REVIEW

Prenatal maternal stress effects on the development of primate social behavior

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Abstract

Prenatal developmental plasticity in response to various environmental and social adversities can affect multiple aspects of offspring phenotype including social behavior strategies with effects that can last into adulthood. Here, we (1) identify adaptive social behavior strategies and their underlying mechanisms as potential targets of developmental plasticity in primates, (2) derive predictions about social behavior outcomes of prenatal adversity from different types of evolutionary models, (3) review the primate evidence for prenatal stress effects on offspring cognitive function, social, and non-social behavior, and (4) discuss avenues for future research. The scarce evidence currently available points towards increased distress behavior, particularly in infant offspring, and reductions of activity, exploration, and affiliative behavior in response to experimental prenatal adversity. Not all effects are stable, the results do not replicate well, and, for the most part, the current data cannot be used to test predictions of evolutionary models because relevant aspects of social behavior were not quantified and not assessed in the complex social environments they evolved for. More comprehensive research in developmental plasticity needs to incorporate sex differences and the interaction of effects from different sensitive periods including adolescence. Moreover, future research needs to assess the role of social buffering in mediating intergenerational effects and trade-offs between the pace of life and social cognitive performance.

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Introduction

Developmental plasticity is the phenomenon that genetically similar organisms can grow into vastly different phenotypes depending on environmental conditions during individual ontogeny (West-Eberhard 2003; Taborsky 2017). Ideally, organisms would always adjust their phenotype to current environmental conditions to maximize returns from interactions with the environment. Yet, plasticity is constrained by the costs of acquiring information and producing and maintaining plasticity, and is limited by information reliability, lag time, and physiological constraints (De Witt et al. 1998). As a result, ontogeny is a sequence of sensitive periods when information about the environment is gathered and phenotypical development changed accordingly, on the one hand, and periods of insensitivity when the current environment does not affect an individual's developmental trajectory, on the other (Hennessy et al. 2015; Trillmich et al. 2015).

During development, information gathering that influences genotype/environment interactions (i.e., the phenotype) may either be direct or indirect. Direct information is gathered by

the individual itself, while indirect information may be gathered by parents. Parents may sample the environment and provide their offspring with information about past, current, or predicted conditions via their own phenotype (Kuzawa and Quinn 2007; Wells 2010; Groothuis and Taborsky 2015). Information gathering starts in utero (von Borell et al. 2019, topical collection on the development of primate sociality). Thus, the ontogeny of overt social behavior (Amici et al. 2019, topical collection on the development of primate sociality) and communication (Briseno-Jaramillo et al. 2018; Fröhlich and Hobaiter 2018, both in topical collection on the development of primate sociality), social cognition (Cui et al. 2018; Rosati et al. 2018, both in topical collection on the development of primate sociality), and social learning (Whiten and van de Waal 2018, topical collection on the development of primate sociality) discussed in this topical collection on the development of primate sociality may be modulated not only by changing needs, postnatal maternal (Maestripieri 2018, topical collection on the development of primate sociality) and sibling behavior (Lonsdorf et al. 2018, topical collection on the development of primate sociality), brain development, sex, and the social niche but also by prenatal effects.

A prenatal effect as conceptualized here for placental mammals is an instant of developmental plasticity that originates in utero and generates an altered phenotype at later life stages. Prenatal stress effects are caused by adverse conditions experienced by the mother that she is unable or reluctant to buffer her offspring against. Adversities that cause prenatal effects include food restriction, predation, and social, heat, or cold stress (Brunton 2013; Tao and Dahl 2013; Berghänel et al. 2017; Piquer et al. 2017). The resulting phenotypic alterations in the offspring include offspring growth (Kapoor and Matthews 2005; Berghänel et al. 2017) and morphology (Dodson 1989; Spitze 1992). Prenatal stress effects have also been described for brain morphology and physiology and cognitive abilities including learning, motor development, metabolism, and stress physiology, as well as anxiety and depression-like behavior (Tauber et al. 2006; Weinstock 2008; Mastorci et al. 2009; Hanson and Gluckman 2014; Moisiadis and Matthews 2014a; Weinstock 2015; McGowan and Matthews 2018) all of which may relate to social behavior.

Many prenatal effects can be elicited by manipulating offspring exposure to maternal hormones (Groothuis and Maestripieri 2013; Hanson and Gluckman 2014; Moisiadis and Matthews 2014a). Maternal hormones including glucocorticoids are either used by the offspring as passive cues to maternal state and the environment experienced by the mother or as a signal from the mother to the offspring if hormone exposure of offspring is under maternal control and evolved for its effects on offspring (Wells 2003; Duthie and Reynolds 2013; Moisiadis and Matthews 2014b; Berghänel et al. 2017; Dettmer et al. 2018).

In the following, we will (1) describe briefly the general argument made in evolutionary models of prenatal stress effects, (2) review adaptive social behavior strategies and the underlying mechanisms as potential targets of developmental plasticity in non-human primates (hereafter primates), (3) derive predictions about social behavior outcomes of prenatal adversity from the different models, (4) review the primate evidence for prenatal stress effects on offspring cognitive skills, social, and non-social behavior together with descriptions of the experimental designs employed, and, finally, (5) suggest future theoretical work and empirical studies into social behavioral outcomes of early adversity and their underlying mechanisms.

Evolutionary models of prenatal stress effects

Here, we define a prenatal stress effect as developmental plasticity originating in utero in response to maternal stress and generating an altered phenotype at a later life stage (Nettle and Bateson 2015). Prenatal stress effects will be evolutionary adaptive if a responsive individual develops a postnatal phenotype with higher fitness than an unresponsive individual that experienced the same prenatal adversity. If the plastic response has only detrimental effects during later life stages, it is not adaptive *developmental plasticity*; this applies even if the response adaptively enhances *immediate* in utero survival (Monaghan 2008; Hanson and Gluckman 2014; Lea et al. 2017). Thus, theoretical accounts of the evolution of prenatal stress effects can be grouped into Developmental Constraints (DC) models and Predictive Adaptive Response (PAR) models based on whether the plastic response has been selected for its immediate survival benefits only with detrimental effects during later life, or for benefits accruing in later developmental stages (Gluckman et al. 2005; Barker 2007; Monaghan 2008; Hostinar and Gunnar 2013; Nettle and Bateson 2015; Lea et al. 2017), although some authors integrate both model types into one (Wells 2010; Berghänel et al. 2017).

Throughout the remainder of the paper we consider three theoretical models. Different authors use different terminology to refer to these ideas. **Developmental constraints** as used here largely correspond to "non-adaptive developmental plasticity" in Nettle and Bateson (2015), "immediate adaptive response" in Hanson and Gluckman (2014), and "silver spoon effects" (Grafen 1988). **External PARs** are the only PARs considered in most of the human literature including Hanson and Gluckman (2014), concern the later "thrifty phenotype hypothesis" (Monaghan 2008), and correspond to "information-based adaptive developmental plasticity" in Nettle and Bateson (2015). **Internal PARs** correspond to "somatic state-based adaptive developmental plasticity" (ADP) in Nettle and Bateson (2015) and have no equivalent in Hanson and Gluckman (2014) who interpreted them as immediate adaptive response.

Developmental constraints models conceptualize processes of non-adaptive developmental plasticity. Accordingly, plastic individuals react to prenatal adversity by reallocating resources to functions increasing immediate survival (Monaghan 2008). Such reallocation leads to reduced investment into growth and development. These constraints to development yield altered phenotypes in later life stages that are always at a disadvantage compared to unconstrained phenotypes later in life (Hanson and Gluckman 2014). As an example for prenatal adversity, we consider maternal food restriction. Without a plastic response, the offspring would continue along the canalized developmental path and invest energy into structural organogenesis during embryonic development and further tissue differentiation, functional organogenesis, and, mainly, growth during fetal development. Thus, under food restriction, the canalized development will lead to an energy deficit impacting not only development but also survival functions. If plastic, the offspring can allocate energy away from growth and development to improve current survival (Monaghan 2008). Although immediately adaptive, the constraint to development and growth will cause lasting detrimental effects on offspring phenotype.

External PAR models propose that offspring uses information on the current environment to predict external conditions later in life. This information is used to adjust offspring developmental trajectory to produce a phenotype that fares better under these future conditions than an unchanged phenotype, a phenomenon called environmental matching (Bateson et al. 2004; Bateson et al. 2014). Here, maternal food restriction will lead to direct prenatal nutrient deficits or indirect maternal hormonal signals that the offspring can use to predict low food availability during further development into adulthood. A plastic response by the offspring will adjust physiology and metabolism in ways enhancing survival into adulthood and reproductive performance in a nutrient-poor environment (Gluckman et al. 2005). The plastic offspring will outcompete non-plastic individuals in nutrient-poor environments but will be disadvantaged in a mismatched environment. Mismatches between early and later environments have occurred, for example, in humans exposed to drastic famines in the womb but not during their later development and adult life. This mismatch between prenatal and later environment has been used to explain the development of systemic diseases like coronary heart disease, diabetes, and obesity in offspring exposed to extreme nutritional restriction (Barker and Osmond 1986; Hales and Barker 2001; Bateson et al. 2004; Roseboom et al. 2006). Whether external PARs evolve will depend largely on the predictability of the environment over relevant time scales (Wells 2007; Nettle et al. 2013; Bateson et al. 2014; Del Giudice 2014b; Hendry 2015). Predictability of the social environment is potentially higher than of the ecological environment (Frankenhuis 2019).

Internal PARs are selected for because they prioritize early reproduction in anticipation of reduced lifespan (Nettle et al. 2013). The prediction does not concern aspects of the external environment, but the future internal somatic state of the offspring. In our example, prenatal maternal food restriction will lead to immediate developmental constraints and an unfavorable current somatic state (Nettle and Bateson 2015). As assumed by DC models, the somatic state shortly after exposure to early adversity can be rather stable and thus predictive of offspring somatic state at later life stages. Since most developmental constraints resulting from early adversity lead to shorter life spans, an adaptive developmental response is to adjust life history pace in order to reproduce earlier and at higher rates (Nettle et al. 2013; Del Giudice 2014a; Nettle and Bateson 2015).

All three model types have been supported by evidence from mammals including humans (see references above) but the relative importance of DC and PARs is still debated (Hanson and Gluckman 2014; Berghänel et al. 2017; Lea et al. 2017; Taborsky 2017; Ellis and Del Giudice 2019; Frankenhuis 2019). Therefore, we present predictions for social behavior outcomes from all three model types (DC, Internal and External PARs) here. It is, however, important to note that the three models are not mutually exclusive, and the proposed mechanisms theoretically may act in concert (Berghänel et al. 2017; Nettle and Bateson 2015).

Adaptive social behavior

Before we can derive predictions for social behavior outcomes of prenatal adversity, we need to review for gregarious primates, what aspects of their social behavior are associated with variation in fitness components. Aspects of social behavior for which the adaptive value has been established for a species should be the target outcomes to investigate in studies of prenatal stress effects on the development of social behavior. We distinguish here between agonistic behavior, affiliation, and the underlying social cognitive skills.

Aggression is a well-integrated part of primate social relationships (de Waal 1992), can vary independently from affiliative tendencies, and is not necessarily associated with reduced gregariousness or sociability. Many primates develop rather linear dominance hierarchies. When group members compete directly over resources within their groups, dominance rank predicts reproductive success and longevity (Bernstein 1976; Ostner et al. 2008; Rodriguez-Llanes et al. 2009; Alberts 2012; Majolo et al. 2012; Pusey 2012). Therefore, behavioral strategies promoting the attainment and maintenance of high dominance rank should be under strong selection and their disruption should be detrimental to fitness.

More recently, variation in **affiliative behavior** has been associated with fitness components as well—sometimes above and beyond the effect of dominance (Silk et al. 2003, 2009, 2010; Schülke et al. 2010; Archie et al. 2014; Kalbitzer et al. 2017; Thompson and Cords 2018). Across different studies, aspects of both the direct and indirect connectedness of an individual in the affiliation network correlate with survival, longevity, offspring production, and offspring survival (Ostner and Schülke 2018). Therefore, the ability to maintain affiliative relationships with many different partners, to connect to partners that are themselves well connected, to connect otherwise unconnected parts of the network, or to establish a few particularly strong, equitable, and stable relationships may all affect individual fitness (Brent et al. 2013; Gilby et al. 2013; Cheney et al. 2016; McFarland et al. 2017; Silk et al. 2018). Consequently, prenatal effects on how often and long an individual engages in friendly interactions, how it chooses, maintains and changes affiliation partners and how it distributes affiliative behavior across partners may have pronounced fitness effects.

Variation in social behavior strategies may result from differences in the underlying cognitive function and hormonal regulation of social behavior (Uno et al. 1994; Chang et al. 2013; Brent et al. 2014; Pearce et al. 2017; Crockford et al. 2018). Gregarious primates possess a series of **social cognitive skills** involved in maneuvering their societies: (1) they can individually identify others by various means, (2) they accumulate knowledge about others' interactions with themselves as well as among each other, and (3) they integrate this knowledge into patterns which allows them, for example, to recognize dominance and use transitive inference, and (4) they use simultaneous recognition along two or more dimensions (Seyfarth and Cheney 2015). These cognitive skills are played out in dominance interactions and conflict resolution where success does not only depend on physical power, but also on information about third-party dominance interactions (Paz-y-Mino-C et al. 2004; Deaner et al. 2005; Berghänel et al. 2011b; Schino and Sciarretta 2016). Skilled individuals integrate information across several interactions into patterns as evident from experiments violating such patterns (Bergman et al. 2003; Seyfarth and Cheney 2015).

Cognitive skills will benefit not only those that eventually use them to attain high social status but also subordinates that need to avoid escalated conflict. Where it is feasible and profitable, individuals may also use coalitionary support to change others' dominance status or the distribution of resources (Chapais 1995; van Schaik et al. 2006). Success in polyadic agonistic conflicts will depend on social knowledge from monitoring others' competitive abilities and supportive relationships, on the ability to communicate during coordination of a joint attack, and on the ability to establish and maintain reliable partnerships (Silk 1992; Noë and Sluijter 1995; Widdig et al. 2000; Perry et al. 2004; Berghänel et al. 2011a;

Ostner and Schülke 2014; Young et al. 2014c; Bissonnette et al. 2015). The coordination that is required for the maintenance of an affiliative relationship and especially for successful cooperation may be facilitated by choosing self-similar partners with regard to their general behavioral tendencies, i.e., aspects of their personality (Schuett et al. 2011; Watt and Ariyomo 2013; Koski and Burkart 2015; Ebenau et al. 2019). Societies structured by dominance relationships lend themselves to the study of social cognition, but it has been argued that a more egalitarian social structure puts an even larger demand on social cognition, because relationships may be more diverse and context specific (MacLean et al. 2008; Freeberg et al. 2012; Joly et al. 2017).

Behavioral phenotypes can be thought of as reaction norms describing how individuals differ in their reaction to variable environmental conditions including the social environment (Dingemanse et al. 2010). Inter-individual differences in both the level of a behavioral expression and the sensitivity to changing circumstances can be rather stable and constitute aspects of animal personality (Dingemanse et al. 2010) that may be the target of prenatal stress effects on social behavior (Sachser et al. 2013). A simple change in the expression of a behavior may increase aggressive tendency, for example, that leads to the individual being aggressive more often or at higher intensities which may be regulated by increased levels of circulating testosterone (Wingfield et al. 1990; Eisenegger et al. 2011). Changes in the sensitivity to changing circumstance will involve not only executive functions but also sensory functions, because such sensitivity requires information gathering from the environment and adjustments in motivation and reward circuits. Thus, the hormonal regulation of behavior via steroid hormones as well as the serotonergic, dopaminergic, and oxytocinergic systems may be targets of prenatal stress effects on social behavior (Groothuis and Maestripieri 2013).

Predicting social behavior outcomes from prenatal stress effects

We do not aim at deriving predictions that allow testing the different models against each other which ideally would involve full-factorial experimental designs (Uller et al. 2013; Taborsky 2017) or natural experiments that mimic these (Douhard et al. 2014; Lea et al. 2015). Our focus here is on social behavioral outcomes of prenatal stress effects and how they are predicted from the different theoretical accounts.

Developmental Constraints models propose that prenatal adversity triggers energy reallocation to immediate survival functions with potential impairment of all other functions including growth and muscle development and regulatory function concerning the fine-tuning of hormone systems, as well as development of cognitive function including social cognition.

We predict the challenged offspring to be smaller and weaker which may hamper the attainment of high **dominance rank** (Maynard Smith and Parker 1976; Bradbury and Vehrencamp 1998; Franz et al. 2015) and the associated benefits in juvenile and later life phases (see above). Smaller and weaker individuals will also be chosen as partners less often, because they have less to give in terms of current and future social support (Perry et al. 2004; Young et al. 2014b). When lacking social support from closely **affiliated partners**, developmentally constrained offspring will be more susceptible to environmental perturbations, because they are not well buffered against stressors (Young et al. 2014a; Wittig et al. 2016). Such a lack of social buffering may make offspring less bold and less explorative because they lack a safe harbor. Developmental constraints will also impair **social cognition** including social monitoring, memory, and decision-making hampering the formation of social bonds or tactical decisions during polyadic interactions (Connor 2007). Impaired cognitive performance may affect foraging efficiency (Janson 2007; Garber and Dolins 2014) exacerbating DC effects on body size and muscle mass. Smaller and weaker DC offspring is more prone to predation which can be balanced by increased vigilance. Reduced foraging efficiency may cause increased foraging time which together with increased vigilance may cause opportunity costs in terms of reduced time and energy for play behavior and **affiliative social interactions** with feedbacks on motor and social skill acquisition and social bonding (Dunbar 1992; Korstjens et al. 2006; Berghänel et al. 2015, 2016). Organizational effects on the immune response may lead to more infections promoting sickness behavior which is characterized by reduced activity and a general reduction of social contact (Dantzer 2004; Müller-Klein et al. 2018). In summary, we predict that DC from prenatal challenges will impair all aspects of offspring adaptive social behavior, i.e., agonistic behavior, affiliation, and underlying social cognition.

External PARS If prenatal adversities affect later-life behavioral phenotypes via External PARs, then predicting the direction of change and the aspects of behavior that should be affected require a comprehensive understanding of the environmental matching process; it is crucial to understand which aspect of the environment the offspring phenotype is matched to. In guinea pigs, for example, regularly occurring increases in population density (Sachser 1986) cause a shift in social organization from widely distributed pairs to small multimale-multifemale groups (Kaiser and Sachser 2009). Such shifts generate a different social environment that female offspring phenotype can be matched to by developing more reactive and aggressive behavior yielding increased reproductive success compared to unchanged offspring (Kaiser et al. 2015).

In contrast to relatively fast-living species like most rodents, effective environmental matching is hampered in most primates. Primates are less adaptable to short-term changes in

environmental conditions due to singleton birth and slow development. As a consequence, population density and the associated changes in the social environment will fluctuate less and need no matching. Environmental matching is less likely to evolve in slowly developing species, because the state of the environment the offspring will be reproducing in is more difficult to predict (Wells 2007; Bateson et al. 2014; Taborsky 2017; Frankenhuis 2019).

Environmental matching is also complicated by the fact that the physiological stress response of HPA axis activation largely is a one-size-fits-all solution to a wide range of environmental perturbations (Selye 1976; Sapolsky 2002; Romero 2004); in experiments with mammals, the same effects on offspring growth can be elicited by prenatal maternal cold stress, heat stress, noise, electric shock, open water maze, handling, strobe light, predator exposure, food restriction, social instability, social isolation, conspecific intruders, or treatment with glucocorticoids, dexamethasone, or ACTH (Berghänel et al. 2017).

If we pick food scarcity and predation pressure as two fundamental stressors and consider general arguments about the costs and benefits of sociality in gregarious primates (van Schaik 1983; Schülke and Ostner 2012; Markham and Gesquiere 2017), adaptive responses to these two stressors should yield rather different outcomes. Offspring developing in a food-impoverished environment will be faced with increased costs of sociality in terms of within-group scramble and contest feeding competition (van Schaik 1989; Schülke and Ostner 2012) promoting less gregarious and more aggressive/competitive behavior coupled with more exploration and risk taking (Miller 2002; Schmitt and Di Fiore 2015). If the source of maternal allostasis is increased predation risk though, the same prenatal maternal stress response of increased HPA activity should elicit in the offspring changes towards a less risk–prone, explorative, more socially tolerant, and gregarious yet less aggressive phenotype (Sheriff et al. 2017). It is possible though that the fetus reads several cues and signals at the same time and integrates this information. For example, high maternal glucocorticoid levels may need to be coupled with nutritional deficits to elicit a response adapted to maternal food restriction. Together, these considerations make it difficult to provide clear predictions for how prenatal adversity should affect offspring behavioral phenotype under the external PAR hypothesis.

One aspect of the environment the offspring will live in that the mother can predict with high reliability is maternal capital, that is her ability to provide for the offspring and to buffer the offspring from short-term environmental fluctuations (Wells 2010). Females may vary in their energy reserves, partly resulting from differences in their ability to accumulate reserves and to maintain homeostasis, and thus may be hit by the same food restrictions in different ways. If the mother has sufficient energy reserves, then maternal capital is to some degree independent from current fluctuations in

environmental capital, that is, resources and energy in the environment, and even from fluctuations in current maternal condition, i.e., whether the mother is currently losing or gaining reserves. In Wells' model, glucocorticoid signaling from mother to offspring concerns maternal investment but not external stressors (Wells 2010). We predict that under prenatally elevated GC levels indicating low maternal capital such early external PARs will produce thrifty offspring that grow slower and conserve energy by reduced motivation and constrained learning capacities as well as less activity, exploration, social play, and active **affiliation**. With increasing independence, the offspring will rely more and more on environmental capital for further development and may utilize this to catch up in development and particularly in size (Wells 2010). These processes may be accompanied by a behavioral shift in juveniles to increased foraging, exploration, and competitive **agonistic behavior** (Dammhahn et al. 2018). The prenatally challenged offspring will not be able to catch up all the way to the unchallenged phenotype though, because the smaller body has less metabolic capacity and catch-up processes will lead to increased metabolic load (Metcalfe and Monaghan 2001; Wells 2010).

Internal PARs might be selected for because they prioritize early reproduction in offspring with low life expectancy (Nettle et al. 2013; Belsky et al. 2015; Nettle and Bateson 2015; Berghänel et al. 2017). For immatures, we predict behavioral strategies and time budgets to shift towards increased energy intake and reduced energy expenditure to fuel accelerated growth and maturation (Dmitriew 2011). Behavioral strategies for accelerated reproduction in adults are more difficult to predict, may differ between the sexes, and can vary with socioecological factors.

The recalibration of infant phenotype will be reflected primarily in increased clinging to the mother and more mother-directed distress calls due to an increased mother-offspring conflict that results from a combination of increased infant needs and reduced maternal investment (Wells 2003; Berghänel et al. 2017). As juveniles, prenatally challenged offspring will spend more time foraging and in more risk-prone exploration (Dmitriew 2011) and will reduce their investment in energy-intensive social behavior like active social play (Dmitriew 2011) and allocate energy towards growth and maturation and away from cognitive development, skill acquisition, and immunocompetence (Coe and Lubach 2008; Palmer 2011; Veru et al. 2014; Berghänel et al. 2015, 2016).

Across several mammal species and a large number of studies, growth is accelerated only after early gestation maternal stress, but decreased after late-gestation stress (Berghänel et al. 2017). The findings suggest that early gestation is a sensitive period for the organizational effects involved in internal PARs and that adversities experienced late during gestation result in non-adaptive responses only (Berghänel et al. 2017). If this pattern is corroborated in further studies, we predict that internal PARs with behavioral outcomes are limited to early gestation stress.

A concept that relates to Internal PARs via its connection to life-history theory and that incorporates predictable inter-individual variation in behavior is the pace-of-life syndrome, POLS (Ricklefs and Wikelski 2002; Biro and Stamps 2008; Réale et al. 2010; Montiglio et al. 2018). The POLS has been broadly defined as "the suite of phenotypic characters associated with the life-history trade-off between current and future reproduction, where phenotypic characters may represent behavioral, morphological, physiological, or other characters adhering to this definition" (Dammhahn et al. 2018). Behavioral traits like boldness, aggressiveness, and activity that may form a behavioral syndrome of correlated characters enhancing resource acquisition at the cost of increased predation risk may be associated, on the one hand, with physiological traits like increased metabolic rate and, on the other hand, with accelerated growth and early maturation which also are a traded-off against a long reproductive life (Réale et al. 2010; Lemaître et al. 2015).

Recent conceptual, empirical, and modeling work has revealed that how exactly physiology is associated with the pace of life and which behaviors are functionally involved in the trade-off may vary between species (Royauté et al. 2018), ecological conditions (Montiglio et al. 2018), and the sexes (Hämäläinen et al. 2018). For example, finding resources, scrounging, handling, or monopolizing requires different behaviors that may all affect predation risk in different ways by changing predator avoidance, detection, escape, or defense (Montiglio et al. 2018). Which of the resource acquisition strategies are most effective in accelerating the pace of life will depend on the type and level of feeding competition experienced (Hoogenboom et al. 2012; Montiglio et al. 2018) which in turn depends on resource characteristics (Schülke and Ostner 2012). Therefore, the current theory on POLSs (Dammhahn et al. 2018; Montiglio et al. 2018) does not allow to derive very general predictions about the social behavior outcomes of an acceleration of life-history pace. The central theme, however, is a shift towards resource acquisition at the cost of reduced safety and maintenance.

Among the most robust findings on POLS are positive associations between **aggression** and growth or aggression and metabolic rate (Careau et al. 2010; Royauté et al. 2018). Increased aggression and risk taking have also been predicted in response to prenatal adversity from the adaptive calibration model, a type of internal PAR (Ellis and Del Giudice 2019). The personality trait aggressiveness is correlated with dominance rank in primates (Konečná et al. 2008, 2012) which could indicate that aggressiveness predicts productivity. Aggressive behavior is associated with dominance rank in complicated ways though (Gutleb et al. 2017), mostly serves to maintain instead of attain high dominance rank (Silk 2002) and rarely is directed towards dominants in more despotic societies (de Waal and Luttrell 1989). The link between

dominance rank and aggressiveness, therefore, may reflect constraints on expression of aggression rather than causality and requires more research, e.g., studies manipulating dominance rank (Snyder-Mackler et al. 2016).

Inter-individual differences in **affiliation** have not been investigated for their association with a POLS. Under the assumption that integration into the affiliation network and the formation of a few close affiliative bonds serve to buffer the individual against aggression from conspecifics and competition for resources while at the same time providing safety from predation and harsh environmental conditions (van Schaik and Aureli 2000; Massen et al. 2010; Ostner and Schülke 2018), affiliation may be traded-off against resource acquisition in a POLS and prenatally challenged offspring should be less affiliative and less well connected. Other trade-offs from an acceleration of the pace of life in reaction to prenatal maternal adversities will concern reduced investments into the development and maintenance of the endocrine, immune, and neural systems which may affect the regulation of behavior and **cognitive skills** in ways discussed above.

The predictions about social behavior outcomes of prenatal maternal stress effects differ only partially between the three types of evolutionary hypotheses about the mechanisms underlying such effects. From Internal PARs we predict an increase in risk taking, aggression, and, perhaps, exploration and activity that lasts into adulthood, whereas these effects should be negative under the DC. The maternal capital based external PAR hypothesis according to Wells (2010) also predicts these effects to be negative but to fade as the offspring ages and increasingly relies on environmental capital; only during possible phases of catch-up growth juvenile offspring may be temporarily more risk prone, explorative, active and aggressive. Additionally, only internal PARs predict prenatally stressed offspring to show reproductive behavior and associated social behavior like pair-bonding at younger ages than unchallenged offspring. Negative effects are predicted from all hypotheses for learning, memory, cognitive performance, social cognitive skills, and affiliative behavior including social play, general affiliative tendency, integration into the affiliation network, and social bond formation.

Primate evidence for prenatal stress effects on offspring behavior

We have searched the literature using google scholar and the keywords primate, prenatal stress, antenatal stress, glucocorticoid, dexamethasone, behavior, consequences, and long-term in different combinations, checked the abstracts of papers retrieved in the literature review for relevance to this article and, if deemed relevant, the reference list of these papers for further studies. We further used published reviews (Lupien et al. 2009; Maestripieri 2009; Pryce et al. 2011; Groothuis

and Maestripieri 2013; Maestripieri and Klimczuk 2013; Meyer and Hamel 2014) and references in a recent meta-analysis of prenatal stress effects on growth in mammals (Berghänel et al. 2017). The non-human primate evidence for prenatal stress effects on different aspects of the phenotype later in life is relatively scarce, especially for behavioral outcomes and if compared to infant developmental plasticity in response to early *postnatal* environments (Maestripieri 2018, topical collection on the development of primate sociality). Because of the scarcity of data and the lack of relevant details regarding statistical results in many publications, we refrained from using meta-analytical techniques. Instead, we interpreted the replicability of positive or negative effects of prenatal stress and grouped outcomes into (1) cognitive skills, (2) non-social behavior outcomes that may have indirect effects on social behavior, and (3) social behavior outcomes (Table 1). We further consider the type of stressor or treatment applied prenatally, the timing of treatment, and the offspring age at phenotyping. Since most studies were motivated from human psychopathology or pharmacology (effects of dexamethasone treatment around delivery), their use for testing evolutionary models of developmental plasticity is limited. We leave the interpretation of the evidence in relation to theory for the "Discussion and outlook" section.

Cognitive skills

Prenatal stress effects on offspring cognitive skills have been assessed in common marmosets (*Callithrix jacchus*) and rhesus macaques (*Macaca mulatta*). In common marmosets, general learning ability (trials to criterion) was not affected by in utero dexamethasone treatment, an artificial glucocorticoid mimicking increased HPA axis activity in response to a stressor (Pryce et al. 2011). These results were not affected by the timing of treatment either early or late (day 42–48 or 90–96 of 144) during gestation. Yet, performance in a skilled motor reaching task improved less over time in prenatally stressed juvenile marmosets compared to a control group with slight differences between juveniles stressed early versus late; late prenatal treatment caused failure to improve in skilled motor reaching over consecutive trials. In early treated juveniles, such detrimental effects were observed only when first grasp success was measured but not if first attempt success in obtaining the reward was quantified (Hauser et al. 2008).

The impairments described for common marmosets may be explained by the early effects of glucocorticoids on neurogenesis and cellular connectivity in the cerebellum which plays a key role in the fine modulation of precise motor control and performance but not so much in learning (Pryce et al. 2011); motor skills of prenatally challenged offspring are found to be corrupted in many studies (Schneider and Coe 1993; Schneider et al. 1999, 2004; Berghänel et al. 2016). Reversal learning is the ability to reverse a previously learned

Table 1 Prenatal maternal stress studies on non-human primates with three classes of outcome variables that concern (a) cognitive skills, (b) non-social behavior, and (c) social behavior. Stressors are either dexamethasone treatment (DEX), a noise startle treatment alone in a dark unknown cage (noise), a high-fat diet, a sham viral infection, naturally fluctuating maternal glucocorticoid levels (mat. GC), or social instability (soc. instability); offspring were phenotyped as infants, juveniles (after weaning), adolescents (neither fully grown nor sexually mature), or adults; the timing of the stressor was labeled as early or late during the gestation period by original authors; behavioral phenotypes were classified into aspects of activity or distress; effects are classified into non-significant versus significantly positive or negative

Species	Stressor	Age class	Age	Timing	Phenotype	Effect	Reference
Callithrix jacchus	DEX	Juvenile	1–12 weeks	Early	Learning	n.s.	Hauser et al. 2007
				Late		n.s.	
				Early	1st attempt success	n.s.	
				Late		n.s.	
				Early	1st grasp success	Negative	
				Late		n.s.	
	DEX	Adolescent	5–12 months	Early	Learning	n.s.	Hauser et al. 2008
				Late		n.s.	
				Early	Reversal learning	n.s.	
				Late		n.s.	
				Early	Motivation	Positive	
				Late		n.s.	
Macaca mulatta	Noise	Adolescent	32–34 months	Early	Learning	Negative	Roberts et al. 2004; Schneider et al. 2001
				Late		n.s.	
Callithrix jacchus	DEX	Juvenile	1–12 weeks	Early	Activity	Positive	Hauser et al. 2007
				Late		n.s.	
	DEX	Adolescent	5–12 months	Early	Activity	n.s.	Hauser et al. 2008
				Late		n.s.	
				Early	Distress	n.s.	
				Late		Positive (1 out of 4 behav.)	
Macaca mulatta	Noise	Infant	1–4 weeks	Early	Activity	Negative	Schneider et al. 1999
				Late		n.s.	
	Noise	Infant	6 months	Late	Activity	Negative	Schneider et al. 2004
					Distress	Positive	
	Noise	Infant	6 months	Late	Activity	Negative	Schneider 1992
					Distress	Positive	
	Noise	Juvenile	18 months	Late	Activity	n.s.	Clarke and Schneider 1993
					Distress	n.s.	
	Noise	Adult	48 months	Late	Activity (homecage)	n.s.	Clarke et al. 1996
					Distress (homecage)	n.s.	
					Activity (novel env.)	Negative	
					Distress (novel env.)	n.s.	
					Activity (new group)	Negative (1 of 4 behav.)	
					Distress (new group)	Positive (1 of 4 behav.)	
					Activity (2 months in new group)	Negative	
					Distress (2 months in new group)	n.s.	
Macaca mulatta	Noise	Infant	1–12 weeks	Average	Activity	n.s.	Bardi and Huffman 2005
					Distress	Negative	
	High-fat diet	Juvenile	4 months	Across	Activity	n.s.	Sullivan et al. 2010
	Viral infection	Infant	6 months	Early	Distress	n.s.	Bauman et al., 2014
				Late		Positive	
		Adolescent	22 months	Early	Distress	Positive	
				Late		Positive	

Table 1 (continued)

Species	Stressor	Age class	Age	Timing	Phenotype	Effect	Reference
Macaca fuscata	mat. GC	Infant	1–12 weeks	Average	Activity	n.s.	Bardi and Huffman 2005
					Distress	n.s.	
Saimiri boliviensis	soc. instability	Infant	2 weeks	Early	Activity	Negative	Schneider and Coe 1993
				Across		Negative	
Callithrix jacchus	DEX	Juvenile	1–12 weeks	Early	Affinitive	n.s.	Hauser et al. 2007
				Late		n.s.	
				Early	Social play	n.s.	
				Late		n.s.	
	DEX	Adolescent	5–12 months	Early	Affinitive	Negative	Hauser et al. 2008
				Late		n.s.	
				Early	Social play	Negative	
				Late		n.s.	
				Early	Grooming	n.s.	
				Late		n.s.	
Callithrix geoffroyi	mat. GC	Juvenile	5–10 months	Early	Social play	Negative	Mustoe et al. 2014
				Late		n.s.	
Macaca mulatta	Noise	Juvenile	18 months	Late	Affinitive	Negative	Clarke and Schneider 1993
					Contact	Negative	
					Clinging	Positive	
		Adult	48 months	Late	Social play (homecage)	Negative	Clarke and Schneider 1997
					Aggression (homecage)	n.s.	
					Submission (homecage)	n.s.	
				Late	Affinitive (new group)	Negative	Clarke et al. 1996
					Contact (new group)	Negative	
	mat. GC	Infant	1–12 weeks	Across	Social play	Negative	Bardi and Huffman 2005
	High-fat diet	Juvenile	4 months	Across	Aggression (intruder test)	Positive	Sullivan et al. 2010
	Viral infection	Juvenile	6–12 months	Early	Affinity to intruder	Positive	Bauman et al., 2014
				Late		None	
Macaca fuscata	mat. GC	Infant	1–12 weeks	Across	Social play	n.s.	Bardi and Huffman 2005

preference in a choice situation. Reversal learning skills were tested on the same common marmoset subjects after they reached adolescence and were not affected by prenatal dexamethasone treatment. Across experiments at juvenile and adolescent age, offspring stressed early during gestation exhibited improved motivation compared to late stressed and control individuals in terms of the number of trials and number of rewards obtained (Hauser et al. 2008).

These motivational differences were interpreted as potentially being driven by changes in the dopaminergic system (Hauser et al. 2008), a speculation supported by a later neuroimaging study on rhesus macaques demonstrating an increase in striatal dopamine receptor density in response to prenatal stress exposure (Roberts et al. 2004). Adolescent rhesus macaques whose mothers were exposed to startle noises in an isolation cage once a day for 8 weeks during early-mid gestation (day 45–90 of 166) were slow learners in a non-matching to sample task with food reward compared both to a control group and to offspring of mothers that experienced stress later (day 90–145) during gestation (Schneider et al. 2001). In an independent study from the same lab employing the same stressor and learning paradigm, the stress timing effect was not replicated; adolescent offspring from the treatment group learned slower than the control group, but here, treatment was applied late during gestation (day 90–145) which did not have an effect on learning in the first study (Roberts et al. 2004).

Thus, there is mixed evidence for an effect of prenatal adversity on juvenile and adolescent **cognitive abilities** assayed as general learning capacities, but indication for impaired precise motor control and for dopamine-mediated increases in motivation to retrieve rewards. Prenatal dexamethasone effects on hippocampal volume neurogenesis in juvenile rhesus macaques (Uno et al. 1994; Coe et al. 2002, 2003) and common marmosets (Tauber et al. 2006) further highlight the potential for prenatal adversity to affect brain structure and function with effects on behavior, though some of these effects did not last into adulthood (Tauber et al. 2008; Michaelis et al. 2009). Studies did not quantify effects on offspring executive function or associated prefrontal cortex brain regions. Social cognitive skills were not tested for their developmental plasticity in reaction to prenatal adversity.

Non-social behavior

The non-social behaviors quantified in prenatal stress studies on primates can be grouped into aspects of activity like mobility, locomotion, exploration, on the one hand, and aspects of distress like self-directed, distress, and stereotypic behaviors, on the other. The marmoset studies of Hauser and colleagues suggest that early but not late dexamethasone treatment yields more active, feeding, mobile, and solitary playing infant offspring (Hauser et al. 2007). None of these activity effects were stable into adolescence, and similarly, distress

behavior, specifically scratching, self-grooming, and tail hair piloerection were not affected by treatment in adolescent marmosets. The only treatment effect on non-social behavior was an increased rate of gnawing on woody parts of the enclosure in the late-treatment group compared to both control and early-treatment group which may be interpreted as increased exploration (Hauser et al. 2008).

During their first month of life, infant rhesus macaques born to mothers exposed to startle noise treatment differed in activity depending on the timing of the stressor; only offspring from the early-treatment group (day 45–90) were less active than controls (Schneider et al. 1999). In the late-treatment group (day 90–145) these effects were not observed during the first month of life (Schneider et al. 1999), but at 6 months of age, late-treatment offspring was less active than controls and exhibited stereotypies more often than controls (Schneider et al. 2004); an early treatment was not part of the study design with 6 months olds. In another sample tested also at 6 months of age, the exploration effect was reversed to a negative late-treatment effect, whereas distress behavior rates remained elevated compared to controls (Schneider 1992). When tested as juveniles (18 months) prenatally stressed offspring were not different from controls in non-social behavioral assays of activity (frequencies of active, inactive, exploratory, locomotion behaviors) and distress behavior (frequency of stereotypy, vocalization, self-directed behavior) (Clarke and Schneider 1993). Young adult rhesus monkeys (48 months) that had undergone late prenatal stress treatment and controls did not show differences in non-social behavior when tested alone in a cage during 3 weeks of separation from their peer group, but were more inactive in a novel environment (Clarke 1996). During the first weeks after formation of a new group, challenged offspring exhibited less active behavior (no effect on inactivity, locomotion, exploration) and more self-clasping (no effect on stereotypy, vocalization, self-groom). Two months after group formation, adolescents were tested in isolation where the treatment group was more inactive, less moving, and less exploratory, but did not differ from controls in the distress behaviors listed above (Clarke et al. 1996). Rhesus, but not Japanese macaque infants (*Macaca fuscata*), exhibit distress behaviors (self-scratching, crying, mother follow) less often the higher the natural prenatal glucocorticoid levels of their mother (Bardi and Huffman 2005); activity behaviors (exploration, locomotion) were not affected by prenatal exposure to glucocorticoids in either species. Juvenile rhesus macaques whose mother were fed a high-fat diet demonstrated an increased latency to touch a novel object but no change in overall activity (Sullivan et al. 2010). Infant offspring (10 mo) of rhesus macaque mothers challenged by sham viral infection showed more distress behavior, stereotypy, and self-directed behavior if treatment occurred late but not if it occurred early in gestation. These stressor timing effects were no longer evident at 22 months

of age when offspring of mothers that were exposed to a sham virus infection early or late during gestation exhibited more distress behavior than controls (Bauman et al. 2014). Squirrel monkeys (*Saimiri boliviensis*) that experienced prenatal social instability (changes in group composition and housing) and were tested at 2 weeks of age showed poor motor ability and impaired balance and were less active compared to controls (Schneider and Coe 1993).

Overall, the results yield an inconclusive picture of prenatal stress effects on non-social behavior. Across studies, **distress behavior** was reduced only once in prenatally challenged offspring, was not affected treatment in six cases, and was increased in seven cases. The observed increases in distress behavior of rhesus macaque and marmoset offspring were not necessarily stable into adolescence or adulthood. Prenatal treatment effects on **activity, mobility, and exploration** were negative in eight cases, positive in one, and absent in nine studies. Results for activity did not replicate well and were not stable across development when subjects were tested in their home cages. Yet, adult rhesus macaques were less active in three test conditions that caused increasing levels of disturbance.

Social behavior

Social behavior has been quantified as an outcome of early or late prenatal stress exposure, dexamethasone treatment, experimental viral infection, extreme changes in diet, or natural variation in maternal perinatal GC levels, in infant, juvenile, and adolescent rhesus macaques and marmosets. Common marmosets exposed to early or late dexamethasone treatment did not show altered durations of social contact with either parent or altered durations of social play with mother, father, or twin as infants (Hauser et al. 2007). The same individuals were tested again as juveniles; only individuals of the early-treatment group approached, followed, and left others less compared to the control group (Hauser et al. 2008). Similar reductions by early treatment were also observed in the initiation of social play and social play itself, although the latter did not reach significance (Hauser et al. 2008). Juvenile marmosets' social grooming was not affected by treatment and effects on aggressive behavior were not reported (Hauser et al. 2008). Further support for the detrimental effects of early prenatal adversity comes from a study on naturally occurring variation in prenatal maternal glucocorticoids in juvenile white-faced marmosets, *Callithrix geoffroyi* (Mustoe et al. 2014); increased prenatal maternal GC levels during the first but not during the last trimester of gestation were associated with decreasing rates of offspring social play (Mustoe et al. 2014).

Rhesus macaques were tested after their mothers were exposed to loud startle noise in a dark isolation cage once a day for several weeks during late gestation (day 90–145).

Offspring born to treatment mothers were phenotyped as young juveniles (18 months) in peer groups in their home cage and two stress conditions (Clarke and Schneider 1993); juveniles from the treatment group were found less often in very close proximity (15 cm) or partial body contact to others (excluding grooming and clinging). Instead, prenatally stressed juveniles were clinging to their peers more frequently when stressed but not in their home cage (Clarke and Schneider 1997). When tested again as adults (48 months) in a newly formed group, the rates of aggression and submission were not affected by treatment, but individuals from the late-treatment group played socially six times less than controls, were in very close proximity and in social contact less often than controls, and showed a larger increase in contact behaviors over the course of the first 3 weeks after group formation as stress faded (Clarke and Schneider 1997). When isolated, vocalization rates of prenatally stressed individuals decrease over time whereas it increased in controls (Clarke and Schneider 1997). When tested again 2 months after group formation, groups were moved to a novel environment for testing; prenatally stressed adults spent less time in very close proximity and partial contact to others; neither the treatment nor the control individuals played or groomed in this test condition (Clarke et al. 1996). This longitudinal study suggests that social proximity, contact, and play but not aggressive behaviors can be less prevalent in prenatally challenged individuals, particularly in stressful situations, and that these effects are stable from an early juvenile age through adolescence in rhesus macaques if challenged during late gestation; early treatment was not part of the experimental design of most studies.

Natural variation in prenatal maternal glucocorticoid levels was negatively associated with infant offspring rates of social play in rhesus, but not Japanese macaques (Bardi and Huffman 2005). Additional information on later aggression as an outcome of prenatal adversity comes from a study on the effects of a high-fat diet fed to rhesus macaque mothers which may be interpreted as a prenatal adversity because it is associated with perturbations in the serotonergic system; young juvenile males (13 months) of the treatment groups were more aggressive than controls in an intruder test (Sullivan et al. 2010).

When challenged by a sham viral infection once early during gestation (day 43ff), but not if challenged late (day 100ff), rhesus macaque mothers produced offspring that emitted social contact calls less in a modified version of a mouse three-chamber social approach assay with an unfamiliar conspecific and approached the other more often and stayed in proximity longer than controls (Bauman et al. 2014). This increase in social contact was interpreted as a social dysfunction because unchallenged individuals are very cautious around unfamiliar partners and typically avoid them (Bauman et al. 2014).

Apart from this three-chamber test, evidence points to a reduction of **affiliative and affinitive behavior** including

social play in prenatally challenged offspring that is rather stable from infancy to adolescence. One of the most prevalent and significant adult social behaviors, allo-grooming, was either not affected by prenatal adversity or (more often) remained untested due to its overall low prevalence in the test conditions, perhaps because these conditions were in themselves stressful. Despite its prominent role in regulating social relationships of gregarious primates, **aggressive behavior** was rarely measured which precludes conclusions about the directionality of prenatal stress effects. In marmosets (Hauser et al. 2007, 2008) and rhesus macaques (Bauman et al. 2014), effects were more pronounced if adversity hit the offspring early rather than late during gestation, but most studies did not include timing of stress exposure in their study design. None of the studies reviewed here assessed the onset of reproductive activity and associated behaviors, the number of affiliation partners, or how individuals distributed their affiliation across partners.

Evidence from studies on humans

Most of the studies on non-human primates reviewed above are experimental manipulations of prenatal maternal stressors or of HPA axis activation. It is currently not entirely clear though, whether exogenous steroid administration generates the same information for the offspring as the mother's evolved response to a social or environmental adversity (Maestripieri and Klimczuk 2013). Likewise, we are unaware of the information the offspring gathers if its mother encounters predators on a regular basis for example and whether this information is similar to the one generated when mothers experience a startle noise in a dark cage in isolation on a daily basis. The first field studies on non-human primates filling this gap are underway but to date have not measured social behavior outcomes in greater detail (Berghänel et al. 2016; Murray et al. 2018), or have focused exclusively on adversities occurring postnatally (Lea et al. 2015; Tung et al. 2016).

Therefore, we turn to studies on humans. One study population has been particularly influential, the cohort born after the Dutch Hunger Winter 1944/45 during the German occupation (Roseboom et al. 2011). The famine was restricted to the West of the country where it hit the entire population irrespective of socio-economic status leaving the East as a control. The famine was harsh and restricted to a 5-month period. Thus, possible effects were not confounded by malnutrition before or after the event as it is the case when comparing low-income and high-income countries or boroughs (Buffa et al. 2018). Offspring that experienced the famine in the womb during late but not early gestation had lower birth weights (Roseboom et al. 2011), and if female were more likely to become obese later in life (Ravelli et al. 1976), fueling the discussion about external PARs and mismatching adult

environments (Hanson and Gluckman 2014). More relevant to the topic of this review is work on psychosocial and behavioral effects of prenatal maternal stress. Early gestation exposure to the Dutch hunger winter increased the risk of schizophrenia, anti-social personality disorders, and affective psychosis for the offspring, and impaired selective attention (de Rooij et al. 2010; Roseboom et al. 2011).

Inspired by the Dutch Hunger Winter studies, prenatal stress effects have been assessed in other "natural experiments" like storms (Laplante et al. 2008), earthquakes (Li et al. 2015; Blanc et al. 2019), and floods (Simcock et al. 2016). Together with retrospective studies selecting human subjects based on records of adverse events like prenatal loss of close relatives (Huttunen and Niskanen 1978; Khashan et al. 2008), samples of mothers with varying degrees of anxiety disorders (O'Connor et al. 2002) or depression (Talge et al. 2007; Kinsella and Monk 2009), and prospective studies (Rijlaarsdam et al. 2016), the natural experiments provide reproducible evidence for a variety of offspring non-social and social behavior outcomes as well as altered social cognition. Prenatal maternal stress increases the incidence of schizophrenia, autism spectrum disorder (Walder et al. 2014; Rijlaarsdam et al. 2016; Varcin et al. 2017), major depression (King et al. 2012), and attention deficit hyperactive syndrome (Li et al. 2010) the symptoms of which include reduced emotional expression (i.e., social signaling), reduced social engagement, and problems with social engagement, creating and maintaining social relationships. Thus, at the extreme, prenatal maternal stress in humans leads to decreased **affinitive** and **affiliative behavior** with consequences for **social integration and bonding**.

Prenatal maternal stress in humans also increases conduct disorder, that is, breaking of rules often associated with increased **aggression** and general risk proneness (Glover 2011). Aggressive anti-social behavior as an outcome of prenatal adversity is more pronounced in boys than girls (van Hazebroek et al. 2018). Moreover, prenatally challenged children exhibit increased anxiety which can be interpreted as greater **vigilance** as well as aspects of increased attention deficit hyperactive syndrome that imply increased **activity and exploration** behavior (Glover 2011; Buffa et al. 2018).

It has been proposed that those systems that develop the fastest will be most sensitive to prenatal adversity which would make the prefrontal cortex a prime target and its function an important outcome of prenatal stress exposure (Neuenschwander and Oberlander 2017). One such function is executive function which is particularly sensitive to stress in adults (Arnstein 2009). The components of executive function, i.e., inhibition of predominant responses, mental set shifting, and maintaining and manipulating information in working memory are all impaired by prenatal stress (Neuenschwander and Oberlander 2017). Such detrimental effects will also impair **social cognitive skills** that require

top-down control. In support of this argument, prenatally stressed 30-month-old children show poorer theory of mind, that is, the ability to attribute mental states, beliefs, and desires to others (Simcock et al. 2017). In line with predictions of the internal PAR hypothesis, executive function in humans seems to be a traded-off against growth in the sense that accelerated weight gain is paid for with impaired executive function (Blair et al. 2019). Thus, results from human studies suggest that prenatal maternal stress can have profound consequences for offspring solitary and social behavior as well as social cognition with outcomes often depending on the timing of the stressor during pregnancy (Davis and Sandman 2010) offspring sex (Sutherland and Brunwasser 2018). The fitness conseqeunces of such effects remain unstudied.

Discussion and outlook

Above, we have laid out the main types of evolutionary models of prenatal maternal stress effects on offspring phenotype and discussed their predictions for the development of social behavior in primates. As far as any general predictions can be made, all models predict impaired social cognitive function and constrained affiliative behavior leading to negative effects on social integration and social bonding. The current evidence from non-human primates reviewed above is scarce, but the one significant effect of prenatal adversity on offspring **cognitive skills** was a negative effect on learning abilities. Social cognitive skills have not been assessed. If they differed from controls, challenged offspring exhibited more distress and sometimes less but never more affinitive, **affiliative**, and play behavior. Often, these effects were stronger after early- compared to late-gestation stress, which matches with previous findings on patterns of accelerated growth indicative of an Internal PAR mechanism (Berghänel et al. 2017), but clearly more studies are needed for a conclusive treatment. Via its conceptual overlap with the POLS framework, only the Internal PAR hypothesis predicts increased risk taking, exploration, activity, and aggression throughout life. The other hypotheses predict the opposite or make ambiguous predictions. In prenatally challenged macaques, **aggression** was increased in one but not another case. The only study assessing both aggression and activity found the behaviors not to be correlated (Sullivan et al. 2010); aggression was increased but activity was unaffected by prenatal stress treatment. Evidence from humans also points towards prenatally stressed offspring exhibiting increased activity and exploration, increased aggression, decreased affiliation, and impaired social cognitive skills, which best matches predictions from the internal PAR hypothesis.

The weak patterns revealed across primate studies largely reflect those on laboratory rodents where prenatal maternal stress exposure yields offspring that is less socially motivated and less socially active (Sandi and Haller 2015); challenged rodent offspring clearly are less aggressive than controls though. Like primates, prenatally challenged rats suffer from learning impairments resulting from hippocampal dysfunction, enhanced sensitivity to drugs of abuse as a response to altered dopaminergic function, and increased anxiety- and depression-like behaviors which, however, are typically not assessed in a social context (Lupien et al. 2009). These rodent studies as well as most of the work on primates reported above were motivated from clinical and biomedical research on psychosocial disorders in humans where one goal is to produce a model for anxiety and depression that can then be studied further. Therefore, the environmental perturbations mothers were exposed to may not correspond to experiences animals have in their natural environment. The paradigms used to assess behavioral outcomes are often highly standardized (Schneider and Suomi 1992; Sandi and Haller 2015) but do not correspond to natural social environments of the species (Groothuis and Maestripieri 2013; Maestripieri and Klimczuk 2013) and often cannot be used to test evolutionary hypotheses for prenatal maternal stress effects (Taborsky 2016, 2017).

Crucial aspects of the development of social behavior have not been investigated yet as responses to prenatal adversity in non-human primates. Adaptations in the offspring to the opportunities and challenges in its social environment concern social cognition (Seyfarth and Cheney 2015), dominance rank acquisition, and behavior in dyadic and polyadic contest (Chapais 1995), as well as integration into the affiliation network and social bond formation (Ostner and Schülke 2018). Whether prenatally challenged offspring affiliates less with all social partners or concentrates its reduced affiliation time on a few important partners as it is known from sickness behavior (Willette et al. 2007; Hennessy et al. 2014) or other ways in which reduced affiliation time affects relevant aspects of social network position remains unexplored.

Notably, affiliation always needs a partner. Levels and patterns of affiliation will not only be affected by the social motivation and social activity level of the prenatally challenged offspring but also by the actions of others. If prenatal stress affects quality-related aspects of offspring phenotype like body size, built, muscle mass, agility, or skill, then its value as a social partner for others will change as well. Coalition partners are often chosen for their dominance rank which partly reflects physical aspects of fighting ability (Noë and Sluijter 1995; Widdig et al. 2000; Perry et al. 2004; Schino et al. 2006; Young et al. 2014b). Others will also seek affiliation with more knowledgeable and resourceful individuals (Fruteau et al. 2009) instead of poor-quality offspring of previously stressed mothers.

Analyses that go beyond the individual level overall behavioral tendency and tap into the dyadic aspects of social behavior phenotypes will reveal whether the social buffering effect (Kiyokawa and Hennessy 2018) may produce

intergenerational effects. Close social bonds among adults have evolved for their function in the contest over dominance rank and access to resources and safety where close partners tolerate and support each other (Massen et al. 2010; Seyfarth and Cheney 2012; Ostner and Schülke 2014). As a consequence, closely bonded individuals are buffered functionally against different social and environmental stressors and show an attenuated physiological stress response (Young et al. 2014a; Wittig et al. 2016; Kiyokawa and Hennessy 2018). The social buffering effect is also prevalent during pregnancy in humans (Skoluda and Nater 2013). If prenatally challenged offspring affiliates less as an adult, it may not form close bonds and react to stressors in the environment with stronger or longer activation of the HPA axis which will trigger adaptive responses in its unborn offspring and carry over across multiple generations. In other words, intergenerational effects emerge via both the lack of social maternal capital and somatic maternal capital, potentially leading to phenotypic segregation within and between populations (Wells 2010) and may help to adapt fast to changing socioecological conditions like increasing within-group contest competition. Thus, behavior may mediate intergenerational organizational effects without causing direct prenatal stress effects on HPA axis activity, sensitivity, or (dys-)regulation itself (McGowan and Matthews 2018). Instead, the same stressor will elicit in the adult prenatally challenged offspring a physiological stress response possibly within the normal reactive scope (Romero et al. 2009) whereas other adults will be buffered by functional support from close partners (i.e., a behavioral mechanism) so that the HPA axis will not be activated and the next generation remains unaffected by the maternal stressor exposure.

Alternatively, social behavior may mediate the effects of prenatal adversity on offspring HPA function much in the same way as dominance rank is proposed to affect physiology (Snyder-Mackler et al. 2016, 2019; Lea et al. 2018); the possible mediating role of sociality was not assessed in these studies though. The fact that different direct and indirect effects may act and may even do so in concert explains why prenatal stress does not affect offspring HPA activity in simple and general ways in primates (Maestripieri 2018, topical collection on the development of primate sociality; Murray et al. 2018).

Similar to the social buffering effects described above, prenatal stress effects may be mediated via the benefits of social integration or weak bonds. If prenatally challenged offspring cannot establish relevant network positions it will forgo benefits in terms of enhanced thermoregulation (McFarland et al. 2015), safety from predation (Ostner and Schülke 2018), or access to information (McFarland et al. 2017). The lack of these benefits will increase reactive homeostasis (Romero et al. 2009) in the adult offspring which then signals to the next generation. The effect is not mediated by behavior in the same way as

the social buffering effect, but the behavior of the prenatally challenged, less social offspring may affect fitness via forgone benefits of general sociality.

Phenotypic shifts in response to prenatal adversity may not only concern affiliation but also aggressive behavior. Increased aggressiveness may evolve as a reaction to prenatal adversity as an internal PAR if this recalibration of phenotype provides the offspring with a benefit over the unchanged phenotype that has suffered the same prenatal adversity. Since such prenatal adversity is thought to yield disadvantaged somatic states, individuals can be expected to have reduced physical power and lower success in conflicts. These disadvantages can be partly compensated by increased aggressiveness or motivation expressed during contests (Hardy and Briffa 2013). Contest behavior will be further affected by inequalities among competitors ensuing from differences in life expectancy and thus in the value of the contested resource that is higher in prenatally challenged offspring making them escalate more often (Hardy and Briffa 2013).

It will be particularly rewarding to test whether prenatal adversity affects offspring social cognition in primates (Davis et al. 2017). Negative effects are expected for how individuals focus their social attention when monitoring others, for social memory and learning, and for the integration of social information into predictions or expectations about others' behavior. Social monitoring can be assessed in gaze-tracking studies presenting photos of close partners versus other individuals, group members versus strangers, higher versus lower ranking individuals, and positive versus negative stimuli (Shepherd et al. 2006; Almeling et al. 2016; Schino and Sciarretta 2016; Rosati et al. 2018). Cognitive integration of social information can be tested for example with playbacks of staged fights and affiliations violating expectations (Cheney et al. 1995; Bergman et al. 2003). Prenatal adversity may also affect trust since trust is associated with risk-aversion in the sense that trust requires delayed gratification (Petersen and Aarøe 2015). It also remains unexplored whether variation in social cognitive skills translates into variation in affiliative and agonistic social relationships and ultimately into differential fitness in gregarious primates (Huebner et al. 2018).

Prenatal maternal effects may also affect dispersal. Depending on the social system, the dispersing sex may have the option to breed in the natal group albeit with fewer reproductive options due to inbreeding avoidance (Muniz et al. 2006). If current reproduction is strongly valued over future reproduction as predicted from internal PARs, challenged offspring may be selected to avoid the risks (Alberts and Altmann 1995a; Isbell and van Vuren 1996) and the delay in reproduction that is associated with dispersal and integration into a new group and start to reproduce in the natal group. If the costs of dispersal are associated with individual quality (Alberts and Altmann 1995b) then prenatally challenged offspring is also predicted to adjust dispersal decisions

accordingly (Perry et al. 2017). Alternative routes of natal dispersal have been described where primate males either migrate at a rather young age and well before being fully grown, or after reaching full body size and fighting power (van Noordwijk and van Schaik 2001). If prenatally challenged offspring never reaches the body size and strength of unchallenged competitors, they may choose to disperse as juveniles or small subadults to avoid aggression upon immigration.

As mentioned at the very beginning of the introduction, gestation is only one sensitive period in the development of primate offspring. It is followed by the early postnatal phase where offspring samples the maternal and external environment for information that the phenotype can be adapted to via organizational effects. The role of maternal behavior during postnatal development has been summarized in other contributions to the special issue (Lonsdorf et al. 2018; Maestripieri 2018, both in topical collection on the development of primate sociality). One empirical study that specifically related early adversity to the evolutionary models discussed here demonstrates that in a population of wild yellow baboons (*Papio cynocephalus*), six early postnatal adversities, in the form of extreme food shortages, high local population density, low maternal dominance rank, weak maternal social integration, maternal loss, and untimely birth of a sibling, had cumulative effects on adult female survival and lifetime reproductive success (Lea et al. 2015; Tung et al. 2016). The results match predictions of the DC hypothesis, but not those of the external PAR hypothesis; adults born into very harsh environments did not outperform others later in life in matching harsh environments, but were always at a disadvantage (Lea et al. 2015). Predictions of the internal PAR hypothesis were not tested. Early adversity also had detrimental effects on later social integration (Tung et al. 2016), as we have predicted for prenatally challenged offspring. In this latter analysis on the same population, cumulative adversity was measured throughout the entire early life from conception through maturation, i.e., implicitly combining prenatal and postnatal effects.

Postnatal organizational effects on primate behavior may be caused by variation in the quantity, quality, and composition of mother's milk as shown in rhesus macaques (Hinde and Capitanio 2010). Milk energy provided early after birth may act as a cue for the offspring to the condition of the mother or the environment at large that offspring behavioral phenotype is adjusted to. The higher the milk energy provided early during lactation, the more active and confident were the offspring later after controlling for several potential mediating effects. Apart from milk energy, its cortisol content is associated with later offspring social play behavior in daughters, but not with general social behavior of offspring in a naturalistic context, i.e., in their social group (Dettmer et al. 2018). When stressed by separation from the mother, offspring were more nervous and less confident the higher milk cortisol levels had been before (Hinde et al. 2015).

Another study on adaptive postnatal developmental plasticity assessed in captive rhesus macaques a gene by environment interaction for variants in a serotonin transporter gene-linked region. A supportive early postnatal environment allowed the carriers of the rare allele to develop higher social competence expressed in more social play than carriers of the frequent allele that had experienced the same early environment (Madrid et al. 2018). Accordingly, carriers of one or the other variant will differ in their adaptive developmental plasticity. Together, these studies demonstrate that early postnatal life is also a sensitive period for organizational effects on later behavioral phenotypes in primates that requires further investigation in relation to evolutionary models.

Although it is generally difficult to predict the timing of sensitive periods solely on theoretical grounds, it has been argued that adolescence also is one such developmental phase often associated with profound organizational effects (Piekarski et al. 2017; Sachser et al. 2018). Developmental plasticity during adolescence allows recalibration of the phenotype to the conditions at the onset of own reproduction (Sachser et al. 2018). Earlier developmental effects may be buffered, fully compensated, masked, reversed or exacerbated during puberty (Taborsky 2017). Such recalibration may be most effective in fast-reproducing litter-bearing species where inter-birth intervals and litter sizes can be manipulated to generate vastly different numbers of offspring in response to conditions in adolescence (Belsky et al. 2015). Yet, in more slowly developing species, more time will have passed between early sensitive periods and environmental conditions may have changed enough to warrant another adjustment of phenotype as seen in humans (Belsky et al. 2015; Ellis and Del Giudice 2019).

Prenatal stress effects on offspring development will differ from developmental plasticity during adolescence in the types and sources of information available to the offspring. Early in life, all information is gathered indirectly mainly from maternal glucocorticoid and nutrient levels which are both under maternal control, may not be true reflections of environmental conditions, and may be integrations across several types of adversities (Wells 2010; Sachser et al. 2018). During adolescence, the offspring can sample the environment directly and may develop differently in reaction to very specific aspects of the environment that are associated with specific stress signatures (Sapolsky 2002; Wells 2019). With the onset of reproduction, the challenges and opportunities of gregariousness change as do certain life-history trade-offs. Immatures need to ensure survival until reproduction, but mature individuals suffering from long-term effects of early adversities may jeopardize survival and maximize investment in current offspring even at pronounced costs to survival. Reproduction is often delayed in one sex after maturation yielding sex differences in the role of adolescence as a time to predictively adapt phenotype (Zimmermann et al. 2017; Sachser et al. 2018). Such sex

differences in life history will also affect how behavioral phenotypes are shaped earlier in ontogeny (Hämäläinen et al. 2018), which is a topic that is yet underexplored in primates.

In conclusion, there is little empirical work on evolutionary models of prenatal developmental plasticity of behavioral phenotypes in primates and most work was motivated by human clinical research on psychosocial disorders. Therefore, crucial aspects of social behavior outcomes as they play out in complex societies over time have not been quantified. These outcomes include changes in aggressiveness and how they relate to contest behavior and rank attainment as well as patterns of dyadic affiliation and the resulting network positions and possible social bonds. Although different types of models often make similar predictions about behavioral outcomes of prenatal adversity, they do make differential predictions about life-history and other energy trade-offs (linked to, e.g., catch-up growth or accelerated life history and reproduction) as well as the differential effects of stress exposure depending on its timing during gestation. Future research should incorporate assessments of sex differences in prenatal plasticity and explicitly integrate later sensitive periods including adolescence to test how different periods of increased plasticity interact to determine phenotypic outcomes. The role of prenatal stress effects on social cognition and resulting constraints in adaptive social behavior and its effects on fitness components will require more work as does the role of social buffering effects in mediating intergenerational developmental plasticity.

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