Physiology & Behavior 164 (2016) 164–181

Contents lists available at ScienceDirect

Physiology & Behavior

journal homepage: www.elsevier.com/locate/phb

Review

Why are there lasting effects from exposure to stress during development? An analysis of current models of early stress

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HIGHLIGHTS

- Stress exposure in early life can affect adult physiology, behavior, and cognition.
- Many hypotheses have been proposed to explain why stress can shape adult phenotype.
- The strengths, limitations, and applications of current hypotheses are discussed.
- Application of current hypotheses may be species and context dependent.

ARTICLE INFO

Article history:
Received 19 January 2016
Received in revised form 29 April 2016
Accepted 17 May 2016
Available online 18 May 2016

Keywords:
Early life stress
Adolescence
Predictive Adaptive Response
Reactive scope
Allostasis
Mismatch

ABSTRACT

The potential for stressful experiences in early life to cause lasting changes in phenotype is well documented, but the functional and evolutionary context of these changes is not well understood. Many hypotheses have been proposed to explain the role of lasting effects of stress exposure during gestation and early development; the purpose of this review is to discuss these hypotheses in the context of human and non-human animal research in the last three decades in order to (i) further dialogues between those approaching early stress from biomedical and evolutionary/ecological perspectives, (ii) outline strengths and limitations of current hypotheses, with respect to species and context-specific effects of exposure to stress in early development, and (iii) address recent evidence suggesting that stress in early development can have beneficial effects in adulthood. It is suggested that the hypotheses discussed are not mutually exclusive, but the applicability of each hypothesis will depend upon the environmental conditions and stability a species, or perhaps even an individual, experiences in their lifetime. Potential investigations to clarify applications of the current hypotheses are discussed, including longitudinal studies that span multiple developmental stages and investigations of species where measures of fitness are possible.

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http://dx.doi.org/10.1016/j.physbeh.2016.05.032
0031-9384/

1. Exposure to stress in early life can cause lasting changes in adult phenotype

The potential for exposure to stress in early development to induce persistent changes in phenotype has been of interest to the scientific community for at least six decades [21,164,238]. Exposure to adverse environmental conditions in early life can modulate fitness and human health outcomes by causing lasting changes in behavior, cognition, and physiology [40,41,155,240,241]. Lasting changes in phenotype caused by early exposure to stress (Box 1) have been well studied in mammals and documented in numerous non-mammalian taxa including fish (*Pomacentrus amboinensis*, [159]; *Gasterosteus aculeatus*, [90]), birds (*Sturnus vulgaris*, *Coturnix japonica*, reviewed in [112]), lizards (*Lacerta vivipara*, [60]; *Pseudomoia pagenstecheri*, [221]), and invertebrates (*Daphnia cucullata*, [1]). Exposure to stress in early life can (i) modulate reproductive outcomes, (ii) have lasting effects on longevity, risk for disease, perceived wellbeing, cognitive performance and cognitive bias, and (iii) shape physiological, cognitive, and behavioral responses to future challenges ([41,46,145,190]; Table 1). Many hypotheses have been proposed to explain the evolutionary role of lasting effects from exposure to stress in early life. The purpose of this review is to discuss these hypotheses in order to (i) encourage dialogues between those contextualizing the effects of early stress in biomedical and evolutionary/ecological frameworks, (ii) outline strengths and limitations of existing hypotheses with respect to species and context-specific effects of early stress exposure, and (iii) address recent findings that suggest stress exposure in early development can have beneficial effects in later life stages.

Box 1
Defining the term "stress"

Defining stress is both a modern and historical challenge, in part because the term is used to refer to three distinct phenomena: (1) stimuli in the environment that are aversive or challenging (also called stressors), (2) behavioral and somatic emergency responses (also called the stress response), and (3) the consequences of over-stimulation of the emergency response [205]. Constructing a definition of stress is further complicated by mounting evidence that these three phenomena can be species-specific. For example, most organisms would collapse under 5000 m of water (nearly 500 times more pressure than above water), but some deep sea organisms tolerate these conditions, including the common fangtooth fish (*Anoplogaster cornuta*). Despite the great pressure tolerance of *A. cornuta*, it may not survive if brought to shallow water inhabited by pelagic fish [259]. Though remarkably different, both extremophiles, such as *A. cornuta*, and mesophiles are likely to experience stress in environments outside their preferred conditions. However, species can also differ in their response to conditions outside a preferred range. For example, the sand cat has extensive adaptations as a desert specialist but is rarely found outside this range, whereas the closely related domestic cat (*Felis catus*) has extensively expanded its range while outcompeting native species, in part because of their broad and rapidly adjusting tolerance range [192,232]. Extreme conditions that exceed a species' tolerance range, such severe environmental changes, are essential to the life cycle of some species, even if the conditions are ultimately fatal (e.g. semelparous species or fire-stimulated seed release [25,33,78,132,52]). Further, the environment can shape the stress response at both the level of the species and the individual; even though aspects of the physiological stress response are highly conserved across taxa and conditions (even between yeast and mammals), responses to stress can vary considerably, especially behavioral responses, both between closely related species and individuals within a species [72,255,268,273]. For example, behavioral responses to stress vary widely from "active" responses, such as tail flagging in squirrels and inflation in pufferfish, to "passive" responses, such as freezing in rats and octopi [24,105,111]. Yet, these opposing behavioral responses are associated with similar physiological responses ([237,270]). Compounding this, aspects of the physiological stress response are activated for both "positive" stressors, such as mating, and negative stressors, such as social conflict [204]. Humans injected with epinephrine will attribute their arousal to either a negative or positive emotional state depending upon the experimental context, which suggests that an individual's interpretation of their stress response is based on external cues and cognitive labels [274]. The issues that complicate the definition of stress include (i) the subjective influence of context, (ii) use of the term for three phenomena, (iii) inter- and intra-species differences, and (iv) the potential for self-report in humans but not other taxa.

2. Does early stress exposure make individuals vulnerable to later stress, or prepare them for it?

Extensive literature describes adverse effects of exposure to stress in early life, yet a growing number of studies have found that early life stress exposure can have effects on behavior, morphology, physiology, and reproductive outcomes that increase performance or potentially enhance fitness (Table 1). The concept that stress can have seemingly beneficial effects is not new; Southam & Ehrlich [227]) termed the phenomenon "hormesis" and Hans Selye [212]) called it "eustress" (though these terms are no longer frequently used). Selye, often referred to as the father of stress, emphasized the potential positive effects of stress and advised that "we must not suppress stress in all forms, but diminish distress and facilitate eustress" [213].

Currently, the available evidence supports both beneficial and detrimental consequences of early stress exposure, and it is contentious whether stress in early development can prepare an individual for subsequent stress or have positive effects later in life. For example, exposure to stress in early life has been robustly shown to increase anxiety across taxa, with potentially pathological consequences [71,86,231,250]. Yet, humans with higher levels of anxiety are more attentive to threats and identify threatening stimuli more quickly [35,148,149]. Highly anxious rats and humans have a larger amplitude startle response ([126]; reviewed in [57]), increased activity of the autonomic nervous system [169], and elevated resting levels of norepinephrine, all of which serve as vital components of the initial stages of the stress response (Box 2, [154,214]). The consequences of these changes can be context specific - an increase in anxiety caused by stress exposure in early life might enhance threat avoidance in a high-threat environment, but prohibit an animal from pursuing available resources in a low-threat environment [125].

The costs and benefits of effects from early stress exposure can depend upon environmental conditions later in life [219]. For example, in viviparous lizards (*L. vivipara*), prenatal-corticosterone exposure can increase attraction to maternal odor and reduce the likelihood of dispersal [60,134]. Lizards that do not disperse are less likely to encounter novel resources or escape competition, but have reduced mortality, which is one of the most important factors determining fitness for *L. vivipara* [172,173]. Yet, some responses to stress may be so extreme as to not convey an advantage in any environment [264]. In humans, early stress exposure can increase adult risk for hippocampal atrophy, cognitive impairment, and mental illness, including posttraumatic stress disorder (PTSD). By definition PTSD impairs functioning and can detract from health and wellbeing through chronic symptoms including flashbacks, dissociation, nightmares and general sleep disruption, memory problems, emotional numbness, and self-destructive behavior [26,139].

Table 1
Studies reporting lasting effects from stress in early development that appear to enhance functioning (organized by the effect type, then age at which the effect was seen).

Reference	*Species*	*Stress exposure*	*Effect type*	*Stage at testing*	*Outcomes of stress exposure*
[156]	Ringed salamander (*Ambystoma annulatum*)	Eggs exposed to chemical predation cues (dissolved in water, twice per day for 3 days)	Behavioral	Larvae	Exhibit antipredator behaviors from birth; low activity; increased shelter-seeking
	Wood frog (*Rana sylvatica*)	Eggs exposed to predation cues and/or alarm cues (dissolved in water, twice per day for 3 days)	Behavioral	Tadpole	Tadpoles classically conditioned to respond to newt predation cue
[226]	Pacific sockeye salmon (*Oncorhynchus nerka*)	Semelparous mothers exposed to physical stress (chased with a net for 3 min, twice per day, for ~40 days)	Behavioral/motor	Fry (~1 month after absorption of the yolk sac)	Faster burst swimming rate (but slower swimming duration)
[173]	Lizard (*Lacerta vivipara*)	Pregnant females exposed to corticosterone until parturition (13.5 μg CORT/day, 15 days on average)	Behavioral	Juvenile	Corticosterone-exposed offspring were more likely to disperse from small/young moms but less likely to disperse from large (more experienced) moms
[8]	Laboratory rat (*Rattus norvegicus*)	Exposure to elevated platform stress as juveniles (26–28 days of age) and adults (60–62 days of age), compared to stress exposure twice in adulthood	Behavioral	Adulthood	Faster swimming and more exploration in a Morris water maze compared to rats reared without stress
[45]	Laboratory rat (*Rattus norvegicus*)	Chronic unpredictable stress in adolescence (physical, social, predation stressors from 30 to 70 days of age)	Behavioral	Adulthood	Adolescent-stressed rats showed enhanced vigilance in low-threat conditions but matched unstressed rats in foraging behaviors; in high-threat conditions adolescent-stressed rats matched unstressed rats in vigilance and foraging behaviors (but adolescent-stressed rats obtained more food [43]), suggesting an increase in foraging efficiency
[34]	Laboratory rat (*Rattus norvegicus*)	Social and physical defeat in adolescence (at 28, 31, and 34 days of age)	Behavioral	Adulthood	Faster attack latency and fewer received attacks in adulthood compared with rats reared in control conditions
[36]	Japanese quail (*Coturnix japonica*)	Unpredictable variable stress beginning two weeks after hatching (5 stressor types, 12 stressors per day, for 8 days)	Behavioral/cognitive	Early life (23–31 days of age)	Accelerated reversal learning in a spatial task compared with birds not exposed to developmental stress
[96]	Domestic chicken (*Gallus gallus*)	Daily isolation for 30 min, (from 4 to 26 days after hatching)	Behavioral/cognitive	Juvenile (50–51 days of age)	Enhanced associative learning compared with chicks not exposed to developmental stress
[80]	Zebra finch (*Taeniopygia gutta*)	Nestlings fed corticosterone in peanut oil twice daily (6.2 μg CORT/day)	Behavioral/cognitive	Juvenile (37-approx 60 days of age)	Corticosterone fed nestlings were faster to solve a novel foraging task in a social setting and showed increased social learning from unrelated adults (but decreased social learning from parents) compared with control nestlings fed only oil
[198]	Humans	Physical abuse in childhood (8–10 years of age)	Behavioral/cognitive	Childhood	Abused children recognized angry faces with less information compared with unabused children
[203]	Humans	Physical abuse in childhood (4–9 years of age)	Behavioral/cognitive	Childhood	Abused children made fewer errors when recalling aggressive stimuli compared with unabused children
[215]	Humans	Physical abuse by the mother (duration and timing of abuse unspecified, but verified by the Department of Human Services)	Behavioral/cognitive	Childhood (~9.5 years of age)	Abused children exhibited greater attention and accuracy on tasks with anger cues compared to unabused children (but also showed greater attention to anger cues when they were task-irrelevant, such that performance was very context specific)
[153]	Humans	Children removed from homes for severe neglect and/or physical or sexual abuse	Behavioral/cognitive	Childhood (~11.5 years of age)	Maltreated children were faster to identify facial emotions (fearful and happy) than control children (no difference detected for neutral faces)
[66]	Humans	Maternal anxiety, nonspecific stress, and depressive symptoms during 2nd and 3rd trimesters	Behavioral/cognitive emotional	Childhood (2 years of age)	Children of mothers with high anxiety/stress/depressive symptoms had more advanced mental and psychomotor development (composite measures including attention, cooperation motor speed & coordination, etc.) but no change in engagement/affect
[243]	Laboratory rat (*Rattus norvegicus*)	Variable stress during juvenile development (once per day, from 27 to 29 days of age, with a different stressor each day)	Behavioral/cognitive	Adulthood	Enhanced auditory fear conditioning compared with unstressed controls
[41]	Laboratory rat (*Rattus norvegicus*)	Chronic unpredictable stress in adolescence (physical and social stressors from 30 to 70 days of age)	Behavioral/cognitive	Adulthood	Adolescent-stressed rats showed accelerated decision making compared with unstressed rats
[53]	Zebra finch (*Taeniopygia gutta*)	Nestlings fed corticosterone in peanut oil for 17 days (6.2 μg CORT/day from 12 to 15 days of age, 8.15 μg CORT/day from 16 to 28 days of age)	Behavioral/cognitive	Adulthood	Nestlings fed corticosterone oil solved a foraging task in fewer trials compared with nestlings fed oil without corticosterone

Table 1 (*continued*)

Reference	*Species*	*Stress exposure*	*Effect type*	*Stage at testing*	*Outcomes of stress exposure*
[43]	Laboratory rat (*Rattus norvegicus*)	Chronic unpredictable stress in adolescence (physical, social, and predation stressors from 30 to 70 days of age)	Behavioral/cognitive	Adulthood	Adolescent-stressed rats showed enhanced foraging performance in the presence of novel threat (but unaffected foraging performance in the absence of threat) compared with unstressed rats
[44]	Laboratory rat (*Rattus norvegicus*)	Chronic unpredictable stress in adolescence (physical, social, and predation stressors from 30 to 70 days of age)	Behavioral/cognitive	Adulthood	Adolescent-stressed rats showed enhanced reversal learning, but no change in associative learning or reference memory in an appetitive task
[221]	Lizard (*Pseudemoia pagenstecheri*)	Gestating females exposed to snake scent in bedding and shelter sites for 39 days (with the scent renewed twice per week)	Behavioral morphological	Neonate	Neonatally-stressed offspring had (i) higher sensitivity to snake odor compared with offspring from unstressed females; (ii) increased body mass; (iii) long tails (potentially to enhance tail autotomy – an antipredator behavior)
[68]	Aspic vipers (*Vipera aspis*)	Water deprivation for 20 days mid-gestation (congruent with summer drought conditions)	Morphological physiological	On the day of birth and 2 weeks after birth	Water-deprived mothers had higher baseline corticosterone and produced offspring with higher growth rates
[90]	Three-spine sticklebacks (*Gasterosteus aculeatus*)	Females chased with predator model for 30 s per day until gravid (about 25 days)	Behavioral morphological physiological	Juvenile	Tighter shoaling (antipredator behavior); larger eggs; higher cortisol levels in eggs
[188]	Laboratory rats (*Rattus norvegicus*)	Maternal separation for 24 h (a model of neglect) at 3 days of age	Cognitive neurological	Adulthood	Maternally separated rats showed enhanced (i) fear memory and (ii) synaptic plasticity following corticosterone exposure
[46]	Laboratory rats (*Rattus norvegicus*)	Pups exposed to low licking mothers (compared with pups exposed to high licking mothers)	Behavioral neurological	Adulthood	Enhanced (i) contextual fear memory and (ii) long-term potentiation following corticosterone exposure (but impaired under basal conditions)
[54]	Zebra finches (*Taeniopygia gutta*)	Male nestlings fed corticosterone for 17 days (6.2 μg CORT/day from 12 to 15 days of age, 8.15 μg CORT/day from 16 to 28 days of age)	Behavioral reproductive	Adulthood	Increased parental provisioning (female driven); higher body condition in offspring; greater number of genetic offspring for males
[190]	Humans	Dutch famine (1944–1945); maternal daily ration less than 1000 cal for at least 13 weeks	Reproductive	Adulthood	Women exposed to the Dutch famine (1944–1945) in utero began reproducing earlier, had more children and twins on average, and were less likely to be childless (at 50 years of age) than women born before/conceived after the famine. No differences in reproductive outcomes were detected in males.
[172]	Lizard (*Lacerta vivipara*)	Pregnant females exposed to corticosterone until parturition (13.5 μg CORT/day, 15 days on average)	Survival	Juvenile	Corticosterone-treated male offspring had greater survival after release (but female survival was unaffected)

CORT = Corticosterone.

Box 2
The vertebrate stress response

Across many vertebrate taxa, including mammals, animals respond to a challenge within seconds by releasing epinephrine and norepinephrine from their adrenal medulla and locus coeruleus – these key catecholamines act as messengers for the sympathetic stress response to prepare the body for immediate action [146]. Following this, the parasympathetic stress response is activated: the hypothalamus sends corticotrophin releasing factor (CRF) to the pituitary, which causes the pituitary to release adrenocorticotropic hormone (ACTH) into the blood to trigger the production of glucocorticoids (cortisol, corticosterone) in the adrenal cortex (this 3-step process is referred to as the hypothalamic-pituitary-adrenal axis and is regulated by the hippocampus and frontal cortex). Glucocorticoids have diverse functions throughout the body, including the mobilization of energy resources [146,161]. They act largely by activating or suppressing the transcription of specific genes [210]. Glucocorticoids also function in their own negative feedback system, by binding to glucocorticoid and mineralocorticoid receptors in the central nervous system, and glucocorticoids play an important role in recovery from challenge [97].

The concept that stress exposure may prepare an individual for subsequent stress crosses disciplines; a phenomenon called "preconditioning" (when small amounts of stress exposure reduce tissue damage caused by a larger, subsequent stressor) has been detected in numerous tissue types, including cardiac, endothelial, liver, kidney, and brain tissue, across several species including dogs, pigs, rabbits, rats, and humans [107,127,180,251,263,267,269]. For example, in 7-day old rat pups, exposure to hypoxia the day before combined hypoxia and ischemia can completely prevent the 34% loss of cerebral hemispheric weight detected in rat pups that were not preconditioned with hypoxia exposure [88]. In adult rats, exposure to a small amount of ischemia (3 min) before a more major ischemic episode (6 min) can prevent neuronal death and damage in the hippocampus [114]. In this study, the first ischemic event initiated a (seemingly preparatory) cascade: preconditioned neurons released adenosine that bound to adenosine A1 receptors, which opened adenosine triphosphate (ATP)-sensitive K+ channels [114]. Preconditioning has been a topic of research for three decades and can be caused by various types of stress, including oxygen deprivation, physical stress, oxidative stress, and heat stress [38,114,122,127,151,183]. The specific effects and mechanisms of preconditioning appear to vary with the type of stress, the type of tissue, and over time; for example, delayed or late cardiac preconditioning protects against myocardial infarction and myocardial stunning while classic preconditioning (on a shorter timescale) protects against only

infarction (reviewed in [73]). Importantly, there is overlap between the mechanisms of preconditioning across different types of stress (such as the activation of heat-shock proteins), suggesting that preconditioning may have preparatory effects that span multiple types of stress [140,184]. Studies exploring preconditioning effects may inform discussions of phenotype-level changes following stress exposure in early life, including (i) investigations of preconditioning and early life stress effects on comparable timescales, (ii) comparisons of preconditioning effects across developmental stages, and (iii) comparisons between mechanisms of preconditioning and phenotypic changes resulting from early life stress.

3. How can we explain the effects of developmental stress?

Numerous distinct hypotheses, developed from both clinical and animal research, address why early life experiences can have lasting effects on phenotype. Hypotheses that address this phenomenon are introduced and discussed below; though this is not a complete list of current and historic hypotheses related to developmental stress, the synopsis presented here is intended to represent the current state of developmental stress research. Many of the hypotheses presented are not mutually exclusive, as they target different questions related to the lasting effects of early stress (defined below for each hypothesis). Additionally, some more recent hypotheses apply ideas from important older hypotheses to new contexts. For example, the Environmental mismatch and the Predictive Adaptive Response hypotheses expand the Thrifty Phenotype hypothesis to better address a greater number of species and environments. It should also be noted that some of the hypotheses discussed, particularly the Allostasis and Reactive Scope models, were not proposed as models of developmental stress, but rather as more general models of how animals deal with stress. Here, these hypotheses are discussed in a developmental framework and compared to hypotheses that more explicitly model ontogenetic change.

3.1. The Thrifty Phenotype hypothesis

The Thrifty Phenotype hypothesis was proposed by Hales and Barker [103] to advance earlier lines of thought by Neel [181]; it addresses the question of why maternal effects cause context-dependent health outcomes and explains the counterintuitive link between prenatal food restriction and Type 2 diabetes in adulthood. The Thrifty Phenotype hypothesis suggests that maternal cues during gestation signal information about resource availability to the offspring, which results in immediate but permanent changes in offspring phenotype. These changes can help the fetus survive acute challenges, but may risk future health outcomes. Hales and Barker suggest that these permanent phenotypic changes are context-specific and can provide advantages in environments similar to the maternal environment, but can be detrimental if the environment differs from the maternal environment [103,104]. Scores of studies addressing these ideas have tracked famine survivors from communities in the west Netherlands who were exposed to extreme rationing during the winter of 1944–1945 as a consequence of an Axis embargo on food transport in WWII. This tragedy has become a natural case study for the effects of temporary food restriction on human development. We now know that exposure to famine during gestation is correlated with low birth weight and adverse health outcomes in food-rich environments, including increased risk of coronary disease, glucose intolerance, breast cancer, obesity, obstructive airway disease, and blood coagulation problems, depending upon gestational stage at exposure ([201,248], reviewed in [206]). Similar effects have been detected in rats; prenatal nutrient restriction, followed by unlimited access to food after birth, can result in hyperphagia and an increased preference for high-fat foods in adult rats, as well as reduced longevity, early pubertal maturation, and reduced sex steroid levels in adulthood ([120,225,249], but see [135]). These findings support Hales and Barker's [103] assertion that exposure to mismatched environments during gestation (food restricted) and adulthood (food-rich) can cause adverse health outcomes.

An early issue facing the Thrifty Phenotype hypothesis was the degree to which environmental pressures and genetic differences mediate lasting changes in phenotype following fetal nutrient restriction (reviewed in [104]). A rich literature now explores gene by environment interactions, including studies examining genetic polymorphisms in humans, twins exposed to varying environmental conditions, transgenerational effects, and studies of animal models that manipulate maternal and offspring diet (e.g. protein, fat, or calorie restriction, reviewed in [145,189]). This seminal hypothesis has been applied successfully for nearly 25 years and its core arguments have been incorporated and expanded in several more recent hypotheses (including the Predictive Adaptive Response [Section 3.4.] and Environmental mismatch hypotheses [see Section 3.5.]). These more recent hypotheses have updated the ideas of the Thrifty Phenotype hypothesis to (i) more readily translate across taxonomic groups, (ii) address how the type and duration of stress can modulate lasting changes in phenotype, and (iii) explain how the effects of early stress can vary across stages of development.

3.2. The Predictive Adaptive Response (PAR) hypothesis

Maternal influences during gestation and early postnatal life have frequently and independently been suggested to prepare offspring for subsequent environmental conditions [12,15,28,93,98,142,173,177,219]. In a keystone paper Gluckman et al. [93] proposed the Predictive Adaptive Response (PAR) hypothesis to address the question of why effects from early environmental conditions can appear after a delay and cause context dependent health and fitness outcomes. The PAR hypothesis states that the effects of early life stress (i) can be advantageous if the environment remains consistent with the early life environment, or detrimental if the environment changes, and (ii) can manifest in later developmental stages - delayed phenotypic changes are referred to as Predictive Adaptive Responses (PARs). To introduce the hypothesis, Gluckman et al. [93] discussed a now classic example of a PAR: meadow vole (*Microtus pennsylvanicus*) coat thickness is determined by environmental conditions during gestation, but does not have a functional role until after weaning [133].

As studies of developmental stress exposure become more frequent, examples of changes that appear after a delay are also increasing [277] – after maternal separation, offspring later exhibit decreased parvalbumin in prefrontal cortex interneurons and decreased synaptophysin in the hippocampus [3,27]. Similarly, after chronic stress in adolescence, rats exhibit decreased hippocampal volume after a delay [117]. Though the mechanisms facilitating delayed effects are unclear, and may differ depending on the species or system affected, it should be noted that responsivity to stress can vary as a result of ontogenetic change [146,228] For example, during the first two weeks of life rodents are hyporesponsive to stress as the HPA axis matures. This hyporesponsive period is marked by attenuated HPA axis responsivity that is maintained by the presence of the dam (and is disrupted by maternal separation [137], reviewed in [146]). A similar hyporesponsive period may exist in humans [100]. Developmental changes in stress responsivity, such as a hyporesponsive period in early life, may contribute to a delay in the appearance of effects from early stress.

Gluckman et al. [93] related their ideas to the earlier Thrifty Phenotype hypothesis; Hales and Barker [103] suggested that a nutrient-restricted fetus will undergo changes that are advantageous in a nutrient-restricted environment, but detrimental in a nutrient-rich environment - Gluckman et al. suggested that fetuses undergo permanent changes (PARs) in *all* nutrient conditions to prepare for matching nutrient conditions later in life (Table 2). Thus, two key differences that distinguish the PAR hypothesis from the Thrifty Phenotype hypothesis are that (1) PARs are permanent phenotypic changes that are induced in all environments (i.e. PARs enable an individual to match moderate, not

Table 2
Features and applications of current models of early life stress (summarized from text, see Section 3 for additional details and citations).

Theory	Question(s) of interest	Distinguishing features	Application considerations
Thrifty phenotype hypothesis	Why do maternal effects cause context dependent health outcomes? How can we explain the counterintuitive link between prenatal food restriction and increased likelihood of Type 2 diabetes in adulthood?	Changes from early stress exposure are permanent, and may be necessary to survive an adverse period (e.g. famine), but lasting changes in phenotype likely risk future outcomes.	Well suited for humans and species with limited plasticity/flexibility in adulthood or relatively stable postnatal food availability.
Predictive Adaptive Response hypothesis	How do early life conditions cause context dependent health and fitness outcomes? Why do early life conditions cause changes that appear after a delay?	Animals create permanent Predictive Adaptive Responses (PARs) to all early life conditions in anticipation of later life conditions, so consistency between early and later life conditions is vital for fitness. Emphasis on effects of early stress that appear after a delay, and the potential for effects to have functions specific to the stage in which they manifest.	Well suited for delayed effects of prenatal stress, especially in species with longer life spans, those that seek different environmental conditions across development (e.g. dispersal, migration), or species with limited plasticity/flexibility in adulthood.
Environmental mismatch hypothesis	Why do animals undergo lasting phenotypic changes in response to stress exposure in early life? Are these changes context dependent? What are the benefits of changes in offspring phenotype for caregivers and offspring?	Changes from early stress exposure are permanent. Emphasis on (i) a match between prenatal conditions and early life conditions/the quality of maternal care, and (ii) the relative importance of maternal fitness vs. offspring fitness in determining the effects of early life stress.	Well suited for immediate effects of prenatal stress, especially in species with parental care or environments that are relatively stable within a single lifetime but not across multiple lifetimes (species with a fast pace-of-life strategy, limited dispersal, and limited plasticity/flexibility in adulthood). Reproductive strategy should also be considered (for example, lecithotrophic species may be more affected than matrotrophic species [178]).
Differential Susceptibility Theory	What makes children vulnerable or resilient to the effects of rearing conditions on later development?	Emphasizes that (i) the behaviors of a child should be interpreted through the lens of the child's environment and (ii) children that are sensitive to their environments have a high-risk but high-reward strategy, and can either capitalize on a good environment or be strongly affected by adverse conditions.	Well suited for humans and species with variability in the quality of parental care and the adult environment, where variable exploitation of the environment is possible, and, possibly to a lesser degree, monogamous species.
Biological Sensitivity to Context Theory	What makes children vulnerable or resilient to the effects of rearing conditions on later development?	Early stress exposure can reinforce a lasting trajectory towards increased sensitivity to environmental conditions.	Well suited for species with variability in the quality of parental care and the adult environment, where variable exploitation of the environment is possible, and species with rapid early development that leads to high levels of independence early in life and minimal changes in stress sensitivity over development.
Allostasis	How do animals maintain stability through unpredictable and predictable challenges?	Changes following stress exposure can be temporary (but cause a lasting increase in Allostatic load). Emphasizes that animals face unpredictable (e.g. social conflict, predation) and predictable challenges (e.g. migration, reproduction) Models aspects of the physiological stress response that are well conserved.	Well suited for species that have both preparatory and reactive responses to stress, ideal for understanding complex physiological states characterized by changes in several physiological processes (i.e. pathologies). Also well designed to model cumulative effects of stress (Allostatic load).
Reactive Scope	How do animals maintain stability through unpredictable and predictable challenges?	Proposed as an extension of Allostasis; changes from stress exposure can be temporary (but cause lasting wear and tear). Distinguishes between unpredictable and predictable challenges, chronic and acute stress, and emphasizes individual variability and changes across development.	Well suited for understanding an aspect of the stress response in isolation with respect to the predictability, chronicity, and severity of stress and age at exposure, which facilitates comparisons between species at the level of individual features of the stress response. Well suited for species with high levels of individual variability.
Arousal-shift hypothesis	How can early stress exposure enhance cognition and why are these effects context-specific?	Proposed as an application of the Environmental mismatch and PAR hypotheses to the Yerkes-Dodson law. Emphasizes the capacity for early life stress to induce changes in cognition that can enhance performance in a high-threat context but have minimal effect on performance in a low-threat context (applies only to tasks of moderate difficulty or greater).	May be best suited for (i) species with high cognitive demands, (ii) species with behavioral and cognitive stress responses that remain relatively stable after early development, and (iii) effects of stress exposure during developmental stages when brain regions supporting cognition are maturing.

just extreme, conditions) and (2) the manifestation of PARs is specific to developmental stage [93,106]. Gluckman et al. proposed that PARs prepare an organism to thrive in a specific range of conditions through its reproductive phase, and the functionality of PARs is contingent upon environmental predictability and consistency. PARs are suggested to lead to disadvantage or disease if environmental conditions exceed predicted ranges (in either direction).

Gluckman et al. [93] also addressed possible PARs in modern humans by expanding on the Thrifty Phenotype hypothesis. They explained that in the context of human evolutionary history, early and frequent reproduction would have been emphasized over health in later life stages, and suggested that some adverse effects of a thrifty phenotype have become evident only because of increases in human longevity (especially those detected after peak reproductive age, which some estimates suggest is around 30 for modern humans [207]). Gluckman et al. [93] also discussed maternal constraints on offspring development that are present even in resource-rich environments and may modulate offspring PARs in modern humans (e.g. maternal pelvis size, maternal age, and parity). It should also be noted that pregnant women frequently suffer from nutrient deprivation (not necessarily linked to caloric intake), even in modern first world countries (e.g. iron deficiency in the United States and Europe, [2,113,141]).

One possible limitation of the PAR hypothesis is that it considers maternal effects from the perspective of the offspring, but not from the

perspective of the mother, yet the ultimate purpose of maternal effects is to increase the fitness of the mother [219,256]. Parent-offspring conflict theory states that selection operates to create offspring that demand more resources than mothers are selected to provide [242,256,257]. Maternal effects should be considered through this lens, rather than solely through the perspective of the offspring, or consideration of only early life stages prior to offspring sexual maturation [152]. Yet, if maternal effects function solely in offspring/maternal conflict, it is unclear why there would species-specific maternal effects in species with no parental care (e.g. *G. aculeatus*, [90]) or maternal effects that manifest after dispersal [133]. These results suggest that maternal effects can benefit offspring outside of the direct maternal-offspring relationship. Regardless of the proportion of maternal effects that function primarily to modify either the maternal-offspring relationship or delayed effects that enhance the fitness of mature offspring, the goal of all reproducing females is to maximize personal fitness [219]. When interpreting the effects of early life conditions on offspring phenotype, a greater emphasis should be placed on possible benefits to the mother [144,219]. This is especially important for rats (a common model for maternal effects), given that rats have a fast pace-of-life strategy that prioritizes fast growth rates and frequent reproductive bouts, such that investment in individual offspring and the likelihood offspring will reach sexual maturity is low, but a mother's capacity for future reproductive bouts is high [37,202]. A fast pace-of-life strategy can bias females to maintain resources for future reproductive bouts [195]. Conflict between a mothers need to simultaneously provision for offspring and maintain resources for future reproductive bouts may complicate application of the PAR. Others have cited a second possible limitation of the PAR hypothesis: in free-living systems, it can be difficult to dissociate possible effects of PARs from extreme viability selection in early life [67], though similar problems may complicate application of most hypotheses of early life stress to free-living systems. Overall, the PAR hypothesis is an insightful expansion of earlier theory that is being actively advanced and broadly applied in ecological and biomedical contexts [94,106].

3.3. The environmental mismatch hypothesis

The Environmental mismatch hypothesis addresses questions similar to the PAR hypothesis; why animals undergo lasting phenotypic changes in response to stress exposure early in development, whether these changes are context dependent, and the benefits of changes in offspring phenotype for caregivers and offspring. To do this, the Environmental mismatch hypothesis emphasizes several key points: (i) maternal inclusive fitness, encompassing both direct fitness across all possible reproductive bouts and indirect fitness from non-offspring kin, (ii) the maternal-offspring relationship and the potential for early phenotypic changes to mitigate the demands of parental care, and (iii) the role of glucocorticoids as a possible mechanism by which mothers convey signals about environmental conditions to their offspring [142,144,217–219].

The Environmental mismatch hypothesis suggests that a match between maternal quality and offspring quality can increase fitness [142]. This suggests that females with low body condition or access to only adverse or resource-limited environments are less able to rear "high" quality offspring, and can increase fitness by producing "low" quality offspring (i.e. low birth weight, slow growth rate). Love & Williams [144] provide a strong supporting example of this: low quality offspring (European starling eggs injected with corticosterone) that are "matched" with low quality mothers (genetic mothers with wing and tail flight feathers removed) show decreased mortality compared with "mismatched" offspring that were not exposed to corticosterone but were reared by their genetic mothers with low maternal quality (feather removal). In addition to increased offspring mortality, mothers in the mismatched condition also had diminished future reproductive success compared with matched mothers [144]. The Environmental mismatch hypothesis has great explanatory power for mammalian and avian species (e.g. starlings, snowshoe hares, root voles, etc.), but these taxa share a proclivity for pair bonding and extended, obligatory parental care – application of the Environmental mismatch hypothesis can be mediated by mating or breeding strategy (income vs. stored capital breeding) and level of parental care [19,143,178,216,219,226]. Sheriff and Love [219] also caution that species are less likely to be responsive to maternal cues about the environment if they (i) disperse, (ii) occupy environments that are consistent or provide stable predictive cues, or (iii) have a slow pace-of-life strategy that makes them more likely to encounter varying environmental conditions.

Other factors to consider when applying the Environmental mismatch hypothesis are developmental changes in phenotypic plasticity (the ability to alter phenotypic traits in response to environmental conditions) and phenotypic flexibility (the ability to generate variable responses or states, that are often reversible, in response to environmental conditions) [196,199]. Highly plastic or flexible individuals would not need to gamble on phenotypic changes that may or may not provide advantages in future environments, because they could continually adjust to current conditions [144]. The potential for plasticity after maturation (e.g. resource requirements, duration, and magnitude) may modulate the applicability of the Environmental mismatch hypothesis for any given species. Similarly, Predictive Adaptive Responses (PARs) may also be obviated in highly plastic species that can readily adjust to changing conditions. Plasticity can be limited because it is costly, the specific costs and benefits of plasticity can vary across species and conditions [7] and extremely plastic species can risk mercuriality by responding prematurely or without adequate information [199]. The ability to be plastic after sexual maturation can be shaped by natural selection, through pressures like the frequency of environmental change on an evolutionary time scale and species-specific mortality and longevity [258]. For example, plasticity can allow an individual in high-resource conditions to return to an average size trajectory after a period of nutrient restriction that stunted growth (compensatory growth). However, limits to plasticity may compromise longevity or symmetry following compensatory growth (e.g. *zebra* finches, rats, Sandhill Cranes [20], reviewed in [7]). In this case, limited plasticity can allow individuals to adjust some aspects of their phenotype to new resource-rich conditions, while other aspects remain "optimized" for the previous, adverse conditions [170,171], which may complicate "snapshot" measures of phenotype. In addition to plasticity associated with ontogeny, it is also possible for species to have environmentally triggered plasticity throughout their lifetime. This can allow an individual to adjust to changing social or physical conditions, such as changes in group composition (e.g. socially induced sex-changes in fish, [13]) or seasonal or environmental changes (e.g. migratory behavior, such as rapid transitions from fresh to saline water in anadromous salmonids, eels, etc.; [6,244]). Thus, applications of the Environmental mismatch hypothesis may be mediated by the timing of windows of plasticity, as well as environmental consistency (temporally and spatially homogenous environmental conditions) and environmental predictability (seasonal changes, timing of food availability, etc.), across the average lifespan of a species ([67,133,223], Table 3).

The Environmental mismatch hypothesis and the PAR hypothesis require consideration of species life history, both for study design and interpretation [143,219]. A distinction between these two hypotheses is that the Environmental mismatch hypothesis suggests that changes induced by early life conditions occur immediately, while PARs can be delayed or have effects that vary across ontogeny, which may facilitate generalization across species that lack parental care or experience variable environments later in life. Overall, the Environmental mismatch hypothesis has great explanatory power for the immediate effects of early stress in species with specific life history traits ([143,219,220]; Table 3). The life history traits modeled in the Environmental mismatch hypothesis have variable overlap with species that can be shaped by early life stress [19,90,226]; future studies investigating the relationship between life history traits and lasting effects from early stress exposure

Table 3
Ideal phenotypes to respond to stress in adulthood according to current models of early life stress.

Hypothesis	Ideal phenotype for a stressful adult environment
Thrifty phenotype hypothesis	Individuals that are exposed to food restriction prenatally may be better prepared for subsequent food restriction, but likely compromise longevity and future health in order to cope with food restriction. Exposure to "mis-matched" food rich conditions after prenatal food restriction can cause additional, severe problems that detract from health, wellbeing, and reproductive capacity.
Predictive adaptive response hypothesis	If an individual is exposed to developmental stress, and is then exposed to an adverse environment later in life *that matches the range of the developmental environment*, then the adaptive responses caused by early stress should enhance fitness, compared to unstressed individuals. (Exposure to stress can prepare an individual for later stress).
Environmental mismatch hypothesis	If an individual is exposed to developmental stress, and is then exposed to an adverse environment later in life (independent of severity), then the phenotypic changes caused by early stress should increase the likelihood of reaching reproductive age and enhance maternal and personal fitness, compared to unstressed individuals. (Exposure to stress can prepare an individual for later stress).
Allostasis/reactive scope	The ideal phenotype to deal with stress is an individual with low wear and tear (minimal prior stress) or an individual with a wide gap between predictive homeostasis and homoeostatic overload (due to individual differences in resilience, seasonal changes, food availability, etc.).
Differential susceptibility theory	Individuals born with a less environmentally responsive phenotype (i.e. "alternative strategists") are most resistant to the negative effects of stress.
Biological sensitivity to context theory	Individuals that experienced moderate conditions (low-stress) in early life will be most resistant to the negative effects adverse conditions later.
Arousal shift hypothesis	Individuals previously exposed to stress should have increased cognitive/behavioral performance under subsequent stress compared to unstressed individuals, especially if the intensity/type of stress is similar.

could determine whether the mechanisms of early life stress effects depend upon life history and expand applications of the Environmental mismatch hypothesis.

3.4. The Differential Susceptibility Theory (DST)

The Differential Susceptibility Theory (DST) proposed by Belsky [16] addresses what makes some children vulnerable and others resilient to the effects of rearing conditions on later development. The DST suggests that individuals are either context sensitive ("conditional strategists") or resistant to environmental/social influences ("alternative strategists"). According the DST, parents cannot predict future conditions and so should produce offspring of both strategies in order to "hedge their bets" and maximize fitness. In this model, conditional strategists thrive in favorable conditions and resource-rich environments, but are also more affected by adverse conditions (including poor parenting) compared with alternative strategists, who are more insensitive to both positive and negative aspects of the environment. The DST suggests that conditional strategists adjust their phenotype in adverse conditions using strategies that once acted to maximize reproductive output, but, in modern society, are often categorized as mental illnesses or undesirable behaviors (e.g. gang membership, depression, insecure attachment, substance abuse, sexual promiscuity, limited parental investment, *sensu* [76]). The DST builds upon the established ideas that susceptibility to rearing influences varies across individuals [10,17], and children can exhibit contrasting externalizing or internalizing strategies [74,75]. The DST provides cogent explanations for variability between closely related individuals exposed to similar environmental conditions, how variability can be reinforced over time, and how phenotypes can be context specific [16].

According to the DST, children with conditional strategies that are exposed to adverse environments can exhibit behaviors that are often categorized as problematic by families, schools, and law enforcement, but serve to benefit the child – Belsky [16] describes an example in which an aggressive, disobedient child attends a crime-ridden school and is benefited "if he or she hit first and asks question later", while a less problematic, less aggressive child is often the victim of violence that threatens their wellbeing or survival. Belsky [16] suggests that the strategy of the aggressive child is potentially high-reward but also high-risk, because the advantages of externalizing behaviors, such as aggression, are countered by costs that vary across environmental conditions. Parallels could be drawn for children in other adverse conditions, including those with negligent or abusive caregivers. Understanding that context may promote problematic behaviors to enhance survival and minimize risk emphasizes that a child's behavior should be interpreted through the lens of the child's environment [16]. These ideas offer a strong framework from which to consider the treatment of behavioral problems in children that can and cannot be removed from adverse environments, and suggests parallels to free living animals that exhibit context-specific behaviors [9,16,31].

Applying the DST in an evolutionary or inter-species context could be facilitated by evidence connecting responsivity to parental care to fitness outcomes. To demonstrate that DST strategies can be shaped by evolution it is necessary to show that conditional and alternative strategies can provide a functional advantage, can result in differential reproductive success, and are heritable [95,138]. Current rates of environmental instability make it difficult to predict how phenotypic changes in response to early life conditions may improve function or affect fitness [51], particularly in species that are subject to viability selection and have long lifespans (or a slow pace-of-life strategy, [47,67,202]). Ellis et al. [76] posited that the production of offspring with dichotomous strategies evolved because it is advantageous for both parents and offspring, because siblings "share 50% of the same genetic alleles", so there are inclusive fitness benefits to having siblings of the opposite strategy. Siblings can increase fitness though shared genes, assuming parental care can increase offspring fitness, however, sibling relatedness can range from 100% for monozygotic twins to 0% for adopted or parasitic siblings, or species with cooperative breeding or alloparental care [69,116]. Alloparenting (parental care from non-genetic parents) is present in several mammalian, avian, and teleost species, including magpie-jays, meerkats, gerbils, prairie voles, sea lions, macaques, and humans [11,18,48,58,116,150,239]. An estimate of siblings sharing 50% of their genome also requires a monogamous mating system; extended monogamy is an uncommon strategy even amongst mammals [116]. Historically humans have been largely non-monogamous and are described as strategic pluralists or serial monogamists (varying by culture [81,85]). The average relatedness of siblings varies between species, depending upon mating and life history strategy (cuckoldry prevalence, group structure, communal vs solitary offspring rearing, etc.), and competitive behavior between offspring can increase as sibling relatedness decreases [29]. This suggests that the degree to which offspring accrue indirect fitness benefits from opposite strategy siblings may be species and context dependent, complicating the application of the DST to non-human species.

Before adaptive arguments can be made it may be necessary to determine whether dichotomous stress reactivity strategies are present in systems where fitness measurements are possible. Extensions of the DST could consider sex-specific responses, age-specific effects (including periods of hyporesponsivity or hyperresponsivity to stress), and the potential for offspring stress responsivity to reinforce parenting styles ([82,115]; but see [175]). In humans, variation in stress responsivity in early life offers great explanatory value for vulnerability and resilience of closely related children raised in the same household. The DST incorporates many disciplines, and though the integration of

disparate fields can be wrought with complications, the DST has much to offer in considering variation in responsivity to stress exhibited throughout development and variation between closely related siblings reared in similar environments.

3.5. The Biological Sensitivity to Context Theory (BSCT)

The Biological Sensitivity to Context Theory (BSCT) suggests that some children are highly reactive to their environment, which causes them to be strongly influenced by poor or favorable conditions, while other children are less sensitive to environmental conditions (similar to the DST conditional and alternative strategies, respectively [22,23,77]). Though the BSCT and DST address similar questions, a key difference between the two theories is that the BSCT suggests that exposure to extreme conditions (positive or negative) can increase responsivity to an environment (i.e. extreme conditions can make an individual more reactive/conditional) while the DST suggests that offspring use either conditional or alternative strategies from parturition. In light of this, Belsky [17] suggested that a combination of influences, both before birth and during rearing, can shape responsivity to environmental conditions. Supporting this, extensive evidence collected in multiple taxa shows that conditions during gestation and early life can affect reactivity of the hypothalamic-pituitary-adrenal (HPA) axis, which plays a key role in regulating the stress response (Box 2, [108,211,218,252]).

Yet, the assertion that exposure to extreme stimulation can increase stress responsivity is met with at least two challenges: (1) stimulation in early life can increase, decrease, or not affect stress reactivity (reviewed in [168,253]) and (2) the effects of stress exposure on subsequent stress reactivity can be dependent upon developmental stage, which suggests that the effects of stress exposure during development are more complex than reinforcing change towards a consistent, lifelong trait or trajectory such as heightened responsivity to environmental cues [146]. The first challenge represents evidence that exposure to prenatal stress often increases HPA reactivity, but depending upon the severity of stress and the age at exposure these effects can be negated or even reversed (reviewed in [91]). For example, in neonatal rats, combined maternal separation and handling by humans typically triggers an increase in corticosterone production and a decrease in body temperature, but reduces HPA reactivity and emotionality after weaning [123,136,166,167,185,186]. Similarly, Richardson et al. [275] showed that dams exposed to repeated restraint stress produced female offspring with prolonged HPA reactivity but male offspring with attenuated HPA reactivity. This same study showed that dams exposed to repeated variable stress (restraint, food shock, and saline injections) produced offspring with no significant change in HPA reactivity [275]. The second challenge, which emphasizes the importance of modeling developmental stage, can be illustrated by the following example: neonatal rats separated from their dam for 24 h on postnatal day 3 exhibit enhanced stress responsivity (plasma adrenocorticotrophic hormone (ACTH)) at postnatal day 20, while pups separated from their dam for 24 h on postnatal day 11 exhibit reduced ACTH responsivity to stress at postnatal day 20 [247]. The effects of exposure to stress during development can be dependent upon timing (associated with windows of plasticity), the intensity and duration of stress, sex, and species/strain (reviewed in [253]).

The BSCT suggests that exposure to stressful conditions increases stress reactivity to enhance fitness in a high-threat environment by increasing vigilance for threats. This is both supported and challenged by current empirical evidence. Supporting evidence shows that exposure to early life stress can increase action of the stress response system in the absence of stimuli (i.e. increased baseline levels of corticosterone and norepinephrine [234]). Changes in stress response systems in the absence of stimuli can indicate that fear has shifted from being a 'state' to a 'trait', thereby affecting cognition, behavior, and physiology in the presence and absence of threat [187]. However, contrasting evidence shows that exposure to stress, particularly in early life, can precipitate internalizing coping strategies, such as dissociation or tonic immobility, in humans and across taxonomic groups [79,109]. In humans, dissociation is the disruption of consciousness, identity, or perception [276]. Individuals are more likely to dissociate if they are forced to be immobile, experience physical pain, or feel powerless. Dissociation is especially common in children, likely due in part to the relative size and power differences between children and their abusers [147]. Dissociation is posited to allow children to bond with abusive caregivers and mentally escape trauma when physical escape is not possible [83]. Data on the prevalence of dissociation in response to trauma in childhood are scarce, but Kirby et al. [129] showed that 85% of adult female psychiatric patients abused before age 14 exhibited high levels of dissociation and Zlotnick et al. [271] found dissociation to be the primary coping method for females exposed to multiple sexual abusers as children. The relationship between dissociation and attention is complex and dependent upon developmental stage, but in children dissociation is negatively correlated with attention (in relation to cognitive inhibition) and dissociation may actually bias attention away from threats [14,49,62,84,124]. Further, children that dissociate when exposed to stress can actually lower their physiological hyperarousal over time [193,194]. Dissociation can become a long-term coping strategy; one of every four adults exposed to abuse in childhood reports three or more symptoms of dissociation that occur "often" or "always or almost always", and dissociative symptoms may be even more extreme in earlier developmental stages, closer to when the abuse occurs [39,179]. Given the prevalence of dissociative responses to early life stress, the relationship between stress reactivity and vigilance may be dependent upon life stage, at least in humans. In the context of the BSCT, the hypothesis that enhanced stress reactivity can increase fitness by increasing vigilance may be most applicable to adults or individuals that are capable of dispelling threats autonomously, rather than individuals in earlier life stages that are highly dependent on caregivers and less capable of independently resolving threats [147].

An extension of the BSCT has been proposed called the Adaptive Calibration Model, which addresses individual differences in stress responsivity and incorporates life history perspectives (within a single lifetime) in humans [63]. The Adaptive Calibration Model (ACM) suggests that individual differences in stress responsivity are caused primarily by conditional adaptation, defined as changes in phenotype in response to environmental conditions. As with the BSCT, the ACM suggests that stress during development can cause long-term changes in an individual's phenotype, "calibrating" an individual to match conditions experienced early in life. The ACM offers insightful exploration of the idea that the stress response system mediates behavior and emotion in order to shape an individual's life history strategy (modulating reproductive timing, offspring number, and parental investment). The ACM suggests that environmental conditions (including adverse conditions such as malnutrition) can cause an individual to adopt a "fast" life history strategy (early, frequent reproduction, low parental investment, shorter life span, i.e. 'grow fast die young') or a "slow" life history strategy (delayed, infrequent reproduction, high parental investment, longer life span, *sensu* [171]). More evidence is need to resolve whether human reproduction in adverse conditions is better described as (i) a purposeful shift to a slower life history strategy characterized by a decrease in offspring number and an increase in parental investment (i.e. an environment-matching effect) or (ii) a decrease in reproductive capacity caused by limited resources, independent of parental investment [246]. The relationship between stress and parental investment can be dependent upon the severity of stress, type of stress, availability of resources, sex of the offspring, and the relative importance of the current reproductive bout ([50,102], reviewed in [5]). For humans, the relationship between offspring number and parental investment is likely complicated by culture-dependent views on parental investment, socioeconomic status, access to contraceptives, and (as suggested by [63]) single vs. dual parental care. Separate from these issues, Boyce and Ellis [23] identified possible limitations of the BSCT derived from

key premises. One such premise is a unilateral relationship between chronic stress exposure and morbidity and pathology. Boyce and Ellis [23] clarified that stress exposure or high stress reactivity can have normative effects or improve health outcomes in some contexts. The question of how and when stress may have beneficial effects is addressed by the PAR and Environmental mismatch hypotheses [93,219].

One key strength of the DST and BSCT is that they emphasize the importance of understanding the broader environment of children exhibiting culturally rejected behaviors. These hypotheses present evidence that reversal of "problem" behaviors may be complicated by continued exposure to an adverse environment, such that interventions may be most successful if they are individualized for a child's overall environment (including family dynamics, school life, neighborhood, etc.). Elimination of externalizing behaviors may be practical only after facilitating a child's transition to a safe environment.

The BSCT has helped transform the way we think about stress responsivity across development, and has much to offer in advancing our understanding of stress with respect to context-specific phenotypes. By incorporating life history perspectives, the BSCT offers a framework for species-specific considerations, including changes in growth and developmental trajectories, trade-offs during resource limited developmental periods, sex-specific responses to stress, and changes in sensitivity to stress across ontogeny.

3.6. The theory of Allostasis

The theory of Allostasis addresses how animals can maintain stability following predictable or unpredictable challenges [164]. In the theory of Allostasis, organisms facing a challenge must balance energy intake and expenditure by maintaining shifting homeostatic set points using allostatic processes (e.g. regulation of catecholamine production, HPA axis), which regulate levels of allostatic mediators (e.g. catecholamines, glucocorticoids). Allostasis – defined as "achieving stability through change" – is similar to the earlier concept of homeostasis, but emphasizes the use of allostatic processes to maintain context-specific set points for biological systems. Allostatic processes can be hyperactive or hypoactive, resulting in allostatic mediators that are respectively atypically high (e.g. high corticosterone in by Cushing's disease) or atypically low (e.g. low corticosterone in Chronic Fatigue Syndrome). Sustained changes in allostatic processes can lead to a change in allostatic state, which refers to a combination of allostatic processes viewed at a phenotypic level. An allostatic state can be characterized by opposing changes in allostatic processes (e.g. Chronic Fatigue Syndrome is an extreme state marked by hypoactive corticosterone production and hyperactive inflammatory cytokines [191]). If an extreme allostatic state is maintained, allostatic overload can occur and cause pathology in two ways: (1) energy demands can exceed available energy or (2) chronic challenges can cause extended or continuous elevation of allostatic mediators, which can result in toxicity or adverse somatic effects (reviewed in [210]). Glucocorticoids are allostatic mediators that can cause the latter type of Allostatic overload - extended elevation of glucocorticoids (caused by persistent social conflict, high predator density, etc.) can decrease hippocampal volume and neurogenesis rate, cause neuronal or glial cell death, suppress immune responses, reduce bone minerals and muscle, and accelerate or alter aging processes [160,200,229,245].

One strength of the Allostasis model is that it incorporates the earlier "glucocorticoid cascade hypothesis", which describes the capacity of excess glucocorticoids to damage brain regions that function in negative feedback regulation of glucocorticoids, which can result in a feed-forward cycle of glucocorticoid production [209] – the Allostasis model terms this process "wear and tear" [161,162]. Using the concept of wear and tear, the Allostasis model explicitly defines when stress is likely to have pathological consequences and, using the concept of Allostatic state, describes pathologies characterized by opposing physiological processes [163]. Another advantage of the Allostasis model is that it highlights a broad range of glucocorticoid functions, including the mobilization of energy stores. Contextualizing glucocorticoids as mediators of energy use and availability facilitates comparisons across diverse species and contexts, and provides a clear link between humans and non-human species. The Allostasis model provides a strong bridge between ecological and biomedical scientists, and addresses discordance between the evolutionary context of the mammalian stress response and the context of the stress response for modern humans [164]. Further, the Allostasis model distinguishes between the effects of a predictable challenges (i.e. migration, reproduction) and unpredictable challenges (i.e. storms, human disturbance). For this reason, the theory of Allostasis has great explanatory power for addressing changes in emergency responses of free-living organisms over time, independent of changes in external stimuli or individual phenotype.

A potential limitation of the theory of Allostasis is its ability to address effects of early stress exposure on future stress sensitivity, including "preconditioning" effects which may have a preparatory function. In the Allostasis model, the lasting effects of stressful stimuli, after stimuli no longer pose a challenge, are described as wear and tear that accumulates throughout an individual's lifetime as Allostatic load (an individual's cumulative stress exposure). This appears to represent a unilateral link between early stress exposure and pathology, and suggests that stress in early life can impair, but not enhance, the capacity to deal with subsequent stress. Yet, early stress exposure can buffer the effects of subsequent stress (see Table 1), which is not yet described in the Allostasis model. Similarly, that the relationship between allostatic load and resilience is currently unclear; for example, Brody et al. [30] showed that allostatic load increases with socioeconomic risk in adolescents, but adolescents from high-risk backgrounds with high allostatic loads exhibit greater self-control and psychosocial competence than adolescents with lower allostatic loads from high-risk backgrounds. The Allostasis model seems to generate fewer clear predictions about effects of stress that (i) cause lasting changes in phenotype, including behavioral effects that can vary dramatically across species, (ii) vary across developmental stage, or (iii) reinforce trajectories, as described in the BCST, though subsets of these features are absent from most models of stress.

3.7. The Reactive Scope hypothesis

The Reactive Scope hypothesis was proposed by Romero et al. [205] to describe how animals deal with predictable and unpredictable stress and to address limitations of the Allostasis model. Briefly, Romero et al. [205] posited that the Allostasis model is limited because of (1) excessive reliance on energy budgets, which can be problematic because energy intake and expenditure is variable and not well understood (though this may be a weakness in our ability to assess Allostatic load without being a limitation in the predictive value of the model), (2) the relationship between glucocorticoids and energy mobilization is not as straightforward as was once thought, which can also complicate measures of energy use (also discussed in [56]), (3) heavy reliance on glucocorticoids over other features of the stress response, (4–5) it is unclear how to incorporate developmental effects and behavioral and cognitive stress responses into the Allostasis model, and (6) individual variation is not directly addressed (a possible weakness of the PAR and Environmental mismatch hypotheses also, but see [165]). Romero et al. [205] also identified three main strengths of the Allostasis model they sought to retain in the Reactive Scope model: (1) accounting for changes in physiological states over time, (2) a clear framework for defining when stress causes pathological damage, and (3) generalizability across species through measures of Allostatic load that are well conserved.

The Reactive Scope hypothesis suggests that individuals are typically in one of two states: (1) predictive homeostasis, facing predictable environmental changes such as circadian or seasonal changes, or (2) reactive homeostasis, facing a threat or unpredictable environmental

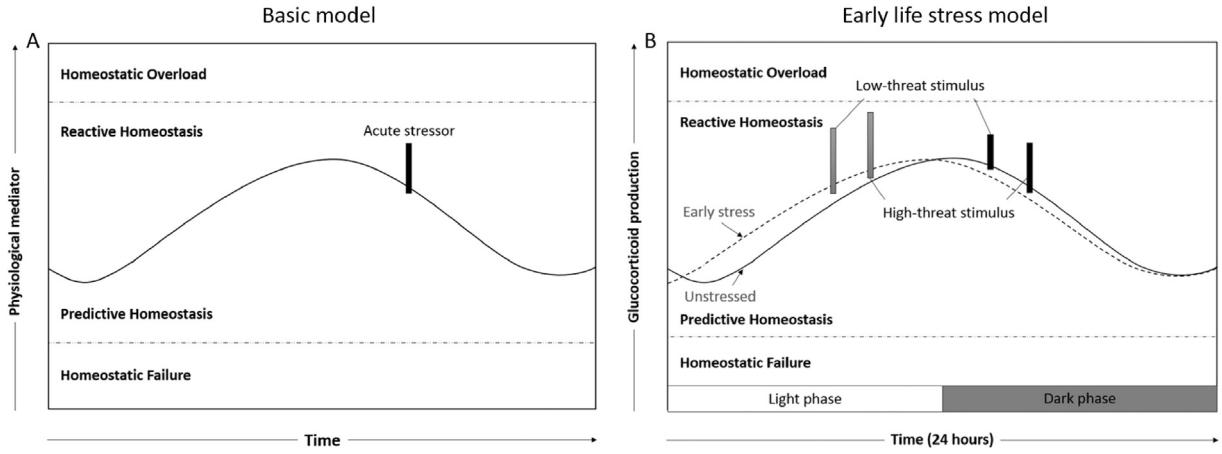

Fig. 1. Panel (A) depicts the basic Reactive Scope model adapted from Romero et al. [205]. According to Romero et al. [205], an individual is in the predictive homeostasis range when production of a physiological mediator (e.g. glucose, glucocorticoids, cytokines) is within a normal range (taking into account seasonal/circadian rhythms of mediator production, which are reflected by the curve of the solid line). The predictive homeostasis range is depicted between the curved solid black line and the bottom dashed line, while the region below the bottom dashed line is the homeostatic failure range. Individuals enter the homeostatic failure range if levels of a mediator necessary for homeostasis cannot be maintained (resulting in immune failure, water balance failure, etc.). When an unpredictable challenge disrupts homeostasis, an animal enters the reactive homeostasis range (above the curved solid line). After the reactive homeostasis range, challenges can cause an individual to enter the homeostatic overload range, above the upper dashed line. In the homeostatic overload range levels of a physiological mediator are so high that the mediator itself causes damage (resulting in obesity, immunosuppression, autoimmune disorders, pathologies, etc.). Panel (B) proposes a model for lasting effects of early exposure to stress (depicted for glucocorticoid production). Early (prenatal) stress can cause a phase shift in daily rhythms of corticosterone production in rats (depicted as a shift in the sine wave; [130]), but early stress may or may not affect basal HPA axis function (reviewed in [254]). The "acute stress bars" in (B) are longer for the early-stressed individuals (the gray bars on the dotted line) to reflect that exposure to early stress can increase responsivity to low-threat stimuli (reducing differentiation between high and low threat; reviewed in [76]). It should be noted that for the Reactive Scope model, each mediator requires its own model.

change (Fig. 1a). When an animal is challenged, levels of homeostatic mediators (e.g. glucocorticoids, heart rate) within a physiological system can exceed the range of reactive homeostasis, causing animals to cross the threshold into homeostatic overload. Excessive production of a mediator that pushes an animal into homeostatic overload can result in pathologies, including diabetes, depression, or autoimmune disorders. Conversely, if an animal is unable to maintain minimum levels of a homeostatic mediator, then the animal can enter homeostatic failure, which is typically fatal (e.g. water balance failure, immune failure). Over time, with increasing wear and tear, the threshold between reactive homeostasis and homeostatic overload can decrease, reducing the range in which an animal can respond to a challenge (i.e. decreasing Reactive Scope).

A strength of the Reactive Scope hypothesis is that it can readily model the effects of acute stress as a rapid transition from predictive to reactive homeostasis [205]. The model also makes clear predictions for the lasting effects of acute and chronic stress over time (wear and tear). Another strength of the Reactive Scope model is that individual differences can be modeled by adjusting Reactive Scope, the range between the upper "overload" and lower "failure" thresholds, or by adjusting the proximity of the predictive and reactive ranges to the overload and failure thresholds. Romero et al. [205] explain that modeling homeostatic mediators separately allows the Reactive Scope model to be readily modifiable across individuals and species, but modeling mediators separately may also be a limitation – while the Reactive Scope hypothesis models mediators individually, Allostatic states describe the relationship between multiple mediators, which can help define specific states or pathologies [224]. By modeling homeostatic mediators separately, the Reactive Scope model may gain greater accuracy for some homeostatic mediators but sacrifice a more integrative phenotypic view and risk an overly simplistic view of mediator function. Mediators may not move linearly, even within a physiological system. For example, the Reactive Scope model suggests that animals in the reactive homeostatic range exhibit increases in learning and memory, but decreases in learning and memory after homeostatic overload. However, aspects of learning and memory can relate independently to stress, for example, exposure to stress in early life can cause lasting increases in contextual fear learning but decreases in spatial learning, though both are hippocampally-dependent [46,188].

With respect to early life stress, in the Reactive Scope model the threshold between reactive homeostasis and homeostatic overload can be affected by early life experiences [205], but the model does not account for effects of early life conditions on actions of the homeostatic mediators. In the Reactive Scope model, early stress exposure can increase Reactive Scope and provide a "buffer" by increasing the gap between homeostatic overload and reactive homeostasis. This increases the amount of stress required to cross the overload threshold and induce pathology. However, it is unclear how the Reactive Scope model addresses the potential for early life conditions to shape regulation or baseline levels of a mediator, and the capacity for lasting changes in a mediator to influence suites of phenotypic traits in adulthood. For example, neonatal rat pups exposed to chronic stress can exhibit lasting increases in corticotrophin releasing factor (CRF) expression, which can effect the hippocampus and learning and memory processes in adulthood, but these effects can be avoided by blocking CRF after the early chronic stress treatment [118]. It is also unclear how the Reactive Scope hypothesis would characterize the effects of early stress on phenotype outside the context of resistance or vulnerability to pathology (particularly for behavioral and cognitive changes). A modified version of the Reactive Scope model, intended to address additional effects of early life stress, including effects on circadian rhythms, basal levels of mediators, and responsivity to subsequent stressors, is depicted in Fig. 1b. It should be noted that because all Reactive Scope models are specific to a single mediator, the proposed modification still cannot capture behavioral and cognitive effects of early stress such as those discussed in Table 1.

Another complication that can arise from modeling mediators individually is that the concept of homeostatic failure can be unclear; homeostatic failure occurs when a mediator level is too low to maintain life such that "death usually follows", which differentiates it from homeostatic overload when a mediator is overproduced, possibly resulting in pathology but not "immediate death" as with homeostatic failure [205]. Yet, complex states like lethargy and post-traumatic stress disorder are described as homeostatic failure by the Reactive Scope model [205]. This is a challenge because such states are often associated with both hypo- and hyper-production of stress mediators, for example, PTSD is characterized by chronic decreases in glucocorticoids, but chronic elevation of CRF and norepinephrine, which suggests that,

depending upon the mediator(s) being considered, individuals with PTSD simultaneously approach both the overload and failure thresholds [61,261,265]. In PTSD, these opposing relationships are so strong that pharmaceutical interventions for PTSD function to increase glucocorticoid levels or inhibit norepinephrine via blockade of the α_1 adrenergic receptor [235,236]. Opposing effects of stress can also be detected within the central nervous system; PTSD is often marked by hyperactivity of the amygdala but hypoactivity of the medial prefrontal cortex (reviewed in [222], effects may differ depending upon PTSD subtype, [55,174]). Although PTSD is characterized by hyperactive stress responses, it is not a disorder of hyper-vigilance – individuals with PTSD exhibit symptoms distinct from threat-detection, including intrusive thoughts or memories, self-destructive behavior, irritability, and detachment/dissociation. In the Reactive Scope model, it is unclear when animals should be categorized as approaching the overload or failure range. If homeostatic thresholds cannot be universally identified and applied to individuals or states, then comparisons between species and contexts may be difficult. Despite this, the Reactive Scope hypothesis incorporates diverse and important features of the stress response, environmental conditions, developmental changes, and species-specific considerations. The Reactive Scope hypothesis has advanced our understanding of ecological stress and has great promise as a framework for exchanging ideas across disparate lines of research.

3.8. The arousal-shift hypothesis

The arousal-shift hypothesis (proposed in [43] [authors include the current author]) attempts to explain the effects of early stress exposure on cognitive performance (Box 3) by marrying lines of thought from the PAR and Environmental mismatch hypotheses with earlier models of the effects of stress on performance described by Yerkes and Dodson [266]. This is an important challenge because there are effects of stress on cognitive performance that can persist after stressful stimuli are no longer present (see Table 1), but these effects are not addressed in many current stress models. The arousal-shift hypothesis does not contradict crucial prior hypotheses, it addresses the potential for developmental stress to affect the relationship between performance and arousal and applies current lines of thinking to the capacity for stress exposure to shape subsequent responses to stress. The arousal-shift hypothesis emphasizes that performance can be shaped by (i) context (threat level, resource availability, etc.), (ii) the difficulty of the task, and (ii) the relationship between testing environment and early environment [32,87,176,262].

Early stress can enhance subsequent cognitive performance in multiple taxa [36,43,44,80,203]. These effects span multiple aspects of cognition, including associative learning, object recall, decision making, associative and contextual fear memory, social learning, and reversal learning (Table 1). These effects may be context-specific, such that performance is enhanced following early stress for high arousal cues or contexts, but not for low arousal cues or contexts [e.g. 42–44,46,153, 203]. The arousal-shift hypothesis suggests that lasting cognitive and behavioral effects of exposure to early stress could be mediated by a shift in the curvilinear relationship between arousal and performance for tasks of moderate or extreme difficulty, such that early stress exposure facilitates subsequent performance at higher levels of arousal (Fig. 2). In the arousal-shift model, exposure to adverse conditions can have context-specific effects on performance, which should be minimal at low levels of arousal. It follows that differences in performance caused by rearing environment may only be detected near the optimal arousal level for unstressed animals, because the shift would cause early-stressed animals to reach their optimal arousal range (the peak of the Gaussian curve) at higher levels of arousal than unstressed animals. When arousal level is high, past the optimal range of arousal for unstressed animals, the proposed shift would allow animals exposed to early stress to maintain high performance. Under this framework, all animals would show a decline in performance after arousal exceeds their optimal range, but animals exposed to stress in adolescence would maintain a performance advantage over unstressed animals at every level of arousal that exceeds the optimal arousal range for unstressed animals (until arousal becomes too high to permit completion of a moderately challenging task regardless of rearing environment).

Supporting the arousal-shift hypothesis, adult rats exposed to chronic stress in adolescence can exhibit increased performance on a problem-solving foraging task in a high-threat context, compared to unstressed adult rats, but rearing condition has no effect on performance of the same task in a low-threat context [42]. Though application of the arousal-shift hypothesis to other species will require empirical investigation, a shift in the relationship between arousal and performance may account for the effects of childhood abuse on the performance of tasks related to face and object identification in humans (Table 1). Abused children are faster to identify emotional faces, but do not show these advantages when arousal levels are lower (with neutral faces [153]). Similarly, abused children show an increased ability to recall threatening objects compared with unabused children, but abuse has no effect on recall of neutral objects [203]. Shackman et al. [215] measured both performance and arousal, and demonstrated that abused children exhibit greater arousal (skin conductance response) when shown their mother's angry face and perform a task more accurately when their mother's angry face is the target compared with unabused children. This study examined vocal and auditory stimuli across congruent and incongruent conditions, and reported numerous stimulus- and context-specific behaviors. In the arousal-shift model, abused children should exhibit no difference in performance compared with unabused children on neutral or low arousal tasks of moderate or extreme difficulty, but abused children should exhibit increased performance on the same task in high-arousal contexts (until the level of arousal becomes too high to permit completion of the task).

Investigation of the arousal-shift hypothesis may inform our understanding of the role of plasticity during early development, but the hypothesis requires more empirical support and mechanistic grounding before it can be applied more broadly - it may be limited to species with high cognitive demands or behavioral and cognitive responses to stress that remain relatively stable after early development. The arousal-shift hypothesis may be best suited to explain the effects of stress exposure during developmental stages in which brain regions supporting cognition are maturating, especially given that much of the supporting evidence was obtained following stress exposure in adolescence or

Box 3
Yerkes-Dodson law

The Yerkes-Dodson law has been in use for over a century, it was derived from a series of experiments in which mice performed visual discrimination tasks paired with weak, moderate, or strong electrical stimulation [266]. Yerkes and Dodson [266] found a linear relationship between the strength of electrical stimulation (arousal) and acquisition of a simple discrimination task, but a curvilinear relationship between arousal and performance of a more difficult task. The Yerkes-Dodson law explains these findings: performance and arousal have a linear relationship for simple tasks, but performance of more challenging tasks can be enhanced by moderate arousal (partly by modulating motivation, [64,182,208]) and decreased by high arousal (partly by reducing the amount of information that can be processed, as described by the Easterbrook hypothesis [4,70]). Yerkes and Dodson's findings have been replicated with modern techniques and statistical analyses in numerous taxa, and the Yerkes-Dodson law has been widely applied to performance across contexts (athletic training: [230]; workplace conditions: [89]; video games: [121]).

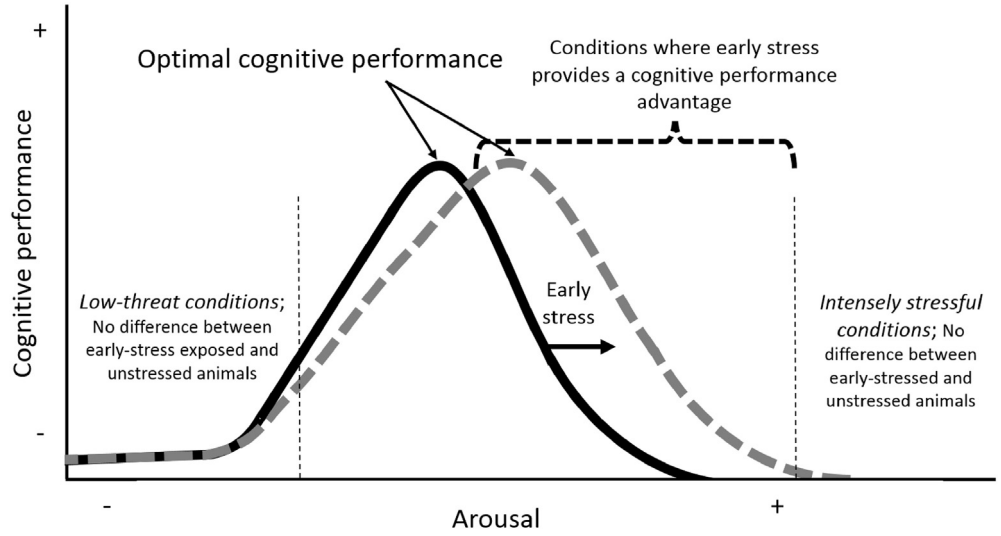

Fig. 2. The Gaussian functions represent a context-dependent relationship between cognition and early stress, adapted from [43]. The dashed grey line depicts a shift in this relationship caused by early stress exposure, which allows previously stressed individuals to perform at higher levels of arousal. These ideas are based on the earlier Yerkes-Dodson Law, which proposes a curvilinear relationship between arousal and cognitive performance for moderately and highly difficult tasks (Box 2).

childhood. Future investigations that map performance as arousal increases, similar to Yerkes and Dodson's original experiments, could reveal whether early stress exposure can cause a shift in the relationship between cognitive performance and arousal that mediates context-specific changes in performance.

4. How can we understand lasting phenotypic changes caused by early stress?

In this review, models of stress in early development are compared to (i) facilitate discourse between those using primarily biomedical or evolutionary/ecological models of early life stress exposure, (ii) explore applications of the current models of developmental stress based on their relative strengths and limitations, and (iii) address recent evidence suggesting that stress exposure in early development can have beneficial effects. The current hypotheses align on several ideas; for example, multiple hypotheses discuss the potential adaptive value of phenotypic changes resulting from exposure to early stress in an evolutionary context – understanding the adaptive value of phenotypic changes resulting from developmental stress exposure has the capacity to further our understanding of the stress response in ecological and modern human contexts. The concept that pathology or adverse life outcomes can be caused by discordance between the role of the stress response in ecological and modern human contexts is also addressed by several hypotheses (including the Thrifty phenotype, PAR, Allostasis, and Reactive Scope hypotheses). When applying these hypotheses, it is important to consider that the lasting effects of developmental stress are dependent on the age at exposure, even within a developmental stage [146], and current models are limited in their ability to account for age-specific responses. Further, application of the current hypotheses will likely depend upon the environmental stability a species, or perhaps even an individual, experiences in their lifetime (Table 2). For example, the Environmental mismatch hypothesis may be best suited for animals that live in relatively consistent, stable conditions (fast pace-of-life species, island populations, etc. [219]), whereas the PAR hypothesis may be better suited for conditions that vary or species that disperse to variable environments. However, the hypotheses presented here are not mutually exclusive. For example, both the PAR and Environmental mismatch hypotheses predict that individuals exposed to early life stress undergo phenotypic changes to prepare for adverse conditions later in life. Individual variation in the ability to undergo such changes may be described by the DST/BSCT.

It is important to consider that the models discussed here differ in their conceptualization of the "ideal" phenotype to minimize negative effects of stress exposure (Table 3). For example, in the Reactive Scope model, an individual with a maximized reactive scope (or the largest physiological range of homeostatic mediator production) is best suited to avoid negative effects of stress. In the Allostasis model, an ideal phenotype is one that can always rebound back to set points (and accrue minimal wear and tear). This is in contrast with the PAR and Environmental mismatch hypotheses, which suggest that ideal phenotypes for dealing with stress exposure are shaped during gestation/early life and have context-specific suitability. In these models, a phenotype will remain ideal only if the predictions made during gestation (or early life) are accurate estimates of ultimate environmental conditions.

In the future, models of stress should account for (i) developmental changes in stress sensitivity and responsivity [228,233], (ii) differences in plasticity and strategies for dealing with stress both across species and between individuals (Box 1), (iii) the duration of stress exposure (prolonged stress can drive negative 'states' to become 'traits' [187]), (iv) the type of stress (e.g. social, physical), and (v) sex differences. Currently, it is known that exposure to stress in early life can affect males and females differently [11,48,161,253], but it is unclear how sex-specific responses to stress in early life may vary with the type of stress, across species (and differences in sexual dimorphism and mating strategy), or across developmental stages (and the waxing and waning of sex steroid production). Additionally, exposure to stress can initiate both biologically specific and non-specific responses, it may be important to understand and investigate these separately [110]. Given that the ultimate goal of all individuals is to maximize personal fitness, a greater emphasis should be placed on the potential benefits of maternal effects for mothers [219]. Finally, it is difficult but necessary to empirically address the fitness impacts of phenotypic changes following early stress exposure [172] and dissociate the effects of viability selection [67]. Challenges remain in understanding the role of lasting phenotypic changes caused by early stress, but important strides have been made by the hypotheses discussed here, and interdisciplinary communication and collaboration can accelerate our progress.

Acknowledgements

The author is grateful to the Huck Institute of the Life Sciences, the Eberly College of Science, and the Pennsylvania Department of Health (Tobacco CURE Funds) for support. The author thanks Victoria

Braithwaite for her invaluable guidance and support, and Michael Sheriff, Benoit Dayrat, David Hughes, Sonia Cavigelli, and David Vandenbergh for their input and discussion. The Department specifically disclaims responsibility for any analyses, interpretations, or conclusions.

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