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Stress and Decision Making: Effects on Valuation, Learning, and Risk-taking

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Abstract

A wide range of stressful experiences can influence human decision making in complex ways beyond the simple predictions of a fight-or-flight model. Recent advances may provide insight into this complicated interaction, potentially in directions that could result in translational applications. Early research suggests that stress exposure influences basic neural circuits involved in reward processing and learning, while also biasing decisions towards habit and modulating our propensity to engage in risk-taking. That said, a substantial array of theoretical and methodological considerations in research on the topic challenge strong cross study comparisons necessary for the field to move forward. In this review we examine the multifaceted stress construct in the context of human decision making, emphasizing stress' effect on valuation, learning, and risk-taking.

Express to anyone that you are "stressed" and you are likely to receive some commiseration, a perception of understanding that belies a more complex reality. As a construct stress is amorphous, easily identified but difficult to define, its nature varying by circumstance and individual. Similarly nebulous can be a decision maker's grasp of cognitive computations involved in large and small daily life choices, so often made under stressful conditions. Therefore, it should come as no surprise that exploring the relationship between the two poses a particularly thorny methodological puzzle. The emergence of the discipline of neuroeconomics [1], coupled with knowledge gained by decades of research on the influence of stress on learning and memory [e.g., 2], have promoted a surge of attention to this very question. While significant advances have been made, the growing literature on stress and decision making (DM) in humans is far from internally consistent. To move towards reconciliation, and in a translational direction, it is important to understand methodological differences that challenge cross-study comparisons. In this review, we explore stress effects on DM-related processes focusing on valuation, learning, and risk-taking.

The Stress Construct

Stress has classically been defined as “the non-specific response of the body to any demand for change”, an adaptive homeostatic function [3]. It is associated with parallel activation of two biological systems: the quick-acting sympathetic-adrenal-medullary (SAM) axis, and the slow-acting hypothalamic-pituitary-adrenal (HPA) axis [4]. Sympathetic nervous system reactivity and associated catecholamine (CA; e.g., nor/adrenaline) release promote peripheral excitation that quickly returns to baseline [i.e., the “fight-or-flight” response; 5]. Concurrent brainstem signals of homeostatic disruption trigger HPA activation and corticosteroid release at a slower pace. Yet, this characteristic description does not convey wide-ranging individual differences based on stressor used (e.g., physiological or psychological) or stressor timing (e.g., when applied and exposure duration), and associated central/neuroendocrine dynamics. Across studies, variability in stress operationalization along these lines has contributed to inconsistencies in the stress-DM literature.

Differences in stressor timing can be conceptualized as an interaction between (at least) three factors: stress-to-task latency, stressor duration, and exposure across the lifespan. Given the different timelines of HPA/SAM reactivity, carefully calibrating stress-to-task latency is critical to link experimental outcomes with SAM and/or HPA physiology. For instance, a few minutes’ difference in latency can be enough to influence stress effects on risk-taking [6]. Similarly, stressors that are repeated or occur long-term (chronic) but not those of short-term duration (acute) have been associated with structural changes in DM-related brain regions in rats [7] and humans [8]. Stress effects on DM may also differ based on lifespan phase of the individual [9]. Adolescents exposed to early life stress, for example, are susceptible to changes in affective/motivational circuits typically involved in DM [e.g., amygdala, prefrontal cortex, and ventral striatum; 10].

Stress-to-task latency is also an issue with respect to cellular and neuroendocrine dynamics. Much stress and DM research has considered only slower genomic effects of cortisol after reaching peak levels, leading to general adoption of designs involving long stress-to-task latency. Yet, both fast-acting catecholaminergic and cortisol-based rapid non-genomic effects can influence brain function in DM-critical regions [e.g., prefrontal cortex, amygdala; 11]. It is noteworthy that as a class corticosteroids include not only glucocorticoids (e.g., cortisol) but also oft-overlooked mineralocorticoids (e.g., aldosterone), which have been linked to optimization of explicit memory retrieval [12]. While stress-DM research is only beginning to explore the latter, evidence suggests that mineralocorticoids play a central role in rapid non-genomic stress effects recently gaining attention in the literature [13].

Another source of variability across studies of stress and DM is the type of stressor used. These can generally be classified as systemic (i.e., physiological homeostatic disruptions like heat, pain, and cold), processive (i.e., psychological or psychosocial), or systemic/processive hybrids. In terms of HPA/SAM activation, systemic stressors are brainstem mediated whereas processive stressors require limbic system engagement [i.e., subjective identification of stimulus as threat; 14]. A related point is that certain stressors, such as those involving social-evaluation or uncontrollability, tend to yield greater peripheral cortisol [15].

This highlights the importance not only of stressor used but also individual differences in subjective appraisals of stressors as contributing factors to variability across findings.

Beyond methodological issues related to stress operationalization, differential stress effects on specific DM computations must be considered. While important and complex frameworks of DM computations have been proposed [e.g., 16], here we will focus on a simplified subset. Specifically, valuation (of anticipated or received decision outcomes), learning (updating of existing valuations), and risk-taking. Stress exposure may modulate any/all of these processes, a proposal that emerging research supports at this early stage; however, it is not always easy to identify the locus of stress' effect.

Stress and Valuation: Reward-related processing

A central axiom of DM research rests on the principle that people act to approach rewards and avoid punishments in their environment [17]. Thus, valuation of the appetitive/aversive nature of anticipated (or received) decision outcomes is a likely candidate for stress' modulation. Indeed, stress-altered sensitivity to rewarding/punishing outcomes (e.g., primary, food; secondary, money) appears to play a role in development of some pathologies including binge eating [18], pathological gambling [19], and anhedonia in depression [20; 21]. Given emerging evidence that valuation is a locus of stress' influence on DM, can a synthesis of research outcomes be reached accounting for methodological differences?

Initial evidence supports the idea that acute stress reduces sensitivity to rewards, including behavioral [22; 23] and neuroimaging studies highlighting an influence in regions including orbitofrontal cortex (OFC), medial prefrontal cortex (mPFC), amygdala and striatum [24–27]. Consistent with this, there have also been demonstrations that chronic (i.e., cumulative early life) stress is associated with blunted ventral striatal reward responses in adulthood [28]. Given methodological differences in stress-to-task latency and decision process (e.g., valuation at anticipation/receipt) across studies, however, it is still early to claim that stress universally blunts reward valuation.

Though reductions in reward-related responses have been observed using different stressors during anticipation [24; 25] and receipt [26; 27], there are some disagreements. For example, one of these studies employed a methodology that permitted exploration of stress effects as a function of decision process [27]. Enhanced responses associated with reward anticipation were observed under stress, in contrast with prior studies [24; 25]. A critical methodological difference is that studies reporting increases at anticipation used a short stress-to-task latency [i.e., immediately prior to task performance; 26; 27], compared to studies employing a longer latency which led to decreases [24; 25]. Thus, cross study comparisons show some consistency in results even with different stressors applied, but are not conclusive given differing cellular and neuroendocrine dynamics associated with stress-to-task latency.

Other recent behavioral research utilizing reinforcement-learning paradigms has demonstrated systemic and processive acute stress effects seemingly opposed to blunted reward valuation. There learning, operationalized as improved choice after repeated positive/negative feedback, was impaired for decisions based on negative outcome feedback but

enhanced for positive (i.e., rewarding) feedback [29; 30]. Thus, a reasonable hypothesis is that "stress triggers increased reward salience" [STARS; 31]. The noteworthy STARS model is consistent with research linking stress and cortisol to increases in extracellular dopamine in rats in mPFC, dorsal and ventral striatum [32; 33], replicated in the human PET literature [34; 35]. This begs the question, however, of how previous reports of blunted valuation for rewards but not punishments can be reconciled with STARS in reinforcement-learning [36].

Beyond potential difficulty in directly comparing fMRI BOLD with PET/animal neurochemical results, it is plausible that different processes were examined. For example, in reinforcement-learning increased reward "salience" may represent enhanced retrieval of representations of previously learned reward associations rather than enhanced valuation of rewards per se. Consistent with this interpretation, in one of the learning studies above stress-blunted reward sensitivity was observed in early trials despite ultimately enhanced reward-based performance [29]. Another possibility is that stress influences different components of a reward outcome [e.g., the affective value, but not the information; 37] or impacts learning via reward but not punishment [38]. It is clear moving forward that novel experimental designs focus on careful manipulation of such factors to dissociate stress effects on the intertwined processes of valuation and learning.

Stress and Learning: The Role of Habit

A logical next question relates to how stress might influence expression of previously learned outcomes. Research across disciplines supports the idea that DM processes can be placed on a spectrum ranging from (I) habitual, stimulus-bound, automatic, and less effortful, to (II) goal-directed, flexible, controlled, more effortful and resource-dependent [39–42]. As learning proceeds over time to establish strong and ingrained prior expectations informing DM, might stress exposure bias choice towards those expectations and away from novel but relevant information (i.e., a goal-directed to habit-based shift)? Consider, for example, how habitual an elevator button press to one's floor at work becomes over time. If one day circumstances require a different floor be chosen after a stressful experience, is a person more likely to choose the goal-directed or habitual button?

Chronic stress may support a shift to habitual responding while promoting an insensitivity to novel goal-directed contingencies. For instance, rats under chronic stress operantly conditioned to respond for two food rewards tend to perseverate in responses associated with the devalued stimulus [classic devaluation studies would suggest a reduction; 7]. Structural neural changes in such animals is also observed, with atrophy in mPFC and dorsomedial striatum [associated with goal-directed DM; 43] but hypertrophy in the dorsolateral DM [associated with habit-based DM; 44]. Taken together, these data suggest that a goal-directed to habit-based shift may possibly become a persistent change under chronic stress.

A nascent human literature involving acute stress using a similar devaluation approach based on primary reinforcers converges with the above [45; 46]. Additionally, an analogous fMRI study involving exogenous administration of hydrocortisone and the β-adrenergic antagonist yohimbe (to mimic combined HPA/SAM engagement) yielded similar outcomes [47]. There, BOLD responses in OFC and mPFC for devalued outcomes were reduced and a

perseverative DM pattern manifested. This implies a key interaction between DM and memory processes, that synergistic HPA/SAM engagement may promote a goal-oriented to habit-based shift [48]. It is notable that mineralocorticoids may also play a critical role [49].

Potential clues about underlying mechanisms can be drawn from well-developed human and animal literatures on stress, learning, and memory. It has been proposed that under stress combined glucocorticoid/noradrenergic activity promotes a mode of hippocampal memory formation by which stress-associated experiences are strongly consolidated and ancillary systems brought offline [50], resulting in a shift in neural resources away from executive towards salience networks that enhance vigilance and fear [51]. This converges with a rich literature on stress-related impairment of prefrontal-based working memory linked to excess catecholamine release [52].

In fact, greater working memory capacity serves as a protective factor against stress-related impairments in model-based learning [a goal-directed form of reinforcement learning; 53]. Critically, stress-influenced brain regions discussed earlier in valuation are consistent with prefrontal and dorsomedial striatum mediated goal-directed processing [54]. It is plausible that valuation impairments may play a role in goal-directed/habit-based shifts under stress (e.g., insensitivity to devaluation). That said, timing of stress exposure is likely to influence which system informs DM most strongly [48], as it is yet unclear how differing stress-to-task latencies may promote or impair shifts on a goal-directed/habit-based spectrum.

Stress and Risk-Taking

Another prominent emphasis in stress-DM investigations is risk-taking, a critical issue given its prevalence in stressful real-life contexts including medicine [55], psychopathology [56], and financial investing [57]. Decision-makers' likelihood to engage in risk varies greatly based on multiple decision-inherent features including uncertainty [i.e., degree of information informing outcome predictability; 58], framing of a decision [as a potential gain or loss; 59], and valuations of outcome valence, magnitude, and probability of receipt [also combined to compute expected utility; 60]. As such, decisions involving risk-taking rely in part on stress-susceptible valuation/learning processes and brain regions already discussed [61–63]. Though, little research has examined the neural substrates of stress in this context necessitating focus on a growing behavioral literature.

Acute stress effects on risk-taking have yielded mixed results in decision making tasks under risk characterized by explicit probabilistic information (e.g., 50% chance of $100). Multiple studies have reported risk-taking increases when decisions are framed as potential financial gains [64–66], though longer stress-to-task latencies have recently been shown to be a factor with respect to decisions under risk [i.e., greater risk taking immediately after stress but reduced risk-taking 45 minutes later; 67] Other studies separating decisions under risk by domain (gain/loss trials) are inconsistent. For instance, acute systemic stress applied in that context has led to an exaggerated reflection effect (i.e., decreased risk-taking for gains but increases for losses) interpreted as a stress-related shift towards habit-based DM [68]. More recent processive stress studies also manipulating decision frame yield different outcomes, reductions (rather than increases) in loss risk-taking and no effects during gains [69; 70].

Beyond a systemic/processive stressor difference, other design elements may partially explain the discrepancy in results. In the former [68], decisions were limited to two domains and equal expected values, a fast-paced time-limited choice period, and a large number of trials. In the latter two [69; 70], decision options were more varied (i.e., gain/loss/mixed and several options), time to choose unlimited, and fewer trials offered, all which reduced repetition and increased variability of choices. Thus, one possibility is that a fast-paced and repetitive methodology promoted sensitivity to a stress-related habit-based shift in the first case whereas a slower-pace, less repetition, and varied expected values in the second two may have promoted a more deliberative strategy and enhanced salience of potential losses.

Compared to decisions based on explicit probabilistic information, in decision making tasks under ambiguity, where such information is not available (e.g., x% of $100) results are different but more consistent. Specifically, multiple studies have reported reduced risk-taking in stressed females but increases in males. This holds true both whether risk-taking is financially disadvantageous [i.e., Iowa Gambling Task; 71; 72] or advantageous [Balloon Analgoue Risk Task; 73; 74]. While one study reported no stress/sex effects at all [66], early evidence appears to support the proposal that in decisions made under ambiguity acute stress increases risk-taking in males while decreasing it in females. A potential explanation for this is that females may be more ambiguity averse in some DM contexts [75], which could be exacerbated under stress.

While these early studies indicate that acute stress can influence risk-taking, its effect varies. When decisions provide little/no probabilistic information (i.e., ambiguous), stressed females may be more risk-averse and males more risk-seeking. With more explicit information to inform choice, stress effects appear to differ based on methodological elements related to stress and decision operationalization. A recent meta-analysis offers insights along these lines, highlighting that (a) stress may promote increased risk-taking/reward-seeking even when this leads to disadvantageous outcomes, and (b) processive stressors yield more reliably stress effects than systemic ones [potentially due to enhanced HPA reactivity; 76]. Notably, no effects of moderating factors such as sex, age, neuroendocrine response, and stress-to-task latency were observed. This is surprising given growing research supporting the importance of such moderators, for example stress-related risk-taking increases in adolescents [77; 78]. Though it is premature to draw strong conclusions given small sample size, likely without adequate representation of moderating factors and great methodological variability across included studies (e.g., uncertainty level), the meta-analysis represents an important step forward and helps shape impending research.

Future Directions

Despite some lack of internal consistency given a wide range of between-study methodological differences, the human stress and DM literature has made great advances over the last few years. For instance, there are consistent observations indicating that stress exposure reduces reward valuation upon receipt of an outcome yet questions remain at anticipation due to differences in stress-to-task latency. A growing consensus supports a propensity to shift towards habit-based from goal-directed systems under stress, potentially associated with facilitation of reward-based reinforcement-learning – but also an

insensitivity to updated environmental contingencies that can be maladaptive in some contexts. At choice, stress can exert an influence at multiple levels ranging from altered valuation/feedback processing and automaticity effects expanded on in this review, to increased impulsivity in decision implementation. While some studies report stress-related modulation of risk-taking and/or disadvantageous choices, drawing strong conclusions would be premature as this literature in particular is subject to great variability in methodology and outcome measures.

Yet, there is room for growth and exciting future directions. In stress operationalization, fruitful future avenues include targeted manipulations of timing and stressor type as previously discussed. Gaining a greater understanding of stress' cellular and neuroendocrine influences on DM, so strongly influenced by timing, will significantly improve our ability to make cross-study comparisons. For example, though some studies discussed here linked reported effects to cortisol reactivity many did not. Exploration of neuroendocrine dynamics little-examined under stress will be critical to gain new insights, including rapid nongenomic versus slow genomic cortisol effects and the role of mineralocorticoids. Also of note are important quasi-independent factors recent research indicates clearly interact with stress such as sex [73], age [9], other stress-influenced neuroendocrine factors [e.g., oxytocin and testosterone; 79], and genetic variants influencing catecholaminergic and executive function [80].

Future studies will also benefit greatly from targeted manipulations of DM computation components such as valuation (e.g., beyond expected value) and learning (e.g., stress effects at different stages of learning). One direction may be carefully decomposing the influence of stress on various DM components, as in a recent study controlling for risk attitudes, loss aversion, and choice consistency which reported no acute stress effects [suggesting stress might influence other processes that contribute to DM; 81]. It is also plausible that acute stress affects subcomponents of receipt of reward-related information that may exert distinct influences on valuation and learning (e.g., affect versus quantitative information). Along these lines, early stress-DM investigations involving decisions with a more complicated structure accounting for factors like intertemporal discounting [82] and loss aversion [83] raise interesting questions as to the locus of stress' influence even within a specific computation (e.g., probability versus magnitude, etc.). Finally, there are exciting extensions with respect to decision making in the social context. For example, prosocial behavior is influenced by stress exposure, with increases in self-interested decisions during social exchange games against strangers [84–86] but generosity towards close others when decisions and stress exposure were close in time [87].

Looking ahead, stress-DM research has great potential to contribute to science in both the basic and applied senses. To reach that point, however, it will be critical to develop a common methodological framework for stress research implementation and reporting. The benefits of moving in a translational direction to inform clinical work and ameliorate everyday lives cannot be overstated. For instance, individuals may have difficulties using emotion regulation strategies under stress [88] which could lead to deficits in decision making such as reduced self-control [89]. Increasing positive emotion [90] or fostering a perception of control in the face of stress could serve as alternative coping mechanisms with

potential consequences for decision making, such as promoting persistence in goal pursuit [91]. Future advances along these lines will move the field in an exciting and valuable applied direction.

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HIGHLIGHTS

- Acute stress can impair valuation of reward information critical to decision making
- Acute stress influences a shift from goal-directed to habit-based decision making
- Effects of acute stress on risk-taking are mixed but promising for future studies