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Developmental stress and social phenotypes: integrating neuroendocrine, behavioural and evolutionary perspectives

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Summary

The social world is filled with different types of interactions, and social experience interacts with stress on several different levels. Activation of the neuroendocrine axis that regulates the response to stress can have consequences for innumerable behavioural responses, including social decision making and aspects of sociality such as gregariousness and aggression. This is especially true for stress experienced during early life, when physiological systems are developing and highly sensitive to perturbation. Stress at this time can have persistent effects on social behaviours into adulthood. One important question remaining is to what extent these effects are adaptive. This paper initially reviews the current literature investigating the complex relationships between the hypothalamic-pituitary-adrenal (HPA) axis and other neuroendocrine systems and several aspects of social behaviour in vertebrates. In addition the review explores the evidence surrounding the potential for 'social programming' via differential development and activation of the HPA axis, providing an insight into the potential for positive effects on fitness following early life stress. Finally the paper provides a framework from which novel investigations could work to fully understand the adaptive significance of early life effects on social behaviours.

1. Background

The ability to effectively interact with conspecifics is a vital skill, which we have only just begun to explore in terms of its impact on fitness (1, 2). In humans, this ability is also highly valued and disorders known to reduce our ability

to interact or understand the emotional cues of others place sufferers at significant disadvantages, with long term impacts on health and wellbeing. Understanding the factors that give rise to individual differences in 'social competence' is therefore of fundamental importance to both our knowledge of human pathologies, but also animal welfare and population structure. Stress is a major regulator of social behaviour and in turn social influences can alter behavioural and physiological responses to stress (3-5). Activation of the vertebrate neuroendocrine axis which regulates the response to stress can cause pleiotropic effects on several aspects of social behaviour, including reduced social interaction, increased affiliative behaviours, increased aggression and altered mating behaviours (6-15). The direction and magnitude of these effects is often related to the context within which the experiment was conducted as well as the type of stressor that was experienced and the life history strategy of the species in question, however there is clear evidence to suggest that stress during all life stages can impact on sociality.

One major driver of social ability in adulthood is the conditions experienced during development, when neural substrates and physiological systems are in their infancy and sensitive to perturbation. Developmental modifications of several of these systems have shown permanent changes in a range of phenotypic trait. This phenomenon, known as 'developmental programming' is at the centre of a large debate as to whether these permanent changes represent constraint on later behaviours or they are a method of creating phenotypes "tuned" to respond to salient environmental cues in a way that maximises fitness in later life (16-19). Due to the powerful effects stress can have on social behaviour in later life, social behaviour represents an excellent model system to investigate the potential for adaptive responses to developmental conditions. This review describes the relationships between stress and social behaviour in vertebrates, discusses evidence for and against 'social programming,' and, finally, provides a framework from which future investigations could robustly evaluate the adaptive role of developmental stress in shaping adult social behaviour.

2. Stress: what is it and how is it regulated?

In order to understand how stressful conditions can drive changes in social behaviour it is important to address the nature of stress itself and define the neuroendocrine axis that regulates an organism's response to adverse conditions. Stress is a part of every organism's life, with a variety of environmental stimuli that can act as 'stressors', i.e. factors that perturb homeostatic processes. The capacity to respond to these stimuli in a way that

restores homeostasis and/or removes the individual from the stressful environment is one of the most important physiological mechanisms underlying survival in vertebrate animals. The stress response is controlled and regulated by the highly phylogenetically conserved hypothalamic-pituitary-adrenal (HPA) axis or stress axis (16, 20). This axis is activated during adverse conditions in both development and adulthood and, in vertebrates, results in the release of glucocorticoids (21, 22). Activation of the HPA axis, often via the amygdala, facilitates a switch of physiological processes and behaviours from non-essential activities to those that promote short-term survival, such as increased locomotion and mobilisation of energy stores (23). Stressful stimuli cause the hypothalamus to release corticotrophin releasing factor (CRF), which works in conjunction with arginine vasopressin (AVP: mammals) or vasotocin (AVT: birds) to promote the release of adrenocorticotrophic hormone (ACTH) from the pituitary gland (23, 24). ACTH then stimulates the synthesis and release of glucocorticoids (GC) from the adrenal cortex, which enter the blood stream to act on target tissues (Figure 1). This cascade essentially describes an endocrine response to 'acute stress', where glucocorticoid levels increase from a baseline circulating level to peak over a period of minutes after a stressor is perceived (25). When stress is experienced chronically elevated baseline levels of the hormone are often seen, and in some cases the acute response becomes dampened (25, 26). The activity of the HPA axis is tightly regulated by classical negative feedback loops that utilise two receptor types; glucocorticoid receptors (GR), and mineralocorticoid receptors (MR), which regulate basal and stress induced hormone levels (27-29). Other physiological systems can also act to alter HPA axis activity. For example, serotonin (5HT) is well known as a positive driver that can increase the amount of glucocorticoids produced by the axis (30). In addition the nonapeptides (AVP and oxytocin (OT)), a hormonal group that has been linked to social behaviours such as pair bonding, affiliation, trust (31, 32) also interact with the HPA axis. Aside from the fundamental role AVP plays in the axis, increased levels of hypothalamic OT can have significant inhibitory effects on glucocorticoid production by reducing levels of the prohormones within the cascade (Figure 1). These interactions across hormonal systems reveal a complex network of neuroendocrine mechanisms, however they also highlight the close relationship between the social brain and the HPA axis.

Due to its fundamental implications for survival, stress has been studied for decades in a range of species and we now have an excellent understanding of what types of stimuli can as act stressors and trigger the HPA axis. Such

challenges include unpredictable changes to weather patterns, including periods of low or high rainfall, storms or significant or rapid changes in temperature (23, 26) and other variables that could be considered to signal environmental quality to an individual have similar effects on the HPA axis, including food availability, predation risk, anthropogenic disturbance levels and pollutants (23, 26, 33-40). Each of these factors has been shown to have the ability to trigger a stress response; however the strength of this response is often related to the environmental context within the experiment or study (34, 35, 41-46). For example, chronic exposure to lead can cause alterations in the amount of corticosterone produced under an acute stress in white storks (*Ciconia ciconia*) (41), however few studies have found active effects of pollutants on baseline levels of glucocorticoids (35, 41). Social challenges and changes in the social environment can also act as highly potent stressors (3, 47, 48), although the relationship between stress and social behaviour is complex, as social situations can also buffer the negative effects of stress in affiliative species (3, 4, 49, 50). Nevertheless social stressors have the power to exert fundamental changes to the HPA axis over both the short and long-term. Whilst the focus of this review is to understand the effects of stress on social behaviour, it is important to recognise that social factors themselves can activate the HPA axis.

3. Social factors as stressors – how do they affect the HPA axis?

Several components of the social landscape, such as parent-offspring interactions, social defeat, aggression, social isolation and competition are key activators of the HPA axis (47, 48, 51-56). These are considered 'social stressors'. Transient changes in HPA activity in sexually mature and juvenile animals have been shown in a range of species in response to adverse social interactions or conditions. Obtaining or maintaining a socially dominant position or rank can act as a stressor, activating the HPA axis, although there is conflicting evidence in favour of this finding (57). Recent work has shown that in baboons, only the alpha male exhibits elevated levels of cortisol (58), whilst there was no significant relationship between GCs and social rank in the other group members. Clearly dominance and stress have a complex interplay, dependent on the life history strategy of the species at hand. However, it is not just interactions with conspecifics that can alter HPA axis activation. Social isolation or separation from a familiar social setting is also a powerful stressor. For example, adult captive starlings (*Sturnus vulgaris*) show increased corticosterone production when they are separated from their group (59), solitary housing of adult prairie voles (*Microtus ochrogaster*) from a sibling cage mate for 4-8 weeks is associated with increased ACTH and corticosterone

production (60), and experimental separation of bonded mating pairs increases baseline corticosterone levels in the zebra finch (*Taeniopygia guttata*)(61). Maternal separation significantly elevates plasma glucocorticoids in rat (54) and vole pups (62). In juvenile weaned prairie voles housing in either social isolation with a familiar or novel conspecific significantly alters several components of the HPA axis in the short term (63). Stowe et al (64) quantified faecal metabolites of corticosterone as an integrated measure of stress and related these to the level of positive social behaviours occurring, such as allopreening and social contact. They found that in the nestling phase allopreening was negatively correlated with these metabolites, however in the post-fledging period birds that showed elevated levels of metabolites sat closer to conspecifics. This corroborates the idea that positive social interactions can reduce 'stress' and reduce activation of the HPA axis, but also suggests that GC elevation may occur to drive more sociality in periods when new bonds are being made, such as in early independence (65). In humans, increases in salivary cortisol can be achieved by inducing negative social interactions, initiating the feeling of rejection or isolation (55). Social defeat and aggressive encounters can also elevate stress hormone levels. Experiencing aggression from conspecifics followed by social defeat can also raise corticosterone in the rat, although sustained defeat over a period of days can lead to glucocorticoid resistance, i.e. a reduction in the sensitivity of target tissues to glucocorticoid actions (47). In addition to short-lived effects on HPA activity and GC production, exposure to social adversity during development can have persistent effects on HPA functioning and regulation (13, 56, 66). For example in rats experiencing high or low quality maternal care can alter the activity of AVP neurones within HPA axis brain regions in response to a forced swim stressor in later life (67). Social stressors can therefore be powerful stressors, with their effects being seen across different life history stages.

4. Stress effects on social behaviours

Although it is clear that social factors can trigger the HPA axis it is certainly true that a wider range of stimuli can affect HPA activity and in turn alter social behaviours. The effects on sociality are widespread and can have significant impacts on fitness, it is therefore vital that we understand the consequences of stress on social behaviour in terms of outcomes and mechanisms. Many of these effects occur over the short-term, causing transient changes in behaviour. However, more persistent effects have been recognised, particularly when stress sis

experienced in early life. It is these effects that are fundamental to our understanding of the potential for adaptive programming of the social behaviour. The following sections give an overview of this evidence, with the aim of determining the potential for both positive and negative impacts on fitness.

Short-term effects

Priming an individual to alter their social situation or behaviour when stressors are present is logical, as several changes to an individual's behaviour could assist in removing either the stressor itself (if it is another animal) or removing /protecting the animal from the stressful event. The vast majority of studies suggest that stressors typically lead to reduced social behaviours, such as social motivation, approach behaviour and interaction, and increased aggression (7, 11, 14, 68-70). However other studies find no immediate effects of acute activation on sociality (reviewed in (14)). The differences here are likely due to the intensity and duration of the stressful events, and there is such a broad range of manipulation strategies, it is difficult to tease apart these effects across studies. It seems sensible that a one off change in stress hormones would not trigger dramatic long-term changes in social behaviour, as this could have persistent effects on the ability to integrate into the population/ group. However, more sustained stressors, stimuli that provoke a more significant response of the HPA axis, or more socially relevant stressors might signal the need to alter social behaviours and avoid conspecifics. For example, chronic social defeat by a dominant individual causes an immediate reduction in hippocampal neurogenesis in mice, which in turn has significant impacts on social avoidance behaviours in several species (71). In this case the experience of repeatedly losing out to conspecifics which could result in later harm or injury should provoke a socially avoidant phenotype as the potential costs of injury outweigh the benefits of social contact in that perceived environment. Studies that have investigated the effect of exogenous stress hormone administration at different ages have provided support for the idea that glucocorticoids can at least partly mediate the effects of stress on social behaviour. In many cases exogenous GC treatments induce very similar effects on social behaviour as those mediated by social and other stressors(11). For example direct GC administration in juveniles immediately diminishes social exploration (14) and acute GC treatments in adulthood increase aggression (11, 14, 68, 72).

These above examples provide evidence for stress 'negatively' impacting on social behaviour, albeit in a potentially adaptive way. However, stressful situations can trigger an increase in social behaviours, promoting affiliative

behaviours specifically, which is thought to link to increased group cohesion facilitating better coping in stressful conditions (73). In some cases social isolation can enhance affiliative behaviours, such as huddling upon reunion with conspecifics. As groups confer significant survival prospects it is unclear if this response is mediated by an increased risk aversion, or increased sociality, or both. One study in prairie voles, suggests that GC production is required for the maintenance of already established pair bonds. In this species treatment with exogenous corticosterone or CRF significantly increases mating partner preferences in males as well as increasing avoidance of novel individuals (74, 75). When GCs are experimentally reduced in males, partner preferences are significantly reduced and males begin to show preferences for mating with novel females (74). However if circulating hormone (CRF) levels are manipulated to very high, almost supra-physiological levels partner preferences are again inhibited, possibly related to the high levels of anxiety that are induced by this treatment (10, 74).Thus, in male prairie voles, very low circulating GC levels inhibit the formation of a partner preference, whereas exposure to stress or an increase in stress-related hormones facilitates social bonding. This could be driven by the need to enhance pair cohesion during stressful events. In zebra finches, a highly gregarious species, increases in GCs also cause more robust preferences for the opposite sex compared to preferences to remain in a group of familiar same sex birds (76). Control birds preferences lie mainly with the group, so increasing stress seems to drive the need for a mating partner. Whist this is potentially different from the vole work in that they did not investigate the effects of stress on existing bonds with the opposite sex, it does suggest that HPA activation could alter the motivation to breed in some species. Changes in residual reproductive value are likely to occur in response to physiological/cellular changes, which in turn result in behavioural shifts in important reproductive behaviours. In the vole study the results then could also be attributable to this motivation. Since breeding in this species only occurs after substantial bonds have been formed over time and strong bonds promote higher reproductive success then it is pragmatic to remain with your already acquired partner to maximise reproductive output under stress. Interestingly chronic elevation of GCs at relatively high levels can supress reproductive behaviours (26, 77, 78), particularly parental behaviours (79, 80), but in this case they drive the need for breeding initiation by altering social preference strengths (76). Changes to social preferences following stress are not confined to a reproductive context. Moderate acute stress in male rats increases social support seeking from same sex cage mates, resulting in reduced aggression and increased sharing of resources (73).

In addition to the transient effects of stress on behaviour, many of these manipulations cause alterations in nonapeptide levels, and this may be the main mechanism through which stress exerts its behavioural effects (3, 81). For example direct manipulations of glucocorticoids as well as the application of chronic stressors can cause immediate increases in oxytocin binding within the hippocampus and other brain regions (82). Whilst the immediate effects of stress on social behaviour are interesting and have obvious implications for fitness, one additional component to consider is the potential for longer lasting effects.

Long-term effects

Early life represents a period of sustained growth, reorganisation and sensitivity in terms of the development and functioning of neuroendocrine systems, particularly the HPA axis (65, 83-86). There is a wealth of evidence to show that stressors experienced in early life stages can significantly alter HPA axis development and impact on a wide range of phenotypic traits. Social behaviour is no exception to this. Pre-natal exposure to elevated GC levels in coho salmon (*Oncorhynchus kisutch*) alters dominance behaviours, creating bolder and more dominant fish in adulthood when faced with a novel conspecific (87). In rodent models pre-natal stress tends to cause significant reductions in social behaviours in adulthood, in a similar way to those described for adult animals in the previous section (88-91). For example, male rats born to mothers who experienced four sessions of restraint per day during the latter stages of pregnancy, exhibit reduced social interactions when repeatedly faced with a single conspecific in a familiar arena in adulthood (88). Interestingly this result was found as part of a social memory test, designed to determine if individuals could recognise a novel conspecific after habituation to a previous one. Pre-natally stressed rats showed reduced interaction across all trials compared to controls. The separate social interaction test carried out after this memory test, where animals were given a novel adult conspecific yielded no interaction differences between treatments, except for an increase in aggression.

Post-natal conditions can also have significant effects on later social abilities. Maternal separation prior to weaning is a potent stressor in mammals. It is used as a proxy for deficient parental care and is akin to social isolation in older animals (11, 70, 92). Individuals exposed to this stress show significant deficits in social behaviours in adulthood. Toth et al (93) exposed juvenile rats to three hours of maternal separation during early lactation. They

found no effects on cognition in juvenile animals; however they did find a reduced propensity to interact with a conspecific in an open field test. Here they only measure social behaviour in a three minute period, once in a single context, but they do quantify several behaviours in order to glean the effects on the potential different strategies for social interactions (93). Dominance behaviours can also be affected by maternal separation stress. Mice repeatedly isolated from their mother are more likely to express a subordinate phenotype; they lose out to competitors in ecologically relevant arenas when resources are limited experimentally (94). Interestingly these behavioural effects were also seen in animals exposed to dioxin pollutants in milk in the same study. Social relationships in adolescence are one of the most important determinants of health into adulthood. At this time there is a significant increase in peer to peer interactions and there is evidence to suggest that adolescents are highly sensitive to stress (65). Stressors applied at this time also seem to have significant inhibitory effects on adult social behaviours, particularly interaction levels with a novel conspecific and increased aggression (13, 65, 95-98).

Stressors experienced at each of these stages can impact upon nonapeptide and serotonergic systems, which again feed into the mechanism by which early life stress can permanently alter social behaviour. For example, Ahmed et al (99) found that pre-natal exposure to elevated glucocorticoids significantly reduces brain serotonin levels in adulthood in chickens (*Gallus gallus*), which coincided with an increase in aggression in the stressed phenotype. Further work in mice has also suggested that pre-natal stress can perturb the development of serotonin neurones (100). Both pre-natal and juvenile stressors have been shown to significantly alter nonapeptide production in later life (88, 101-105). Maternal separation of mouse pups for three hours a day in the first two weeks of life causes opposite effects on the two main nonapeptides within the hypothalamus in adulthood; AVP was seen to be significantly upregulated, whereas OT was downregulated (106). These neuroendocrine changes caused increased aggression towards a novel conspecific, no other social behaviours were recorded, however reduced OT is directly linked to reduced sociality (81).

The potential for social programming.

Overall the evidence provided in the previous sections suggests that early life stress decreases measures of social motivation, reduces the expression of social behaviours, increases aggression and promotes the development of anti-social behaviours, but the specific consequences depend on the timing and nature of the stressor (11, 14).

Although these may be problematic from a human health or an animal welfare perspective, from an evolutionary perspective such responses to early life have often been interpreted as mechanisms through which early adversity prepares an individual for similar environments in later life (13, 14). The idea that early life creates phenotypes that are able to cope better in adverse environments was posited several years ago in the form of the thrifty phenotype hypothesis and later in terms of 'developmental programming'. These hypotheses suggest that during development, individuals are physiologically (and thus behaviourally) "tuned" to respond to salient environmental cues in a way that maximises fitness in later life(19, 83, 107, 108).The adaptive significance of this programming may only become evident under certain contexts in later life, such as when later environments match this experienced in development (109, 110). For example, in mites pre-natal exposure to the risk of predation produces offspring that are less active when living in high predation areas (111). This result provides evidence for a possible adaptive response, in that it allows parents to communicate cues about the likely state of the post-natal environment (i.e. high risk of predation) to their offspring before birth, thereby allowing them to adjust their behaviour accordingly. In the case of 'social programming' alterations to the HPA axis during development that last into adulthood need to impact on social behaviours, potentially via their effects on other neuroendocrine systems, in a way that could enhance their ability to cope in a socially stressful environment throughout life.

This idea is more easily reconciled for aggression or behaviours that could lead to a more dominant social position, enabling animals to better find or retain resources due to superior competitive skills. However, in highly social species significant reductions in social interactions would on the face of it seem to be maladaptive. Instead such reductions could be the outcome of a constraint imposed by developmental stress. However in certain contexts reduced conspecific contact can reduce the risk of parasitic or disease infections (112-114). Reductions in social interaction or motivation in the short-term may have advantages, however the social deficits described above are persistent and manifest in adolescence and adulthood. During these periods animals of most species need to interact with novel conspecifics and social species rely on group living to maximise foraging and breeding success, at least for part of their life cycles. Reductions in social interaction ability or motivation at these stages could significantly impact on fitness. Recent work in the highly social zebra finch has provided evidence for more positive effects of developmental stress on sociality (9). During post-natal development siblings were fed either a

physiological dose of corticosterone or vehicle daily for a period of two weeks. Once birds reached adolescence, after nutritional independence, the population was transferred in two free flying rooms with equal numbers of each treatment group and Radio-frequency identification (RFID) technology was used to estimate social networks and measures of gregariousness and social ability were inferred from these networks. The authors found that developmentally stressed birds had more central network positions as well as much wider associations with unrelated adults than their control siblings (9, 115). This work did not attempt to replicate the types of social interaction tests commonly used in the literature, providing a single individual to interact with, instead it used a more ecologically relevant tool to determine how well an individual copes in a group. Recently there has been great interest in the use of social networks and several studies have now suggested that central positions in networks can indeed have positive fitness benefits in a range of species (1, 2, 116-119). Indeed work in birds has also suggested that network centrality is related to specific behavioural syndromes or individual personalities (117) and such syndromes have been linked to HPA axis activity, and developmental conditions (120-124). In addition Levin and colleagues (125) recently showed that the magnitude of the acute stress response is positively related to social network position in free living barn swallows (*Hirundo rustica*). The use of more ecologically relevant behavioural tests may indeed yield further results to support the idea that early life has useful effects on social behaviour specifically. However, further work using innovative technologies, such as RFID tags are required in free living animals if we are to understand social behaviour in a 'real world' context. These technologies can also be used to investigate how perturbations to social networks can also impact on the HPA axis. Although this body of work hints at a programmed effect on sociality that could reap significant rewards, an integrative approach is essential to determine the true nature of developmentally driven social behaviours.

5. The importance of evaluating a social phenotype in different contexts.

Sociality is a complex multivariate trait and we can only really determine the adaptive significance or even existence of 'social programming' if we quantify a suite of social behaviours that define that trait fully. Individual social traits are likely to be highly inter-related, but the strength of these relationships may change with social context, sex, environmental conditions or age. In many cases the laboratory setting provides an unrealistic arena to measures these complexities. Some studies do attempt to use more ecologically relevant settings, with enriched caging, social

housing or aviary settings; however much of the research relies on tests that do not take context into account (14, 70, 95). In many cases studies only conduct a single to limited number of tests.

It may be more fruitful to quantify social behaviour in terms of an individual's 'social phenotype', where the expression of a range of ecologically relevant behavioural responses to different social and environmental contexts is considered. Hence, the social phenotype represents the suite of specifically social behaviours used by an individual across a range of social environments. Certain aspects of the social phenotype may become more important at specific life history stages, for example during breeding, and the ability to modulate the phenotype may have significant implications for health and fitness. One alternative hypothesis is that social phenotypes represent a form of behavioural syndrome, sometimes known as personality, and remain relatively stable in different contexts (121, 126). A central idea in behavioural syndrome research is that the strength of the behavioural correlations generate trade-offs. Behavioural syndromes could therefore manifest maladaptive behaviour in some contexts, thereby reducing fitness. The ability to be flexible and alter traits within an overarching phenotype might enhance the ability to maximise fitness in a range of environments (Figure 2). To date the ability of early life to alter flexibility in social traits has not been studied, and more work investigating the context dependency of social traits is required. A series of predictions can be generated in order to understand how early life experience could impact on fitness by altering the ability to cope in different adult social environments.

For example, if we consider that adult environments vary in their social landscape from aggressive to affiliative, we may expect most individuals from a benign developmental background to show increased fitness in more affiliative areas, due in part to the enhancement of fitness through lower aggression levels, which can cause harm and alter time activity budgets as well as resource allocation strategies (Figure 2 Red line). If early life stress creates permanent deficits in social motivation and interaction, but increases in aggression as suggested by much of the rodent literature, it would be expected that these individuals will show the opposite relationship to that described above (Figure 2 Green line); fitness should be higher in aggressive later environments, due to increases in dominance and competition for resources. In this case the individual is programmed to cope with aggressive social challenges. However, permanent reductions in affiliative behaviours become maladaptive in affiliative social environments. Developmental stress may also program other types of social phenotype. Permanent increases in

gregariousness following post-natal stress have been suggested by recent avian work. In this case fitness is predicted to be lower in aggressive environments as increased association might lead to increased defeat, altered resource allocation and potential harm. However due to their increased affiliative behaviour such individuals may outperform those from a benign environment in affiliative environments, hence they show a steeper 'reaction norm' slope (Figure 2 Blue line). Each of these potential phenotypes described above trades off affiliation against aggression. This brings us back to the idea of a flexible social phenotype. If early life stress could program a phenotype that is able to alter their affiliative and aggression levels depending upon the adult social environment this could potentially stabilise their fitness across differential social environments (Figure 2 orange line). More work is required to test these predictions.

Some studies have investigated more than one social context during their study. For example, Green et al (98) subjected rats that had experienced social instability stress prior to sexual maturity to three different social tests: 1) interaction behaviours following placement of the test and an novel rat in a familiar open field apparatus, 2) approach of a test rat to a conspecific sitting behind a plexiglass pane in a novel environment and 3) social approach in the same apparatus following acute stress via restraint. The authors found that the earlier instability stress only altered the interactions with a novel rat in a familiar environment, reducing contact time and play significantly. No other test provided evidence for social deficits. However, control rats, that had experienced no stress during adolescence showed a significant reduction in social approach behaviour following restraint stress, compared to the unstressed situation. Previously stressed rats did not exhibit the same reduction. The use of a stress versus non stress context in this study gives an insight into how the animals cope in different environments. The lack of a stress initiated reduction in interaction levels in the developmentally challenged animals is interesting. Could this be due to an increase in boldness following unpredictable social stress? Or are the animals programmed in such a way that they use the stressful cue in a different way to the control rats?

Another example of the measurement of a social phenotype is a study conducted in zebra finches (127). Here the authors investigated the relationships between nonapeptide neuronal activation and a range of social and non-social phenotypic traits. Specifically they quantified; 1) group size preference, using a simple choice chamber presenting groups of 2 and 10 birds as potential interaction stimuli at each side of the chamber; 2) social

preferences, using the same apparatus but adjusting the stimuli to be either a novel or familiar conspecific and finally 3) they conducted colony observations on groups of 8 birds placed into a novel breeding enclosure. Here they recorded a wide range of behaviours encompassing allopreening, singing behaviour, nesting behaviour, following, aggression and pair bonding. They uncovered a very complex set of relationships centred on sex differences, for example males showed a positive relationship between gregariousness and VT neuron activation, whilst females showed a negative association. One of the most important aspects of this work, however, is the ability to uncover these complexities in a robust way and the ability to determine how different aspects of a social phenotype trade off against each other in different contexts. Using this type of framework, further insights could be drawn about the importance of developmental stress in mediating these trade-offs and the whole social phenotype.

6. Conclusions

It is clear that stress and sociality have a complex relationship: social interactions can act as stressors as well ameliorate stress responses and activation of the HPA axis by a range of stimuli can cause both transient and persistent effects on social behaviours. Many of these behavioural responses to stress are mediated by changes to other hormonal systems, such as the nonapeptides; however stressors can also alter the activity of the HPA axis and it seems likely that the resultant effects on behaviour are due to a combination of direct effects on stress and social hormone production. The idea that developmental conditions act as a cue to later environmental quality, and that developing animals adjust their physiology and behaviour in order to match their phenotype to those conditions is an established hypothesis, with limited consensus. Whilst the social behaviour literature hints at this possibility there is still a great deal of work to be done in order to fully test this idea in a social context. Exploration of social phenotypes in different contexts could be the key to unlocking this, as well as experiments that track the neuroendocrine responses of these phenotypes to differential environments. Whilst this potentially calls for large scale and long-term comparative studies, it could indeed provide robust evidence for social programming and distinguish between adaptive and constrained hypotheses relating to developmentally driven social phenotypes.

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Figure legends

Figure 1. Schematic diagram of the hypothalamic pituitary adrenal (HPA) axis and the physiological cascade that ensues following the perception of a biological stressor. Briefly upon detection of the stimulus the paraventricular nucleus (PVN) within the hypothalamus secretes corticotropin releasing factor (CRF), which travels to the pituitary gland stimulating the release of adrenocorticopic hormone, which in turn stimulates the adrenal cortex to secrete glucocorticoid stress hormones. Once levels become high binding to intracellular receptors (glucocorticoid (GR) and mineralocorticoid (MR) receptors) in the pituitary, PVN and hippocampus serve to shut down the response and adrenal glucocorticoid production reduces. The hippocampus therefore has inhibitory effects on the HPA axis. The amygdala however has known stimulatory effects acting at the level of the hypothalamus. Another stimulatory process is achieved via serotoninergic (5HT, serotonin) neurons which project from the raphe nuclei directly to the hypothalamus and hippocampus, In addition the nonapeptide oxytocin (OT, mesotocin (MT) in birds/reptiles) is known to inhibit the hormonal cascade at the level of AVP and ACTH production which reduces the amount of glucocorticoids produced. –ve arrows depict inhibition of targeted nuclei, whilst +ve arrows depict activation routes.

Figure 2. Potential outcomes of social programming by developmental conditions (early life stress ELS) for social species, i.e. those species that commonly live in groups for part of their life cycle and rely on group living to enhance their ability to find food, mates and breeding areas. When faced with adult environments that vary in their social landscape from aggressive to affiliative individuals from a benign background will tend to show increased fitness in more affiliative areas (RED). Several rodent models have suggested that ELS creates adult phenotypes that are more aggressive and less interactive (affiliative). If this is the case when social behaviours are measured across a range of contexts then fitness is predicted to show the opposite relationship to that of those from the benign environment, here labelled ELS constraint (GREEN). If ELS confers permanent increases in gregariousness as suggested by recent avian work (ELS Gregarious; BLUE) then fitness is predicted to be lower in aggressive environments as increased association might lead to increased defeat, altered resource allocation and potential harm. However due to their increased affiliative behaviour such individuals may outperform those from a benign environment in affiliative environments. Finally if ELS can program phenotypes that are more flexible in their social abilities (ORANGE), so individuals are more sensitive to the social environment around them it is predicted that fitness should be stable across each adult environment.

Media summary

Social interaction is a vital activity in most species, including our own. Several factors can alter the way in which individuals interact, including elevated stress levels. Interestingly, if individuals experience stress during early life this can have long-term effects on their sociability into adulthood. The question posed by this review is if these behavioural responses, driven by changes in the physiology of an organism, can actually prepare an individual for adverse social situations in later life. Answering this question could gain us insight in to not only animal social behaviour, but also our own.