = .01, \eta^2 = .07$, partial $\eta^2 = .10$. The greatest differentiation in terms of the hemodynamic processes underlying blood pressure reactivity were present in Pseudo Cynicism scores greater than $\pm 2SD$ from the mean, while these were no differences for Pseudo Cynicism scores within $\pm 2SD$ (figure 4.21). Extremely low Pseudo Cynicism scores (Repressed Hostility) were associated with a passive coping physiological response

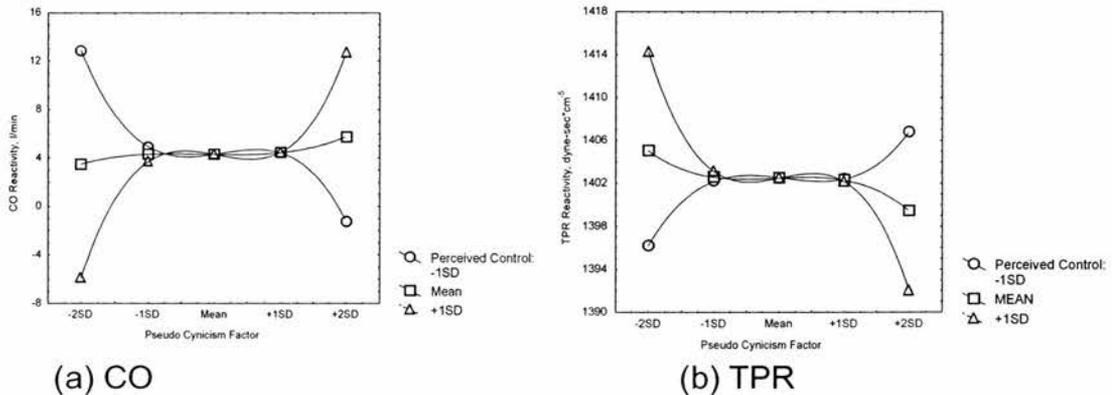


Figure 4.21 Pseudo Cynicism³ x Perceived Control_{btw} interaction for Cardiac Output (CO) and Total Peripheral Resistance (TPR) reactivity.

(decreased CO and increased TPR reactivity) when Perceived Control_{btw} was high (+1SD) but active coping physiological response (increased CO

and decreased TPR reactivity) when Perceived Control_{btw} was low (-1SD). This interaction between Pseudo Cynicism and Perceived Control_{btw} was in the reverse direction for extremely high scores on the Pseudo Cynicism Factor.

For Cynicism Factor x Perceived Control_{wtn} interaction was not significant: SBP, $F(1, 105.11) = .06 \times 10^{-2}$, $p = .98$, $\eta^2 = .08 \times 10^{-7}$, DBP, $F(1, 105.34) = .001$, $p = .98$, $\eta^2 = .08 \times 10^{-7}$, MAP, $F(1, 108.33) = .01 \times 10^{-2}$, $p = .99$, $\eta^2 = .01 \times 10^{-2}$, HR, $F(1, 82.00) = .03 \times 10^{-2}$, $p = .98$, $\eta^2 = .04 \times 10^{-2}$, CO, $F(1, 103.73) = .05 \times 10^{-2}$, $p = .98$, $\eta^2 = .05 \times 10^{-2}$, and TPR, $F(1, 106.84) = .01$, $p = .90$, $\eta^2 = .01$. Pseudo Cynicism Factor x Perceived Control_{wtn} interaction was also not significant: SBP, $F(1, 105.11) = .47$, $p = .50$, $\eta^2 = .004$, DBP, $F(1, 105.34) = .002$, $p = .97$, $\eta^2 = .01 \times 10^{-7}$, MAP, $F(1, 108.33) = .67$, $p = .42$, $\eta^2 = .006$, HR, $F(1, 82.00) = .17$, $p = .68$, $\eta^2 = .002$, CO, $F(1, 103.73) = 1.45$, $p = .23$, $\eta^2 = .01$, and TPR, $F(1, 106.84) = .26$, $p = .61$, $\eta^2 = .002$.

The Cynicism Factor was not moderated by Perceived Control for cardiovascular reactivity; Hypothesis 4 was not supported. For the Pseudo Cynicism Factor, some differences in terms of hemodynamic processes (CO and TPR) were found for Pseudo Cynicism scores $\pm 2SD$ from the mean. When Perceived Control_{btw} was low, individuals scoring extremely high in Repressed Hostility (low Pseudo Cynicism scores) had active coping physiological responses (increased CO and decreased TPR reactivity) while extremely high in Pseudo Cynicism had passive coping physiological responses (decreased CO and increased TPR reactivity).

These hemodynamic patterns between extreme repressed hostile individuals and extremely pseudo cynical individuals were reversed when Perceived Control_{btn} was high.

For both Cynicism and Pseudo Cynicism Factors, no interactions involving Perceived Control_{wtn} were significant. Hypothesis 4 was not supported as Cynicism Factor-Reactivity was not moderate by Perceived Control_{btw} or Perceived Control_{wtn}.

Task Control, Cynicism & Pseudo Cynicism Factors. Cynicism Factor and cardiovascular reactivity was not moderated by Task Control for SBP, $F(1.85, 103.60) = 2.26, p = .14, \eta^2 = .04$, DBP, $F(1.83, 104.42) = 2.01, p = .16, \eta^2 = .03$, MAP, $F(1.88, 105.50) = 2.23, p = .14, \eta^2 = .04$, HR, $F(1.43, 79.86) = .32, p = .58, \eta^2 = .005$, and CO, $F(1.80, 104.63) = 1.30, p = .26, \eta^2 = .02$. The Cynicism x Task Control interaction was of borderline significance for TPR, $F(1.86, 105.91) = 3.75, p = .06, \eta^2 = .06$. Low Cynicism scores was associated with an increase in TPR reactivity during More Variable Pauses (figure 4.22).

Pseudo Cynicism x Task Control interaction was also not significant: SBP, $F(1.85, 103.60) = 1.05, p = .31, \eta^2 = .02$, DBP, $F(1.83, 104.42) = .61, p = .44, \eta^2 = .01$, MAP, $F(1.88, 105.50) = .52, p = .47, \eta^2 = .008$, HR, $F(1.43, 79.86) = 1.26, p = .27, \eta^2 = .02$, CO, $F(1.80, 104.63) = .66, p = .42, \eta^2 = .01$, and TPR, $F(1.86, 105.91) = 1.03, p = .31, \eta^2 = .02$.

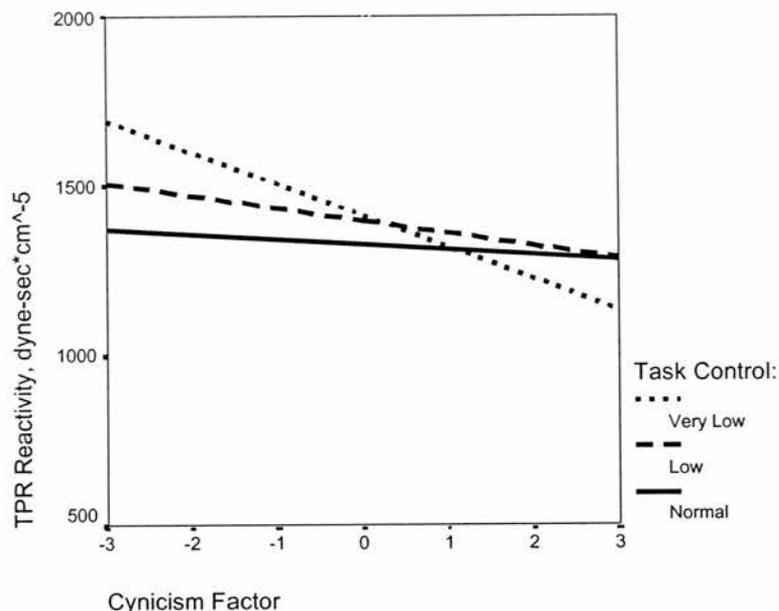


Figure 4.22 Cynicism Factor x Task Control interaction for Total Peripheral Resistance (TPR) reactivity

Hypothesis 4 was not supported - Cynicism-Reactivity relationship was not moderated by Task Control. High Cynicism was not associated with greater cardiovascular reactivity during low control. There was a trend for TPR reactivity but the pattern of the Cynicism x Task Control interaction was in the opposite direction for Hypothesis 4 when Cynicism was operationalised as the 12-item Cynicism scale and the Cynicism Factor. Some effects for CO and TPR reactivity were found for participants scoring greater than $\pm 1SD$ from mean for Pseudo Cynicism Factor.

Hypothesis 5: *Cynicism and Cardiovascular Reactivity is moderated by State Anger.*

A Cynicism x State Anger interaction was hypothesised to be significant for cardiovascular reactivity. Participants with high Cynicism scores have greater reactivity when self-reported state anger was high.

In order to have a less ambiguous interpretation of the results involving State Anger, the associations between the various forms of Cynicism and State Anger Baseline and State Anger given after the three Task Control conditions were analysed. The results for State Anger Baseline are analysed and reported first. The following between subjects model was tested using State Anger Baseline as the dependent variable (non significant quadratic and cubic terms were removed in the final regression model):

Between Subject model

- 1) Cynicism
- 2) Cynicism²
- 3) Cynicism³

For the 12-item Cynicism, the Cynicism main effect, $F(1, 57) = 2.77, p = .10, \eta^2 = .05$ was not significant. GNAT Cynicism was not significantly associated with State Anger Baseline, $F(1, 57) = .69, p = .41, \eta^2 = .01$. For Cynicism and Pseudo Cynicism Factors, the following model was tested (non-significant quadratic and cubic terms were removed from the final regression model):

Between Subject model

- 1) Cynicism Factor
- 2) Pseudo Cynicism Factor
- 3) Cynicism² Factor
- 4) Pseudo Cynicism² Factor
- 5) Cynicism³ Factor
- 6) Pseudo Cynicism³ factor

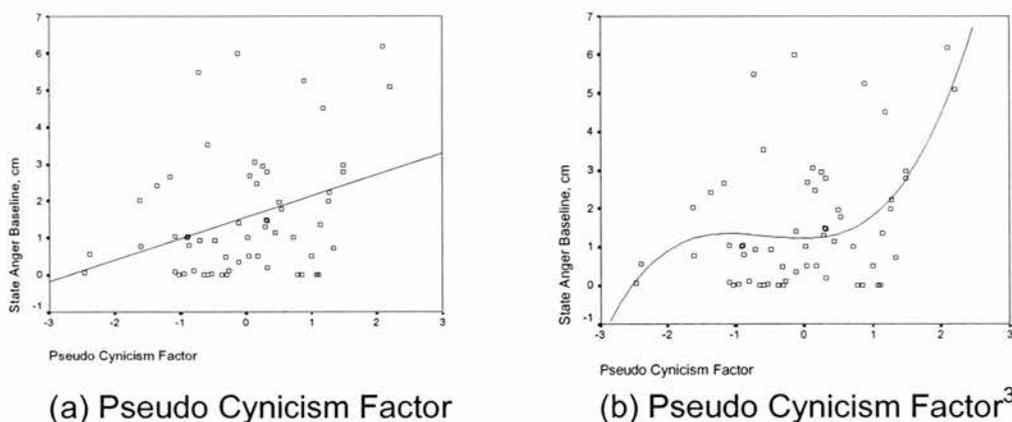


Figure 4.23 Relationship between (a), Pseudo Cynicism Factor, (b) Pseudo Cynicism Factor³ and State Anger Baseline

Cynicism Factor was not associated with State Anger Baseline, $F(1, 54) = 1.19, p = .28, \eta^2 = .008$. A Pseudo Cynicism Factor main effect was significant, $F(1, 54) = 20.15, p = .004, \eta^2 = .13$. A Pseudo Cynicism³ main effect was of borderline significance, $F(1, 54) = 3.86, p = .06, \eta^2 = .05$. The relationship between Pseudo Cynicism Factor and State Anger Baseline was also positive (figure 4.23). The results were congruent with the earlier analyses that indicated higher correlation between the Pseudo Cynicism Factor and negative affectivity.

Given the significant associations between the Pseudo Cynicism Factor and State Anger Baseline, there could be an attenuation of the Cynicism x State Anger interaction when the Pseudo Cynicism Factor was used due to the law of initial values (Wilder, 1967). Specifically, participants with high Pseudo Cynicism scores may give lower state

anger ratings after a stressful Synwork condition than a participant with a low Pseudo Cynicism score. This was because high Pseudo Cynicism participants gave higher ratings of State Anger during the Baseline, so their subsequent ratings after stress may have a 'ceiling' effect. To investigate whether this was true for the current data set, the linear, quadratic and cubic terms of the baseline were entered as independent variables. The State Anger ratings given after each Task Control condition was entered as a 3-level repeated measure dependent variable. If the law of initial values was in effect, the association between State Anger Baseline and State Anger ratings given after Synwork should be quadratic. The quadratic and cubic terms of State Anger Baseline were not significant: quadratic term, $F(1, 55) = .42, p = .52, \eta^2 = .01$, and the cubic term, $F(1, 55) = .02, p = .89, \eta^2 = .03 \times 10^{-2}$. The linear, quadratic and cubic terms' interaction with Task Control were not significant using Greenhouse-Geisser epsilon correction, p 's .61 to .82. The only significant result was a State Anger Baseline main effect, $F(1, 55) = 27.27, p = .03 \times 10^{-8}, \eta^2 = .49$, and a Task Control main effect, $F(1.94, 106.41) = 37.04, p = .01 \times 10^{-14}, \eta^2 = .40$. The significant State Anger Baseline main effect was positive indicating that higher State Anger Baseline predicted higher mean State Anger ratings for the three Task Control conditions. The Task Control main effect for State Anger was analysed in Table 4.10 and will not be repeated here. Thus, there was no evidence in the current data set to indicate that high State Anger Baseline ratings attenuated State Anger ratings given after Synwork.

Next, self-reported state anger given by participants after each Task Control condition was entered as a 3-level repeated measure dependent variable (by Task Control). State Anger Baseline and its interactions were entered as covariates (non-significant interactions involving the covariate and quadratic or cubic terms of Cynicism were removed from the final regression model). Regardless of how Cynicism was operationalised (12-item Cynicism scale, GNAT Cynicism or Cynicism Factor), State Anger Baseline main effect was significant. For 12-item Cynicism, State Anger Baseline was $F(1, 56) = 27.59, p = .02 \times 10^{-8}, \eta^2 = .33$, for GNAT Cynicism, $F(1, 56) = 27.71, p = .02 \times 10^{-8}, \eta^2 = .33$ and for Cynicism and Pseudo Cynicism Factors, $F(1, 55) = 27.22, p = .03 \times 10^{-8}, \eta^2 = .33$.

As State Anger given by participants after Variable and More Variable Pauses conditions were significantly higher (table 4.10), Cynicism x Task Control interaction was also reported as Cynicism may be positively related to State Anger only after Synwork that was less controllable. For 12-item Cynicism, Cynicism main effect was not significant, $F(1, 56) = .09, p = .76, \eta^2 = .001$, and the Cynicism x Task Control interaction was not significant, $F(1.26, 108.66) = .65, p = .52, \eta^2 = .007$. For GNAT Cynicism, Cynicism main effect was $F(1, 56) = .33, p = .57, \eta^2 = .004$, and the Cynicism x Task interaction was $F(1.95, 108.98) = .69, p = .50, \eta^2 = .007$. For Cynicism Factor, the Cynicism main effect, $F(1, 55) = .29, p = .59, \eta^2 = .004$, and Cynicism x Task Control interaction, $F(1.94, 106.81) = .96, p = .39, \eta^2 = .01$, were not significant.

For Pseudo Cynicism, Pseudo Cynicism main effect and its interaction with Task Control was also not significant, $F(1, 55) = .05, p = .83, \eta^2 = .09 \times 10^{-2}$, and , $F(1.96, 106.81) = .08, p = .92, \eta^2 = .09 \times 10^{-2}$ respectively.

Though the Pseudo Cynicism Factor was positively associated with State Anger Baseline, Cynicism and Pseudo Cynicism measures were not significantly associated with State Anger ratings given after Synwork.

To avoid confusion with State Anger Baseline, State Anger ratings given after Synwork is labelled 'State Anger Synwork'. As was done for Perceived Control, two forms of State Anger Synwork are created. State Anger Synwork ratings for the three conditions of Task Control were averaged to index the mean State Anger Synwork for each participant. This was labelled as 'State Anger Synwork_{btw}'. The original values of State Anger were entered as a continuous variable in the within subject regression model was labelled as 'State Anger Synwork_{wtn}'. The following model (non significant quadratic and cubic terms for Cynicism were removed from the final regression model) was tested:

Between subject model

- 1) Cardiovascular Baseline (covariate)
- 2) Cynicism
- 3) Cynicism²
- 4) Cynicism³
- 5) State Anger Synwork_{btw}
- 6) Cynicism x State Anger Synwork_{btw}
- 7) Cynicism² x State Anger Synwork_{btw}
- 8) Cynicism³ x State Anger Synwork_{btw}

Within Subject model

- 1) State Anger Synwork_{wtn}
- 2) Cynicism x State Anger Synwork_{wtn}
- 3) Cynicism² x State Anger Synwork_{wtn}

4) Cynicism³ x State Anger Synwork_{wtn}

Any significant interactions involving the covariate from previous analyses was also included into the model. In addition, Cynicism and Pseudo Cynicism Factors were tested together in the same model.

5a) Cynicism x State Anger Synwork_{btw} and Cynicism x State Anger Synwork_{wtn} are significant for cardiovascular reactivity. Participants with high Cynicism scores have greater reactivity when State Anger Synwork is high.

12-item Cynicism. Cynicism x State Anger Synwork_{btw} interaction was not significant for SBP, $F(1, 54) = 2.95, p = .09, \eta^2 = .04$, partial $\eta^2 = .06$. HR, $F(1, 53) = .64, p = .43, \eta^2 = .004$, , partial $\eta^2 = .09$, and CO, $F(1, 50) = .57, p = .45, \eta^2 = .004$, partial $\eta^2 = .01$. Cynicism x State Anger Synwork_{btw} was significant for DBP, $F(1, 54) = 5.94, p = .02, \eta^2 = .07$, partial $\eta^2 = .17$, MAP, $F(1, 54) = 4.84, p = .03, \eta^2 = .05$, partial $\eta^2 = .13$, and TPR, $F(1, 54) = 5.41, p = .02, \eta^2 = .06$, partial $\eta^2 = .09$. Figure 4.24 shows that participants with high Cynicism scores had greater vascular-mediated (TPR-mediated) DBP and MAP reactivity when State Anger Synwork_{btw} was high then when State Anger Synwork_{btw} was low. Of interest to note is that participants with low Cynicism scores had greater DBP, MAP and TPR reactivity when State Anger Synwork_{btw} was low than high Cynicism participants when State Anger Synwork_{btw} was high.

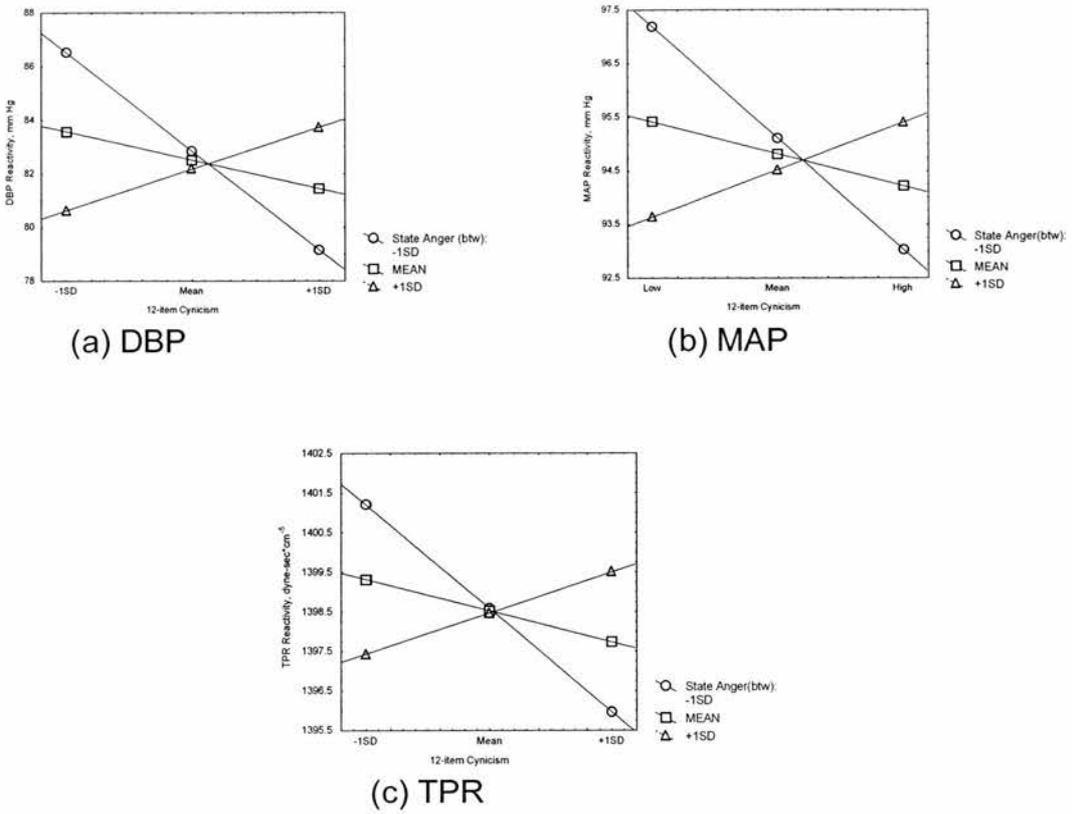


Figure 4.24 12-item Cynicism x State Anger Synwork_{btw} for mean Diastolic Blood Pressure (DBP), Mean Arterial Blood Pressure (MAP) and Total Peripheral Resistance (TPR) reactivity.

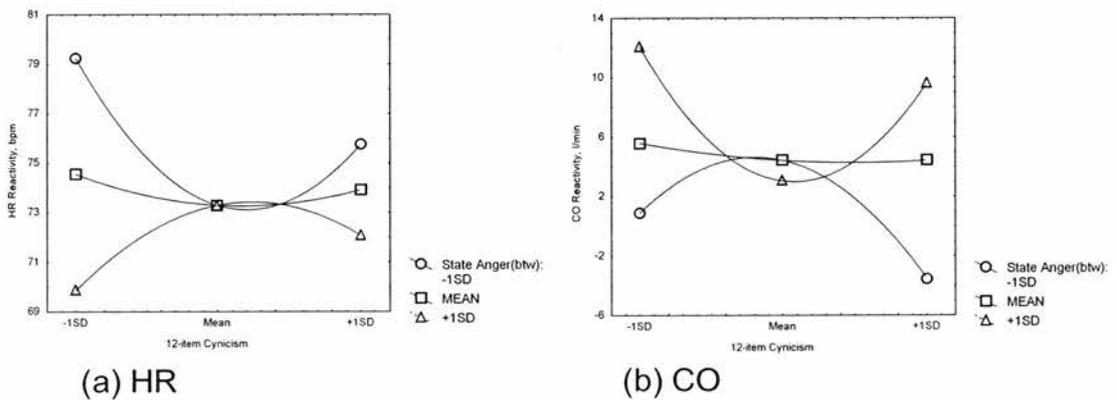


Figure 4.25 Mean Heart Rate (HR) and Cardiac Output (CO) reactivity by 12-item Cynicism and State Anger Synwork_{btw}

A significant Cynicism x State Anger Synwork_{btw} interaction involving the quadratic term of Cynicism was found for HR. Specifically for HR, a Cynicism² x State Anger Synwork_{btw} was significant, $F(1, 53) = 9.34, p = .003, \eta^2 = .06, \text{partial } \eta^2 = .14$. For CO, a Cynicism² x State Anger Synwork_{btw} for CO was also significant, $F(1, 50) = 3.50, p = .02, \eta^2 = .04, \text{partial } \eta^2 = .10$ (figure 4.25). For low 12-item Cynicism scores, HR reactivity was the greatest when State Anger Synwork_{btw} was low. Both high and low 12-item Cynicism scores had the same pattern of CO reactivity compared to Cynicism scores at the mean. Both high and low Cynicism scores were associated with greater CO reactivity when State Anger Synwork_{btw} was high. When State Anger Synwork_{btw} was low, CO hypo-reactivity occurred. Scores at the mean did not differentiate with different levels of State Anger Synwork_{btw}.

State Anger Synwork_{btw} main effect was not significant: SBP, $F(1, 54) = 2.28, p = .14, \eta^2 = .03, \text{partial } \eta^2 = .05$, DBP, $F(1, 54) = 1.32, p = .24, \eta^2 = .02, \text{partial } \eta^2 = .04$, MAP, $F(1, 54) = 1.94, p = .17, \eta^2 = .02, \text{partial } \eta^2 = .05$, HR, $F(1, 53) = .64, p = .43, \eta^2 = .004, \text{partial } \eta^2 = .01$, CO, $F(1, 50) = 1.29, p = .26, \eta^2 = .01, \text{partial } \eta^2 = .02$, and TPR, $F(1, 54) = .003, p = .96, \eta^2 = .03 \times 10^{-3}, \text{partial } \eta^2 = .05 \times 10^{-3}$.

For State Anger Synwork_{wtn}, the Cynicism x State Anger Synwork_{wtn} interaction was not significant: SBP, $F(1, 106.02) = .004, p = .95, \eta^2 = .04 \times 10^{-3}$, DBP, $F(1, 106.26) = .32, p = .57, \eta^2 = .003$, MAP, $F(1, 109.27) = .17, p = .68, \eta^2 = .002$, HR, $F(1, 83.42) = .02 \times 10^{-2}, p = .99, \eta^2 =$

$.02 \times 10^{-4}$, CO, $F(1, 104.63) = .02$, $p = .89$, $\eta^2 = .02 \times 10^{-2}$, and TPR, $F(1, 107.76) = 1.33$, $p = .25$, $\eta^2 = .01$.

State Anger Synwork_{wtn} main effect was also not significant: SBP, $F(1, 106.02) = .53$, $p = .47$, $\eta^2 = .005$, DBP, $F(1, 106.26) = 1.24$, $p = .27$, $\eta^2 = .01$, MAP, $F(1, 109.27) = .56$, $p = .45$, $\eta^2 = .005$, HR, $F(1, 83.42) = .03 \times 10^{-2}$, $p = .96$, $\eta^2 = .03 \times 10^{-3}$, CO, $F(1, 104.63) = .15$, $p = .70$, $\eta^2 = .001$, and TPR, $F(1, 107.76) = .06$, $p = .81$, $\eta^2 = .06 \times 10^{-2}$.

GNAT Cynicism. For GNAT Cynicism, Cynicism x State Anger Synwork_{btw} interaction was not significant: SBP, $F(1, 54) = .003$, $p = .95$, $\eta^2 = .04 \times 10^{-3}$, partial $\eta^2 = .06 \times 10^{-3}$, DBP, $F(1, 54) = .64$, $p = .43$, $\eta^2 = .008$, partial $\eta^2 = .02$, MAP, $F(1, 54) = .16$, $p = .69$, $\eta^2 = .002$, partial $\eta^2 = .003$, HR, $F(1, 50) = 1.38$, $p = .25$, $\eta^2 = .008$, partial $\eta^2 = .02$, CO, $F(1, 50) = .66$, $p = .42$, $\eta^2 = .005$, partial $\eta^2 = .01$, and TPR, $F(1, 50) = .74$, $p = .39$, $\eta^2 = .008$, partial $\eta^2 = .01$.

For HR and CO, some interactions involving the nonlinear terms of Cynicism with State Anger Synwork_{btw} were found to be significant. For HR, Cynicism² x State Anger Synwork_{btw} was significant, $F(1, 50) = 11.19$, $p = .002$, $\eta^2 = .07$, partial $\eta^2 = .16$. High GNAT Cynicism was associated with greater HR reactivity when State Anger Synwork_{btw} was low. When State Anger Synwork_{btw} was high, high GNAT Cynicism was associated with decreased HR reactivity (figure 4.26). For CO, Cynicism³ x State Anger Synwork_{btw} interaction was significant, $F(1, 50) = 7.03$, $p = .01$, $\eta^2 =$

.06, partial $\eta^2 = .11$. For TPR, this interaction was of borderline significance, $F(1, 50) = 3.88, p = .05, \eta^2 = .05$, partial $\eta^2 = .06$.

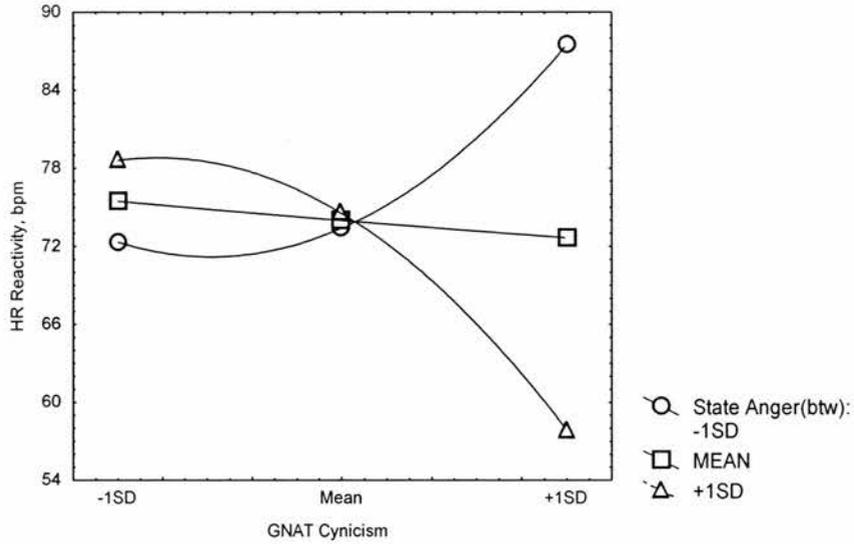


Figure 4.26 Heart Rate (HR) Reactivity by GNAT Cynicism and State Anger Synwork_{btw}

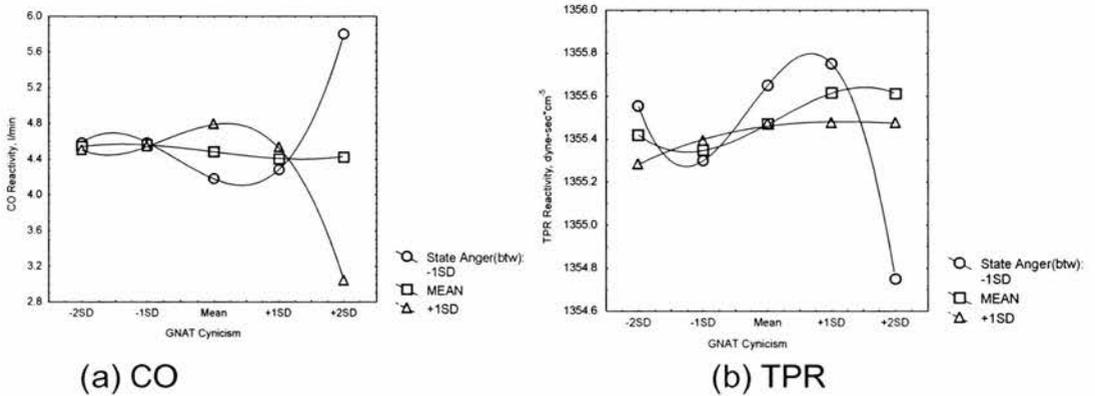


Figure 4.27 Cardiac Output (CO) and Total Peripheral Resistance (TPR) reactivity by GNAT Cynicism and State Anger Synwork_{btw}

The Cynicism³ x State Anger Synwork_{btw} interaction for CO and TPR show that GNAT Cynicism greater than +2SD from the mean had active coping physiological response (increase CO and decreased TPR)

when State Anger Synwork_{btw} was low (figure 4.27). When State Anger Synwork_{btw} was high, passive coping physiological response (decreased CO and increased TPR) was present.

The State Anger Synwork_{btw} main effect was reported for the 12-item Cynicism scale and the effect sizes were identical when tested in the GNAT Cynicism model. Hence, the results were not reported.

GNAT Cynicism x State Anger Synwork_{wtn} interaction was not significant: SBP, $F(1, 105.44) = 2.33, p = .13, \eta^2 = .02$, DBP, $F(1, 110.55) = .04, p = .85, \eta^2 = .03 \times 10^{-2}$, MAP, $F(1, 106.82) = 2.22, p = .14, \eta^2 = .02$, HR, $F(1, 86.19) = 1.03, p = .31, \eta^2 = .01$, CO, $F(1, 104.75) = .03, p = .87, \eta^2 = .02 \times 10^{-3}$, and TPR, $F(1, 100.69) = .91, p = .34, \eta^2 = .001$. State Anger Synwork_{wtn} main effect was reported for the 12-item Cynicism and will not be repeated here.

Cynicism and Pseudo Cynicism Factors. For Cynicism Factor, Cynicism x State Anger Synwork_{btw} interaction was not significant for SBP, $F(1, 50) = 3.20, p = .08, \eta^2 = .03$, partial $\eta^2 = .05$, DBP, $F(1, 47) = 1.64, p = .21, \eta^2 = .02$, partial $\eta^2 = .03$, MAP, $F(1, 51) = 1.64, p = .21, \eta^2 = .02$, partial $\eta^2 = .03$, and HR, $F(1, 48) = .01 \times 10^{-2}, p = .99, \eta^2 = .01 \times 10^{-2}$, partial $\eta^2 = .02 \times 10^{-2}$. Cynicism x State Anger Synwork_{btw} was significant for TPR, $F(1, 44) = 5.40, p = .02, \eta^2 = .06$, partial $\eta^2 = .09$, and borderline significant for CO, $F(1, 50) = 3.60, p = .06, \eta^2 = .03$, partial $\eta^2 = .05$. The pattern of results obtained for Cynicism Factor x State Anger Synwork_{btw} interaction for TPR reactivity was similar to that obtained when the 12-

item Cynicism scale was used (figure 4.24) with a few notable differences. When 12-item Cynicism was used, Cynicism was negatively related to TPR reactivity when State Anger Synwork_{btw} was low. When the Cynicism Factor was used, the negative relationship between Cynicism and TPR reactivity when State Anger Synwork_{btw} was low has been reduced. When the 12-item Cynicism scale was used, low Cynicism and low State Anger_{btw} had the greatest TPR reactivity. When the Cynicism Factor was used, high Cynicism and high State Anger_{btw} now had the greatest TPR reactivity (figure 4.28), which was more in line with Hypothesis 4. In addition, the Cynicism x State Anger Synwork_{btw} CO reactivity is of borderline significance showing the reverse pattern to that obtained for TPR. When 12-item Cynicism was used, the interaction was

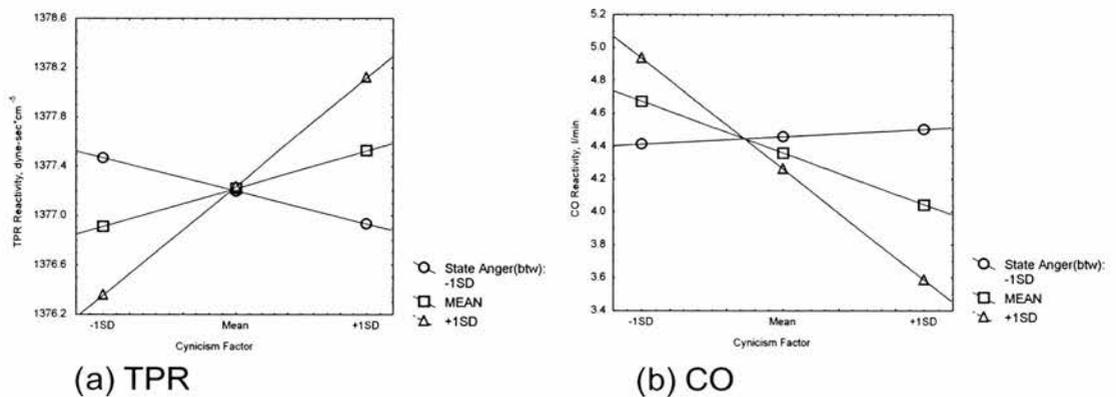


Figure 4.28 Cynicism Factor x State Anger Synwork_{btw} interaction for Total Peripheral Resistance (TPR) and Cardiac Output (CO) reactivity.

not significant. When Cynicism Factor was used, the effect size for this interaction for CO was 7.5 times larger than when 12-item Cynicism scale

was used. The effect size for this interaction for TPR reactivity was identical whether Cynicism Factor or the 12-item Cynicism scale was used. Furthermore, when 12-item Cynicism was used, the larger increase in DBP and MAP for low Cynicism participants with low State Anger Synwork_{btw} was due to greater TPR reactivity. However, when the Cynicism Factor was used, there was no significant Cynicism x State Anger Synwork_{btw} interaction for DBP or MAP. There was no hemodynamic dysregulation of blood pressure for Low Cynicism scores when Cynicism Factor was used.

For State Anger Synwork_{wtn}, Cynicism Factor x State Anger Synwork_{wtn} interaction was not significant: SBP, $F(1, 105.11) = .63, p = .43, \eta^2 = .006$, DBP, $F(1, 112.13) = .22, p = .64, \eta^2 = .002$, MAP, $F(1, 108.33) = 1.26, p = .26, \eta^2 = .01$, HR, $F(1, 85.56) = .34, p = .56, \eta^2 = .004$, CO, $F(1, 104.08) = .03, p = .86, \eta^2 = .03 \times 10^{-2}$, and TPR, $F(1, 106.95) = 1.26, p = .26, \eta^2 = .01$. As State Anger Synwork_{wtn} main effects have been reported when the 12-item Cynicism scale was used, the effect sizes were not significant and identical and will not be reported here.

For the Pseudo Cynicism Factor, the Pseudo Cynicism Factor x State Anger Synwork_{btw} interaction was not significant: SBP, $F(1, 50) = 1.81, p = .18, \eta^2 = .02$, partial $\eta^2 = .03$, DBP, $F(1, 47) = 2.29, p = .14, \eta^2 = .02$, partial $\eta^2 = .04$, MAP, $F(1, 51) = .21, p = .65, \eta^2 = .002$, partial $\eta^2 = .03$, HR, $F(1, 48) = 2.37, p = .13, \eta^2 = .02$, partial $\eta^2 = .04$, CO, $F(1, 50) = .19, p = .67, \eta^2 = .001$, partial $\eta^2 = .003$, and TPR, $F(1, 44) = .57, p = .45,$

$\eta^2 = .006$, partial $\eta^2 = .09$. For HR, the Pseudo Cynicism Factor² x State Anger Synwork_{btw} interaction was borderline significant, $F(1, 48) = 3.80$, $p = .06$, $\eta^2 = .03$, partial $\eta^2 = .06$. Participants with low Pseudo Cynicism scores had greater HR reactivity during low levels of State Anger Synwork_{btw}. When State Anger Synwork_{btw} was high HR reactivity hypo-reactivity was present (figure 4.29).

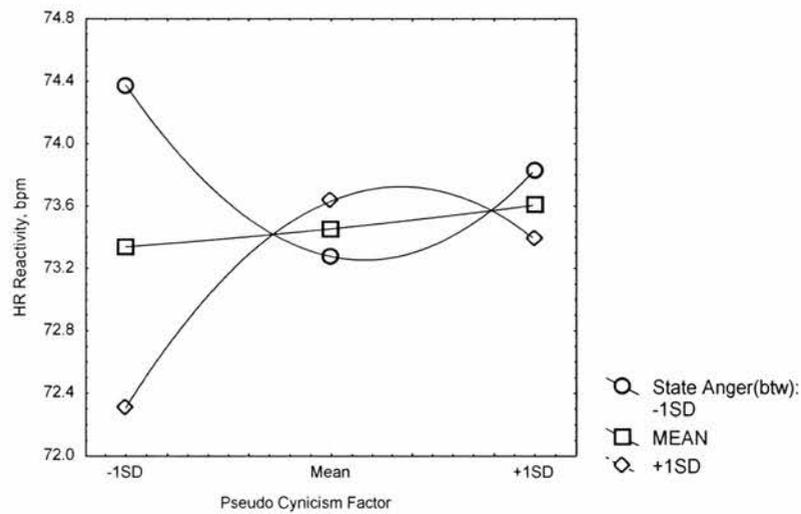


Figure 4.29 Pseudo Cynicism² x State Anger Synwork_{btw} interaction for Heart Rate (HR) reactivity

For State Anger_{wtn}, Pseudo Cynicism Factor x State Anger Synwork_{wtn} interaction was not significant: SBP, $F(1, 105.11) = 1.96$, $p = .16$, $\eta^2 = .02$, DBP, $F(1, 112.13) = .12$, $p = .73$, $\eta^2 = .001$, MAP, $F(1, 108.33) = 1.05$, $p = .30$, $\eta^2 = .01$, HR, $F(1, 85.56) = .91$, $p = .34$, $\eta^2 = .01$, CO, $F(1, 104.08) = .03 \times 10^{-2}$, $p = .99$, $\eta^2 = .03 \times 10^{-4}$, and TPR, $F(1, 106.95) = .13$, $p = .71$, $\eta^2 = .001$.

Hypothesis 5a was supported for blood pressure reactivity when Cynicism was assessed using the 12-item Cynicism scale. Though high 12-item Cynicism scores were associated with greater blood pressure and TPR reactivity, low Cynicism scores had greater (or as much) TPR reactivity when State Anger Synwork_{btw} was low. The absence of the Cynicism x State Anger Synwork_{btw} interaction for blood pressure reactivity when Cynicism was assessed using the Cynicism Factor suggests that this blood pressure effect for the 12-item Cynicism was not due to Cynicism. When Cynicism was assessed using the 12-item Cynicism or Cynicism Factor, high Cynicism scores were associated with greater TPR reactivity during high State Anger Synwork_{btw} than low State Anger Synwork_{btw}. Hypothesis 5a was supported for State Anger Synwork_{btw} but not for State Anger Synwork_{wtn}.

Synwork Scores

As was done for Study 2, the following model was tested to see if Synwork Scores offer a better model fit than Task Control:

Between Subject Model

- 1) TPR Baseline
- 2) Score_{btw}
- 3) Cynicism Factor
- 4) Pseudo Cynicism
- 5) Cynicism Factor²
- 6) Pseudo Cynicism²
- 7) Cynicism³
- 8) Pseudo Cynicism³

Within Subject Model

- 1) Score_{wtn}

TPR reactivity was used as significant Task Control main effect was found. $\text{Score}_{\text{btw}}$ main effect was not significant, $F(1, 50) = .32, p = .60, \eta^2 = .002$, partial $\eta^2 = .002$. $\text{Score}_{\text{wth}}$ main effect was also not significant, $F(1, 108.69) = .59, p = .44, \eta^2 = .005$. Hence, Task Control offered a better model fit than Synwork Scores.

Table 4.17 Summary of Main Results by Cynicism Measure*

	12-item Cynicism	GNAT Cynicism	Cynicism Factor	Pseudo Cynicism Factor
1) Cynicism main effect				
Cynicism	n.s.	n.s.	n.s.	n.s.
Cynicism ²	HR	n.s.	HR: $p = .05$	SBP, DBP, MAP
Cynicism ³	CO	TPR	CO, TPR	HR, TPR
2) Cynicism x Perceived Control				
Cynicism x Perceived Control _{btw}	n.s.	n.s.	n.s.	n.s.
Cynicism ³ x Perceived Control _{btw}	n.s.	n.s.	n.s.	CO, TPR
Cynicism x Perceived Control _{wn}	n.s.	n.s.	n.s.	n.s.

* abbreviations of dependent variables listed in table are significant, n.s. = not significant ($p > .05$), ' $p = .05$ ' = borderline significance, SBP = Systolic Blood Pressure, DBP = Diastolic Blood Pressure, MAP = Mean Arterial Pressure, HR = Heart Rate, CO = Cardiac Output, TPR = Total Peripheral Resistance

Table 4.17 Summary of Main Results by Cynicism Measure*

	12-item Cynicism	GNAT Cynicism	Cynicism Factor	Pseudo Cynicism Factor
3) Cynicism x Task Control	TPR	n.s.	TPR: $p = .05$	n.s.
4) Cynicism x State Anger				
Cynicism x State Anger _{btw}	DBP, MAP, TPR	n.s.	CO: $p = .05$, TPR	n.s.
Cynicism ² x State Anger _{btw}	HR, CO	HR	n.s.	HR
Cynicism ³ x State Anger _{btw}	n.s.	CO, TPR	n.s.	n.s.
Cynicism x State Anger _{win}	n.s.	n.s.	n.s.	n.s.

*abbreviations of dependent variables listed in table are significant, n.s. = not significant ($p > .05$), ' $p = .05$ ' = borderline significance, SBP = Systolic Blood Pressure, DBP = Diastolic Blood Pressure, MAP = Mean Arterial Pressure, HR = Heart Rate, CO = Cardiac Output, TPR = Total Peripheral Resistance

Study 3 Discussion

This chapter addresses another source of measurement error in the Cook-Medley Cynicism scale - the problem of false negatives (Repressed Cynicism) and false positives (Pseudo Cynicism). The main aim of this discussion is to summarise and interpret the results obtained in this study. Chapter 5 (Main Discussion) discusses the overall results obtained from the 3 studies in this dissertation, relates that to published studies, and the implications.

The current method of identifying Repressed Cynicism uses the Cook-Medley Hostility scale and the Marlowe Crowne Social Desirability scale (Cook-Medley/Marlowe-Crowne method). Repressed Cynicism reflects some discrepancy between explicit and implicit Cynicism. Repressed Cynicism is characterised by low explicit cynicism and high implicit cynicism. Whether defined as a form of high Cynicism or not, the cardiovascular reactivity of Repressed Cynicism is likely not to be as low as low cynical individuals. Thus, not correcting or identifying Repressed Cynicism individuals in any sample can produce null or conflicting results.

The Cook-Medley/Marlowe-Crowne method of detecting Repressed Cynicism is problematic because it fails to differentiate between Repressed Cynicism and low hostility-high social desirability traits. An alternative to the Cook-Medley/Marlowe-Crowne method is to develop another way to assess Cynicism that is less susceptible to social desirability and use that to 'correct' the Cook-Medley Hostility scale. The Go/No Go Association Test (GNAT) version of Cynicism assessment is

proposed as such an alternative. This chapter details the development of an implicit assessment of Cynicism (GNAT Cynicism). GNAT Cynicism is then used together with the Cook-Medley 12-item Cynicism scale to reduce measurement error in the Cook-Medley 12-item Cynicism scale. These various forms of Cynicism assessment is then used to predict cardiovascular stress reactivity in order to investigate the role of Cynicism as well as measurement error in predicting cardiovascular stress reactivity.

This study also investigates the role of State Anger and Control as moderators of the Cynicism-Reactivity relationship using a similar experimental design as Study 2. However, in this study, Synwork Task Control was counterbalanced. The amount of blood pressure reactivity elicited from the various Task Control conditions was slightly lower in this study than those obtained for Study 2. For instance, the Normal Task Control elicited an average increase of 10.07mm Hg for MAP in Study 2 while average MAP reactivity was 8.46mm Hg for the study in this chapter. The overall hemodynamic pattern obtained for Study 2 and 3 using this paradigm reveal that Task Control, operationalised as response-outcome contingency is associated with a passive coping physiological response (decrease CO and increased TPR). This study counterbalances the three Task Control conditions, and a significant TPR effect was found. This shows that the borderline TPR main effect for Task Control obtained in Study 2 is unlikely to be due to the upward drift of TPR associated with prolonged use of the Finapres. TPR reactivity for Task Control was of borderline significance in study 2 and significant in

this study. However, the effect size for the Task Control main effect for TPR reactivity is the same for both studies (i.e., $\eta^2 = .08$). In addition, the pattern of TPR reactivity is also similar. In Study 2, a significant Task Control main effect was found for CO reactivity while in this study the no such significant Task Control main effect for CO reactivity was found. Further analysis revealed that CO reactivity for the Normal-Variable-More Variable Pauses conditions found in Study 2 are replicated in this study. Counterbalancing eliminated the Task Control main effect for CO found in Study 2. Like Study 2, there was also a downward drift of CO and upward drift of blood pressure and TPR over the course of the whole experimental procedure in this study. The elimination of the CO effects for Task Control via counterbalancing indicated that CO effect is likely to be influenced by the downward drift of CO reactivity over extended use of the Finapres. In contrast, counterbalancing did not eliminate Task Control effect for TPR reactivity - the effect size for Task Control main effect for TPR was the same for both Studies 2 and 3. This indicated that Task Control effect for TPR reactivity is unrelated to the upward drift of TPR over extended Finapres usage.

Like Study 2, Study 3 also found that Synwork Variable and More Variable Pauses conditions were quite similar in terms of Synwork scores obtained by the participants. Participants' ratings of Perceived Control between these two conditions are also similar. Unlike Study 2, Perceived Control and Task Scores are no longer significantly higher in the More Variable Pauses condition than the Variable Pauses condition. Counterbalancing Task Control conditions has eliminated this pattern.

This shows that More Variable Pauses condition is not *more* controllable than the Variable Pauses condition. However, TPR reactivity was not significantly different between Variable and More Variable Pauses Task Control conditions. This questions whether the Variable and More Variable Pauses Task Control conditions actually differ in controllability.

The equivalent TPR reactivity obtained between Variable and More Variable Pauses is likely to be due to a technical oversight for the Faulty Mouse device (appendix 4). A failure to control for the frequency in which the random pauses are generated by the Faulty Mouse device may have resulted in almost equivalent average response-outcome contingency between the Variable and More Variable Pauses Task Control conditions²². Random pauses generated by the Faulty Mouse device does not standardise the number of random pauses it generates. During the Variable Pauses condition random pauses were set from 25ms to 300ms while during the More Variable Pauses condition, random pauses were 25ms to 500ms. If both Variable and More Variable Pauses conditions are of the same duration (e.g., 3mins), then more random pauses can be generated during the Variable Pauses condition since the average random pause generated is shorter (mean = 162.5ms). If more short random pauses are generated during the Variable Pauses condition then this can result in an equivalent average amount of disruption to the computer mouse as in the More Variable Pauses condition. For 400ms, there can be two random pauses generated during the Variable Pauses

²² This point has been verified with the Faulty Mouse creator, Mr. Malcolm McCandles (personal communication, 2004).

condition ($162.5\text{ms} \times 2 = 325\text{ms}$) or one random pause generated in the More Variable Pauses condition (262.5ms). Notice how close the average duration of the pauses are between the two Task Control conditions. Therefore, the lack of gradation in terms of TPR reactivity by Task Control condition is most probably due to a technical oversight in the development of the Faulty Mouse device.

This does not invalidate the chief points made using the Faulty Mouse device. The hypotheses tested involving Task Control do not require the response-outcome contingencies to be lowest in More Variable Pauses condition, though a gradation of Task Control would be stronger evidence for the role of response-outcome contingency in predicting active/passive physiological response. As long as there are at least two levels of control (one high and another low Control), the hypotheses involving Task Control can be adequately (but not ideally) tested.

The results still support the proposal that Task Control should be re-operationalised as response-outcome contingency rather than the presence/absence of coping behaviours. It is important to keep in mind that response-outcome contingency in this dissertation refers to the contingency between the participants' manipulation of the computer trackball and the movement of the mouse cursor on the computer screen and not the amount of Synwork scores obtained. In Study 2, one of the aims of fixing the order of Task Control was to enable participants to have practice with Synwork having Variable Pauses. This practise was to enable them to improve on their coping strategies for Synwork with More

Variable Pauses. While Synwork Variable Pauses and More Variable Pauses had equivalent TPR reactivity, Perceived Control and Synwork work scores increased. This meant that efficacy of their coping behaviours (Synwork scores) and participants' perceived efficacy of their coping behaviours (Perceived Control) increased for the Synwork with More Variable Pauses. Thus, a discrepancy between the efficacy of the participants' coping behaviours and response-outcome contingency was created. That is, while the participants' coping behaviours with the Synwork improved, response-outcome contingency remained the same (rather than originally hypothesised to decrease). The results from this Study 2 and 3 showed that response-outcome contingency (Task Control) always provides a better model fit than Synwork scores (coping behaviour efficacy). Even when a discrepancy exists between Task Control and Synwork Scores (as in Study 2), Task Control still fitted the model better. This meant that response-outcome contingency (Task Control) accounts for active/passive physiological responses better than efficacy of coping behaviours (Synwork Scores) or perceived efficacy of coping behaviours (Perceived Control). This is evidence suggesting that response-outcome contingency rather than coping behaviours efficacy should be the defining characteristic of active/passive coping physiological response. However, the poor model fit of Perceived Control cannot be generalised to the corresponding subjective form of response-outcome contingency - response-outcome expectations (Bandura, 1977). In this dissertation, Perceived Control is the participants' appraisal of the efficacy of their own

coping behaviours for Synwork (i.e., the extent they can influence the outcome of the computer task).

Task Score should not be taken as an index of response-outcome contingency. While it is reasonable to expect a positive association between the two, Task Score can be independent of response-outcome contingency. This is because Task Score is also a performance-based index and performance-based indices do not always correlate with response-outcome contingency. For example, a faulty copier machine copies a document half the time when the 'print' button is pressed, which indicates a response (pressing the 'print' button) outcome (copier working) contingency is .50. To obtain a desired outcome of 10 sheets in 3minutes becomes more unlikely. However, an individual can increase the number of responses (pressing the 'print' button repeatedly in quick succession) in order to achieve the outcome. In this instance, the response-outcome contingency of the copier remains the same but the outcome is achieved because the number of responses has increased. Hence, outcome measures do not always indicate response-outcome contingency. However, looking at the relationship between Task Control (response-outcome contingency) and active/passive coping physiological response, it can be inferred that both Variable and More Variable Pauses conditions have equal levels of response-outcome contingencies, though both are lower than the Normal Control condition.

Another chief focus of this chapter is the conceptualisation, development and testing of a GNAT version for Cynicism assessment. The correlation of the GNAT Cynicism and the other psychometric scales

showed that it is less susceptible to social desirability (non-significant correlation with the Marlowe-Crowne Social Desirability scale) and has convergent construct validity (significant correlation with Cook-Medley 12-item Cynicism scale). The magnitude of the correlation between the implicit Cynicism (GNAT Cynicism) and the explicit Cynicism measure (12-item Cynicism scale) closely resemble the correlation obtained by Greenwald and his colleagues for the implicit-explicit correlation for racial stereotype attitudes (Greenwald et al., 2003). Greenwald's research on the implicit association for racial stereotypical attitudes is an appropriate comparison because like Cynicism, it is a negative evaluation of others that is also susceptible to positive self-presentation bias. Constructs that are more susceptible to positive self-presentation bias (e.g., racial prejudice, sexism) tend to have moderate to low explicit-implicit correlation (Greenwald et al., 2002).

The GNAT Cynicism also has better discriminant construct validity than the 12-item Cynicism scale in that it is not significantly correlated with negative affect (DS16 Negative Affectivity subscale) and STAXI's Trait Anger Reaction. The non-significant correlation between GNAT Cynicism and GNAT Trait Hostility also supports the proposition that the attitude of regarding others as unreliable (Cynicism) does not imply that the self is benign (Trait Hostility). It is because 'Cynicism does not imply low Trait Hostility' that Nosek and Banaji's GNAT (Nosek & Banaji, 2001) version of assessing implicit association was used instead of Greenwald's IAT.

The design differences between the GNAT and the IAT makes it applicable for its use in Implicit Cynicism assessment but it is also one of its limitations in that it is less time efficient as the IAT. The IAT assessment saves times because two implicit associations are tapped in a single block while the GNAT has to use two blocks in order to replicate the same results in IAT. This limits the number of implicit association the GNAT can tap as it lengthens the GNAT assessment twice compared to the IAT. Extending the GNAT assessment can increase tedium and decrease the participant's accuracy in detecting the target stimuli.

Greenwald et al. (Greenwald et al., 2003) reported that the revised computation of the IAT effect by dividing the IAT effect by the pooled standard deviation of all the participant's response times helps to prevent penalising slow responders with high IAT effect sizes. Greenwald et al. (2003) also suggests that the inclusion of errors (in a revised format) and the re-scoring of extreme response times increase the implicit-explicit correlation. In this study, including errors into the IAT computation decreased rather than increased the implicit-explicit correlation for Cynicism. The difference between the results obtained by Greenwald et al.'s (2003) and this study is likely to be due to the different design of the GNAT compared to the IAT. The IAT had several practise trails before the actual test trail (figure 4.1) while the GNAT is limited to one practise trial. Hence, errors in the practice trial for the IAT just prior to the actual test block (Block 3) may indicate weak implicit association but the errors for the practice block in the GNAT is probably due to unfamiliarity with the target stimuli. Revising errors to be included in the computation of the

implicit association effect is also not feasible for the GNAT used in this study because Greenwald et al.'s (2003) study did not have a time limit for the participant to respond. This resulted in some response times in their study that were as long as 10^6 ms! Their error latencies do not have a ceiling while the error latencies for the GNAT used in this study are all 1 second as the stimuli for the GNAT were presented on the computer monitor for a maximum duration of only 1 second. The shorter time limit of the GNAT ensures that the participant pays attention to the stimuli displayed on the screen. Future researchers developing the GNAT Cynicism may wish to increase this time limit in order to obtain a 100% hit rate in order to get a more accurate assessment of the implicit association.

The encouraging results obtained with GNAT Cynicism in terms of its convergent and discriminant construct validity compared to the 12-item Cynicism scale should not be taken to imply that it is a replacement for the 12-item Cynicism scale. There are several reasons against such a proposition. Firstly, the modest correlation ($r = .30$) between GNAT Cynicism and 12-item Cynicism implies that the GNAT Cynicism is measuring a similar but not identical construct compared to the 12-item Cynicism. The moderate correlation does not make the GNAT Cynicism a suitable replacement for the 12-item Cynicism scale. Secondly, though the results for the GNAT Cynicism are encouraging, its validity and reliability still needs further work. For example, how does the GNAT Cynicism relate to the Structured Interview Hostility assessment? Dovidio et al. showed that an implicit measure of racial prejudice is related to

nonverbal behaviours and not related to verbal behaviours (Dovidio, Kawakami, & Gaertner, 2002), which suggests that GNAT Cynicism will not be significantly associated with the Structured interview-derived Hostility measure. But this needs to be empirically tested for the HBI that was used in Study 2. In addition, though a test-retest reliability of .69 for implicit measures has been reported (Bosson, Swann, & Pennebaker, 2000), the test-retest of the GNAT Cynicism still needs to be empirically tested. Thus, further research into the validity and reliability is still warranted in addition to a continuation to improve the design of the GNAT Cynicism. Thirdly, the ability of the GNAT Cynicism to predict cardiovascular disease or other health outcomes is absent compared to the number of studies that have reported significant ability of the 12-item Cynicism scale to do so. The increasing interest in Cynicism arises from evidence showing that it is predictive of future cardiovascular health status and mortality (Everson et al., 1997; Julkunen et al., 1994; Matthews et al., 2004). Hence in terms of health consequences, the GNAT Cynicism does not have the base of empirical evidence compared to the 12-item Cynicism. Fourthly, the GNAT Cynicism itself is not error free as well. It is also susceptible to false positives and false negatives. Given that the GNAT Cynicism is not measuring an identical construct as the 12-item Cynicism, it is incongruous to suggest that the GNAT Cynicism is a “better” measure of Cynicism. Hence, as was discussed for the 12-item Cynicism, the GNAT Cynicism, conversely speaking, also suffers from false positives and false negatives. For GNAT Cynicism, Repressed Cynicism represents false positive while Pseudo Cynicism

represents false negative. (For the 12-item Cynicism, Repressed Cynicism represents false *negative* while Pseudo Cynicism represents false *positive* [see figure 4.5].)

The introduction of the GNAT Cynicism was to develop another method of Cynicism assessment that is less amenable to positive self-presentation bias. In this study, this was successful. The proposal was then to use the GNAT Cynicism and the 12-item Cynicism together to derive a measure of Cynicism that has less measurement error. Specifically, to reduce the problem of false negatives (Repressed Cynicism) and false positives (Pseudo Cynicism) in the Cook-Medley 12-item Cynicism scale. In this chapter, this was accomplished via factor analysing these two measures together. Factor analysis revealed a 2-factor solution with the two measures of Cynicism correlating at .80 with the first factor. This was named as a 'Cynicism Factor'. This factor is a suitable replacement for the Cook-Medley 12-item Cynicism scale because of its high correlation with this scale. The second factor was labelled as the 'Pseudo Cynicism Factor', which represents the measurement error of the 12-item Cynicism scale. At this point, it is perhaps a bit misleading to label Factor 2 as measurement error. Describing Factor 2 as 'measurement error' gives the wrong impression that either the GNAT Cynicism or the 12-item Cynicism is the 'correct' measure of Cynicism. This is because low scores on Factor 2 represents Repressed Cynicism (low 12-item Cynicism and high GNAT scores). Repressed Cynicism refers to individuals who inhibit explicit admission of cynicism due to positive presentation bias. If operationalised as low

Factor 2 scores, it then implies that GNAT Cynicism is the 'correct' index of Cynicism while the 12-item Cynicism is not. This is misleading. As was discussed earlier, GNAT Cynicism and 12-item Cynicism scale do not measure identical constructs, so it is incongruous to conclude which is a better measure of Cynicism. Both measure different types of Cynicism, which in turn are susceptible to different sources of error. The justification for defining Factor 2 as 'measurement error' is that the researcher defines measurement error. In this case, the focus is on Cynicism (Factor 1). Therefore any residual variance from the factor analysis can be correctly referred to as 'measurement error'. Factor analysis also reduced the correlation between Cynicism and negative affectivity. This indicates that discriminant validity in Cynicism assessment can be improved via the method adopted in Study 3.

Factor 2 (Pseudo Cynicism Factor) represents a disparity between two different forms of Cynicism assessment. Factor 2 (Pseudo Cynicism Factor) represents the direction of this disparity. Thus, low Pseudo Cynicism scores represent negative 12-item Cynicism and positive GNAT Cynicism scores while high Pseudo Cynicism scores represent the reverse disparity. Fortunately, these disparities are not frequent as they account for only about 35% of the variance from GNAT Cynicism and Cook-Medley Cynicism contribute to such disparities. Repressed Cynicism may represent an attempt by the participant to present himself/herself in a positive light, or a lack of awareness of their implicit cynical attitudes (Barefoot, 1992; Barefoot & Lipkus, 1994). Implicit attitudes are considered more to be more spontaneous and time efficient

(Bargh, 1994). Pseudo Cynicism may represent a situation where a person has an explicit attitude of Cynicism but has yet to internalised such attitudes into a more spontaneous implicit form. It is also unlikely that Factor 2 measures Cynicism, given the opposite factor loadings from both the GNAT Cynicism and 12-item Cynicism scale. The correlation with negative affectivity and basal levels of State Anger suggests that this Factor measures a form of negative affectivity.

By interpreting the results from the various forms of Cynicism assessment (12-item Cynicism, GNAT Cynicism, Cynicism Factor, and Pseudo Cynicism Factor), it will be possible to identify the significant cardiovascular reactivity results that are attributable to Cynicism, measurement error or both. It is this interpretation that is discussed in the next few paragraphs. There are three core hypotheses being tested in this chapter: the relationship between Cynicism and Cardiovascular Reactivity, the Cynicism-Reactivity relationship moderated by Control, and the Cynicism-Reactivity relationship moderated by State Anger. The discussion of the results and its implications will follow these three main categories.

Cynicism and Cardiovascular Reactivity

Cynicism as measured by the Cook-Medley Hostility scale has been found to be associated with a higher risk of developing cardiovascular disease. Some researchers proposed that people with high levels of Cynicism have greater cardiovascular reactivity to stress. This places greater stress on their cardiovascular system and with time,

may hasten the process of atherosclerosis due to endothelium damage (Williams & Barefoot, 1988). However, there has been an emergence of null and significant negative findings being published for the Cynicism-Reactivity hypothesis (Burns & Katkin, 1993; Carroll et al., 1997; Felsten, 1995; Felsten, 1996; Fichera & Andreassi, 1998; Sallis et al., 1987; Spoth et al., 1992; Weidner et al., 1989). The findings for this study and study in Chapter 3 are congruent with the pool of studies showing null results between Cynicism and reactivity.

One reason for null results could be that the relationship between Cynicism and cardiovascular reactivity could be nonlinear. A few studies that have reported significant results for the Cynicism-Reactivity effect have selected groups of extreme Cynicism scores (Smith et al., 2000; Suarez et al., 1998; Suarez & Williams, 1989). If the relationship between Cynicism and reactivity is only present for extremely high and extremely low Cynicism scores, and not present for the majority of participants scoring close to the mean, a non-significant linear effect and significant cubic effect for the Cynicism-Reactivity could help explain the discrepant findings. The results from this study suggest that this is unlikely to be the reason for blood pressure reactivity. The nonlinear terms for the 12-item Cynicism were all not significant for blood pressure reactivity. However, the absence of significant results for blood pressure reactivity reflect different underlying hemodynamic processes for participants with Cynicism scores greater than $\pm 1SD$ from the mean. Participants with 12-item Cynicism scores $>+1SD$ have greater CO reactivity while participants with 12-item Cynicism scores $<-1SD$ have

reduced CO reactivity. The GNAT Cynicism predicted the reverse pattern for TPR reactivity. The effect size (η^2) for TPR reactivity between GNAT Cynicism and Cynicism Factor remained unchanged (.06). The presence of these same effects with identical effect sizes for CO and TPR for Cynicism Factor suggests that this is due to Cynicism. The significant effect for CO reactivity for the 12-item Cynicism increased from .04 to .08 when the Cynicism Factor was used. This indicates greater power for CO reactivity with a reduction in measurement error for Cynicism. In addition, a significant quadratic trend for heart rate was also found between 12-item Cynicism and the Cynicism Factor with comparable effect sizes (.04 and .03 respectively). Greater HR reactivity was found for participants scoring Cynicism scores (on the 12-item Cynicism scale and Cynicism Factor) $>+1.5SD$. The role of heart rate reactivity and the risk of cardiovascular disease is at present unclear. While there has been a number of experimental studies done with nonhuman primates showing that high heart rate reactivity is significantly associated with atherosclerosis (Bassiouny et al., 2002; Kaplan et al., 1987; Manuck et al., 1983; Manuck et al., 1988; Manuck et al., 1997), no studies have generalised these findings for humans. In fact, Fauvel et al. reports a significant positive association between resting baroreflex sensitivity and increased heart rate reactivity (Fauvel et al., 2000)²³. The Baroreflex arc modulates heart rate in order to reduce blood pressure variability. So increased heart rate reactivity could mean increased cardiac sympathetic

²³ Although not the focus of this dissertation and thus not reported, this dissertation also found significant positive associations between BRS and HR reactivity in Chapter 3 in

activity (which may increase cardiovascular disease risk) or increased baroreflex modulation of heart rate via increased cardiac parasympathetic activity to reduce blood pressure variability (which may decrease cardiovascular disease risk). Hence unless the source of this heart rate reactivity is ascertained, it would be difficult to interpret the health implications this finding for the quadratic relationship between Cynicism and heart rate reactivity. A re-analysis of study 2's dataset for Normalised HF HR (to index cardiac parasympathetic activity) and baroreflex sensitivity failed to reveal any significant effects for this quadratic term of the 12-item Cynicism scale. Hence, the reason for this quadratic relationship between Cynicism and heart rate reactivity obtained in this chapter is inconclusive.

The derivation of the Cynicism and Pseudo Cynicism Factors was to determine the sources of the significant effects and the influence of Cynicism, Repressed Cynicism and Pseudo Cynicism on cardiovascular reactivity. If Repressed Cynicism do not represent a true low Cynicism score, then their cardiovascular stress reactivity could be an average of the sample, or greater than the sample mean. Without the reduction or identification of Repressed Cynicism, a researcher can obtain null or negative findings depending on the influence of Repressed Cynicism in his/her sample. The results of this study support this finding. The quadratic relationship between Pseudo Cynicism and blood pressure reactivity show that Repressed Cynicism is related to greater blood pressure reactivity. This is particularly evident for two participants in this

sample who had extreme Repressed Cynicism scores (i.e., scores less than -2SD on the Pseudo Cynicism Factor, [figure 4.12]). For these two individuals with extremely high Repressed Cynicism, blood pressure reactivity was largely vascular-mediated. These two individuals have a 12-item Cynicism score of '2'. The result for this study suggests that Repressed Cynicism can contribute to the null or negative findings of the sample.

The investigation of Pseudo Cynicism (high explicit and low implicit Cynicism) has not been the focus of research. The results involving Pseudo Cynicism indicate that Pseudo Cynicism should be as much of a concern for the measurement error of the 12-item Cynicism scale as Repressed Cynicism. This is because participants with extremely high Pseudo Cynicism scores ($>+2SD$) also tend to have greater blood pressure reactivity. The source of this blood pressure reactivity is unclear as TPR reactivity does not increase and there was a lack of significant finding for CO reactivity. If the researcher has a number of participants with high Pseudo Cynicism in his/her sample, this may inflate the Cynicism-Reactivity relationship to be significant and provide an inaccurate estimate of the "true" effect size. The significant findings for blood pressure reactivity for Repressed Hostility and Pseudo Cynicism gives credence to the proposition that false negatives and false positives can produce misleading patterns for the Cynicism-Reactivity relationship.

It is also of interest to note that both Cynicism Factor and Pseudo Cynicism Factor show similar patterns of TPR reactivity with identical effect sizes (.06). The difference between the two being that while TPR

reactivity was found to be associated with blood pressure reactivity for Repressed Hostility, no such blood pressure effects were present for the Cynicism Factor. This suggests that TPR reactivity for Pseudo Cynicism may be associated with cardiovascular hemodynamic homeostatic dysregulation while Cynicism is not.

Overall, the results for the Cynicism main effect from this study suggest that it is important to reduce the influence of participants with extremely high Repressed Cynicism and Pseudo Cynicism in the sample for the assessment of Cynicism. As was mentioned, extreme Repressed Cynicism and Pseudo Cynicism scorers tend to be relatively few; the majority of low 12-item Cynicism scorers tend to be low in both implicit and explicit Cynicism. Such a definition of Cynicism is perhaps the least problematic as it avoids the issue of choosing whether implicit or explicit Cynicism assessment is 'superior'. Since Cynicism and Pseudo Cynicism Factor are both uncorrelated, the researchers may proceed to investigate the effects of Pseudo Cynicism Factor on cardiovascular stress reactivity in the same statistical model as the Cynicism Factor. However, a limitation of analysing Pseudo Cynicism Factor is that it is unreliable. This is because the main focus of using factor analysis in this chapter was to produce a composite measure of Cynicism and reduce the collinearity between GNAT Cynicism and 12-item Cynicism to include both in the same regression model. The factor analysis resulted in about 35% of the variance between these two measures being used to derive the Pseudo Cynicism Factor. This means that if the focus (or one of the focus) of the research is on Pseudo Cynicism Factor then its measurement error, when

it is derived using the current method, is 65% (that used to derive the Cynicism Factor). In this dissertation, the focus is on comparing and contrasting the results obtained from the three forms of Cynicism assessments (i.e., 12-item Cynicism, GNAT Cynicism and Cynicism Factor) for cardiovascular reactivity.

Nevertheless, this study found a lack of significant findings between blood pressure reactivity and Cynicism, whether it is operationalised by the 12-item Cynicism scale, GNAT Cynicism or the Cynicism Factor. A number of researchers who have noted the emergence of null findings for Cynicism main effect have investigated and suggest the role of moderators for the Cynicism-reactivity relationship (Bishop & Robinson, 2000; Bongard et al., 1998; Suarez & Williams, 1989; Why et al., 2003). Two of these moderators investigated in this dissertation are control and state anger.

Though discussed separately, control and state anger are related. Appraisal theories of emotion have shown that one of the characteristics of anger arousal is the controllability of an aversive event or obstruction of a desired goal (Berkowitz, 2000). Obstruction of a goal is an element that defines the construct of 'control' (Skinner, 1996). Averill conducted surveys and the common reason given for anger arousal is frustration (Averill, 1983). Frustration can be conceptualised as goal obstruction (Dollard et al., 1939). Thus, reducing task controllability to create an obstacle to goal attainment can increase state anger. The correlation between Perceived Control and State Anger found in this study and Study 2 support this.

A number of studies have indicated that anger and control moderate the Cynicism-reactivity relationship (Everson et al., 1995; Smith & Brown, 1991; Suarez & Williams, 1989). However the methodology used by these studies implicate interpersonal conflict with control and state anger. For state anger inducement, the harassment procedure is used (Everson et al., 1995; Suarez & Williams, 1989) while for control, a task involving social conflict is used (Smith & Brown, 1991). There is evidence suggesting that Cynicism functions within a social context; high hostile individuals have greater ambulatory blood pressure during social interaction (Brondolo et al., 2003; Gyll & Contrada, 1998) and this is partly due to the fact that their interpersonal skills are perceived as less effective than low hostile individuals (Watkins & Eisler, 1988). It is not known whether state anger and control that is created experimentally using a procedure that does not involve interpersonal conflict would indicate that anger and control (induced from non-interpersonal causes) can moderate the Cynicism-Reactivity Hypothesis. Such an investigation will help to disentangle the role of interpersonal conflict from anger and control in moderating the Cynicism-Reactivity hypothesis. The current method of using a computer task (Synwork) with a mouse that had adjustable efficiency (to manipulate task controllability) was to arouse non-interpersonal state anger and control.

*Cynicism and Reactivity moderated by Control*²⁴

In study 2, Perceived Control was not found to moderate the Cynicism-Reactivity relationship. One reason for this lack of significant findings for Perceived Control as a moderator could be a lack of power due to the use of Likert scales. In this study, Perceived Control was assessed using a visual analogue scale. Even so, this study did not find any significant Cynicism by Perceived Control interaction for cardiovascular reactivity.

Could it be that using a visual analogue scale only increases error variance rather than increase power? This is an unlikely explanation as Russell and Bobko found that a visual analogue scale has comparable reliability as a Likert scale (Russell & Bobko, 1992). Rather, research has consistently shown that reducing a continuous scale into categories reduces power and increases Type 1 error (Cohen, 1983; MacCallum et al., 2002; Maxwell & Delaney, 1993). So it is more likely for Likert scales to be unreliable compared to continuous scales. The results from this study also suggest that visual analogue scales have greater power. The Cynicism by State Anger_{btw} interaction was significant in this study and not in Study 2. In Study 2 where the Likert scale was used, neither of the interactions between Cynicism and Perceived Control, and between Cynicism and State Anger_{btw} were significant. These points indicate that a

²⁴ In this dissertation both Perceived Control_{btw} and Perceived Control_{wtm} are analysed. In this discussion, Perceived Control (without any subscripts) refers to both forms. The same is done when discussing State Anger.

visual analogue is likely to have greater power rather than increased error variance.

In study 2 when Perceived Control was assessed using a 7-point Likert scale, Perceived Control_{wtn} predicted the same variance as Task Control. In addition, Task Control predicted about 60% more variance than Perceived Control_{wtn}. However when Perceived Control was tapped using the visual analogue scale, both Perceived Control_{wtn} and Task Control do not tap common variance consistently. When Task Control was entered into the regression model first, there was often an attenuation of Perceived Control_{wtn}'s effect size (table 4.16). In Study 2, Perceived Control_{wtn} was set to '0'. Moreover, Perceived Control_{wtn} for Study 2 predicted CO reactivity, Perceived Control_{btw} for this study predicted CO reactivity.

Discrepancies between Perceived Control and Task Control predicting common or unique variance is contributed by the type of scale used to assess Perceived Control. As was discussed in the introduction (chapter 1), there has been research evidence to support the importance of both perceived control and actual control. Perhaps part of the controversy lies in the way Perceived Control is assessed. If Perceived Control is assessed using a Likert scale then its ability to predict cardiovascular reactivity can be compared to actual control since they both tap the same variance. If Perceived Control is assessed via a visual analogue scale, Perceived Control and actual control do not tap the same variance. If so, it is then not possible to compare whether Perceive

Control is 'superior' to actual control or not as both are predicting different variance. The results obtained between study 2 and study 3 suggests that how Perceived Control is tapped influences the sort of significant results obtained. Clearly more research is required to investigate into the reasons for such discrepancies for visual analogue scale-derived versus Likert scale-derived Perceived Control.

Such discrepancies between Perceived Control and actual control (Task Control) moderated by the mode in which Perceived Control is measured should not distract the reader from the general congruence between Perceived Control and Task Control. That is, Variable and More Variable Pauses are associated with low Perceived Control (no matter how it is measured) compared to Normal Task Control. The concordance between Perceived Control and Task Control show that people are generally accurate in actual control assessment with minor discrepancies.

When Task Control was used to index actual control, a significant Cynicism by Task Control interaction was found. The presence of this significant for both the 12-item Cynicism and Cynicism Factor indicate that this effect is likely to be due to Cynicism. However, the pattern of this interaction was the reverse of the hypothesis. Instead of participants with high Cynicism having greater TPR reactivity, participants with low Cynicism have greater TPR reactivity as Task Control got lower. The effect size obtained for this effect for 12-item Cynicism scale in this study is much larger (.07) than that obtained in study 2 (.02). Perhaps it can be said that a comparison of TPR reactivity between the two studies is unfair as significant Task Control differences were found for CO reactivity in

Study 2. A comparison of the effect sizes for CO reactivity between the two studies also revealed smaller effect sizes for Study 2 than Study 3 (.001 & .03 respectively). This could be because there was some time difference in Study 2 between the personality assessment and the laboratory stress testing. In Study 3, the personality assessment occurred prior to the laboratory stress testing. The time difference between the personality assessment and laboratory stress testing for Study 2 may affect the reliability of the 12-item Cynicism in accordance with its test-retest reliability. Therefore, there is a need to ascertain the test-retest reliability of this 12-item Cynicism scale. Having Cynicism assessment prior to the experiment may also serve to prime the participants' Cynicism and Cynicism-related behaviours. Subjects who had their attitudes on racial stereotype primed showed more hostility towards a confederate of that ethnic group (Bargh, Chen, & Burrows, 1996). In this study, having participants perform the GNAT Cynicism and 12-item Cynicism assessment may have primed Cynicism. This may increase the likelihood of detecting the Cynicism-Reactivity effects. Another reason for the significant Cynicism by Task Control interaction for TPR reactivity found in this study could be due to variations due to the sampling process. Replication will enable one to ascertain the stability of this effect.

The general finding for control (whether perceived or actual) is that it does not moderate the Cynicism-Reactivity relationship. Some results obtained from the 12-item Cynicism scale and Cynicism Factor indicate that low but not high Cynicism is associated with greater TPR reactivity.

These TPR effects did not result in any corresponding changes in blood pressure.

Cynicism and Reactivity moderated by State Anger

Reducing Task Controllability decreased Perceived Control and also increased State Anger. This form of State Anger induced is not due to interpersonal conflict. This is to investigate whether State Anger aroused through non-interpersonal means moderate the Cynicism-Reactivity relationship. In this study State Anger was also measured using the visual analogue scale.

For the 12-item Cynicism scale, significant Cynicism by State Anger_{btw} interaction for blood pressure was found. The pattern of this interaction supported the hypothesis that State Anger moderates the Cynicism-Reactivity relationship. For participants with high Cynicism, they had greater blood pressure reactivity when State Anger_{btw} was high compared when it was low. Of interest to note was that participants who have low 12-item Cynicism scores have greater (or as much) blood pressure reactivity when State Anger_{btw} was low (figure 4.23). The results for TPR indicate that the Cynicism by State Anger_{btw} interaction for blood pressure is mediated by TPR. Girdler et al. also reported greater TPR stress reactivity among high hostile men (Girdler et al., 1997). However when the Cynicism Factor was used, the Cynicism by State Anger_{btw} interaction was significant only for CO and TPR reactivity. The pattern for this interaction obtained using the Cynicism Factor is similar compared to that obtained for the 12-item Cynicism with the exception that participants with high Cynicism Factor scores have the greatest TPR reactivity when

State Anger_{btw} was high (figure 4.27). The greater TPR reactivity for low Cynicism scorers during low State Anger_{btw} is reduced. In addition, for Cynicism Factor, Cynicism by State Anger_{btw} interaction for CO is of borderline significance. Both TPR and CO regulate blood pressure adequately resulting in a lack of significant Cynicism by State Anger_{btw} interaction for blood pressure, which was present for 12-item Cynicism. The plethora of significant results obtained for 12-item Cynicism and GNAT Cynicism involving nonlinear Cynicism terms (table 4.17) was also not present for the Cynicism Factor. The nonlinear Cynicism effects are due to extreme scores (scores greater than $\pm 2SD$) on the 12-item Cynicism and GNAT Cynicism. These extreme scores are likely to be influenced by false positives and false negatives in these two Cynicism measures. As the Cynicism Factor reduces the influence of these data points, these effects are no longer significant.

The current study lends support for the hypothesis that the Cynicism-Reactivity relationship is moderated by Anger. However in this study there was support for only the weaker version of this moderation hypothesis. This is because there were no significant Cynicism by State Anger for State Anger_{wtn}. Cynicism by State Anger_{wtn} interaction represents a stronger test of the moderation hypothesis because State Anger is manipulated via Task Control conditions. Variable and More Variable Pauses conditions are associated with significantly higher levels of State Anger than the Normal Task Control condition. Therefore, if Cynicism-Reactivity is moderated by State Anger, increasing the State Anger for a person with high Cynicism will increase cardiovascular

reactivity. Conversely, lower levels of Anger will also be associated with lower cardiovascular reactivity. Though Cynicism is a personality trait that cannot be manipulated or randomised, State Anger can be manipulated. However, there was no evidence to support this stronger version of the moderation hypothesis. A weaker version, Cynicism by State Anger_{btw} was found. This is a weaker test of the moderation hypothesis because it requires different individuals (with similar levels of Cynicism) with different levels of State Anger predict different levels of cardiovascular reactivity. This raises questions about the status of the moderation hypothesis because both Cynicism and State Anger are not manipulated. If everyone received the same stressor (averaged Synwork with three levels of Task Control), why did two persons with equivalent levels of Cynicism report different levels of State Anger? This opens the probability of a third factor (e.g., anger expression) interacting with Cynicism if that third factor is associated with increased ratings of State Anger_{btw}. At present there have been no known studies that have differentiated between effects due to Cynicism by State Anger_{btw} and Cynicism by State Anger_{wtn}. Studies conducted in the laboratory test the Cynicism by State Anger_{btw} interaction (Felsten, 1995; Suarez & Williams, 1989). Brondolo et al. is the only known study to use a sophisticated statistical method (Mixed Model) to model the effects of Cynicism, social interaction on ambulatory blood pressure and heart rate. They reported a significant interaction between Barefoot et al.'s (1989) Cynicism subscale (a closely matched comparison with the 12-item Cynicism scale used in this dissertation) and negative ratings of the social interaction in predicting DBP reactivity

(Brondolo et al., 2003). Specifically, as high hostile rated the social interaction as more negative, their DBP levels increased as well. Though Brondolo et al. measured mood, they did not report any effects on Cynicism by mood interactions in predicting blood pressure reactivity.

In addition, though this study found support for the Cynicism by State Anger_{btw} interaction for TPR reactivity, there are some differences with past studies. Some researchers proposed that high hostile individuals are more reactive only when they reported higher State Anger than low hostile individuals (Suarez & Williams, 1989; Suls & Wan, 1993). Based on the results of this study and other published studies, there was no evidence that this is a necessary and sufficient condition for a significant Hostility-Reactivity effect. In this study, State Anger given by participants after the Synwork sessions were not significantly related to Cynicism. Despite this lack of significant association between Cynicism and State Anger, the interaction for TPR was found to be significant and for CO, the interaction was of borderline significance. Felsten's study also reported that even when high hostile individuals reported higher levels of state anger due to harassment, high hostile individuals do not have greater cardiovascular reactivity (Felsten, 1995). Everson et al.'s study also replicated the Hostility by Harassment interaction for blood pressure reactivity (Everson et al., 1995). High hostile participants gave significantly higher ratings of irritation during the harassing task than the neutral task compared to low hostile participants. However irritation (or any of the other mood ratings) was not related to cardiovascular reactivity for high hostile individuals. Contrary to Suarez and Williams' (1989)

proposition, high hostile participants having higher anger than low hostile participants is not a necessary and sufficient condition for anger to moderate the Cynicism-Reactivity relationship. Perhaps a way to resolve these problems entails the assessment of the hemodynamic processes (CO and TPR) underlying blood pressure. The same blood pressure reactivity can often reflect different hemodynamic processes (Sherwood & Turner, 1992). It is possible that the discrepant findings for the Cynicism by State Anger interaction for blood pressure reactivity can actually reflect the same hemodynamic processes found in this chapter. However, as reviewed in Chapter 1, there is yet no consistency in terms of Hostility and the underlying hemodynamic profile of blood pressure regulation.

Nevertheless, the conditions whereby a significant Cynicism by State Anger interaction for blood pressure reactivity can be elicited remain uncertain. This may be due to the fact that mood ratings per se have not been found to relate to cardiovascular reactivity consistently, which raises questions about the reliability of mood ratings. Regardless of how State Anger was assessed, whether by Likert scales (Study 2) or visual analogue scales (Study 3), State Anger_{btw} and State Anger_{wtn} main effects were not significant. Schwartz suggests that current methods of operationalising mood may be unreliable and have reduced variance (Schwartz, 1999). Based on Schwartz's (1999) recommendation, steps were taken in this study to maximise power, reliability and variance for the measurement of Perceived Control, State Anger and Cynicism in this study. ANCOVA was done instead of using gain scores to operationalise reactivity in order to increase the reliability of reactivity and overall power.

Three Task Control conditions were included to elicit different levels of State Anger so that Normal Task Control can be a 'control' condition for testing the effect of State Anger_{wtn} on cardiovascular reactivity. The use of visual analogue scales increased the variance for mood ratings for Study 3 compared to Study 2. In addition, variance for mood ratings was also increased via having Task Control as a repeated measure. Thus, variance for state anger is obtained within each participant as well between each participant. The effect sizes of State Anger_{wtn} and State Anger_{btw} main effects for TPR reactivity are extremely small ($.03 \times 10^{-2}$, and $.06 \times 10^{-2}$ respectively). In Study 2, the respective effects sizes were .03 and .01. Feldman et al.'s meta-analysis revealed that effect sizes for anger mood predicting cardiovascular reactivity range from .0064 to .12 (Feldman et al., 1999). The results of study 2 and 3 suggest that the lack of significant results between state anger and cardiovascular reactivity is only marginally attributable to (i) the use of gain scores, and (ii) the variance of mood ratings. This does not exclude other problems with current methodology of mood assessment (e.g., post-treatment measurement of mood instead of concurrent mood measurement with stress). The lack of significant State Anger main effects but significant Cynicism by State Anger_{btw}, raises the possibility that participants with high Cynicism who give higher State Anger ratings may have specific traits that increase their likelihood of experiencing and reporting state anger.

Conclusion

Using Study 2's design, this study also found that Task Control operationalised as response-outcome contingency is related to passive/active coping response. Like Study 2, TPR reactivity between Variable and More Variable Pauses conditions were not significantly different. The reason for this is due to a technical oversight in the creation of the Faulty Mouse device. Specifically, the number of random pauses generated by this device was not fixed. However, the overall pattern of results revealed that response-outcome contingency provided a better model fit than Synwork Task Scores and Perceived Control. This indicates that Task Control should be defined by response-outcome contingency rather than efficacy of coping behaviours.

This chapter also detailed the development of the GNAT Cynicism as a form of implicit assessment of Cynicism. The convergent and discriminant construct validity for the GNAT Cynicism was good. The GNAT Cynicism was then used to predict cardiovascular stress reactivity during the Synwork. Factor analysis for both 12-item Cynicism scale and GNAT Cynicism derived two factors on which the first is labelled as the Cynicism Factor. The other factor was labelled Pseudo Cynicism factor representing the axis of Repressed Cynicism (low 12-item Cynicism and high GNAT Cynicism) and Pseudo Cynicism (high 12-item Cynicism and low GNAT Cynicism).

Results obtained when Cynicism was operationalised as the 12-item Cynicism, GNAT Cynicism and Cynicism Factor were compared to

discern whether the effects were due to Cynicism or measurement error. In addition, nonlinear effects of Cynicism were also explored.

The analysis revealed some significant nonlinear effects for the Cynicism-Reactivity relationship. Participants who had Cynicism scores above +1SD tend to have active coping physiological responses while those with Cynicism scores below -1SD tend to have passive coping physiological responses. Of interest is that 12-item Cynicism predicted the pattern for CO, GNAT Cynicism predicted the pattern for TPR, while the Cynicism Factor predicted the same pattern for both CO and TPR. Result from the Pseudo Cynicism Factor also suggests that that high Repressed Cynicism and Pseudo Cynicism are associated with greater blood pressure reactivity. Thus a failure to take assess or reduce the influence of these traits when assessing Cynicism may lead to null or conflicting results. The overall pattern for the Cynicism main effect is that extreme scores on the 12-item Cynicism are likely to be the source of the Cynicism-Reactivity relationship.

Perceived Control, assessed using a visual analogue scale, did not moderate the Cynicism-Reactivity relationship. Studies that have reported significant results showing that interpersonal control moderates the Cynicism-Reactivity have used tasks involving interpersonal conflict (Smith & Brown, 1991). The results from this chapter suggest that it is not control that is the crucial element in moderating the Cynicism-Reactivity but it is more likely to be anger. Using a computer task and manipulating the controllability of this task, non-interpersonal state anger was successfully increased among participants in this study. Participants with

high Cynicism Factor scores and who reported high anger had greater TPR reactivity and lower CO reactivity (passive coping physiological response) than high cynical participants who reported less anger. Those with low Cynicism scores showed the reversed pattern. Comparing the results obtained for the 12-item Cynicism scale and the Cynicism Factor revealed that when the influence of Repressed Cynicism and Pseudo Cynicism traits was not reduced in the 12-item Cynicism, low Cynicism participants had as much or greater blood pressure reactivity and TPR reactivity during low anger than high Cynicism participants during high anger. When the influence of Repressed Cynicism and Pseudo Cynicism was reduced via using the Cynicism Factor, high Cynicism with high state anger had the greatest TPR reactivity and the interaction between Cynicism and state anger was no longer significant for blood pressure. Thus, the 12-item Cynicism by State Anger interaction for blood pressure reactivity can be influenced by Repressed Cynicism and Pseudo Cynicism.

However, the Cynicism by State Anger effect was only present in the weaker version. There is an absence of Cynicism by State Anger_{wtn} interactions for cardiovascular reactivity. This means that manipulating the state anger of a person with high Cynicism did not influence their cardiovascular reactivity in the expected direction as predicted by the anger moderation hypothesis. Furthermore, State Anger was not related to cardiovascular reactivity, which questions the reliability of the mood ratings. This suggests that participants who have high Cynicism and who report high anger ratings may belong to a subset of Cynicism who are

more likely to admit their anger experiences. That is because increasing the state anger of the other individuals with high Cynicism do not increase their cardiovascular stress reactivity.

The next chapter (Main Discussion) will discuss the overall conclusions for this dissertation and will delve into greater detail some of the points raised in this discussion.

CHAPTER 5

GENERAL DISCUSSION

Hostility has been found to be related to cardiovascular disease risk and mortality. Researchers have proposed that high hostility individuals are at greater risk of developing cardiovascular disease because they have greater cardiovascular stress arousal (the Cardiovascular Reactivity Hypothesis). Another pathway could also be that high hostile individuals have a profile of autonomic nervous system activity (high sympathetic, low parasympathetic) and reduced baroreflex sensitivity that increases blood pressure variability, increasing their risk of cardiovascular disease (the Cardiovascular Variability Hypothesis).

There is evidence supporting both Hypotheses as pathways linking Hostility to cardiovascular disease. High hostile individuals have been reported having greater cardiovascular reactivity when harassed (Everson et al., 1995), engaging in debates (Smith & Allred, 1989) and having social interactions (Brondolo et al., 2003). Reduced sympathetic activity due to sympathetic down-regulation and reduced parasympathetic modulation of heart rate among high hostile individuals have also been reported (Fukudo et al., 1992; Hughes et al., 2003). However, there are also a number of studies that have reported null findings between Cynicism and cardiovascular stress reactivity (e.g., Felsten, 1995). A few other studies have even reported negative findings. Specifically, some studies have reported results that indicated low Cynicism and not high

Cynicism is associated with greater cardiovascular reactivity (Bongard et al., 1998; Carroll et al., 1997; Durel et al., 1989; Siegman et al., 1991).

Two explanations for such occurrence of discrepant findings for Hostility-Reactivity link are suggested and explored in this dissertation. Firstly, the measurement of Cynicism is imprecise. Measurement error leads to low power and can also result in null or negative findings. A review of the literature in chapter 1 and 2 showed that there is lack of empirical agreement on the internal psychometric structure of the scale. In addition, the current method of measuring Cynicism using the Cook-Medley Hostility scale also confounds several constructs with Cynicism (Eckhardt et al., 2004). Secondly, the Hostility-Reactivity link could be moderated by other variables. Two variables investigated in this dissertation are Control and State Anger. The two are related as appraisal theories of emotion have found that low Control through goal obstruction can increase State Anger. The findings and implications for these two explanations are now discussed.

Measurement Error in Hostility - a focus on Cynicism

Hostility can be distinguished into attitude (Cynicism), affect (Anger) and behavioural (Aggression) components. This dissertation used the Cook-Medley Hostility scale to measure Cynicism (Cook & Medley, 1954) and the Structured Interview-derived Hostile Behaviour Index (HBI) via the Interpersonal Hostility Assessment Technique (Haney et al., 1996). Though these two methods of Hostility assessment are susceptible to different sources of measurement error, the focus in this

dissertation has been on improving the measurement precision of the Cook-Medley Hostility scale. This is because this scale has been found to be significantly associated with cardiovascular disease in a meta-analysis and it represents the more common Cynicism assessment used. In addition, most of the null and negative results found for the Hostility-Reactivity effect have used this form of Cynicism assessment. This section of the discussion centres on the reduction of measurement error for the Cook-Medley Hostility scale. The next section discusses the effects of measurement error on the Cynicism-Reactivity effect.

One of the problems with this scale is that there has been no consistent empirical evidence for its internal psychometric structure. This is compounded by the problem when different researchers use different subset of the items for their own research. If there is no known stable internal structure for this scale, the selection of subset of items as measuring a particular aspect of Hostility (e.g., Cynicism) cannot be justified. Yet this is one of the ways ambiguity in terms of the construct assessed by this questionnaire can be reduced. In Chapter 2 of this dissertation, four factor analytic studies were reviewed. The overall pattern of item loadings for the 50-items of the Cook-Medley Hostility scale was not consistent above chance level (i.e., occurring in more than 2 of the 4 studies reviewed). There was no support for Greenglass & Julkunen's 9-item model (Greenglass & Julkunen, 1989), or Barefoot et al.'s 27-item model (Barefoot et al., 1989). Only 12 items loaded consistently onto Factor 1 and 1 item loaded consistently onto Factor 2. The item content analysis done in the introduction of Chapter 2 revealed

that this 12-item Cynicism scale consists of mainly items identified as measuring Cynicism by Barefoot et al. (1989) with the exception of one item. The 12 items appear to measure the respondent's assessment of others as unreliable and selfish without implicating an evaluation of the respondent. The high correlation between the 12-item Cynicism version with the 50-item Cook-Medley Hostility scale (.79) indicates that the 12-item Cynicism version is a suitable substitute for the full 50-item scale. Correlations with Marlowe-Crowne Social Desirability Scale (Crowne & Marlowe, 1960), DS16 Negative Affectivity scale (Denollet, 2000), Desirability for Control scale (Burger & Cooper, 1979), and STAXI (Spielberger, 1988) revealed that using the 12-item Cynicism did not reduce the correlation between Cynicism and social desirability and trait anger reaction. However, compared to the unstable components of the Cook-Medley Hostility scale (represented by the 12-item Alternative), the 12-item Cynicism scale was less correlated to negative affectivity, and desirability for control. This indicated that using the 12-item Cynicism showed a slight improvement in discriminant validity for Cynicism. There are also other potential advantages with using the 12-item Cynicism scale. Firstly, since this is obtained from an empirical review of four factor analytic study with a combined sample of over 8,000, it has greater reliability. This is also supported by the confirmatory factor analysis done on another sample of 205 male participants in Chapter 2 supporting the psychometric structure for this scale. Secondly, based on the content analysis done by Barefoot et al., researchers can be more certain that what they are measuring Cynicism and not Cynicism with other unstable

Cynicism-related constructs. As discussed in Chapter 1, negative affectivity is singled out as a more serious issue for the discriminant construct validity of Cynicism because negative affectivity encompasses a broader range of negative affect (and related cognition) that are not congruent with the construct of Hostility. Negative affectivity includes anxiety and depression as well as anger (Watson & Clark, 1984). It can be argued that anger is related to Cynicism because anger is the affect component of Hostility. However, anxiety and depression should not be related to Cynicism, as it does not fit into the construct of Hostility. Hence a reduction in correlation between Cynicism and negative affectivity is a desired outcome of improving the discriminant construct validity of the Cook-Medley Hostility scale.

Improving the internal structure based on empirical evidence does not rectify another measurement problem associated with this scale - the problem of false negatives and false positives. High cynical participants may under-report their levels of trait cynicism due to impression management (social desirability) or a lack of introspective awareness (Barefoot & Lipkus, 1994). These individuals are referred to as repressed cynical individuals. Repressed Cynicism is of concern as they can influence the results within the sample to produce null or negative findings (see next section). At present, there does not exist a satisfactory method of detecting and reducing the influence of Repressed Cynicism in the sample. The Cook-Medley/Marlowe-Crowne method confounds high and low cynical individuals in operationalising Repressed Cynicism. This is because the Marlowe-Crowne is not measuring Cynicism.

Using the Structured Interview derived HBI is also not optimal as the correlation between HBI and the Cook-Medley scale tend to fluctuate widely from sample to sample. Recent data suggest that the HBI has low (.20 and below) correlation with the Cook-Medley Hostility scale. In chapter 2, HBI and the 50-item Cook-Medley correlated at -.17, HBI correlated with the 12-item Cynicism version at -.07. Unpublished data from Bishop et al.'s study also revealed that the 50-item Cook-Medley scale correlated with HBI at .14 (N = 243) and the 12-item Cynicism scale correlated with HBI at .08 (Bishop et al., 2001). Barefoot et al. reported a correlation of .24 between HBI and the 27-item Cook-Medley scale (Barefoot et al., 1993). Other studies have reported correlations of .38 (Räikkönen et al., 1999) as well as .57 (Davidson & Hall, 1995) between the 50-item Cook-Medley Hostility scale and the Potential for Hostility. The HBI is a recently revised version of the Potential for Hostility that concentrates on vocal stylistics. Compared to the Potential for Hostility, it appears from the studies that the HBI has a lower correlation with the 50-item Cook-Medley Hostility scale. This correlation is further reduced if a smaller number of questionnaires items (12-item or the 27-item version) from the Cook-Medley Hostility scale is used. Furthermore, verbal stylistics is a form of behaviour and this puts the HBI as measuring the behavioural component of Hostility (i.e., Aggression). Separating the Hostility construct into the Attitude, Affect and Behaviour allows a more multifaceted definition of Hostility. This allows for a more comprehensive view of what Hostility is and does not necessarily imply that the components are correlated with one another. If HBI measures verbal

Aggression and the Cook-Medley Hostility measures Cynicism, then the HBI is an unsuitable candidate to correct for measurement error in the Cook-Medley Hostility scale as it is not measuring the same Hostility dimension as the Cook-Medley Hostility scale.

In Chapter 4, an implicit form of Cynicism (GNAT Cynicism) assessment was developed and tested. In general, the GNAT Cynicism has a better profile in terms of discriminant construct validity than the Cook-Medley Hostility. The GNAT is not correlated with social desirability and negative affectivity. As discussed in the discussion section of Chapter 4, the encouraging results of the GNAT Cynicism should not be taken to imply that it is a superior measure of Cynicism than the 12-item Cynicism scale. In short, the reasons for not using the GNAT Cynicism as a substitute are: (i) it is moderately correlated with the 12-item Cynicism scale indicating that it is measuring a similar but not identical construct, (ii) the test-retest reliability and convergent construct validity for the GNAT Cynicism needs further research, and (iii) research into the ability of the GNAT Cynicism to predict health is absent compared to the Cook-Medley Hostility scale. Rather, the GNAT Cynicism was developed in this dissertation as a means to reduce measurement error for Cynicism assessment. This is done with the implicit acknowledgement that both GNAT Cynicism and the 12-item Cynicism measures different forms of Cynicism (i.e., implicit and explicit respectively).

Factor analysing the GNAT Cynicism and 12-item Cynicism produced two factors accounting for 100% of the variance in both these scales. One factor labelled as the Cynicism Factor and another factor as

the Pseudo Cynicism Factor. The high correlation between the 12-item Cynicism scale and the Cynicism Factor (.80) makes the Cynicism a suitable candidate for the 12-item Cynicism. The Cynicism Factor further improved the discriminant construct validity of Cynicism assessment via reducing the association between Cynicism and negative affectivity. About 35% of variance from GNAT Cynicism and the 12-item Cynicism scale was used to derive the Pseudo Cynicism scale. This scale is associated with negative affectivity. The convergent construct validity for the Pseudo Cynicism measuring a form of negative affectivity is also indicated by its significant correlation with Baseline State Anger.

The general trend of results from this dissertation showed that improving the measurement precision of Cynicism through an evidence-based selection of a subset of 12 items and using an Implicit Cynicism measure to derive a Cynicism Factor decreased the correlation between Cynicism and negative affectivity. The next few sections discuss the effect of this improvement in the discriminant construct validity in Cynicism assessment in the pattern of results for cardiovascular reactivity/variability.

Hostility and Cardiovascular Reactivity/Variability

Some researchers have proposed that high hostile individuals are at a higher risk of developing cardiovascular risk because they have greater cardiovascular stress arousal. In addition, high hostile individuals could also have a pattern of cardiovascular variability associated with greater cardiovascular disease risk (i.e., high sympathetic activity, low

parasympathetic activity and reduced baroreflex sensitivity) (Suarez & Williams, 1992). In this dissertation, assessing cardiac output, total peripheral resistance, sympathovagal cardiac activity and baroreflex sensitivity allows a deeper understanding of the regulation of the cardiovascular system during stress.

The results of Studies 2 and 3 revealed that there was no linear relationship between Cynicism and cardiovascular reactivity/variability. On the contrary, there was a negative trend for heart rate reactivity and Baroreflex Sensitivity in Study 2 for Cynicism. In Study 2, HBI was also measured and this behavioural form of Hostility was also not related to cardiovascular reactivity, sympathovagal cardiac activity and baroreflex sensitivity during stress. Since the 12-item Cynicism and HBI are uncorrelated and measure the two aspects of Hostility, it was hypothesised in Study 2 that the combination of high Cynicism and high verbal Aggression (a multidimensional assessment of Hostility) was related to a poorer cardiovascular health profile during stress. Again, no such significant results were present. The results from Study 2 and 3 join the emerging trend of published studies reporting null and negative findings for the Hostility-Reactivity effect. Study 2 also extends the research on Hostility and sympathovagal cardiac activity and baroreflex sensitivity.

Besides using both HBI and the 12-item Cynicism scale to tap Hostility, Study 3 of this dissertation also explored the possibility of nonlinear effects between Cynicism and cardiovascular reactivity. This is because a few studies that have reported significant Cynicism-Reactivity

effects have pre-selected participants with extreme Cynicism scores (Smith et al., 2000; Suarez et al., 1998; Suarez & Williams, 1989). This suggests that the relationship between Cynicism and reactivity may be cubic - positive slope between Cynicism and reactivity for extreme Cynicism scores but a 'flat line' for Cynicism scores close to the mean. The cubic term for Cynicism was not significant for blood pressure reactivity. The results showed that participant with extreme Cynicism scores (typically $>\pm 1.5$ SD from the mean) were more likely to have greater heart rate reactivity. Participants with extremely high Cynicism score tend to have increased cardiac output reactivity and participants with extreme low Cynicism scores tend to have reduced cardiac output reactivity. This cubic relationship between Cynicism and cardiac output reactivity was not sufficient to create differences in blood pressure regulation. The dissertation did not find support for the explanation that Cynicism-Reactivity relationship for blood pressure reactivity is present in participants with extreme Cynicism scores.

Pre-selecting participants with high and low Cynicism scores provides a misleading effect size estimate for the linear effect between Cynicism and reactivity when the effect analysed is likely to be nonlinear. When pre-selecting participants based on Cynicism scores, Cynicism can only be analysed as a dichotomised variable. Dichotomising continuous variables increases Type 1 error (MacCallum et al., 2002). There are studies that have reported null findings when Cynicism is analysed as a continuous variable but when dichotomised, the results of Cynicism became borderline significant or significant (Julkunen et al., 1994;

Weidner et al., 1989). A number of studies reviewed in Chapter 1 have used extreme Cynicism scores or a categorised version of Cynicism. Until more research is done with the actual raw continuous Cynicism scores, it is difficult to get a reliable estimate of effect sizes to help plan for sample sizes in future studies.

Another explanation for the null findings in the Cynicism-Reactivity investigated in this dissertation was measurement error in Cynicism assessment. In this respect, using the 12-item Cynicism scale did not result in significant findings for blood pressure reactivity. Using GNAT Cynicism (implicit Cynicism) and Cynicism Factor also did not reveal any significant results for the Cynicism-Reactivity linear main effect. This does not necessarily invalidate these Cynicism measures as prediction of reactivity is not construct validation (Tomaka, Blascovich, & Kelsey, 1992). Congruent with a meta-analysis (Suls & Wan, 1993), perhaps it is likely that the Cynicism-Reactivity main effect is less than .05. The sample sizes used in Studies 2 and 3 may be too small to detect such small effect sizes.

Reducing the influence of false negatives (Repressed Cynicism) and false positives (Pseudo Hostility) within a sample was a more revealing endeavour. In Study 3, participants with high Repressed Cynicism and High Pseudo Cynicism had greater blood pressure reactivity. This was not present for high and low Cynicism Factor scores. At this point, it is appropriate to address the question - is Repressed Cynicism a form of *high* Cynicism? In this dissertation Repressed Cynicism is defined as participants with low 12-item Cynicism score and

high GNAT Cynicism score. As discussed in Chapter 4, claiming that Repressed Cynicism is a form of high Cynicism implies that the GNAT Cynicism is the accurate assessment of Cynicism. This runs into problems when the reverse occurs (i.e., high 12-item Cynicism and low GNAT Cynicism - Pseudo Cynicism). Since GNAT Cynicism cannot substitute 12-item Cynicism, whether Repressed Cynicism is a form of high Cynicism or not, cannot be satisfactorily answered at present. However, what can be argued is that Repressed Cynicism is *not* a form of low Cynicism. If both GNAT Cynicism and 12-item Cynicism measure two aspects of Cynicism then both has to be taken into account in deriving an estimate of Cynicism as is done in factor analysing these two variables.

The non-detection or non-identification of Repressed Cynicism is particularly problematic for Cynicism assessment. Not identifying and reducing the influence of participants with high Repressed Cynicism in a sample can result in null or negative findings. This is illustrated in Table 5.1 taking two participants sampled in Study 3. Pseudo Cynicism was derived from factor analysis. Therefore, it is standardised. The two participants with high Repressed Cynicism (extremely low Pseudo Cynicism Factor scores) have low 12-item Cynicism scores but average MAP (yp28b, table 5.1) or above average MAP reactivity (yp20b).

If by sampling error, a researcher has a number of repressed cynical participants in his/her sample, depending on the cardiovascular arousal of these individuals, null or negative findings can result. Table 5.1 is only presented here as a possible explanation for the emergence of null or negative findings in the Cynicism-Reactivity literature and cannot

Table 5.1 Two Participants with high Repressed Cynicism and their corresponding MAP Reactivity (Standardised Scores)

ID	12-item Cynicism	Pseudo Cynicism	Mean MAP Reactivity
yp20b	2 (-1.73SD)	-2.39	127.01 (+2.59SD)
yp28b	2 (-1.73SD)	-2.46	98.57 (+.25SD)

be taken as definite prove as there are relatively few extremely repressed cynical individuals in this sample. Though few, these individuals influence the pattern of some of the other results that will be discussed later. Of importance is that, prior to the development of the GNAT Cynicism, there was no satisfactory method of identifying and reducing the influence of such cases in a sample.

Hostility-Reactivity/Variability moderated by Control

As was mentioned throughout this dissertation, both Control and State Anger are related. Perceived Control consists of the relation between agent, means and ends (Skinner, 1996). Appraisal theories of emotion have highlighted that reducing the means to goal attainment can increase state anger (Stein & Levine, 1990). Studies 2 and 3 manipulated the controllability of a computer mouse and found that Perceived Control was related to State Anger arousal, particularly when there was low Task Control. The point of manipulating this form of control was to investigate the role of non-interpersonal control and state anger as moderators of the

Hostility-Reactivity relationship. This is because there has been a number of studies that have suggested that the Cynicism-Reactivity effect is present only in situations of social conflict. Current method of anger inducement and control manipulation in the laboratory used interpersonal conflict tasks that confound interpersonal conflict with anger/control manipulation.

Actual Control. It was proposed that Task Control (actual control) can be operationalised as a continuum of response-outcome contingency rather than the 'classical' definition using presence/absence of coping behaviour. The paradigm used here operationalised response-outcome contingency as the contingency between the participant moving the trackball mouse and the movement of the mouse cursor on the computer monitor. Other forms of response-outcome contingencies are also available - moving a pen (response) and writing (outcome), and pressing the 'Send' button (response) to send a facsimile (outcome). In these two instances, reduced response-outcome contingencies need not be equipment malfunction (e.g., pen tip blocked with ink) as was operationalised in this dissertation but can also refer to physical disabilities. For instance, a stroke patient may have difficulty controlling his/her fine hand movements for writing purposes. In this instance, the contingency between the patient's hand movement (response) and writing (outcome) may be low at the beginning but may improve with rehabilitation. Hence response-outcome contingency encompass a range of tasks not restricted to the paradigm used in this dissertation.

Results from Study 2 and 3 showed that response-outcome contingency can produce active and passive physiological responses even when coping behaviours are present. Moreover, even when coping behaviour was efficacious (high Synwork scores), low response-outcome contingency was still related to passive coping physiological response. In Study 2, the Task Control condition order was fixed and the average Task Scores obtained in each Task Control condition were significantly different from zero. This is due to a practice effect as Task Control condition gradually went from controllable to uncontrollable. Counterbalancing eliminated this practice effect. Even when coping behaviours were efficacious in producing Task Scores that were significantly different from '0' in Study 2, response-outcome contingency always provided a better model fit than Task Scores accounted for about 60% more variance than Task Scores. This was true in both Studies 2 and 3. The participants' perceptions of how efficacious their coping behaviours were (Perceived Control) also offered a poorer fit of the model than response-outcome contingency.

Actual control defined by the presence/absence of coping behaviour ('classical' definition of actual control) is much more restrictive and confounds the coping behaviour with Task Control. As Studies 2 and 3 show, presence of coping behaviour (efficacious or not) is not predictive of an active coping physiological response. The use of the classical definition of actual control can also lead to contradicting evidence that may lead researchers to suggest that Perceived Control is more predictive of the active/passive physiological response than actual

control. This can happen even when there are no discrepancies between the two if actual control is re-defined as a response-outcome contingency.

Certain classically defined active coping like the Stroop colour-word and mirror-tracing tasks have been shown can be associated with a passive coping physiological response (Hurwitz et al., 1993; Waldstein et al., 1997). The mirror-tracing task is a classically defined active coping task but it elicits passive physiological response (increased TPR reactivity while CO decreases or remain unchanged) (Kasprowicz, Manuck, Malkoff, & Krantz, 1990). However, response-outcome contingency for individuals unfamiliar with the mirror tracing task can be low. This is because using a mirror to draw something requires a reversal of hand movements. For people who are unfamiliar with this type of task, initiating the hand movements (response) may not produce the desired lines one wishes to draw (outcome). The Stroop task is also a task with low response-outcome contingency as it represents the reflex-like properties of automated a cognitive process (reading) competing against a less automated skill (colour naming) (MacLeod, 1991). As reading is a well-practised skill among individuals who have received more years of education, reading is a skill that is better rehearsed than naming colours. The Stroop effect is the result of competition between a well-practised skill (reading) and a less well-practised skill (naming colours). With time limitation reading takes precedence, as it is more automated and is executed faster. However, the outcome of the Stroop task is to say the name of the colour in which the word is printed. Hence, a response (colour-naming) does not always relate to the outcome (saying the colour)

as often the automated skill of reading causes the participant to read the word instead. Hence it can be viewed as a task with low response-outcome contingency.

Hence, task control operationalised as the presence/absence of coping behaviour runs into problems with the mirror-tracing and Stroop tasks as these are active coping tasks eliciting a passive coping physiological response. Task control operationalised as response-outcome contingency can explain such effects. In addition, though manipulating response-outcome contingency elicited the corresponding active and passive physiological response, there were no differences in terms of sympathovagal cardiac activity and baroreflex sensitivity.

Overall, Hostility assessed by the 12-item Cynicism and the HBI was not moderated by Task Control in predicting cardiovascular reactivity or variability. Study 3 found a significant effect for TPR reactivity which suggested the reverse - low, not high, cynical participants have greater TPR reactivity as actual control was reduced. The presence of the same result when the Cynicism Factor was used suggests that this result is due to Cynicism and not measurement error. However, no such corresponding effect for blood pressure or CO was found.

Perceived Control. Two forms of Perceived Control were analysed - Perceived Control_{btw} and Perceived Control_{wtn}. Perceived Control_{btw} was the average Perceived Control ratings for the three Task Control conditions and is used to predict the average cardiovascular reactivity/variability. Perceived Control_{wtn} retains the original values given

by the participants and is entered as a continuous within subject variable in replacement of Task Control.

When a 7-point Likert scale was used to assess Perceived Control, Perceived Control_{wtn} was positively related to CO reactivity. When a visual analogue scale was used, Perceived Control_{btw} predicted CO reactivity. A visual analogue scale has been found to be as reliable as a Likert scale (Russell & Bobko, 1992). An average score is more generalisable (Kamarck, Debski, & Manuck, 2000). Thus, Perceived Control_{btw} is more generalisable than Perceived Control_{wtn}. It is likely that the Perceived Control_{btw} significant effect for CO reactivity represents a general relationship between high Perceived Control and high CO reactivity. Perceived Control assessed via a Likert scale predicted the same variance as Task Control while Perceived Control assessed using a visual analogue scale did not predict the same variance as Task Control. It is unclear why this is so. This pattern of results seem to suggest that a Likert scale-derived Perceived Control is more likely to detect changes in Perceived Control as a within subject (i.e, random effect) while visual analogue-derived Perceived Control tend to be an accurate assessment of between subject differences. Further research is required to determine the causes of such differences between Perceived Control derived from these two methods. But neither Perceived Control_{btw} nor Perceived Control_{wtn} assessed using a Likert scale or visual analogue scale moderated the Hostility-Reactivity relationship. Both also did not moderate the Cynicism-Reactivity relationship. This shows that control is not the predictor of cardiovascular reactivity among high hostile

participants. Smith and Brown used a social conflict task and confounded both control/anger and social conflict (Smith & Brown, 1991). The results of this study suggest non-interpersonal control does not moderate the Hostility-Reactivity relationship.

Task Control offers a better model fit than Perceived Control even when a discrepancy exists between the two. Thus, response-outcome contingency is associated with active and passive coping physiological response. A presence of efficacious coping behaviour does not necessarily elicit active coping physiological response. The relation between Perceived Control and Task Control, and Perceived Control and CO reactivity appears to be dependent on how Perceived Control is measured. Specifically, whether it is measured by Likert or visual analogue scale. Task Control and Perceived Control (regardless of how it is assessed) have not been found to moderate the Hostility-Reactivity effect.

It is probable that the lower predictive power of Perceived Control (compared to Actual Control) can be attribute to how it was measured. Specifically, the item 'To what extend could you influence the outcome of the task?' was used in Studies 2 and 3 to tap Perceived Control. 'Outcome' here could refer to the Synwork task scores or the movement of the mouse. As Task Control was operationalised as response-outcome contingency that involved the participant's control of the mouse cursor on the monitor, perhaps an item that specifically taps this could increase the predictive power of Perceived Control. That is, an item such as 'To what extend could you influence the movement of the mouse to obtain the

desired outcome for the task?' perhaps could increase its predictive power further. In this dissertation, the formulation of the item to tap Perceived Control avoided the mention of the (dys)functional capabilities of the mouse. This is to avoid priming the participants to the functional abilities of the computer mouse. This is particularly important when participants gave ratings of Perceived Control only after experiencing the Normal Task Control condition. This is reasonable considering that priming participants to the functional capabilities of the mouse before it malfunctions might arouse highly cynical participants' suspicions about the mouse being deliberately manipulated. If participants suspect that the mouse's dysfunction was intentional, this could potentially reduce their level of engagement in the task during the Variable and More Variable Pauses conditions. The measurement of Perceived Control is a complex and delicate issue often encompassing many considerations (Skinner, 1995).

Hostility-Reactivity moderated by Anger

The Hostility-Reactivity effect was found to be significant in situations where anger was provoked (Smith & Pope, 1990). The main method used to induce state anger in the laboratory to produce this effect is the harassment paradigm. The harassment paradigm entails a confederate making disparaging remarks at the participants while the participant is executing the various laboratory stressors. This paradigm confounds both social conflict and anger inducement.

Through manipulating the controllability of the computer mouse, low Perceived Control and State Anger is induced. Anger induced in this manner is non-interpersonal and not related to social conflict. In Study 2, Anger Recall was also included as one of the stressors. Anger Recall involves a recollection of a personally anger-inducing social conflict event. Thus State Anger aroused from Anger Recall is interpersonally related. If State Anger moderates the Hostility-Reactivity relationship, it should do so in non-interpersonal circumstances as well. Both the 12-item Cynicism and HBI were used to assess Hostility and State Anger was found not to moderate the Hostility-Reactivity effect. When State Anger was high, participants scoring high in both 12-item Cynicism and HBI were also not more cardiovascular reactive. Null results were present in for both the Anger Recall and control-manipulated computer task.

The reason for the presence of null results is partly attributed to the use of the Likert scale in State Anger assessment. When State Anger was assessed using a visual analogue scale in Study 3, State Anger_{btw} moderate the Cynicism-Reactivity relationship. Significant interaction between Cynicism and State Anger_{btw} was also found for blood pressure reactivity. However when the Cynicism Factor was used, this was absent. This suggested that the significant effects for blood pressure were not due to Cynicism. The pattern for high cynical participants was in the predicted direction. When State Anger_{btw} was high, high cynical individuals had greater TPR reactivity than when State Anger_{btw} was low. Of interest to note was that when the 12-item Cynicism was used, low cynical participants had as much or greater TPR reactivity when State

Anger_{btw} was low. This indicates that in situations where State Anger_{btw} is low, Cynicism (assessed using the 12-item Cynicism scale) is negatively related to TPR reactivity. The same effect was present for both DBP and MAP reactivity as well. Some of the effects of this interaction with State Anger_{btw} for low cynical participants appear to be caused by Repressed Cynicism. When the Cynicism Factor was used, the significant interaction for blood pressure was non-significant. In addition, the high TPR reactivity for low Cynicism Factor scores during low State Anger_{btw} was slightly attenuated. This shows that if there is a number of repressed cynical participants within a sample, then a low anger provoking situation has the potential to produce a negative finding. Hence, the identification and reduction of the influence of Repressed Cynicism within a sample has the potential to reduce the occurrence of conflicting results.

The Cynicism by State Anger_{btw} interaction for TPR represents a weaker form of the moderator hypothesis. This is because State Anger_{btw} is an averaged State Anger ratings across the three Task Control conditions. While such an average score increases its generalisability, it reduces State Anger from a within subject manipulated variable to a between subject un-manipulated variable. This is because the same treatment evoked different levels of State Anger_{btw} between participants whereas State Anger_{wtm} increased during Variable and More Variable Pauses Task Control manipulations. Hence, it is likely that the significant State Anger_{btw} and Cynicism interaction found in Study could refer to a specific subgroup of high cynical individuals (i.e., high cynical individuals who tend to report high anger).

A number of studies have tried to investigate the role of anger expression styles in moderating the Cynicism-Reactivity relationship (Bongard et al., 1998; Dembroski et al., 1985). However, it is still unclear whether Anger-In (anger directly internally) or Anger-out (anger directly externally) measured should be the moderator as there is evidence to indicate that both forms of anger expression are also related to health outcomes (Gold & Johnston, 1990).

One of the limitations of this dissertation is that the findings are only generalisable to males since only an all-male sample was used. Secondly, the lack of significant linear effects between Hostility and cardiovascular reactivity or variability is not contradictory to findings that Hostility is associated with cardiovascular disease. It only makes it unlikely that the Hostility-Cardiovascular disease link is mediated by cardiovascular reactivity/variability. There are other pathways to explain for the risk of high hostile individuals at developing cardiovascular disease - the Psychosocial Vulnerability model (O'Neil & Emery, 2002). Thirdly, 'purifying' the Cook-Medley Hostility to measure Cynicism precisely may result in occurrence of more replicable null results. This is because, as reviewed earlier, the 50-item Cook-Medley Hostility scale correlates with neuroticism and negative affectivity. Other non-anger components of negative affect (e.g., depression) has been found to predict cardiovascular disease and mortality as well (Wulsin & Singal, 2003). Therefore, it could be that the significant effects found for Cynicism is actually due to negative affectivity and not Cynicism. Hart and Hope demonstrated this for the Psychosocial Vulnerability model (Hart &

Hope, 2004). This model proposed that high hostile individuals are at greater risk of disease and mortality because they have less psychosocial resources to cope with stress (e.g., less social support). Hart and Hope showed that statistically controlling for neuroticism/negative affectivity reduced the effect size between Cynicism (assessed using the Cook-Medley Hostility scale) and stressful life events by 50%. It also reduced the effect size between Cynicism and Perceived Social Support by 82%! Blalock's comments made more than two decades ago are of relevance even now (Blalock, 1979):

"I believe that the most serious and important problems that require our immediate and concerted attention are those of conceptualization and measurement, which have far too long been neglected."

Summary

Research evidence suggests that high hostile individuals are at greater risk of developing cardiovascular disease because they have a profile of cardiovascular stress arousal that is associated with greater cardiovascular disease risk. However, there is an emergence of studies that have reported null or negative findings. This dissertation addresses the presence of such discrepant findings in two ways (i) improving the measurement precision of Hostility assessment and (ii) investigates the role of control and state anger in moderating the Hostility-Cardiovascular

stress arousal relationship. This is done through a new paradigm that re-defines actual control as a continuum of response-outcome contingencies rather than the presence/absence of coping behaviours. In addition, measurement precision was improved via including an attitudinal and a behavioural form of Hostility assessment in one study, confirmatory factor analysis of the Cook-Medley Hostility scale, and the development of an Implicit Cynicism assessment method. The results indicated that response-outcome contingency definition of actual control provides a better model fit for cardiovascular reactivity even when a discrepancy exists between the efficacy of coping behaviour and actual control. In addition, measurement error in Cynicism assessment can potentially result in null or negative findings. Non-interpersonal control (perceived or actual) do not moderate the Hostility-Arousal relationship. Between participant differences in non-interpersonal induced state anger moderate the Cynicism-Reactivity effect. High cynical participants did not report greater state anger reactivity due to stress. The use of visual analogue scale partly contributes to the detection of this effect. The results indicated that state anger, but not perceived control, moderates the Cynicism-Cardiovascular stress arousal even in the absence of social conflict.

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Appendix 1. Polychoric Correlation Matrix for the 12-item Cynicism Model

	CYN50	CYN58	CYN76	CYN81	CYN104	CYN110	CYN254	CYN286	CYN346	CYN352	AGGR 414	CYN470
CYN50	--											
CYN58	.114	--										
CYN76	.260	.389	--									
CYN81	.151	.393	.488	--								
CYN104	.166	.198	.110	.362	--							
CYN110	.179	.331	.339	.713	.455	--						
CYN254	.065	.314	.219	.360	.340	.320	--					
CYN286	.051	.103	.140	.302	.219	.300	.211	--				
CYN346	.250	.056	.220	.150	.330	.202	.086	.220	--			
CYN352	.173	.303	-.031	.012	.246	.177	.136	.264	.109	--		
AGGR 414	.037	-.157	-.009	-.117	.106	.196	-.095	.230	.230	.017	--	
CYN470	-.042	.248	.100	.455	.486	.458	.192	.205	.013	.092	-.039	--

Appendix 2. Polychoric Correlation Matrix for the Alternative 12-item Model

	SOAV 19	AGGR 27	OTH 46	HATT 99	HATT 124	HAFI 136	SOAV 265	OTH 347	SOAV 386	AGGR 406	HAFI 419	OTH 457
SOAV 19	--											
AGGR 27	.189	--										
OTH 46	.034	.077	--									
HATT 99	.249	.245	.043	--								
HATT 124	.336	.285	.100	.417	--							
HAFI 136	.089	.172	.350	.043	.387	--						
SOAV 265	.149	.097	.135	.389	.509	.097	--					
OTH 347	-.173	-.033	.150	.293	.232	.170	.306	--				
SOAV 386	.097	.212	-.197	-.244	-.087	-.090	-.126	-.431	--			
AGGR 406	.288	.147	-.143	.344	.278	.279	.121	.015	.081	--		
HAFI 419	.099	.384	.072	.115	.434	.222	.181	.290	.067	.012	--	
OTH 457	.012	-.157	.003	-.082	.234	.164	.353	.210	-.065	.116	.159	--

Appendix 3 Post-task (1-5) and Pre-task (6) Appraisal items

1. How would you rate your performance on the task?

1	2	3	4	5	6	7
Very Poor			Average			Very Good

2. To what extent were you able to influence the outcome of the task?

1	2	3	4	5	6	7
Not at all			50% of the time			All the time

3. Did you find the task challenging?

1	2	3	4	5	6	7
Not at all			Moderately			Very Much

4. Did you find the task difficult?

1	2	3	4	5	6	7
Not at all			Moderately			Very Much

5. Did you find the task engaging?

1	2	3	4	5	6	7
Not at all			Moderately			Very Much

6. The next task will be the same as the one you have just done. To what extent do you think you can do well on the next task?

1	2	3	4	5	6	7
Not at all			Maybe			Very Sure

Appendix 4 Technical Description and Schematic Diagram of the Faulty Mouse Device

For the Faulty Mouse, the main part of the PIC program is the interception and re-transmission of mouse packets to a personal computer. This part of the program uses a common technique known as bit banging. If the program detects that the intercepted mouse packets are wrongly formatted it re-starts the interception process again. The interception process in itself exhibits random like behaviour. To extend the random behaviour of the interception process a random delay is introduced between the re-transmission of mouse packets. This is generated by the use of a special PIC register that is used as an instruction counter, and by the manual setting of switches S2, S3, S4 and S5. The instruction counter is an 8-bit counter, and it is incremented after every instruction has been executed. Being an 8-bit counter, it counts up to 255, resets itself to zero, and then begins counting again. When the PIC has been successful in intercepting and re-transmitting a mouse packet, it reads the status of the switches, S2, S3, S4, S5 and then copies the value of the instruction counter into a general purpose register. It is important to note that the complexity and uncertain nature of the entire process, interception, re-transmission, reading of the switches and the determination and generation of the of delay between intercepted mouse packets means that the value held by the instruction counter is purely arbitrary. The instruction counter produces pseudo-random numbers. The status of the switches S2, S3, S4, S5 and the random number now held in the general purpose register are used to form simple

programs loops that decrement the general purpose register, and so in conjunction with the interception process produces random delays between mouse packets. S2, S3, S4, S5 set the range of the delay generated as indicated below:

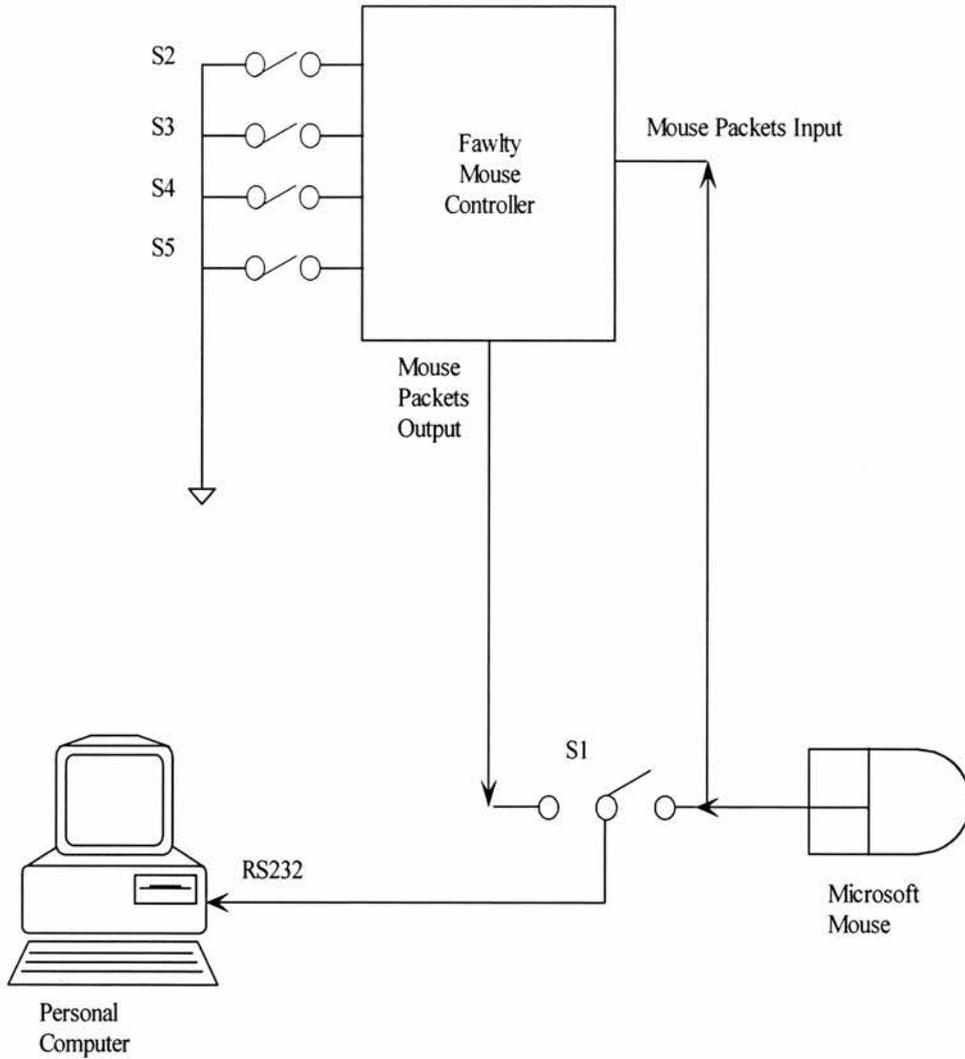
When S5 is set low, the delay is between 25 and 600ms

When S4 is set low, the delay is between 25 and 500ms

When S3 is set low, the delay is between 25 and 400ms

When S2 is set low, the delay is between 25 and 300ms.

It is important to note that switch S5 has highest precedence when set low, and S2 has the lowest precedence when set low. In practice the delays above are adjusted within the software until the Faulty Mouse produces relative step like changes in the way it (mis)behaves.



Block Diagram: Fawly Mouse

At the heart of the Fawly Mouse is Microchip PIC microcontroller. It intercepts Microsoft RS232 mouse packets, of 3 bytes in length, and introduces a randomised delay between mouse packets. To the mouse user, the effect is very similar to when the roller spindles become clogged with dirt and as a consequence the mouse ball begins to stick. The effect can be reduced or increased by opening and closing the switches S2 to S5. S1 selects normal or faulty mouse mode

Appendix 5 Does Cook-Medley/Marlowe-Crowne method identify Repressed Hostility?

This appendix uses the data from Study 2 and 3 to investigate whether the Cook-Medley/Marlowe-Crowne method identifies Repressed Hostility. Alpha was set at .05. There are two hypotheses:

Hypothesis 1: Using the dataset from Study 2, it is predicted that individuals with low Cook-Medley scores and high Marlowe-Crowne Social Desirability scores are more likely to have higher Structured Interview-derived Hostile Behaviour Index (HBI). This is because the HBI is not significantly related to Social Desirability ($r(59) = .20, p = .14$) and is thus less susceptible to positive presentation bias than the Cook-Medley Hostility scale. The HBI measures the behavioural aspect of Hostility. To investigate this pattern, a significant Cook-Medley by Marlowe-Crowne interaction is hypothesised.

The whole 50-item Cook-Medley Hostility scale was used as this is the standard Cook-Medley/Marlowe-Crowne method of identifying Repressed Hostility. Regression was used where the following predictors were entered in this order: Cook-Medley Hostility scale, Marlowe-Crowne Social Desirability scale, and the interaction term for these two scales. The dependent variable was HBI.

The results revealed a Cook-Medley x Marlowe Crowne interaction that was of borderline significance, $F(1, 55) = 3.90, p = .05, \eta^2 = .06$.

Using the method introduced by Aiken and West (Aiken & West, 1991), this interaction is shown in Figure 6.1. Figure 6.1 shows that participants with low Cook-Medley scores and high Marlowe-Crowne Social Desirability scores (Cook-Medley/Marlowe-Crowne method of identifying Repressed Hostility) do not have higher HBI. The reverse was true - these participants had one of the lowest levels of hostile behaviours.

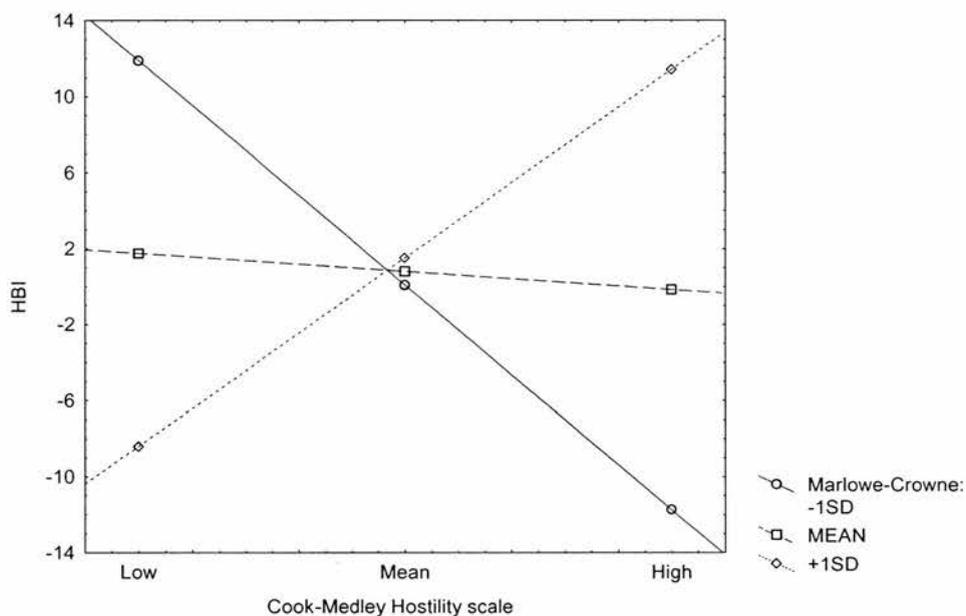


Figure 6.1 Cook-Medley by Marlowe-Crowne interaction on Hostile Behaviour Index (HBI)

Hypothesis 2: Using the dataset from Study 3, it is hypothesised that participants scoring low in Cook-Medley Hostility scale and high in Marlowe-Crowne Social Desirability scale will have higher GNAT Cynicism scores. This is because GNAT Cynicism measures the implicit form of Cynicism that is not significant related to social desirability. A significant Cook-Medley x Marlowe Crowne interaction is hypothesised to be significant.

Regression was used to test this hypothesis. Predictors were entered in the following order: Cook-Medley Hostility scale, Marlowe-Crowne, and the interaction term between these two scales. The dependent variable was GNAT Cynicism.

The result for the Cook-Medley x Marlowe-Crowne interaction was not significant, $F(1, 56) = .83, p = .37, \eta^2 = .01$.

The results from these analyses revealed that the Cook-Medley/Marlowe-Crowne method of operationalising Repressed Hostility did not identify participants with more hostile behaviours or higher implicit Cynicism. The pattern of results for Hypothesis 1 showed the Cook-Medley/Marlowe-Crowne method identified participants with one of the lowest levels of hostile behaviours. The conclusion of these analyses is not a definite proof that the Cook-Medley/Marlowe-Crowne fails to identify Repressed Hostility. But given that this method of identifying Repressed Hostility resulted in selecting groups with the lowest hostile behaviours as assessed by a gold standard of hostility assessment leads one to raise serious doubts about the construct validity of this method in identifying Repressed Hostility. How can the Cook-Medley/Marlowe-Crowne method be said to identify Repressed Hostility if, (i) they have one of the lowest levels of hostile behaviours, and (ii) do not have high implicit Cynicism (GNAT Cynicism)? The findings suggest that a pattern of low Cook-Medley Hostility scores and high Marlowe-Crowne Social Desirability scores measures what it measures - participants with low Hostility and high Social Desirability.

Appendix 6 Covariates included in the Regression models tested in Chapter 4

The following lists the between subject models by Cynicism (or Pseudo Cynicism) measure. This provides information as to what covariates are retained into the between subject models. Within subject models are not reported here as there are no covariates.

12-item Cynicism

Systolic Blood Pressure, Diastolic Blood Pressure, Mean Arterial Pressure, Heart Rate, Total Peripheral Resistance:

1) SBP Baseline

Cardiac Output:

- 1) CO Baseline
- 2) Cynicism
- 3) Cynicism²
- 4) Cynicism x Baseline
- 5) Cynicism² x Baseline

GNAT Cynicism

Systolic Blood Pressure, Diastolic Blood Pressure, Mean Arterial Pressure, Heart Rate, Cardiac Output, Total Peripheral Resistance:

1) Baseline

Cynicism and Pseudo Cynicism Factors

Mean Arterial Pressure, Heart Rate, Cardiac Output, Total Peripheral Resistance

1) Baseline

*Systolic Blood Pressure, Diastolic Blood Pressure**

- 1) Baseline
- 2) Pseudo Cynicism
- 3) Pseudo Cynicism²
- 4) Pseudo Cynicism³
- 5) Pseudo Cynicism x Baseline
- 6) Pseudo Cynicism² x Baseline
- 7) Pseudo Cynicism³ x Baseline

*The significance of (7) for DBP was borderline ($p = .06$) but not significant for MAP ($p = .07$).