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Review

Parental precaution: Neurobiological means and adaptive ends

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ABSTRACT

Humans invest precious reproductive resources in just a few offspring, who remain vulnerable for an extended period of their lifetimes relative to other primates. Therefore, it is likely that humans evolved a rich precautionary psychology that assists in the formidable task of protecting offspring. In this review, we integrate precautionary behaviors during pregnancy and postpartum with the adaptive functions they may serve and what is known of their biological mediators, particularly brain systems motivating security and attachment. We highlight the role of reproductive hormones in (i) priming parental affiliation with young to incentivize offspring protection, (ii) focusing parental attention on cues of potential threat, and (iii) facilitating maternal defense against potentially dangerous conspecifics and predators. Throughout, we center discussion on adaptive responses to threats of disease, accident and assault as common causes of child mortality in the ancestral past.

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1. Introduction and overview

Humans are unique among our closest primate relatives in the sheer amount of parental care required to raise offspring to reproductive age (see Pillsworth and Haselton, 2006, for a review). Compared to other species, human offspring are vulnerable and heavily dependent on parents for many years of their lives. From birth, chimpanzee offspring, for example, are able to hold onto their mothers' backs as they search their surroundings for food and are fully self-sufficient at 5 years of age (Lancaster et al., 2000). Human offspring, by contrast, are unable to lift their heads or unfold their hands until almost 3 months of age. During their extended period of dependency, human offspring need help to avoid a number of deadly hazards, including disease, accidents and hostile humans.

Human progeny are also precious in the sense that humans produce relatively few offspring during their lifetimes. For example, the average woman living in a traditional, natural-fertility culture, begins her reproductive career at around 20 years of age and produces only 2.4 offspring who survive to adulthood (Hewlett, 1991). Losing one of those children as a result of accident or dis-

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ease could reduce a mother's lifetime reproduction by nearly 50%. Because human offspring are so vulnerable and so precious, evolution should have favored the development of a rich parental precautionary psychology to protect viable offspring from harm.

This review is the first application of precautionary psychology theory to parenting. We relate the brain mechanisms theorized to subserve security motivation (Szechtman and Woody, 2004) to parental manifestations of precaution. Our presentation also extends prior considerations of the role of security motivation systems in generating precautionary behavior by highlighting the crucial contributions of reward systems. Perinatal hormones, particularly oxytocin, are discussed as they incentivize offspring protection, the development of detailed knowledge of infant cues, and maternal defense against potentially threatening conspecifics or predators. In addition to mobilizing heightened precautionary efforts, we also cite the importance of parental reward in attenuating otherwise maladaptive levels of parental anxiety.

The review adopts a vertically integrative perspective on parental precautionary psychology which relates evolutionary ends to neurobiological means. In Section 2, we outline chronic adaptive challenges to offspring in the ancestral past to predict which threats should be reflected in modern humans' parental worries and precautionary behaviors. In Sections 3–6, we synthesize results from the study of parental neurobiology with Szechtman and Woody's (2004) model of security motivation, highlighting the crucial importance of parental love in both incentivizing and buffering security-motivated worry. The discussion next turns to specific expressions of precautionary parental behavior during pregnancy, postpartum and beyond. Section 7 summarizes disease-avoidant shifts in food and social preferences during early pregnancy, Section 8 addresses ways of compensating for increased vulnerability to attack during late pregnancy, Section 9 discusses parental preoccupations with a variety of potential infant threats during the postpartum period, and Section 10 focuses on mechanisms of maternal defense against conspecifics who pose risk to offspring. Within each developmental phase, we discuss the possible biological underpinnings of precautions directed at preventing offspring from coming to harm (Table 1 summarizes pregnancy and Table 2 summarizes the postpartum period). Finally, in Section 11, we relate the assembled data on parental precaution to the issues involved in disentangling co-opted adaptations designed for precaution in other domains from adaptations designed specifically for parental precaution in response to particular classes of hazard.

2. Ancestral hazards and parental precaution

Parental precaution systems should be disproportionately attuned to threats which regularly harmed offspring in the ancestral past, much like evolved fear–acquisition systems are attuned to recurrent threats such as dangerous animals and falling from heights (e.g., Seligman, 1971; for a review of developmental precautionary psychology, also see Boyer and Bergstrom, this issue). In this section, we explore the classes of threats likely to have negatively or even lethally impacted children in the environment in which humans evolved.

One class of danger which appears to have posed a serious risk to offspring in the ancestral past is harm inflicted by fellow humans, particularly unrelated males whose reproductive interests conflicted with those of mothers and their existing children (Hahn-Holbrook et al., 2010a). Infanticide committed by unrelated males is commonplace among nonhuman animals (Van Schaik and Kappeler, 1997). Hrdy (1999) compares the prevalence of male strangers committing infanticide across primate species with evidence gathered from the Ache Indians. Among this Paraguayan hunter-gatherer society, out-group tribe members constituted the single greatest cause of mortality for children aged 4–14, accounting for 56% of deaths, and 16% of the deaths of children 0–3 years of age (Hill and Hurtado, 1996). The presence of this pattern in modern traditional societies is consistent with the notion that human history was fraught with infanticidal attacks.

This ancient threat is echoed in the worries of modern parents. For example, parents report that one of their foremost anxieties is that their child will be abused or killed by strangers (Kantrowitz, 1997; Kidscape, 1993). In reality, stranger abuse and homicide account for less than 1% of the actual harm that befalls children in the United States (Center for Disease Control and Prevention, 1982). By contrast, automobile accidents account for 41% of all non-natural child deaths each year (UNICEF, 2001). Modern parents worry over these two potential sources of harm to children at comparable rates despite the drastic differences in their frequencies in the modern world (Stickler et al., 1991). Critics of "stranger danger" hysteria typically blame the mass media for sensationalizing incidents of child assault by strangers (e.g. Skenazy, 2008). Although there is likely to be some merit to the media-influence hypothesis, the question of why this gambit is so effective remains. Given the low probability of child harm due to strangers, why do these scenarios, rather than far more likely and equally severe threats to children, pique the concern of contemporary audiences?

Hahn-Holbrook et al. (2010a) review evidence that stranger anxiety, which emerges cross-culturally at around the time infants begin to crawl, is an evolved mechanism facilitating caution towards strangers which persists throughout the lifespan, albeit muted by habituation and executive inhibition as children age. For example, infants who display overt stranger anxiety at 8-months (e.g. crying or grimacing) go on to display implicit anxiety (e.g. heightened cortisol) when approached by strangers at year 3 (Zimmermann and Stansbury, 2004). In a comparison of implicit versus explicit negative attitudes toward strange out-group members in 6-year-olds, 10-year-olds and adults, Baron and Banaji (2005) likewise found that self-reported aversion decreased with age, but implicit aversion varied little across age groups. In addition, male strangers, who are believed to have posed greater threats in the ancestral past (Hrdy, 1999), are disproportionately feared by both adults (Navarrete et al., 2009) and infants (Benjamin, 1961; Feinman, 1980; Skarin, 1977). Infants display more fear of male strangers even if those infants' primary caregivers were male, discounting the alternative hypothesis that the aversion against males simply results from familiarity with females (Lamb et al., 1982). Taken together, these findings indicate that adults' predisposition to fear strangers, especially males, echoes a genuine threat posed to children in the ancestral environment, leading modern parents to index strangers as salient threats to their offspring today.

Beyond stranger violence, other causes of child mortality in traditional societies provide clues of recurrent ancestral threats and thereby predict the domains of focus for parental precaution. Detailed demographic studies of mortality among hunter-gatherer populations have been published for the !Kung (Ju/'hoansi) of Botswana–Namibia (Howell, 1979), the Ache of Paraguay (Hill and Hurtado, 1996), the Agta of the Philippines (Early and Headland, 1998), and the Hiwi of Venezuela and Columbia (Hill et al., 2007). In comparing the causes of death for children across these populations prior to the colonial era, disease was the most important, accounting for anywhere from 20 to 85% of mortality (Hill et al., 2007). Further, in all populations sampled, infancy was the most vulnerable phase of development. For example, the likelihood of dying during the first year of life for a Hiwi infant was 14% for males and 26% for females. Excluding parental infanticide, the most common causes of death among Hiwi infants were congenital defects (30%), infectious diseases (27%), stranger/out-group violence (7%), negligence/accidents (6%), and within-group violence (3.5%). Between the ages of 1 and 9, the mortality rate drops considerably. In the older age group, diseases still accounted for roughly 66% of deaths

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Table 1
Taxonomy of precautions during pregnancy.

Offspring threat	Function	Psychological or behavioral shift	Possible biological mediators
Disease	Prevent congenital defects	Increased olfactory acuity, disgust proneness, aversion to eating foods likely to harbor pathogens (Fessler, 2002; Flaxman and Sherman, 2000, 2008; Hook, 1974; Profet, 1988)	hCG (see Jueckstock et al., 2010); Progesterone (Flaxman and Sherman, 2000)
	Prevent congenital defects	Preference for social cues (facial features: Jones et al., 2005; in-group membership: Navarrete et al., 2007) indicative of decreased likelihood of pathogen exposure	Progesterone (Jones et al., 2005)
	Decrease infant exposure to pathogens after birth	Intensive cleaning/nesting of home environment late in pregnancy (Leckman et al., 2004)	Progesterone (Broida and Svare, 1983) acting on the medial preoptic area (Jacobson et al., 1980) in association with security motivation circuits involved in ritualistic behavior (Swain et al., 2007; Szechtman and Woody, 2004)
Attack	Avoid predators during period of poor mobility	Reduced foraging, staying near home (Bauwens and Thoen, 1981)	Unknown
	Increase protection from kin	Odor preference for MHC similarity thought to index kin (Roberts et al., 2008; Wedekind et al., 1995)	Changes in estradiol and progesterone associated with pregnancy (Roberts et al., 2008; Wedekind et al., 1995)
Accidents	Reduce risk of injury due to poor mobility	Heightened risk aversion	Unknown
	Reduce risk of home accidents after birth	Intense nesting (e.g. organizing/cleaning) late in pregnancy (Leckman et al., 2004)	Progesterone (Broida and Svare, 1983) acting on the medial preoptic area (Jacobson et al., 1980) in association with security motivation circuits involved in ritualistic behavior (Swain et al., 2007; Szechtman and Woody, 2004)

among the Hiwi, with accidents and homicides each accounting for approximately 14%. These data indicate that modern parents should be expected to promote protection of children from diseases, accidents, and stranger violence, particularly during infancy. Because congenital defects accounted for substantial rates of infant mortality and can be caused by mothers' exposure to pathogens during gestation (Fessler, 2002), pregnant mothers should also exhibit intense contagion precautions. In the next several sections, we introduce neurobiological mechanisms which may mediate the psychological processes involved in parental precaution in these domains.

An evolutionary perspective also predicts sex differences in parental precaution. Women must invest heavily in each offspring they produce because of the energetic costs of pregnancy and breastfeeding. Due in part to these costs, women's reproductive budgets are limited compared to those of men. Moreover, because gestation happens internally within women's bodies, women are certain of their relatedness to offspring, whereas fathers are not, leading to greater on-average returns on maternal investment than paternal investment (see Pillsworth and Haselton, 2006, for a review). All of these factors typically lead to greater parental investment by mothers than fathers (Trivers, 1972), including, we propose, investments in precautionary behaviors.

Fathers nevertheless play a critical role in safeguarding offspring. For example, in traditional societies the absence of a father (due to death or divorce) is associated with as much as a two-fold increase in the likelihood of child death (Hurtado and Hill, 1992). There is also evidence from nonhuman species for the importance of protection from fathers. Hrdy (1999) describes male gorillas picking up their offspring when intruding males display aggressive behavior, evidently to guard progeny against potential attacks from unrelated males (Hausfater and Hrdy, 1984). Van Schaik and Kappeler (1997) have proposed that the evolution of year-round male–female association in primates may reflect the necessity for fathers to ward off infanticidal male interlopers. Fathers are likely to display a range of other precautionary protective behaviors in defense of young beyond guarding against conspecifics. Unfortunately, however, the preponderance of research relevant to parental precaution has studied mothers. Although the evidence for precautionary behavior and neurobiology in fathers is less abundant than for mothers, we make reference to this work when it is available.

Table 2
Taxonomy of postpartum precautions.

Offspring threat	Function	Psychological or behavioral shift	Possible Biological Mediators
Disease	Detect early signs of infant illness	Heightened scrutiny and memory of infant cues (Fleming et al., 1997; Leckman et al., 2004; Stallings et al., 2001)	Oxytocin increases opioid signals to appraisal centers, e.g. amygdala, hippocampus, neocortex (Depue and Morrone-Strupinsky, 2005); cortisol (Fleming et al., 1997; Stallings et al., 2001)
Attack	Preempt potential conspecific/predator attack	Increased willingness to engage in defensive aggression (Gammie et al., 2008; Hansen and Ferreira, 1986; Hahn, 2010)	Changes in estrogen, GABA, and oxytocin associated with lactation (for reviews, see Lonstein and Gammie, 2002; Campbell, 2008)
Accidents	Prevent inadvertent harm (e.g. drowning, falls)	Maintain close proximity to child, anxiety about potential risks, monitor child, organize surroundings (Leckman et al., 2004)	Oxytocin increases opioid signals to appraisal centers, e.g. amygdala, hippocampus, neocortex (Depue and Morrone-Strupinsky, 2005); security motivation circuits involved in ritualistic behavior (Swain et al., 2007; Szechtman and Woody, 2004)

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3. Neurobiology of parental precaution

Precautionary psychology investigates ways people detect and respond to cues of potential danger (e.g. predator proximity) as opposed to imminent danger (e.g. predator attack). These two types of hazard stimuli appear to elicit distinct behavioral and psychobiological processes (Woody and Szechtman, this issue). The *security motivation system* is hypothesized to be functionally designed to avert possible threats (Boyer and Lienard, 2006; Szechtman and Woody, 2004). In this section, we review psychobiological data suggesting that parental precaution co-opts mechanisms associated with the security motivation system, much as parental love has been depicted as largely subserved by reward circuitry common to romantic love (Bartels and Zeki, 2004). To demonstrate this co-optation, key neurobiological regions linked to the security motivation system (for a fuller discussion, see Szechtman and Woody, 2004; Woody and Szechtman, this issue) are synthesized with parallel findings generated in studies of parents' responses to cues of potential infant threat. In addition, we highlight the contributions of reward-oriented affiliative motivation circuits to the production of adaptive parental precaution.

Motivation systems are comprised of inter-related circuits which serve three basic functions: *appraisal*, *response selection*, and *satiety*. Initially, stimuli must be appraised as significant to relevant affective and motor response pathways. Once a particular motivational pathway is energized by appraisal circuits, goal-relevant responses are selected. Finally, feelings of satiety terminate the motivational state. For example, a security-motivated individual may appraise an ambiguous sound as indicative of potential hazard, which promotes anxiety about the origin of the noise and the selection of cautious checking behaviors, ultimately culminating in feelings of safety once the potential threat is determined to have been managed. We organize discussion of the motivational circuitry underlying parental precaution according to appraisal, response selection, and satiety functions in the three sections which follow.

To preview, parents' hazard appraisal regions identify cues of potential threat to offspring and mobilize precautionary affective and behavioral responses. Infants themselves are appraised as rewarding attachment-objects, motivating parental care and attention. This reward-appraisal appears to incentivize defense of the child against potential threats via security motivation processes. In addition, affiliative parental reward enhances learning of idiosyncratic infant attributes, including indications of possible problems. Upon appraisal of potential hazards to young, parental response selection regions initiate anxious feelings and motor responses suggestive of efforts to resolve threats, but which may also stem from a reward-motivated predisposition to approach infants when the infant is the source of the threat cue (e.g. the infant cries or appears ill). Parenthood is stressful, in part, because parents' precaution systems naturally generate worry over potential future harm coming to their children. The reward response derived from affiliative interactions with offspring buffers otherwise deleterious levels of heightened anxiety related to parenting, helping to satiate precautionary parental drives.

4. Appraisal mechanisms

The appraisal system detects potential threats and initiates security-motivated response selection pathways related to feelings of anxiety and behaviors such as checking, organizing or cleaning (Boyer and Lienard, 2006; Szechtman and Woody, 2004). Alternatively, in affiliative contexts, appraisal centers initiate feelings of affection and related approach-oriented behavior (Strathearn et al., 2009). As we will discuss, these dual articulations of the brain's appraisal network appear to interact, such that the reward connotations of young incentivize parental precautionary expenditures as well as learning of offspring cues of potential hazard.

Key elements of the appraisal network include the prefrontal cortex, amygdala, hippocampus, and bed nucleus of the stria terminalis (Depue and Morrone-Strupinsky, 2005; Szechtman and Woody, 2004). Prefrontal processing enables the forecasting of future outcomes, including potential threats to infants (Swain et al., 2007), and relays these interpretations to the amygdala, which functions as an "alarm" complex to relay signals of threat (Baxter and Murray, 2002; Bechara et al., 2000; Gottfried et al., 2003). The hippocampus receives inputs from the amygdala prerequisite to the formation of contextual emotional memories—a vital function for deriving cues of potential danger (Davis and Shi, 1999; Selden et al., 1991; Winocur, 1997). The bed nucleus of the stria terminalis is a major output pathway of the appraisal network (Depue and Morrone-Strupinsky, 2005). In what follows, we briefly discuss how each of these regions, in concert with reward activity related to parenting, may subserve parental precaution.

Parents appraise their infants, particularly in the early months postpartum, as intensely rewarding (Swain, 2008). Oxytocin is a reproductive neuropeptide which may help to explain these dramatic reward responses. Oxytocin is produced in the hypothalamic paraventricular nucleus and projected to appraisal centers (e.g. amygdala, hippocampus) as well as response selection areas (e.g. nucleus accumbens) related to affiliative motivations. High levels of oxytocin are released by vaginocervical stimulation at the time of parturition, during lactation, and in early interactions with offspring (Carter and Altemus, 1997; Gimpl and Fahrenholz, 2001). Immediately after birth, for instance, mother–infant skin contact elevates maternal oxytocin levels (Nissen et al., 1995). Oxytocin intensifies the reward associations of co-occurring stimuli by increasing the release of opiates (Depue and Morrone-Strupinsky, 2005). Oxytocin-enhancement of opiate release may account for rat dams' notable preference for pups over cocaine in the first few days after parturition (Mattson et al., 2003). Although highly rewarding attachment-objects are typically regarded as eliciting pleasurable reactions, sources of reward trigger heightened distress when the attachment object is in jeopardy (Panksepp, 1981). Thus, the opioid activity associated with offspring should be expected to increase the degree of alarm elicited by prospective offspring threat, particularly in the early months after parturition when offspring generate the most intense reward activation.

Heightened "alarm" reactions in the amygdala have been documented in several imaging studies of parents' reactions to infant cries, which may be regarded as cues of potential threat. Swain et al. (2003) exposed primiparous parents to sound recordings of their infants' cries and found notable activation in both the amygdala and insula (linked with aversive affect), suggesting an appraisal of alarm likely to initiate a precautionary response. In a comparison of the responses of parents and non-parents to infant cry versus laugh recordings, Seifritz et al. (2003) observed that infant crying elicited greater right amygdala response than infant laughing among parents, whereas non-parents showed the reverse pattern. This indicates a change in amygdala function accompanying parenthood such that cues of potential child hazard are appraised as more motivationally salient. The amygdala was also found active in response to infant cry stimuli when compared with inter-stimulus rest periods in a study of primiparous mothers 1–2 months postpartum (Lorberbaum et al., 2002; also see Sander et al., 2007). Strathearn et al. (2008) found that images of crying infant faces significantly activated the amygdala relative to a no-face baseline in a study of 28 primiparous mothers, whether the images were of their own infants or unknown infants. While these findings of amygdala response to infant cry are suggestive, it is important to caution that some researchers have failed to detect significant

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differences in amygdala reactivity to infant cry (Bartels and Zeki, 2004; Nitschke et al., 2004; Swain et al., 2008), possibly because the amygdala habituates rapidly to infant stimuli (Kim et al., 2010).

Beyond increasing appraisals of alarm at the prospect of infant threat, the reward-incentive associated with infants may also facilitate the acquisition of detailed knowledge of offspring cues. Oxytocin release appears to intensify parental interest in and memory of interactions with infants. In mice, for example, oxytocin has been found to improve long-term potentiation in hippocampus-mediated spatial learning and memory, an effect hypothesized to assist mothers in tracking pups (Tomizawa et al., 2003). In humans, oxytocin receptors are densely expressed in regions which project to the amygdala, hippocampus, and thalamus, as well as diffusely projecting to acetylcholinergic targets in neocortex. These acetylcholinergic targets are known to modulate memory formation in concert with the amygdala and hippocampus, indicating that oxytocin modulates parental learning indirectly by influencing acetylcholine (Depue and Morrone-Strupinsky, 2005; McGaugh, 2000).

As important as reward-incentive appears to be for learning about young, hormone data indicates that a certain level of parental anxiety also helps parents to acquire high-resolution representations of their offspring. In an early study of the contribution of parental stress, primiparous human mothers with higher levels of cortisol (indicative of stress) were found more accurate in discriminating their own infants from others by smell (Fleming et al., 1997). Notably, these high-cortisol mothers also displayed more affectionate maternal behavior—pointing once again to the interconnectedness of parental anxiety and love. In a follow-up study which attempted to directly measure the contribution of mild stress reactions to maternal care, primiparous mothers in the first 2 days postpartum were exposed to sound recordings of infant hunger cries versus infant pain cries and compared to nulliparous women (Stallings et al., 2001). The investigators found that mothers reported more sympathy with the pain cries, exhibited increased heart rate, had higher levels of salivary cortisol and tended toward emotions which favored approach. Nulliparous females, by contrast, expressed less sympathy, exhibited lower heart rate, possessed less salivary cortisol, and were inclined toward infant avoidance. As predicted, mothers also displayed greater ability to perceptually discriminate hunger from pain cries. The degree of accuracy in discriminating types of cry positively correlated with salivary cortisol levels, mirroring the previous finding regarding infant discrimination by smell (Fleming et al., 1997). Moreover, the most sympathetic mothers in the sample showed the greatest cortisol reactivity when exposed to infant cries, as well as the most dramatic subsequent reduction in cortisol. It is possible that this pattern reflects a relatively greater maternal glucocorticoid stress reaction to cues of potential harm to offspring, followed by greater parasympathetic stress alleviation related to their rewarding attachment relationships with young.

The bed nucleus of the stria terminalis relays signals from the amygdala to the hypothalamus, where both anxiogenic hormones related to security concerns and anxiolytic hormones related to reward and affiliation are emitted (Depue and Morrone-Strupinsky, 2005). This may account for observations that the bed nucleus of the stria terminalis triggers both nurturant maternal behavior and anxious responses (Mayes et al., 2009). As an output of the hazard appraisal system, the bed nucleus of the stria terminalis is thought to regulate anxiety during the postpartum period (Lonstein, 2007).

The medial preoptic area of the basal forebrain is closely connected with the bed nucleus of the stria terminalis, and strongly linked with motivating parental care behaviors (Lorberbaum et al., 2002). Lactation has been found to increase oxytocin receptor binding in the bed nucleus of the stria terminalis and the medial preoptic area of rat dams (Bosch et al., 2010), likely attuning maternal responsiveness to young as social attachments with them develop. For example, parturient dams who were administered an oxytocin antagonist into the medial preoptic area failed to retrieve or assume a nursing posture over their pups (Pedersen et al., 1994). As output regions from the appraisal network to the response selection centers, the bed nucleus of the stria terminalis and the closely related medial preoptic area may trigger species-typical, stereotypical parental responses, or flexible, planned responses drawing on higher executive centers, via distinct output pathways. In rodents, descending connections from the bed nucleus of the stria terminalis/medial preoptic area region have been observed to modulate basic parental behaviors such as nursing, grooming and carrying reflexes, whereas ascending connections with the mesolimbic and mesocortical dopamine systems appear to enable selection of more flexible, situationally appropriate responses, including reacting to cues of potential hazard (Numan and Insel, 2003). In the following section, we review the contribution of response selection regions to the production of both security and affiliation-motivated behaviors which help protect offspring from potential harm.

5. Response selection mechanisms

Shifting focus from the appraisal to the response selection network, precautionary parental behaviors are orchestrated via cortico–striato–thalamo–cortical circuits (O'Donnell, 2005; Swain, 2008; Williamson, 2006) theorized by Szechtman and Woody (2004) to subserve security motivation. When a parent acts to protect their child from possible harm, these motor responses are thought to be disinhibited via a striatal circuit through the ventroanterior/ventrolateral thalamic nucleus to motor regions of cortex, triggering specific behaviors (e.g. infant cleaning) by selectively disinhibiting target motor areas (Lukhanina, 1995; Szechtman and Woody, 2004). In support of this model, degeneration of medial thalamic nuclei has been found to impair maternal behavior in rats and hamsters (Maclean, 1990).

Affective parental responses appear subserved by a closely parallel striato–thalamo–cortical pathway. Once signalled by dopaminergic inputs from the amygdala or ventral tegmental area (e.g., activation associated with child cues), the nucleus accumbens region of the ventral striatum inhibits target areas of the ventral pallidum, thereby disinhibiting regions of the mediodorsal thalamic nucleus thought to relay affective signals related to children to the prefrontal association cortex (Swain, 2008). This parallel relay of affective and motor signals has been theorized to correlate affective reactions to online stimuli with behavioral responses (Li et al., 2004; Groenewegen and Trimble, 2007).

Oxytocin establishes response selections related to the care of offspring (for a review, see Gimpl and Fahrenholz, 2001). Lesion of the oxytocin-generating cells of the paraventricular nucleus has been found to suppress maternal behavior in postpartum dams (van Leegoed et al., 1987), while intracerebroventricular administration of oxytocin into virgin female rats induces full maternal behavior within mere minutes (Pedersen and Prange, 1979). In humans, Feldman et al. (2007) correlated mothers' plasma oxytocin levels with infant interactions and self-reported infant-related thoughts and behaviors. Oxytocin levels both before birth and during the postpartum period were related to a clearly defined set of maternal bonding behaviors, including infant gazing, vocalizations, affectionate touch, and frequent checking. The co-occurrence of precautionary checking and affiliative behaviors in mothers with higher oxytocin levels reported in this study underlines the importance of reward in parental precaution.

Parents' responses to infant cry have been empirically linked with the limbic regions and striato–thalamo–cortical basal ganglia circuits implicated in both security and affiliative motivation (Boyer and Lienard, 2006; Swain, 2008; Swain et al., 2003; Szechtman

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and Woody, 2004). For example, Swain et al. (2003) exposed primiparous parents at 2–4 weeks postpartum to cry stimuli recorded from either their own or an unfamiliar infant. The sounds of parents' own infants crying elicited greater activation in the midbrain and basal ganglia in comparison to the sounds of unfamiliar babies. Mothers showed a more intense level of activation than fathers, consonant with evolutionary predictions of relative parental investment. Other imaging studies of human mothers exposed to infant cues of varying modalities (e.g. images, cry recordings) have replicated findings of reward activation in response selection regions including the ventral tegmental area, substantia nigra, and nucleus accumbens (Bartels and Zeki, 2004; Squire and Stein, 2003; Strathearn et al., 2009). In Lorberbaum et al.'s (2002) infant cry study, for example, cry stimuli activated the ventral tegmental area, hypothalamus, septal and striatal regions linked with the selection of maternal responses in humans and considered fundamental for rodent maternal behavior (also see Strathearn et al., 2008).

The ventral striatum component of the response selection network has been conceptualized as a region for the integration of emotional information (via afferents from the amygdala), contextual information (via hippocampus) and executive information (via prefrontal cortex). The outputs of the ventral striatum combine these sources to influence brain areas involved in the production of motivated behavioral and affective responses (Groenewegen and Trimble, 2007). In rodents, lesions of the nucleus accumbens region of the ventral striatum impair maternal behavior (Hansen, 1994), whereas pup cues trigger reward-related activity in the nucleus accumbens (Champagne et al., 2004). Dopamine activity in the ventral striatum is often identified with reward-seeking behavioral tendencies, but is also implicated in avoidant reactions linked with stress and anxiety (Reynolds and Berridge, 2002; Schultz, 2007), responses which are each closely linked with parental behavior in both nonhuman animals and humans (Swain, 2008).

Strathearn and colleagues' recent studies illustrate the importance of reward-related reactivity in eliciting parental responsiveness (e.g. as measured by activation of the ventral striatum) to cues of potential harm. In an event-related imaging study (Strathearn et al., 2008), 28 primiparous mothers were exposed in 2-s intervals to images of the faces of their own or an unknown infant, aged between 5 and 10 months. The infants were photographed smiling, crying or neutrally expressive. Regardless of face valence, regions linked with response selection, including the ventral tegmental area/substantia nigra regions (indicating reward), ventral striatum, and primary motor area were activated in response to images of own-infant faces compared with unknown infant faces. A region-of-interest analysis revealed that reactivity in nigrostriatal reward-related regions interconnected by dopaminergic neurons was proportional to the positivity of the infant's affect for each contrast between the known versus unknown infants: smiling faces elicited greater responses than neutral faces, which elicited greater responses than crying faces. Thus, images of mothers' own infants automatically initiated behavioral response selection mechanisms regardless of their facial affect, but smiling infants evoked the most rewarding activity.

Strathearn and colleagues subsequent (2009) experiment examined the effect of individual differences in maternal attachment on responsiveness to the same classes of known versus unknown smiling, neutral or crying infant face photos. 30 primiparous women were administered the Adult Attachment Interview and scores were correlated with (i) activity in reward areas and stress areas (e.g. insula) within the mothers' brains upon exposure to infant face stimuli and (ii) the peripheral release of oxytocin during infant contact (measured in a separate session approximately 4 months prior to brain scanning). Mothers with secure attachment relationships showed greater activation of reward centers while viewing images of their infant smiling or crying, as well as greater peripheral oxytocin release during the infant interaction session. Remarkably, in securely attached mothers, oxytocin release during infant contact measured 4 months previously positively correlated with brain activity in the ventral striatum during the scanning session, as well as activation in appraisal centers containing oxytocin projections (amygdala, hippocampus, bed nucleus of the stria terminalis). In contrast, mothers with insecure attachment relationships did not evince ventral striatum reactivity or high levels of oxytocin during prior infant interaction, instead showing heightened insula reactivity in response to images of their own-infant crying.

Interpreting Strathearn et al.'s findings from the perspective of parental precaution, insecurely attached mothers failed to evince automatic impulses toward behavioral responses to cues of potential offspring harm—despite displaying stronger appraisals of those cues as aversive. Conversely, securely attached mothers showed the greatest ventral striatum response suggestive of adaptive precautionary checking and care behaviors—yet the least reflexive aversion to cry cues. This pattern clearly demonstrates the insufficiency of anxious, security-motivated responses alone to account for parental precaution. Indeed, highly anxious parents who garner low levels of reward from offspring interaction tend toward avoidant, abusive or overbearing parenting styles with adverse child outcomes (Mayes et al., 2009; Moses-Kolko et al., 2008; Numan and Insel, 2003). Similarly, mothers whose attachment systems have been "hijacked" by pleasurable substances such as cocaine also tend toward neglectful or physically abusive treatment of their children (Chaffin et al., 1996).

Strathearn and colleagues' (2009) finding that securely attached mothers do not display significant aversion to imagery of their infants' crying should not be mistaken for evidence that these mothers lacked concern when exposed to cues of potential infant harm. Given the precautionary themes characteristic of parental preoccupations with child safety (Leckman et al., 1999; see Section 9), it seems that a certain measure of heightened stress reactivity in early parenting, particularly in response to cues of potential child threat, facilitates care and defense of offspring (Stallings et al., 2001). The early postpartum period is characterized by relatively high levels of cortisol and emotional lability (Fleming et al., 1987), but affiliation with infants during this period simultaneously increases parental reward (Strathearn et al., 2008, 2009) and attenuates parental anxiety (Neumann and Bosch, 2008).

6. Satiety mechanisms

Szechtman and Woody (2004) noted that security motivation differs from other motivational states in that it must be terminated by endogenously generated satiety signals—the environment is literally incapable of providing conclusive safety cues regarding potential hazards (e.g. the lion may yet lurk). Serotonin released in conjunction with the production of precautionary behaviors (e.g. checking, cleaning) has been proposed to provide the satiety-like signal of safety needed to abate security motivation (Woody and Szechtman, this issue). In support of this premise, serotonin release from the brainstem has been observed to down-regulate both threat-avoidant and reward-seeking motivational states (e.g. Blundell, 1991; Linnoila and Virkkunen, 1992), and has been linked with parasympathetic activity related to feelings of satisfaction and calmness (Depue and Morrone-Strupinsky, 2005).

Applying the same logic to parental security motivation, the organism must produce satiety signals indicating that sufficient precautions have been taken to safeguard the child. Deficiencies in serotonin transmission should therefore lead to failure to terminate parental precautionary behaviors and feelings of anxiety. Indeed, in primates, low serotonin has been correlated with

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greater maternal protectiveness and vigilance (Fairbanks et al., 1998; Lindell et al., 1997; for a review, see Maestripieri, 2008). In humans, low serotonin levels have been implicated in pathological levels of parental precaution. New parents typically experience intrusive thoughts regarding their child's safety, but these precautionary intrusions typically last no more than 1 h per day and, although distressing, do not interfere with normal functioning. For a small portion of new parents, however, these preoccupations and anxieties deepen into postpartum obsessive–compulsive disorder (Moses-Kolko et al., 2008; Swain et al., 2007). Parents with postpartum obsessive–compulsive disorder suffer clinically extreme fixations on infant safety and related behaviors such as frequent checking, washing and cleaning (Maina et al., 1999). Serotonin reuptake inhibitors have been found effective in treating postpartum obsessive–compulsive symptoms (Aouizerate et al., 2005) as well as ameliorating non-parental obsessive–compulsive disorder (see Woody and Szechtman, this issue), further corroborating the overlap between parental and non-parental articulations of security motivation mechanisms.

We have reviewed evidence that reward-associations function to inspire parental incentive to safeguard infants, yet the anxiogenic qualities of parental reward appear to buffer parental worry. How can parental reward incentivize security motivation to protect infants on the one hand, and down-regulate security-motivated anxiety on the other? It would seem that there are two aspects of parental reward: the desire to interact with infants, and the satisfaction derived from these interactions. This pattern resembles the classic distinction between anticipatory and consummatory reward (Dillon et al., 2008). Consider, for instance, the value a hungry person might attach to a particularly tasty lunch morsel. Prospective threats to the cherished lunch (e.g. being stolen or dropped) should be expected to evoke a heightened motivation to protect it (Panksepp, 1981). Consuming a delicious lunch, however, produces pleasant feelings of enjoyment and satisfaction. Analogously, an intensely attached parent should be expected to worry over and take steps to prevent harm to cherished offspring (e.g. being stolen or dropped), and affiliative interactions with young should elicit feelings of enjoyment and satisfaction which assuage parental anxiety. These two aspects of parental reward appear cyclical, as satisfying interactions with young reinforce their anticipatory reward value.

To conclude, parental precautionary behaviors appear largely to co-opt the brain circuits associated with security motivation described by Szechtman and Woody (2004; also see Swain, 2008). Equally importantly, parental neurobiology is characterized by dramatic shifts in reward motivation which underlie offspring affiliation. Parental reward appears to incentivize precautionary expenditures to learn about and protect the cherished attachment object (Strathearn et al., 2009; Swain et al., 2007). We have reviewed evidence confined almost exclusively to parents' reactions to infant cries, as these studies provide the clearest data on precautionary processes available. The lack of imaging studies employing a broader array of cues of potential threat to offspring significantly limits our discussion. Future studies of parental precaution should explore how parents respond to more indirect indications of potential hazards, particularly hazards from within the domains which posed the greatest adaptive challenges in the ancestral past (e.g. responses to sick individuals in close proximity to infants).

In the balance of the review, we address ways in which strategic shifts in the parental precaution system (e.g. changing reactions to food, faces or kin) may adaptively track with the demands of successive reproductive phases. We relate the hypothesized biological mediators of these changes during pregnancy and postpartum to the security and affiliative motivation framework where evidence is available.

7. Pregnancy and pathogen-avoidance

Many potentially fatal birth defects are avoidable if mothers minimize contact with contaminants during pregnancy. Indeed, a growing body of evidence suggests that pregnant mothers become more avoidant of potential sources of contagion, such as foods more likely to carry pathogens (Fessler, 2002; Flaxman and Sherman, 2000, 2008; Hook, 1974; Profet, 1988). The most commonly reported pregnancy-related food aversions concern meats (Flaxman and Sherman, 2000), which are highly susceptible to pathogens unless prepared using sanitary methods likely unavailable to our ancestors. These pathogens (e.g. *Escherichia coli*) are especially harmful when contracted during early pregnancy, before the fetus develops key immune defenses (for a review of studies linking various pathogens to adverse fetal, birth and developmental outcomes, see Fessler, 2002).

The selective pressure posed by this period of fetal vulnerability has prompted proposals that nausea and vomiting during pregnancy, commonly termed pregnancy or morning sickness, is an evolved response which promotes strategically adaptive shifts in dietary preferences (Fessler, 2002; Flaxman and Sherman, 2000, 2008; Hook, 1974; Profet, 1988). Endured by mothers the world over, 50–90% of all pregnancies are accompanied by nausea and vomiting in the early months (Gadsby et al., 1993). Pregnancy sickness usually begins roughly 4–6 weeks following conception, peaks at between 8 and 12 weeks, and subsides at approximately 14–15 weeks of gestation (Weigel and Weigel, 1989). In up to 20% of cases, however, nausea and vomiting may continue until delivery.

Fessler (2002) argues that during early pregnancy, when mothers' immune systems are temporarily weakened to permit the growth of a foreign organism (i.e., the fetus) and the added caloric demands of pregnancy are still minimal, changes in the olfactory system mediate nausea and disgust reactions to pathogen-rich foods (but see Forbes, 2002). In support of this prophylactic function, cross-cultural rates of pregnancy sickness were found to correlate with higher average consumption of foods likely to harm the developing fetus (e.g. meats, sugars, alcohol) and lower average intake of safer foods (e.g. cereal grains) in a survey of 21 countries (Pepper and Roberts, 2006). Further, women with pregnancy sickness are less likely to experience miscarriages or preterm deliveries and are at reduced risk of intrauterine growth retardation (for a review, see Furneaux et al., 2001). As one might expect, however, severe levels of pregnancy sickness predict low birth weights, especially when persisting into later pregnancy (Zhou et al., 1999).

Although a relatively large body of research addresses the physiological underpinnings of pregnancy sickness, the specific hormonal mediators are still debated (for a review, see Jueckstock et al., 2010). Perhaps the best candidate is human chorionic gonadotropin (hCG), which peaks at around 12–14 weeks, the period of gestation when pregnancy sickness is most often reported (Furneaux et al., 2001). Masson et al. (1985) took measures of hCG, schwangerschaftsprotein 1, progesterone and oestradiolin 116 pregnant women experiencing varying degrees of nausea and vomiting. They found that only elevated levels of hCG correlated with reported levels of nausea and vomiting. As all pregnant women experience elevated levels of hCG yet some do not experience pregnancy sickness, it is likely that hormone–receptor interactions modify the effects of hGC.

Progesterone has also been proposed to mediate pregnancy sickness (Fessler, 2002). In support of this link, mothers of twins have higher levels of progesterone and report greater levels of pregnancy sickness (Johnson et al., 1994). Progesterone administration can induce gastric dysrhythmias (a symptom of pregnancy sickness involving rhythm disruption in the intestines) in non-pregnant women (Walsh et al., 1996). Despite these suggestive findings, progesterone's role remains unclear as both low (Jarnfelt-

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Samsioe et al., 1986) and elevated levels (Yoneyama et al., 2002) have been reported in women experiencing pregnancy sickness (also see Masson et al., 1985; O'Connor et al., 1998, for null results). It is also important to note that pregnancy sickness typically disappears around mid-pregnancy, while progesterone levels steadily increase over pregnancy, a basic pattern which is difficult to reconcile with the premise that progesterone mediates pregnancy sickness. Whether or not progesterone mediates pathogen-avoidance through pregnancy sickness, however, menstrual cycle shifts in progesterone have been closely correlated with prophylactic behavioral efforts to avoid pathogen contact from the environment, such as increases in hand washing or avoidance of touching public toilet seats (Fleischman and Fessler, 2009). Aside from progesterone, a range of other hormones (such as estrogen, adrenocorticotropic hormone, cortisol and prolactin) have also been studied in relationship to pregnancy sickness, often with conflicting or null results (for a review, see Jueckstock et al., 2010).While the pathogen resistance function of pregnancy sickness appears fairly well-established, some have argued that this trait could be a by-product of genetic conflict between mother and embryo (Forbes, 2002). The proximal mechanisms responsible for pregnancy sickness must be rendered in higher resolution to draw conclusions about the evolutionary origins of this trait. Additional research is also necessary to clarify how shifts in hormone levels might engender increases in negative appraisals of risky foods (e.g. meats) and other potential sources of infection (e.g. public toilet seats). The correlation between hormonal changes and heightened precautionary behavior implies modifications to the neural structures implicated in the appraisal of disease hazards. Suggestively, the amygdala has been linked with both perinatal hormones and odor preferences. In humans, the amygdala is known to mediate aversion to foul odors (LeDoux, 1996); in rats, the amygdala has been found to mediate nulliparous females' aversion to pup odors (Numan and Sheehan, 1996). The neuroendocrine changes accompanying pregnancy have been found to remap pup smells from aversive to neutral or even rewarding connotations for dams (Numan, 1994). Given that perinatal hormones appear to recalibrate odor preferences for pups in rodents, we conjecture that increases in progesterone or hCG may similarly engender aversions to pathogenic food odors by modifying appraisal circuitry, perhaps by activating receptors involved in eliciting disgust reactions.

Beyond prophylactic shifts in food preferences, there is emerging evidence that pregnant women also become more sensitive to potential sources of harmful pathogens in other people. Jones et al. (2005) found that pregnant women preferred healthy faces more than non-pregnant women. Moreover, they found that non-pregnant women in the luteal phase of their menstrual cycle, when progesterone is high, significantly favored healthy faces relative to women in the follicular or ovulatory phases. Jones et al. posit that the heightened progesterone levels associated with both pregnancy and the luteal phase of the menstrual cycle (when the body is prepared for implantation of a conceptus) may lead women to disfavor facial cues of ill health as an adaptive precaution to protect offspring. Although this finding is provocative, no studies to our knowledge have directly investigated the role of endocrine factors in biasing pregnant mothers against subtle cues of disease in fellow humans, a prediction that also follows from the pregnancy prophylaxis hypothesis. Recent rodent studies linking oxytocin with social identification, including the detection of pathogen-bearing conspecifics, provide a potentially relevant animal model (for a review, see Kavaliers et al., 2005). However, oxytocin levels heighten in association with parturition, and thus do not appear directly germane to shifts in preferences for healthy faces observed earlier in pregnancy.

Another shift in appraisals of potentially pathogen-bearing conspecifics during pregnancy has been proposed by Navarrete et al. (2007). They argue that the period of compromised immunocompetence associated with the first trimester of pregnancy necessitates increased aversion to out-group conspecifics due to the likelihood that out-group members might transmit novel pathogens to which mothers would be particularly vulnerable. Navarrete and colleagues presented 206 pregnant women at varying stages of pregnancy with two essays, one written by a foreigner expressing critical attitudes and one by an American author expressing complimentary attitudes about the United States (the participants' home country). Consistent with their disease-threat hypothesis, in-group preference correlated with the course of heightened vulnerability to disease in early pregnancy ($r = .15$, $p = .03$). It should be noted, however, that there was only a marginally significant trend linking out-group dislike with early pregnancy ($r = .12$, $p = .07$). Thus, the results provide more support for in-group chauvinism during the first trimester than out-group aversion.

It is not clear that the primary function of increased group chauvinism during pregnancy would be to avert disease-transmission. As the authors acknowledge, signaling favoritism toward one's in-group during early pregnancy may yield multiple benefits attendant to coalition-enhancement which transcend disease-avoidance functions, such as acquiring extra provisioning and protection from allies. However, these alternative benefits should be expected to become, if anything, more useful over time as later pregnancy renders mothers less mobile. Instead, Navarrete and colleagues found that pro-U.S. bias steadily decreased as gestational weeks elapsed. The concentration of bias in early pregnancy therefore lends credence to their disease-avoidance interpretation. A final caveat is in order, however. Manipulations which have elicited exaggerated group favoritism measured with the same "pro-U.S. versus anti-U.S." instrument used in this study have been found to elicit analogously exaggerated ratings of pleasant versus aversive aesthetic stimuli unrelated to group favoritism, following experimental manipulations unrelated to disease or coalitional relations (Holbrook, 2010). This raises the possibility that the cognitive architecture underlying the heightened in-group favoritism effect reported by Navarrete and colleagues may not be dedicated to either coalitional reckoning or disease-avoidance, but rather reflect a slightly heightened sensitivity to positively and negatively valenced stimuli (across various domains) during the first trimester. Further research is needed to establish the discriminative validity of the group chauvinism effect.

"Nesting" activities in advance of parturition may also relate to pathogen-avoidance. Pregnant females across many species create nests for their offspring. Rodents, for example, typically seek the lowest available sheltered location in which to accumulate soft materials into semi-enclosed areas (Broida and Svare, 1983), and domestic cats also seek private, covered locations in which to build nests from surrounding materials (Feldman, 1993). Although no empirical studies have directly investigated nesting behaviors in humans, anecdotal data imply that a preponderance of human mothers experience a comparable impulse. As one health care professional recounted, "Probably 99 percent of the pregnant women I see have it. They stack and restack their baby's clothes, clean their houses like mad, and pack and repack their hospital bag" (Skolnik, 2003). Human parents often undertake large-scale renovation projects and elaborately 'baby-proof' their homes in preparation for the new child (Feygin et al., 2006; Leckman et al., 2004).

Interestingly, progesterone has also been implicated in nesting behavior. In some strains of mice, pregnant or nulliparous females injected with progesterone engage in more intense nest building behavior (Broida and Svare, 1983). Progesterone likely acts on selective regions of the medial preoptic area in nesting mothers, as lesions of the dorsal region of the medial preoptic area disrupt nesting and pup retrieval behaviors in lactating rats, yet leave other key maternal behaviors such as nursing and crouching

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intact (Jacobson et al., 1980). Given the evidence that progesterone mediates pathogen-avoidance (Fleischman and Fessler, 2009), and that contagion posed perhaps the most severe hazard to young in the ancestral past (Hill et al., 2007), maternal nesting behavior may serve primarily to support disease-avoidance. Consistent with the notion that precautionary mechanisms operative during pregnancy might sensitize mothers to potential pathogens, women who develop obsessive–compulsive disorder during pregnancy disproportionately report contamination obsessions and washing or cleaning rituals (Wisner et al., 1999). In addition, nesting may serve other precautionary functions: minimizing the risk of accidents, concealing young from potential attack, and accumulating resources to avert scarcity. Future research might test the specificity of the relationship between nesting and disease-avoidance by experimentally comparing nesting impulses following exposure to pathogenic (e.g. sick baby) versus non-pathogenic (e.g. dropped, attacked, or hungry baby) threat primes.

In sum, offspring are acutely vulnerable to pathogens their mothers may encounter while they are in the womb, particularly during early pregnancy. In line with evolutionary theory, pregnant mothers display adaptive shifts in diet, social preferences and nesting which appear to reduce the risk of disease contraction.

8. Pregnancy and vulnerability to attack

In addition to disease susceptibility, pregnant females are at greater risk of attack by predators or conspecifics due to hampered mobility (Bauwens and Thoen, 1981). The impaired mobility associated with pregnancy has led to the evolution of compensatory behavioral strategies in many species. For example, White-faced Capuchins reduce time spent foraging during pregnancy, as predators are more likely to lurk in food-rich environments (Rose, 1994). These monkeys offset their lost calorie consumption by decreasing energy expenditure, spending more time stationary and in safer areas (Rose, 1994). Pregnant bighorn sheep adopt the opposite strategy, migrating to food-rich areas which are relatively dense with predators. To overcome the compounded risk of attack plus reduced mobility, pregnant bighorns appraise cues of potential predator presence with acute sensitivity, becoming twice as likely as males to flee in response to indications that predators are near (Berger, 1991). Yet another strategy has been observed in pregnant lizards, who engage in more freezing and less fleeing when attacked, ostensibly because their ability to run has been curtailed (Bauwens and Thoen, 1981). To our knowledge, no research has systematically investigated changes in human mobility, foraging behavior or reactions to cues of potential assault during pregnancy. Anecdotally, however, pregnant women often report an aversion to leaving their homes during late pregnancy (Skolnik, 2003). In hunter-gather societies, reduced mobility in association with reproduction has been found to decrease foraging and increase reliance on supplementary provisioning from mates or kin (Quinlin and Quinlin, 2008).

Heightened vulnerability to attack or mobility-related mishaps during pregnancy may have selected for adaptations prompting expectant mothers to affiliate with and derive heightened provisioning and protection from kin (Roberts et al., 2008; Wedekind and Füri, 1997). Olfactory changes during pregnancy, for example, appear to lead women to affiliate with family. The major histocompatibility complex (MHC) is a highly polymorphic gene complex that encodes cell-surface receptors which play a critical role in the initiation of immune responses. Several theorists have argued that preferences for others' body odors are mediated, in part, by the relative genetic similarity or dissimilarity of their MHC (for a review, see Thornhill et al., 2003). In line with this view, normally ovulating women prefer the scents of men who have rare MHC alleles (e.g., Thornhill et al., 2003), arguably because reproducing with highly dissimilar and varied MHC genes yields pathogen-resistant offspring (Penn et al., 2002). Interestingly, women using contraceptive pills, which hormonally mimic early pregnancy, preferred the scents of individuals with similar MHC genotypes to their own (Wedekind and Füri, 1997; Wedekind et al., 1995). Similar results were found by Roberts et al. (2008), who used a longitudinal design to show that initiation of contraceptive pill use was associated with a shift toward preference for the body odors of those with similar MCH genes. Analogous shifts in preferences for the odors of kin during pregnancy have been observed in rodents (Manning et al., 1992). No studies to date of which we are aware have examined the scent preferences and corresponding MCH matches of pregnant women, thus connections between pregnancy and preferences for scents of kin in humans have not yet been directly tested. This is a clear direction for future study; current theories relating MHC to pregnancy should be considered preliminary.

9. Postpartum precautionary preoccupations

Human children are at their most vulnerable during infancy, suggesting that parents should have evolved to disproportionately focus their precautionary expenditures during this time period. Indeed, roughly three-quarters of parents of newborns report extraordinary levels of preoccupation with thoughts of potential harm to their children (Abramowitz et al., 2003a,b). Approximately 95% of mothers and 80% of fathers report recurrent intrusive thoughts of the possibility of harm to their baby in the early weeks postpartum, with little change over the first 3 months (Leckman et al., 1999). The prevailing parental preoccupation themes echo the hazards infants faced in the ancestral past: disease, accidents due to parental neglect, and conspecific attack (Leckman et al., 1999). These thoughts often trigger meticulous cleaning and repeated checking of both the infant and the infant's surroundings (Swain, 2008).

Winnicott (1956) suggested that mothers' borderline obsessive levels of postpartum preoccupation with their infants, which he termed "the primary maternal preoccupation complex," heighten their abilities to anticipate infant needs and interpret cues indicative of potential problems. Although both mothers and fathers typically experience parental preoccupations, mothers exhibit considerably more (Leckman et al., 1999). In a functional imaging study conducted by Swain et al. (2004), for example, interview data showed that mothers were significantly more preoccupied with their infants than fathers, an observation which was echoed by mothers' greater amygdala and basal ganglia activation in response to infant stimuli. From an evolutionary perspective, the primary maternal preoccupation complex may reflect women's greater incentive to invest in offspring (Trivers, 1972).

In an imaging study of postpartum parental worry as a function of vaginal versus Cesarean birth method, Swain et al. (2008) compared mothers' brain responses to the sounds of their infant crying at 2–4 weeks after delivery. Mothers in the vaginal birth group exhibited significantly more reactivity to the sounds of their infants' cries in alarm centers (e.g. amygdala) and behavioral response selection circuits (e.g. caudate, thalamus, hypothalamus). As the researchers hypothesized, the influx of oxytocin release accompanying vaginal delivery may account for the observations of heightened sensitivity to the sounds of their infants crying. These results should be approached with caution, however, as the sample of women who had Cesarean births elected to do so for their personal convenience, a potentially serious confounding factor which might explain the differences observed. Independent of mode of delivery, mothers' self-reported levels of worry about their infant's welfare positively correlated with activations in the left and right

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lenticular nuclei upon exposure to cry, consistent with the function of these regions in a range of repetitive anxious thoughts and behaviors (Mataix-Cols et al., 2004).

The neurobiological mechanisms discussed in Sections 4–6 appear likely to subserve the acute motivation to protect children from potential harm observed during the postpartum period. The proximal means by which modern parents' preoccupations come to echo threats common in the ancestral past, however, remain unspecified. It is likely that concerns over disease and accidents are largely culturally preserved and transmitted, given the continuing risk to children posed by these hazards in the industrialized world today (Anderson and Smith, 2005; UNICEF, 2001). The disequilibrium between the threat posed by strangers in the environments in which humans evolved and the marginal threat strangers pose in the contemporary modern world, however, raises the possibility of an innate representation of strangers as a source of threat (Hahn-Holbrook et al., 2010a). In the following section, we discuss mechanisms which may have evolved to enable mothers to confront conspecifics who posed a potential hazard to their offspring during the postpartum period.

10. Postpartum maternal defense

The term *maternal defense*, also referred to as *maternal aggression* or *lactation aggression*, describes a period of heightened aggression toward non-kin conspecifics and predators which typically coincides with the course of lactation (see Lonstein and Gammie, 2002, for a review). Maternal defense qualifies as a precautionary behavior insofar as lactating mothers frequently aggress against conspecifics whose presence implies potential threat but who pose no imminent hazard to offspring (Elwood et al., 1991; Flannelly and Kemble, 1988). Maternal defense has been documented in rats and mice (Lonstein and Gammie, 2002), prairie voles (Bales and Carter, 2003), hamsters (Ferris et al., 1992), lions (Grinnell and McComb, 1996), deer (Smith, 1987), domestic cats (Schneirla et al., 1963), rabbits (Ross et al., 1963), squirrels (Taylor, 1966) and domestic sheep (Hersher et al., 1963). Among primates, lactating Japanese and Rhesus Macaques display more aggression than females at any other reproductive stage (Maestripieri, 1994; Schino et al., 2004; Troisi et al., 1988).

Despite the prevalence of lactation aggression in other mammals, the topic has only recently received empirical attention in humans. To test whether lactating mothers display heightened levels of aggression, Hahn-Holbrook et al. (2010b) compared groups of breastfeeding, formula-feeding and nulliparous females' willingness to deliver aversive sound bursts to an unduly hostile confederate in a competitive game based on an aggression paradigm developed by Bushman (2002). Aggression was operationalized as the combined volume and duration of sound bursts. As predicted, breastfeeders produced significantly more aggressive retaliatory sound bursts than either formula-feeders or nulliparous women.

Given the significant risk hostile conspecifics appear to have posed to offspring in the ancestral past (Hahn-Holbrook et al., 2010a), selection should have favored maternal defense to preempt potential assault in humans. Indeed, aggressive maternal defense behaviors in nonhuman animals are routinely elicited by cues of potential rather than imminent threat, such as the mere presence of an unrelated conspecific in the vicinity of the nest (Elwood et al., 1991; Flannelly and Kemble, 1988). In Hahn-Holbrook and colleagues' study of lactation aggression in humans, however, the aggressive confederate was actively rather than potentially hostile. In addition, the infants in the study spent most of the time in an adjacent room and were not present during the competitive game during which aggression was measured. Consequently, follow-up studies are needed to ascertain whether breastfeeding mothers display still more heightened aggression to deter conspecifics who are framed as potential offspring threats. Oriented within the security motivation framework, lactating mothers may appraise threatening conspecifics as more potentially hazardous to young but simultaneously less formidable as opponents. Imaging methods might be employed to test this hypothesis by comparing the amygdala reactivity of lactating mothers exposed to images of aggressive, unfamiliar men approaching the mothers versus images of such men approaching their infants.

The neurobiological mechanisms underlying maternal defense are not well understood. Studies of rodents species implicate a number of brain regions (see Lonstein and Gammie, 2002, for a review). For example, Gammie and Nelson (2001) compared lactating mice who displayed maternal defense with those who did not using two indirect markers of neural activity (cFOS and pCREB). Maternal defense was associated with increased expression of cFOS in the claustrum, bed nucleus of the stria terminalis, medial preoptic area, paraventricular nucleus, medial amygdala, and cortical amygdala; pCREB expression was increased in the ventrolateral septum and the ventrolateral caudal area. As the authors noted, these regions may be required to operate in concert to produce maternal aggression, but the potentially unique contributions of subcomponents are difficult to parse considering their dense interconnectivity. Thus, it is unclear whether increased cFOS or pCREB expression in each area was related to sensory input from the intruder or pups, or motor output necessary to produce maternal aggression.

Serotonin, GABA, and estrogens have all been linked to maternal defense. For example, injection of the serotonin 5-HT1 receptor agonist, fluprazine, decreases maternal aggression in mice, consistent with serotonin's role in signaling motivational satiety (Parmigiani et al., 1989). Hansen et al. (1985) observed that administrations of GABA antagonists reduced maternal aggression in lactating rats, who derive high levels of central GABA release and metabolism from pup suckling. Other researchers, noting that suckling suppresses gonadotropin release and thereby inhibits ovarian release of estrogens, have proposed that low estradiol levels may underlie lactation aggression. In line with this view, ovariectomizing pregnant mice hastens the onset of maternal aggression, whereas estrogen replacement delays the onset of maternal aggression (Ghiraldi et al., 1993).

Other biological models propose that lactation disinhibits aggressive behaviors toward potentially threatening conspecifics by triggering the release of stress-attenuating hormones (i.e., oxytocin and prolactin) which reduce mothers' fear to attack (Gammie et al., 2008; Hansen and Ferreira, 1986). Prolactin and oxytocin facilitate milk ejection and production, respectively, in response to infant suckling (Carter and Altemus, 1997). Several lines of evidence indicate that both hormones also reduce anxiety. Experimentally elevating prolactin in virgin female rats to mimic postpartum levels reduces neuronal, hormonal and behavioral responses to stress (Donner et al., 2007). In humans, women with higher plasma prolactin levels four days postpartum report less anxiety (Nissen et al., 1998). Likewise, women who possess higher plasma oxytocin levels four days postpartum report less anxiety (Nissen et al., 1998), and basal levels of oxytocin negatively correlate with anxiety (Uvnäs-Moberg et al., 1990). Oxytocin inversely correlates with stress hormone levels in the blood plasma of human mothers during suckling (Chiodera et al., 1991). In a neuroimaging experiment, Kirsch et al. (2005) intranasally administered either oxytocin or a placebo to human males, then measured brain reactivity in the amygdala while exposing participants to threatening stimuli (e.g. angry male faces). As predicted, oxytocin administration significantly depressed activation of the amygdala in response to threat cues.

Considerable research demonstrates the fear-buffering effects of lactation (for a review, see Mezzacappa, 2004). Numerous

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studies of lactating rodents document relatively depressed sympathetic reactivity in response to an array of stressors: the acoustic startle paradigm, the open field test, the elevated maze, the defensive burying paradigm, the punished drinking paradigm, and the light/dark choice test (Ferreira et al., 1989; Fleming and Luebke, 1981; Hard and Hansen, 1985; Lonstein and Gammie, 2002; Maestripieri and D'Amato, 1991). Lactating rodents also display dampened responsiveness to corticotropin-releasing hormone (for a review, see Gammie et al., 2008). For example, in comparison to non-lactating dams, lactating rats injected with corticotropin-releasing hormone show decreased stress-related neural activity and less acute startle response (de Costa et al., 1997). Converging research in humans also supports the stress-reducing properties of lactation (see Mezzacappa, 2004, for a review). For example, Mezzacappa et al. (2005) compared exclusively breastfeeding, exclusively formula-feeding, mixed-feeding and non-maternal participants' cardiovascular responses during mental arithmetic and cold pressor stressor tasks. Exclusive breastfeeders displayed less heart rate increase and less shortening of the milk pre-ejection period (an indicator of stress) in response to mental arithmetic, and the frequency of everyday breastfeeding negatively correlated with heart rate in response to both stressor tasks. A number of other studies report converging correlations of breastfeeding with greater calm, more positive moods and less anxiety in comparison to formula-feeding (Altshuler et al., 2000; Carter and Altemus, 1997; Fleming et al., 1990; Groer, 2005).

Fear has been found to reduce aggression in rodents, typically impelling flight or freezing behaviors instead (Boccia and Pedersen, 2001; Erskine et al., 1978; Maestripieri and D'Amato, 1991). Human females tend to experience more fear than males in physically aggressive contexts, which may account for sex-differences in physical aggression (Campbell, 2006). Consistent with the hypothesis that decreased fear facilitates the increases in aggression which coincide with lactation, breastfeeders in the study conducted by Hahn-Holbrook et al. (2010b) had significantly lower systolic blood pressure reactivity to the aggressive encounters compared to baseline than formula-feeders or nulliparous women. Further, systolic blood pressure during the aggressive encounters inversely correlated with aggression in the sample as a whole. These findings support the hypothesis that lactation proximally mediates human maternal defense, at least in part, by attenuating stress.

Oxytocin levels, which are linked with stress-reduction, have been directly correlated with maternal defense in nonhuman animals (for a review, see Campbell, 2008). For example, Bosch et al. (2005) measured oxytocin release before, during and after interaction between a rat dam and a female intruder, finding that oxytocin release positively correlated with aggressive behavior. Interestingly, the researchers also found that highly anxious dams released higher levels of oxytocin within the central amygdala and the paraventricular nucleus—key brain areas related to the regulation of fear—during maternal defense. In a follow-up study, the researchers blocked oxytocin receptor sites within the central amygdala and the paraventricular nucleus, observing that blockade of either site reduced maternal aggression. Conversely, Bosch's team found that local infusion of oxytocin within either the amygdala or paraventricular nucleus increased maternal attacks. As supportive of the role of oxytocin in maternal defense as these findings may appear, it is important to note that other rodent studies have reported null or even inhibitory effects of oxytocin on maternal defense, possibly resulting from testing with incommensurate procedures (Campbell, 2008). Studies of the influence of prolactin on maternal defense have consistently produced null or contradictory results, which is somewhat surprising given that elevated postpartum prolactin levels correlate with the period of heightened maternal aggression (for a review, see Lonstein and Gammie, 2002).

Researchers have only recently begun to investigate the relationship between oxytocin and aggression in humans, likely because the well-known function of oxytocin in promoting parenting and social attachment has discouraged psychologists from extending the link between oxytocin and aggression to humans. The influential "tend and befriend" model of oxytocin, for example, predicts that oxytocin would actually decrease aggressive behavior, instead promoting "tending" to offspring and "befriending" of potential allies to increase the mothers' probability of survival (Taylor, 2006). Consistent with the tend and befriend approach, Lee et al. (2009) found an inverse correlation between oxytocin levels in cerebrospinal fluid and self-reported life history of aggression. However, their self-report measure of aggression was unlikely to capture instances of aggression in defense of offspring or kin, which are often categorized separately from more common forms of offensive aggression. Alternatively, Campbell (2008) advocates the "specific attachment model," which portrays oxytocin as facilitating affiliation with offspring, mates and kin, such that oxytocin may intensify aggressive behavior on their behalf when threatened. In support of the specific attachment view, De Dreu et al. (2010) intranasally administered oxytocin or placebo to randomly assigned groups of males and observed increased aggression in defense of the experimentally defined in-group among participants who received oxytocin. On the basis of this result, De Dreu and colleagues advise updating the popular "tend and befriend" slogan to "tend and defend," a phrase with self-evident applicability to maternal defense and other forms of parental precaution. Further research focusing on females and measuring naturally released oxytocin is required to clarify the role of oxytocin in maternal defense in human.

11. Characterizing adaptations for parental precaution

We have reviewed evidence of numerous shifts in parental sensitivity to cues of potential threat. During pregnancy, for example, mothers evince preferences for foods which are less likely to carry pathogens, faces which appear healthy, kin odors and in-groups, all of which have been argued to adaptively reduce the chance of catching diseases which might seriously harm the gestating fetus. In addition, enhanced kin and in-group favoritism during pregnancy may function to recruit allies for defense against hostile conspecifics or predators, as well as provide access to shared material resources. Later, during the postpartum period, parents display seemingly adaptive preoccupations with averting threats of disease, accidents and stranger violence. This overall pattern of adaptive parental shifts across multiple domains raises complex questions about the evolutionary origins of the underlying psychological mechanisms. Which shifts reflect psychological adaptations specifically engineered by natural selection for these functions, and which reflect serendipitous benefits of traits which evolved for other reasons? Fitness-enhancing effects may arise via by-products of adaptations designed for other functions, or even from by-products of other by-products (Buss et al., 1998; Thornhill, 1997). To qualify as a candidate adaptation for a given function, a mechanism must exhibit design attributes precisely corresponding to that function and not to others, like a key fitting a lock (Tooby and Cosmides, 2005; Williams, 1966). To ground consideration of these issues, we present recent evidence that a brain region linked with detecting conspecific-borne pathogens is modulated by oxytocin, then examine whether a hypothetical increase in oxytocin in this area which helps mothers avoid unhealthy individuals would qualify as a parental adaptation.

Oxytocin is important for social recognition of familiar conspecifics and kin in rodents (Kavaliers et al., 2005). Consistent with the role of oxytocin in social cognition and the notion that the

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medial amygdala is dedicated to processing socially relevant cues, the highest levels of oxytocin receptor gene expression observed in the mouse amygdala have been found within the medial amygdala (Gould and Zingg, 2003). In rodents, the odors of parasitized conspecifics, as well as familiar conspecifics or kin, have been found to activate the medial amygdala (Kavaliers et al., 2005; Meredith and Westberry, 2004). Oxytocin appears integral to detecting and avoiding diseased conspecifics in rodents, and oxytocin-mediated sensitivity to pathogens in conspecifics appears related to social recognition, not an indiscriminate boost in olfactory perception or memory abilities, as oxytocin knock-out rodents have been found equally able to detect and avoid predator (i.e. cat) odors and to learn to associate novel odors with predator odors (for a review, see Kavaliers et al., 2005).

To summarize, the rodent data support the existence of an oxytocin-modulated region of the amygdala used in the detection of kin, familiar conspecifics, and diseased conspecifics. Suppose, for the sake of argument, that oxytocin were found to mediate sensitivity to cues of ill health in fellow humans by acting within the medial amygdala, and that mothers were better able to avoid pathogens as a result of perinatal increases in oxytocin. Would the perinatal upsurge of oxytocin in the medial amygdala constitute a maternal adaptation for avoiding diseases? As meticulously crafted as such a mechanism would seem, it would not be clear that parenting-related increases in oxytocin within the medial amygdala were designed to accentuate mothers' detection of sick people. Oxytocin increases in this region would likely yield adaptive effects on aspects of parental social behavior transcending disease-detection, such as bonding with offspring or affiliating with kin or social allies. Therefore, a hypothetical increased sensitivity of mothers to pathogenic individuals mediated by increased oxytocin action in the amygdala might be better interpreted as a fortuitous by-product (e.g. of a parental adaptation to promote bonding) or as a token of a broader function (e.g. of a parental adaptation to enhance social recognition, including kin, familiars, and those who are ill). On the other hand, if further research were to reveal that perinatal oxytocin increases in this area somehow mediate heightened disease-detection but not social recognition or bonding, such findings would provide convincing evidence of a domain-specific parental precaution adaptation dedicated to disease-avoidance.

We have emphasized the possible evolved functions of the mechanisms which subserve various forms of parental precaution throughout this review. Importantly, and as the example above illustrates, the question of whether these mechanisms are specialized adaptations designed for specific purposes of parental precaution requires further evidence. Looking forward, we may indeed discover adaptations geared toward domain-specific forms of parental precaution. For example, considering the correspondence between pregnancy sickness and the period of offspring vulnerability to disease, the evident discriminativity of nausea and vomiting to the domain of food consumption, and the tendency for mothers with pregnancy sickness to prefer safer foods and have healthier child outcomes, pregnancy sickness is a good candidate for an adaptation specific to parental precaution against food-borne pathogens. Alongside specialized parental adaptations, mechanisms evolved to serve more general security and affiliative functions may be efficiently co-opted to generate adaptive parental precautionary effects.

12. Conclusion

In the environment in which our species evolved, offspring were severely threatened by congenital defects related to diseases contracted during gestation. Of those who survived birth, children were disproportionately vulnerable during the first year of life (Hill et al., 2007). The most severe postpartum threats appear to have been disease, accidents and conspecific attack (Hahn-Holbrook et al., 2010a; Hill et al., 2007). Today's empirical picture reflects these ancestral challenges. Pregnant mothers demonstrate a heightened aversion to potential sources of pathogens, postpartum parental preoccupations with infant safety revolve around disease, accident and conspecific attack, and concerns with preventing harm to young are most salient during the early months following birth.

In addition to outlining the evolutionary logic of parental precaution, we have also advanced plausible proximal mechanisms which may mediate some of these adaptive behaviors. Szechtman and Woody's (2004) proposed security motivation system provides a promising neurobiological framework within which to situate precautionary parental behaviors, but parental precaution appears to require the integral contributions of reward systems as well. The theoretical role of oxytocin in both maternal bonding and maternal defense, for example, illustrates the intersection of affiliation and security concerns in enabling parental precaution. Healthy levels of precautionary anxiety over potential infant threat require affiliative motivation to incentivize offspring protection, focus attention on offspring cues, and temper the potentially debilitating levels of stress associated with assuming parental responsibility. Looking ahead, the interactions between parental affiliation and security motivation systems present a general area demanding further research (see Swain, 2008). Ultimately, understanding parental precaution will require psychobiological models which integrate the motivational circuitry underlying both love and worry.

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