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ARTICLE

Applying Socioendocrinology to Evolutionary Models: Fatherhood and Physiology

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Owing to humans' unique life history pattern, particularly comparatively short interbirth intervals, early weaning, and prolonged support of multiple dependents, human females have greater reproductive value and higher lifetime fertility, on average, than do their Great Ape counterparts.[1–4] As hominin females began weaning their young early and "stacking" dependents of various ages, they must have had cooperative allomaternal care partners already in place or been successful at concurrently soliciting help to ensure a high rate of survival of their offspring.[1–6] Following Hrdy, I define allomaternal care (and its derivatives, such as "allomothers" and "allomothering") as "care from anyone other than the mother," which thus encompasses a wide range of individuals, including fathers.[7] Who the likely allomother candidates mothers were and what form that cooperation took remain intriguing, difficult-to-answer questions, which are limited, in some capacity, by the lines of evidence available to us. Here, I present a framework for the ways in which we can integrate neurobiological-endocrine and social-behavioral data ("socioendocrinology")[8] to contribute to this dialogue in terms of evaluating fathers' roles.

Although the nature and extent of mother-father cooperation varies within and across societies, human males and females often combine their efforts to raise offspring. This has led scholars to posit an evolutionary origin for humans' propensity to form prolonged female-male social and economic bonds.[6,9,10] The notion that invested human fathering comprises a derived set of characteristics stems from the absence of meaningful paternal care in our closest relatives, the Great Apes, and a similar dearth among the Old World monkeys (Cercopithecoids). The dichotomous distinction of humans as a species with paternal care versus most other Old World primates as species in which paternal care is absent probably is overly rigid, since many of the latter show behavioral plasticity in terms of their willingness to engage with and respond to infants. That said, the high ceiling of human fathers' physical caregiving, in combination with their often extensive contributions to the familial energetic economy via provisioning, has no direct analogy among Old World primates.[3–6,9,10]

In part related to the facultative expression of human paternal care, scholars have recently proposed "cooperative breeding" models, which have greatly expanded the scope of this discussion, taking it beyond a relatively narrow focus on the nuclear family. Using diverse lines of evidence, these models highlight the complex roles played by grandmothers, other kin (especially females), and siblings in assisting mothers with direct caring for young, provisioning, and other forms of economic-familial productivity.[1–3] While models vary in the extent to which they tend to deemphasize the importance of adult males, they have brought our attention to cross-cultural data, showing that fathers can vary widely in terms of their reliability as partners and caregivers. These observations have raised significant questions as to whether fathers, or adult males, generally, were particularly critical to the emergence of humans' life history profile and, if so, in what ways men may have invested in their offspring.[1–3,5,6]

Along with some of my colleagues, I have argued that cross-species evidence demonstrates that male reproductive physiology is shaped by natural selection to respond to parenthood and the needs of offspring when biparental care evolves. If human males played a significant parenting role in the course of hominin evolution, we would expect their biology to have undergone selection to attune their physiological systems to father-child interaction and the demands of parenthood.[5,6,8] Drawing heavily on a cross-species comparative perspective, I review the state of knowledge regarding four neurobiological-endocrine pathways, oxytocin (OT), vasopressin (AVP), prolactin (PRL), and testosterone (T), which are germane to human sociality, particularly partnering and parenting, and discuss their relevance

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to evolutionary perspectives on the biology of fatherhood.

LIFE HISTORY TRADE-OFFS AND PHYSIOLOGICAL MECHANISMS

Theoretically, for species in which parental investment has evolved, organisms face trade-off decisions in terms of how to partition reproductive effort (the fraction of total energetic expenditure devoted to reproduction) between current and future parental investment and parenting versus mating.[5,8,10,11] For primate males, behaviors that appear ostensibly as "parenting" might be mating effort; alternatively, the behavioral repertoires of the two aspects of reproductive strategy might overlap so much that distinctions become challenging.[12,13] However, when life history trade-offs between mating/competing and parenting/nurturing do occur, such life history allocation "decisions" are mitigated physiologically.[14]

Applying cross-species comparative and evolutionary perspectives to these systems helps us conceptualize the ways in which selection organizes and refines functional biological pathways as novel demands arise and life history tactics and trade-offs are modified.[14] This framework provides an empirical basis for the development of testable hypotheses regarding the relationships between socioecological selective pressures, the evolution of life history strategies, and mechanistic physiological function. As a primer for the relevance of this approach to human socioendocrinology and hominin evolution, I will briefly describe a well-characterized animal model that demonstrates the linkages between neuroendocrine mechanisms, sociality, and life history strategies.

Oxytocin (OT), Arginine Vasopressin (AVP), and Vole Social Behavior

Much of our current state of knowledge about the neurobiological pathways thought to underlie pair bonding comes from comparisons of OT and AVP between closely related rodent species: montane and meadow voles (generally promiscuous species with maternal care) compared to prairie and pine voles (generally monogamous or pair-bonded species with biparental care).[15] Over decades of research, scientists have shown that these species vary substantially in OT and AVP receptor locations and densities throughout key brain areas related to sociality (mating, bonding, and parenting), with at least some of these differences already apparent at birth.[15,16] These species also differ in DNA sequencing for a critical AVP receptor, AVPR1A, which experimental results suggest might contribute to the observed variability in receptor expression in the brain and pair-bonding behaviors across these species.[17] Prairie vole males also showed alterations in AVP release and production after becoming fathers, patterns that were not mirrored by the nonpaternal montane vole.[15,16] Thus, predictably, there is evidence that selection has affected existing neurobiological substrates to produce divergent reproductive behavioral phenotypes in closely related species.

Because human paternal care, if it evolved, is a derived characteristic, similarities between human paternal socioendocrinology and that of species in other taxa likely represent convergent evolutionary processes.[5,6,19]

In the vole example, behavioral ecological and laboratory research demonstrated that the species had diverged in their mating and parenting behaviors, providing impetus for a search for neurobiological-endocrine pathways that accommodate these variable life history strategies.[15] If we extend this model to the question of human paternal care and its evolutionary heritage, we have to reorient the framework. Because we know the physiological mechanisms that underlie the transition to fatherhood and paternal behavior in diverse species among which paternal care is known to have evolved (that is, "the biology of fatherhood"), we have a foundation to test whether similar selective pressures have shaped human male physiology.[18,19]

Because human paternal care, if it evolved, is a derived characteristic, similarities between human paternal socioendocrinology and that of species in other taxa likely represent convergent evolutionary processes.[5,6,19] There is little reason to expect human male physiological systems to respond to fatherhood and nurturant paternal care unless such behaviors were sufficiently expressed and advantageous to reproductive fitness during our evolutionary past.[5] When supported with ample cross-species evidence, along with considerations of cross-cultural variation and biobehavioral plasticity, this framework can help inform our models of cooperative breeding and allomothering in human evolution.

Prolactin (PRL)

Although PRL is fundamental to lactation and, given proper steroid hormone (estrogen, progesterone) priming during pregnancy, influences the expression of mammalian maternal behaviors, it evolved early in the vertebrate lineage, in advance of the first mammals. It serves enormously diverse physiological purposes across vertebrate taxa, including: osmoregulatory (fish and amphibians), somatotropic (reptiles, amphibians, fish), metamorphic or developmental (amphibians), and reproductive (fish and amphibians) effects.[20,21] PRL is similarly pleiotropic among mammals and birds.[21] The totality and range of functions it serves across vertebrates indicates that this hormone can be flexibly coopted to serve novel physiological and behavioral functions as environmental conditions change and new niche opportunities emerge.[21]

As is well known, the phylogenetic history of a species can influence or constrain what novel traits are possible or likely to emerge through the process of natural selection. In other words, species are not a blank slate on which selection operates; a shared evolutionary history between taxonomic groups increases the likelihood that similar adaptations will evolve in parallel as species independently converge on common niches. Hence, given PRL's lability and other relevant neurobiological-endocrine commonalities across vertebrate taxa, it is perhaps unsurprising that cross-species evidence indicates that PRL has independently evolved to promote paternal investment in three separate lineages (fish, birds, mammals), providing the impetus to test the hypothesis that similar neuroendocrine substrates were selected for in the hominin lineage.[5,6,21]

Approximately 90% of bird species show biparental care, and the roles of PRL in diverse forms of avian parental care have been explored. Although there are obvious differences in life history pace between birds and humans, generalized similarities in prolonged mother-father dual investment provide a basis for analogies between paternal PRL in birds and its potential role in human fatherhood.[6,21] Critically, elevated avian paternal PRL seems to be linked to various direct and indirect investment behaviors, differing by species and not being restricted to a narrow range of care. For example, male birds often show elevated PRL before or during periods in which they physically care for young (for example, when incubating eggs) as well as when they contribute to provisioning. In addition, some seabird males maintain high PRL during long periods of absence from their partners and young, which might increase the likelihood of males returning and performing care when they do. The diverse modes of PRL stimulation and maintenance in birds, together with its seeming facilitatory effect on indirect forms of male care (such as provisioning) are useful models for future studies of human fathers.[21] Research to date has largely linked human paternal hormones, particularly testosterone, to hands-on, physical care behaviors.[22–25] The importance of such behaviors might be underappreciated in evolutionary models of hominin paternal care and cooperative breeding, but paternal contributions to the familial economy and other forms of indirect care are comparatively more common cross-culturally.[5,6,9,10] The physiological correlates of such parental behaviors are understudied in both human mothers and fathers.

Compared to the literature on birds, far fewer studies have explored connections between PRL and mammalian male behaviors, but results are consistent with a role for PRL in promoting paternal care. For example, in multiple rodent species in which males help care for young, fathers' PRL was higher after the birth of their offspring.[26] Among New World monkey species with biparental care, experienced fathers had higher PRL than did inexperienced males (cotton-top tamarins)[27] and fathers showed elevated PRL during the postpartum period and after carrying infants (common marmosets).[28,29] However, experimental models in which fathers' PRL is suppressed have yielded mixed results in terms of its imperativeness for marmoset paternal care.[30]

There has been little research on human paternal PRL. The first study to compare PRL based on men's parenting status found no differences between fathers and nonfathers in Jamaica.[31] In contrast, my colleagues and I recently showed that Filipino fathers had higher PRL relative to nonfathers, with fathers of infants ($\leq$ 1 yr old) having the most elevated PRL. Among nonfathers, partnering status and romantic commitment levels had no independent effect on men's PRL. These findings are broadly consistent with the idea that PRL might have a facilitatory effect on human paternal investment, converging with the patterns from the taxa discussed earlier.[25] Data from other cultural settings also suggest that PRL promotes a nurturant paternal phenotype. Canadian fathers with higher basal PRL reported more concern when hearing infant cries than did lower PRL men, and Israeli fathers with greater PRL engaged in longer bouts of exploratory play with their infants during observations.[32,33] Some potential inconsistencies between "baseline" PRL and its acute responsiveness have emerged from other research designs. Specifically, two studies found short-term (within 20-30 min) declines in PRL during father-child interaction.[34,35]

Importantly, since we still have relatively few data on PRL and human fatherhood, future studies need to explore how the patterns linking elevated PRL to fatherhood and specific forms of paternal care or sensitivity vary across cultural boundaries and between individual fathers. It remains unknown what neurobiological pathways facilitate upregulation of fathers' baseline PRL production through time; that is, a systematic adjustment to the day-to-day homeostatic regulation of PRL. During lactation, mammalian mothers' dopamine neurons (which primarily regulate PRL) are less responsive to negative feedback from PRL, enabling mothers to maintain very high PRL and otherwise normally functioning dopamine circuits.[36] The recalibration of maternal dopamine and PRL pathways during lactation represent an elegant exemplar for the ways in which physiological set points and feedback loops are recalibrated based on life history status. It remains to be seen whether similar circumstance-specific, impermanent neurobiological-endocrine mechanisms might exist in fathers, such as during the infancy period.[21]

We also know very little about other, more generalized socioendocrine roles of PRL. For example, men and women both show short-term (up to an hour) PRL spikes after orgasm. My colleagues and I found that nonfathers with higher PRL reported more recent and lifetime sexual partners, which ran counter to clinical and experimental data indicating that PRL can dampen sexual behavior or libido.[21] Humans also exhibited PRL increases after positive interactions with dogs.[37] Thus, PRL might have implications for diverse forms of sociality and bonding beyond

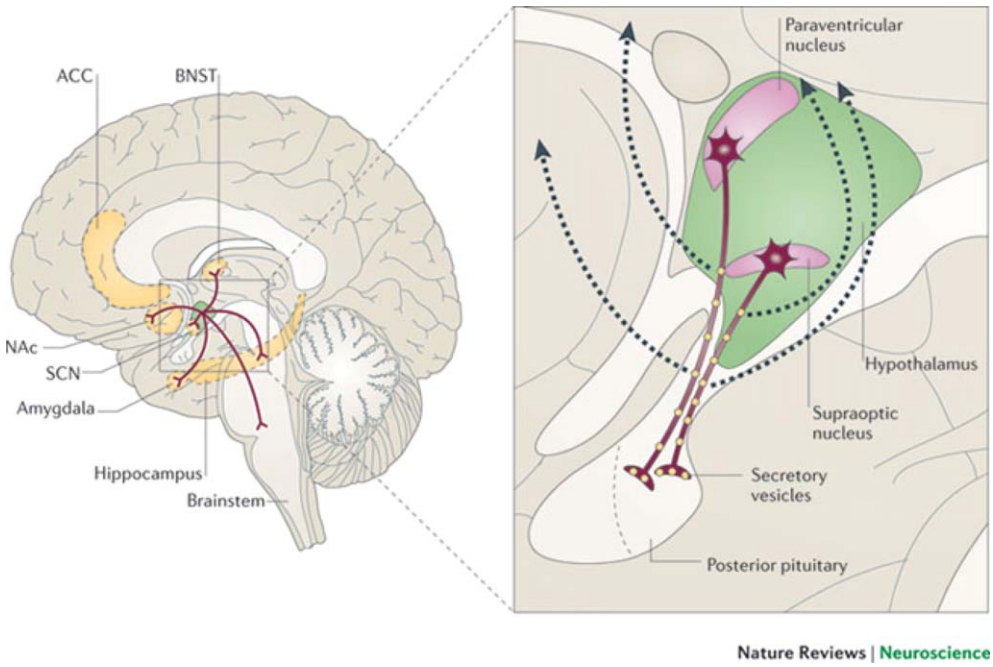

Figure 1. **Oxytocin (OT) and Arginine Vasopressin (AVP) neurobiological-endocrine pathways**. Legend adapted from Meyer-Lindenberg and colleagues.[122] OT and AVP are synthesized in neurons of the hypothalamus and processed along axonal projections to the posterior lobe of the pituitary. There, they are stored in secretory vesicles and released from the posterior pituitary into peripheral circulation (inset). In addition, OT and AVP are released into the extracellular space, resulting in not only local action. but also diffusion through the brain to reach distant targets (dotted arrows). Furthermore, smaller neurons in the hypothalamus also produce OT and AVP and project directly to other regions in the brain. Both peptides have peripheral and central functions. However, the central and peripheral releases are not necessarily associated. The distribution of OT and AVP receptors in the human brain has not yet been fully explored. ACC, anterior cingulate cortex; BNST, bed nucleus of the stria terminalis; NAc, nucleus accumbens; SCN, suprachiasmatic nucleus. Reprinted by permission from Macmillan Publishers Ltd: (Nature Reviews Neuroscience) (Meyer-Lindenberg et al., 2011), copyright (2011). (Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.)

parenting. More expansive research on the hormone's broad socioendocrine functions (allomothering, partnering, friendship) will help us determine the extent to which PRL has effects specific to paternal psychobiology.

Oxytocin (OT) and Arginine Vasopressin (AVP)

OT and AVP are closely related neuropeptides that act as neurotransmitters or neuromodulators when released by hypothalamic neurons located centrally in the brain and as hormones when released into the peripheral circulation by hypothalamic neuronal projections extending to the posterior pituitary (Fig 1, Box 1). Both peptides represent intriguing possibilities as socioendocrine signals that may interface with paternal behavior because of their wide-ranging social effects across vertebrate taxa (nonmammals express homologous peptides).[15]

While OT plays critical reproductive roles in mammalian females, including initiating labor, contributing to lactation, and facilitating maternal behavior, its role in paternal behavior is poorly characterized.[20] A study of prairie voles (biparental care) indicated upregulation of hypothalamic OT among new fathers. However, in a separate rodent model with contrasting results, California mice males showed prolonged elevation of peripheral OT following pair-bond formation, but levels decreased late in their partners' pregnancies. Fathers' caregiving did not differ based on OT.[38,39] Among nonhuman primates, it was recently found that experienced common marmoset fathers had higher hypothalamic OT secretion than did nonfathers,[40] potentially indicating regulation of OT specific to fatherhood and independent of more generalized social roles for the peptide. In a separate study, central OT administration to marmoset fathers increased their willingness to share food with their young.[41]

Much of our existing knowledge on OT in human fathers derives from longitudinal research in Israel that has demonstrated bidirectional relationships between peripheral OT and paternal nurturant behaviors (but see Box 1 for discussion of the limitations of peripheral and intranasal OT). Specifically, fathers with higher baseline OT engaged in greater stimulatory physical play behaviors with their children, while men performing more tactile exploratory play with their infants showed larger short-term (~15 min) OT spikes than did other fathers.[32,42] Mothers and fathers had comparable absolute OT levels and exhibited equivalent stability in OT over multiple years and fathers' OT increased over the first six months postpartum in a similar fashion to maternal OT. Parents' OT was also positively correlated (that is, fathers with higher OT had partners who likewise had

Box 1. Methodological Concerns Regarding Measurement of Human Peripheral Oxytocin (OT) and Arginine Vasopressin (AVP) and Intranasal Administration of the Peptides

Although studies assessing endogenous OT and, to a lesser extent, AVP in humans are increasing, there are three principal methodological challenges in assessing the peptides' effects on human social behavior. First, the release of OT/AVP in the brain (centrally) and into the peripheral circulation (Fig. 1) is variably synchronized; that is, these separate neuronal sources of the peptides do not uniformly act in concert. Rather, the release of OT/AVP in the brain, in the peripheral circulation, or simultaneously in both varies by stimuli. Consistent with this idea, recent human studies show no correlation between cerebrospinal fluid (central) and plasma (peripheral) OT/AVP, though the former is an imperfect method to assess the immediate, localized actions of these peptides in the brain in response to specific stimuli.[117,118] Second, the peripherally released peptides do not readily cross the blood-brain barrier, meaning the hormone levels we can access in blood, saliva, and urine can exert no direct effect on the brain. Third, the release of OT/AVP from central neuronal populations is likely the principal mechanistic pathway through which the peptide affects sociality (rather than through peripheral endocrine effects of the hormones).[118,119] In total, the measurement of peripheral OT/AVP, as is almost universally done in human studies, might not provide direct insight into what stimuli elicit central release of these peptides and how they exert psychobiological effects via actions in the brain. Human psychobiology research can partially mitigate these concerns by acknowledging the diversity of neurobiological-endocrine pathways that can contribute to peripheral release of OT/AVP or can be affected by peripheral OT/AVP with subsequent effects on psychobiology.[118] In human studies with null findings (no correlations between the peripheral peptides and behavior/cognition) a role for OT/AVP cannot be ruled out, given the decoupling of central and peripheral OT/AVP, limiting the utility of the latter. These considerations are critical if the long-term goal of this research paradigm is to define causal pathways.

These concerns are less robust in nonhuman animal models in which more invasive techniques can be used to assess endogenous central OT/AVP production, release, and binding, and in which OT/AVP can be injected directly to brain sites, but still should be kept in mind when evaluating studies of human sociality and OT/AVP.[118,119] A growing number of studies are attempting to overcome these issues in humans by administering intranasal OT and AVP, based on evidence that AVP and some other protein hormones can exploit a weakness in the blood-brain barrier in the human nasal cavity that allows access the brain's ventricular areas and cerebrospinal fluid.[120] To my knowledge, the extent to which intranasally administered OT/AVP can access brain areas relevant to sociality, such as the limbic system or prefrontal cortex, remains unexplored, particularly in humans, although there is evidence from rodent models that other intranasally administered proteins can access such brain regions.[118,121] Though often glossed over, these are critical methodological limitations. The assumption that intranasal OT/AVP acts directly on the human brain is supported only by correlational evidence (especially for OT). Experimental verification is required to ensure that the effects of intranasal OT and AVP do not occur through peripheral pathways. Yet these experimental approaches are intriguing. Well-designed studies indicate that intranasal administration of OT/AVP can substantially impact psychobiology, even if the specific physiological pathways remain murky.

elevated OT) and between both parents and their toddlers, while parents with higher OT reported greater attachment to their infants.[43–45] In an experimental model, fathers who received intranasal OT exhibited more encouragement, social reciprocity, and affectionate touch with their infants than did those given a placebo.[46] However, fathers given OT before a socially challenging parent-infant paradigm showed elevated cortisol as compared to themselves with placebo. Fathers' heightened stress- or challenge-based cortisol response when receiving OT highlights the likelihood that OT enhances the salience of social stimuli rather than acting as a uniform prosocial neuroendocrine factor.[47] In a recent neuroimaging study from a separate research group, fathers' neural responses to infant cries were not related to their circulating OT, which may reflect limitations of peripheral measures (Box 1), but could also align with the likelihood that OT's empathy promotion is context-specific.[48]

Collectively, the findings from the ongoing research in Israel demonstrate prominent triadic (father-mother-infant) socioendocrine OT regulation within immediate family systems and, combined with other psychobiological findings, suggest that OT could play a mechanistic role in facilitating extended kin and nonkin allomothering.[37,43–45,49,50] The work by Feldman and colleagues also provides substantive evidence that OT is similarly "parenting-promoting" as well as "parenting-responsive" in both human mothers and fathers. OT appears to stimulate somewhat different mothering and fathering behaviors (such as

affectionate touch versus exploratory play) in this study setting.[43] It seems possible that these patterns represent neurobiological-endocrine expressions of gender norms and socialization of men and women as parents rather than sex-based genetic propensities. This premise could be tested cross-culturally, particularly drawing on the growing field of neuroanthropology.[51]

There are well-documented correlations between OT and prosociality that reach beyond parenting, such as romantic partners having elevated OT in conjunction with reciprocal or affectionate behavior, and humans and dogs showing upregulated OT after positive interactions.[37,50] Moreover, experimental studies, although they have various methodological limitations (Box 1), have demonstrated that subjects receiving exogenous OT commonly show increases in prosociality, such as elevated trust, empathy, cooperation, and generosity.[49] However, increasingly nuanced studies have shown that these effects are not ubiquitous, but vary based on contextual and individual factors, such as whether generous behaviors are directed toward "in-group" or socially familiar individuals. Indeed, in some cases, OT administration outrightly promotes antisocial tendencies, such as envy of another's financial gain.[49]

Thus, OT is not a panacea when it comes to promoting positive social behavior. If OT increases the physiological and/or cognitive salience of social cues, the very nature of which is surely influenced by our developmental experiences, political and economic circumstances, and cultural context,[52] there are many parenting situations in which OT-heightened social focus might not yield nurturing or empathic parenting outcomes. The study by Weisman and colleagues discussed earlier moves us closer to this sort of context-specific modeling, with artificially elevated OT causing fathers to respond to a challenging parent-infant interaction (that does not typically stress the parent) with heightened cortisol.[47] As a further example, in experimental research with females without children, exogenous OT facilitated activation of brain circuitry related to empathy in response to infant cries, while also dampening areas of the limbic system commonly associated with anger and anxiety.[53] However, the same researchers found that intranasal OT increased patience and restraint in response to infant cries, but only among subjects who, themselves, did not experience harsh parenting during development.[54] While these studies of nulliparous women provide limited understanding of human parental psychobiology, they help reinforce the idea that the behavioral and psychoemotional implications of any "paternal physiology" profile (high PRL, high OT, low testosterone) will depend on socio-cultural and political economic context and be moderated by developmental histories.

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Given my overall focus here, it is critical to acknowledge that OT's effects on paternal behavior, when observed, likely occur via broad, pan-human prosocial neurobiological-endocrine pathways. Rather than being derived characteristics related specifically to fathering, these physiological networks probably reflect the importance of OT to mother-infant sociality throughout our mammalian phylogenetic history and the cooption of those pathways for more general purposes during our evolutionary past as group-living primates and cooperative hominins.

Arginine Vasopressin (AVP)

Compared to OT, much more is known about AVP's diverse array of behavioral effects on males in many vertebrate species, including its implications for aggression, sexual behavior, and mate choice.[55] The AVP-vole literature, which I have discussed briefly, provides much of the foundation for hypotheses regarding AVP and human sociality. In addition to the endogenous increase in neuronal AVP production and release among monogamous male voles following mating or cohabitating and after the birth of pups, AVP injections in the brain not only caused prairie vole males to exhibit pair-bonding behavior, even in the absence of mating, but also led parentally inexperienced males to behave paternally. AVP receptor blockage prevented these phenotypes.[15,16] In total, AVP helps facilitate or reinforce males' participation in a sequence of social events (mating, cohabitating, and parturition or pup exposure) that are key components of species-typical pair-bonding and paternal care phenotypes among monogamous voles.[15,16]

To date, little research to date has explored the role of AVP in primate male social behavior, particularly as related to reproduction and parental-alloparental effort. In a study of marmoset neurobiology, fathers, as compared to nonfathers, were found to have higher levels of AVP dendrite spines and elevated expression of AVP receptor 1a (AVPR1a) in their prefrontal cortices. Marmoset fathers with young infants, which require the most care, had the greatest AVPR1a expression, suggesting infant contact may have contributed to upregulation of these pathways.[56] There is some preliminary evidence for translation of this effect across species, as human fathers with younger children were shown to have elevated peripheral AVP (but see Box 1).[31] Elsewhere, in a brain imaging study, mothers and fathers had comparable peripheral plasma AVP, but when they viewed videos of their infants, fathers' AVP was more strongly linked than mothers' to neural activity in brain areas thought to support bonding processes.[57]

In a recent study, however, high AVP mothers and fathers both engaged in greater stimulatory touch with their infants during observed laboratory-based interactions. Elsewhere married men and women with lower AVP exhibited more negativity when interacting with one another.[58,59] These results are consistent with a potential prosocial effect of elevated AVP regardless of biological sex. There are too few data on human parental AVP at this point to determine whether this peptide has effects that are specific to fathers. Based strictly on a comparative perspective, one might not expect AVP's behavioral effects to be similar across the sexes, because the neuropeptide's effects in some brain areas are contingent on or amplified by testosterone (T).[55] That same critique could be made regarding a potential role of AVP in human paternal behavior, given the commonly observed pattern of low T in fathers and partnered men. For example, prairie vole paternal responsiveness appears to be related, in part, to T's stimulation of AVP production and AVP neural fiber densities.[16] AVP neurobiological-endocrine pathways and implications for prosocial behavior, such as engaged parenting, may differ in humans.

While the more generalized implications of AVP for human sociality are also understudied, existing results provide a preliminary, emerging perspective on the neuropeptide's relevance to the processing of social cues. In experimental research designs, men receiving intranasal AVP exhibited poorer visual recognition of others' emotions, particularly when observing male faces and those with negative valence, and responded agonistically to pictures of unfamiliar males (a pattern not observed in women).[60,61] Results elsewhere suggest that AVP administration facilitates men's retention or processing of both positively and negatively imbued social information, perhaps via effects on prefrontal cortex-limbic area connections in the brain.[62,63] Exogenous AVP may also circumstantially increase men's willingness to reciprocate prosocial behavior, such as if another subject initially signals cooperative intent.[64]

Thus, there is accumulating evidence that AVP heightens attention to social stimuli, potentially promoting prosociality under certain limited conditions, while increasing psychosocial stress under others.[65] Relevant to parenting, it is easy to imagine context-specific circumstances in which enhanced attention to infant cues could facilitate responsive behaviors, protective actions, or stress-related negative outcomes. Given the limited breadth of the literature, it is unclear whether AVP is uniquely upregulated among human fathers or has any parenting-specific effects beyond its more generalized socioendocrine, social-attunement implications. To flesh out these issues, much additional socioendocrine research is needed, with the integration of multiple biomarkers (for example, OT, AVP, and T) into single study designs, particularly for studies of human parenting.

Testosterone (T)

T is a steroid hormone that is produced primarily by the hypothalamic-pituitary-gonadal (HPG) axis in male mammals and those of other vertebrate taxa. T contributes to basic reproductive processes such as spermatogenesis, physical characteristics such as skeletal musculature, and behavioral patterns such as competition with conspecifics, which likely play a role in reproductive success.[10,11,13,14] To the extent that such T-influenced traits negatively impact long-term survival and/or contribute to behavioral phenotypes such as unreliability (for example, investing heavily in extra-pair matings), volatility (for example, being intolerant of or negatively responsive to young), or aggressiveness, elevated T might not be conducive to male parental care or prolonged female-male partnerships. Consequently, among species that have evolved biparental investment, it is thought that the physiological systems affecting T production commonly undergo selection such that invested fathers are likely to experience reduced T during periods in which offspring needs and mother-father cooperation demands are paramount.[5,6,18,22]

For example, there is extensive cross-sectional evidence that male birds' T is higher during the breeding season compared to periods in which males and females pair and cooperatively raise young.[18] Experimental avian data are also consistent with a mating-parenting trade-off framework, as fathers receiving exogenous T pursued extra-pair matings and male-male competition at the expense of investment in their young and to the detriment of offspring survival.[18,66] Artificial exposure to elevated T and its effects on competitive or mating behaviors and energy expenditure also reduced males' long-term survival for at least one species.[66] These ornithological studies provided the primary impetus for testing whether T similarly mediates such reproductive trade-offs in other taxa.

There is less evidence from rodent models that low T facilitates paternal care or that elevated T interferes with it. The strongest support for such a model comes from a longitudinal field study of striped mice, a species among which males can move between multiple reproductive strategy phenotypes. When striped mice males transitioned from solitary roamers to breeders or caregivers, their T decreased, whereas when males became isolated roamers (no care), their T increased.[67] In contrast, among California mice, T is necessary for paternal behavior, acting via T-to-estrogen aromatization.[68] Research on other rodent species has yielded inconclusive or conflicting results as to whether low T is consequential to paternal care expression.[26] At the very least, the variable results in the rodent literature suggest that selection has found multiple, derived neuroendocrine pathways that produce a paternal phenotype rather than a monolithic downregulated paternal T profile.

Studies of paternal physiology in nonhuman primates (primarily New World monkeys) also provide equivocal evidence as to whether T necessarily declines when males transition to parenthood and if T has any implications for caregiving. Among black tufted-ear marmoset fathers, T reached its postpartum nadir when paternal caregiving peaked, and

males that provided more care had significantly lower T. In a closely related species, common marmosets, there was no relationship between paternal care of infants and T.[29,69] In contrast, T increased in cotton-top tamarin fathers throughout their partners' pregnancies and peaked in the early postpartum period, coinciding with a spike in their partners' luteinizing hormone that signaled ovulation, 2-3 weeks after giving birth. The postpartum surge in T likely reflects a behavioral shift toward breeding, but there is no evidence that it interferes with paternal care in this species.[70] This tamarin exemplar is an important reminder that extensive paternal care is a derived characteristic across mammalian taxa and, consequently, that male-female partnerships and paternal care need not be inextricably linked to a socioendocrine phenotype characterized by low T as life history pace and physiological systems coevolve through evolutionary time.[71]

While these marmosets and tamarins provide useful comparative socioendocrine data, their life histories differ dramatically from those of humans, particularly with respect to their faster pace of reproduction and tendency to produce twins and triplets. Moreover, though these tamarins and marmosets do not practice strict monogamy, their group structures rarely include more than a few adult males and females who breed. Also, many members of their groups are closely related kin, including mature sons and daughters of the breeding adults.[72] Thus, their socioecology noticeably diverges from the large multi-male, multi-female groups common among humans. This has significant implications for cross-taxa differences in paternal socioendocrinology, in light of differences in the frequency with which males engage in potentially reproductive, cooperative, affiliative, and competitive social interactions with unrelated males and females besides the mother of their current offspring and nonkin young. While the Great Apes are most comparable to humans in terms of life history, males of those species provide very little substantive, impactful care to their offspring.[6,10,19] Gorilla males are known to playfully interact with and groom young, though these behaviors do not occupy a large percentage of their time,[73] so socioendocrine correlates of low-cost paternal care could be measured in these species. However, there are currently no Great Ape data on T and paternal care or sociality between adult males and young apes from which direct comparisons can be made. However, there are relevant data on the Hylobatids, the "lesser apes," and olive baboons, a long-lived, large bodied primate species with multi-male, multi-female group composition.

. . . these data suggest that the specific context of father-offspring interaction and direct paternal care may be a social pathway whereby T is reduced in ape species, which parallels findings in humans.

Male olive baboons are known to form "friendships" with lactating females and their infants. In one study, such friendship-forming males experienced significant declines in T.[74] It remains unclear whether this is paternal care, although data from other baboon species indicates that it often is, or perhaps a mating strategy. However, it suggests that reduced T might have improved males' effectiveness in a social relationships with mother-infant pairs, particularly buffering them from psychosocial stress.[12,74,75] Life-course changes in male T from other baboon species have likewise been interpreted as consistent with this perspective; that is, older males having low T during periods in which they perform more care and engage less in competition for dominance.[75] Preliminary studies on Hylobatids recently found that captive siamang fathers experienced a significant decline in T over the course of their offsprings' infancy, coinciding with stark increases in father-offspring interaction. In contrast, gibbon males, which do not care for young, showed no T change over the seven-month postpartum period, despite having a relatively high frequency of father-offspring proximity during the study.[76]

A recent study of free-living white-handed gibbons produced noteworthy complementary results, finding that males had higher fecal androgens if they resided in male-female pairs (compared to multi-male groups) or in groups with a dependent infant.[77] Gibbons' high androgens in these contexts might indicate that they are in unstable social conditions (that is, male-female pairs) or require extra vigilance against infanticide, which results in males being primed to aggressively defend the group.[77] In total, these data suggest that the specific context of father-offspring interaction and direct paternal care may be a social pathway whereby T is reduced in ape species, which parallels findings in humans.

When the full breadth of nonhuman mammalian evidence is considered, these studies indicate that an avian analogy, which is the major theoretical foundation on which empirical studies of paternal biology have been based, is not uniformly applicable to mammalian species with biparental care.[18] In particular, among those rodent and New World monkey species showing static or elevated T in the postpartum period, many resume breeding shortly after their partners give birth, being shorter-lived species with comparatively "faster" life histories than humans.[71] Thus, the role of elevated T in stimulating mating-related behaviors appears to have precluded selection for reduced paternal T and evolution has found other neuroendocrine avenues through which paternal care can be facilitated, such as the conversion of T to estrogen in California mice.[68] As a consequence, analogies to these mammalian species are somewhat constrained because humans are not seasonal breeders and have significantly longer interbirth intervals and slow-

developing dependent offspring.[2–4] Recent evidence shows that siamangs and baboons,[74–76] both of which provide comparatively better approximations of human life history, have the capacity to decrease T production when they invest in mother-infant pairs. This suggests that natural selection may have similarly shaped human male physiology as our species evolved a cooperative breeding strategy in which fathers were likely candidates to assist mothers with raising young.

PARENTING AND T AMONG HUMAN MALES

Humans facultatively adjust their reproductive strategies, primarily via behavioral flexibility, over reproductive life spans that last many decades. T might be a key physiological factor that helps males accommodate a range of behavioral repertoires as new social demands arise, particularly if biparental care evolved in the *Homo* lineage.[5,6,10] For hominin fathers who initially cooperated with mothers to raise young, reduced T could have had a range of fitness implications, including, especially, a behavioral shift away from competition and pursuit of many short-term partners and toward increased paternal sensitivity and attentiveness, enhanced male-infant interactions, and reduced maternal parenting burdens (psychoemotionally, energetically, time wise).[5] This suite of behaviors could have facilitated bonding, attachment, and men's long-term commitments to their offspring while enhancing the likelihood that a female-male pair would partner again to produce subsequent young with a shortened interbirth interval.[5,6] Female mate choice and other aspects of female reproductive strategies would likely be critical to the question of whether such a biobehavioral paternal phenotype would lead to fitness benefits for both members of the reproductive dyad, but particularly for males with regard to cuckoldry and long-term continuity of the partnership.[78,79]

The human psychobiological literature on men's T is consistent with the idea that downregulated baseline T could be generally beneficial in the context of invested, nurturing parenting. Although T relates to broad measures of "aggressive behavior," only modestly at best, high T males show tendencies toward angry moods and direct more attention to stimuli that mimic social threats and dominance challenges.[80] Men with elevated T also exhibited heightened brain activity in the amygdala, which contributes to emotional processing, when presented with threat-related images, suggesting that individuals with high T attach more subconscious salience to such stimuli and are perhaps more likely to respond with reactive aggression.[81,82] Indeed, high T seems to reduce prefrontal cortical connectivity with the amygdala, with implications for conscious control of vigilance and social behavior.[83] These integrative hormonal-brain activity studies indicate that men with high T might be particularly oriented to male-male competition and challenge, and primed to react aggressively in such situations or others involving social threat, which could reduce men's focus and availability or capability (i.e. lost time, risk of injury, reduced survival) to invest in young. This remains to be tested.

. . . attention should be paid to psychobiological theory that can move us past simple mating versus parenting dichotomies.[13]

Supporting this model from other behavioral angles, correlational studies have found that men with greater T reported more lifetime sexual partners and also expressed more interest in sexual and romantic opportunities outside their current relationship.[84–86] Among U.S. military personnel, higher T men had greater divorce rates,[87] but other studies have found mixed results for T-romantic relationship quality (low T, low quality[88]; high T, low quality[89]; no significant correlation[90]). Characteristics of the family system, such as psychosocial stress of both partners, potentially moderate whether men's T has positive or negative implications for relationship dynamics.[91] In some studies, men with elevated T were also more likely be extroverts, sensation seekers, and risk takers, all of which are personality-behavioral profiles that have been either linked to men's reproductive success or are hypothesized to contribute to it.[92,93] By extension, such characteristics are also hypothesized to conflict with men's willingness to act as committed parents or their effectiveness in doing so. Broadly consistent with this premise, preliminary evidence indicates that fathers with low sensation-seeking scores have reduced T in the early postpartum period.[88] My colleagues and I also recently demonstrated that men experiencing larger T declines across marriage or fatherhood transitions engaged in less frequent intercourse with their partners, which aligns with some tenets of this framework.[71] Yet many aspects of this model (high baseline T-parenting incompatibility) are based on limited correlational evidence from diverse animal species, some of which do not exhibit biparental care, and thus require verification in humans. Particular attention should be paid to psychobiological theory that can move us past simple mating versus parenting dichotomies.[13]

It is well known that there is between-culture variation in the formation and patterning of romantic and reproductive partnerships and in the dynamics of parenting roles (and the importance of allomothers). Thus, it is perhaps unsurprising that the extent to which T differs by partnering or parenting status seems also to vary substantially according to cultural context.[94] For example, multiple studies in the U.S. have shown that married nonfathers and married fathers have comparable T, with both groups having lower T than single nonfathers. In other U.S.-based studies, partnered individuals' T varied by commitment level.[85,86,94] Additional research by Gray and colleagues suggests that T-partnering–parenting dynamics manifest differently among

polygamous cultural groups.[94,95] At our research site, we observed graded T variation related to partnering and parenthood. Filipino men transitioning from being single nonfathers to being newly partnered new fathers (over a five year period) showed declines in T that were substantially greater than subjects who remained single nonfathers during the same time frame; newly partnered nonfathers' T was intermediate between the two.[22]

Although we need much more cross-cultural integrative data in this domain, there is growing evidence that culturally defined parenting roles may be critical to paternal socioendocrine variation. A seminal study compared the relationships between T and fatherhood in subjects from two neighboring East African groups, the Hadza (foragers, fathers involved in direct care) and the Datoga, (pastoralists, fathers not principally involved in care). Hadza fathers had decreased T relative to nonfathers, whereas Datoga fathers did not. Thus, based on these results, variation in T according to fatherhood status may differ by cultural norms of parenting and, specifically, degrees of involvement with direct care.[23] Similarly, in separate studies conducted in Senegal and the U.S., fathers who were highly invested in their children, as reported by the children's mothers, had lower T compared to fathers who were less invested.[24,96] Our research showed that Filipino fathers who reported performing daily child care had reduced T as compared to that of fathers who reported no caregiving. Fathers who shared a sleeping surface with their children (cosleeping) had lower T than men who slept away from their offspring.[22,25,97] In a study of Canadian men, fathers with past experience in directly caring for infants had reduced baseline T, and fathers with low T reported greater sympathy and need to respond when hearing recorded infant cries.[98]

In total, it appears that men's commitment to offspring care helps explain both inter- and intra-cultural variation in the extent to which fathers have lower T than other men. Along with data from certain nonhuman primates, particularly siamangs, these data appear to support an adaptation-oriented model in which human fathers' physiology is sensitive to involvement in direct care. However, that notion is potentially difficult to reconcile with rates of direct paternal care across cultures, which are generally quite low, especially compared to mothers.[6,10] I have previously suggested the possibility that the selective environment for direct male care might have differed from the cultural contexts and divisions of labor that we observe today.[5] It is also important to bear in mind that the quality of paternal care and its salience for offspring health and development could have been critical to males' fitness, even if, on average, the absolute quantity was not exceedingly high. Among Old World primates in general, the threshold for HPG responsiveness to care might be low but specific, meaning that it takes a particular context (though not a high quantity) of male-young prosocial interaction and bond to elicit reduced T, rather than mere sensory proximity. Under that scenario, an important question is whether such flexible responsiveness of the HPG to male-young interaction is adaptive (and shared or derived in various lineages) or an epiphenomenon or "spandrel" emerging from primates' neurobiological-endocrine history of group living and sociality. An additional possibility is that a separate, to-date unmeasured correlate of fathers' care, such as the extent to which fathers engage in competition with other males, is the driver of low paternal T.

In that vein, a recent study of Polish fathers found that paternal T differed based on number of children, but the direction was moderated by socioeconomic status (SES): high SES+ more children, lower T; low SES+ more children, higher T. The authors suggested that this difference could reflect a constellation of factors varying across SES gradients, including the importance of male-male competition for resources, as well as variable parenting roles and timing of reproduction.[99] Similarly, in a large study of military personnel, young married fathers had higher T than did married nonfathers, which could align with aspects of competition, protectiveness, or family systems germane to military life.[100] Also, a recent study of a contemporary forager-horticultural population (Tsimane of Bolivia) found that males' T acutely spiked during physically demanding tree chopping and plot clearing, a physiological response that the authors suggest could enhance men's subsistence productivity.[101] Because male contributions to household, familial, or community productivity and energy budgets are key in many ecological settings, these emerging data add an intriguing layer of complexity to discussions of paternal biology. Suffice it to say that this research area is rife with opportunities for biological and cultural anthropological integration. The seeming plasticity of the psychobiology of human partnering or fatherhood raises important questions regarding the applications of evolutionary or adaptation perspectives to these phenomena.

NATURAL SELECTION, PHENOTYPIC PLASTICITY, AND EVOLUTIONARY ORIGINS OF PATERNAL PHYSIOLOGY

Males of many Old World monkey species are highly interested in and/or form tolerant relationships with young. These behaviors range from the "friendships" that olive baboon males form with mother-infant pairs to the carrying and transport of young by Chacma baboons, the use of infants as social tools by various macaque and baboon species ("agonistic buffering"), and other prosocial, tolerant, or utilitarian forms of interaction by male monkeys.[12,74] Great Ape males generally do not form extensive social bonds with young, but both chimpanzee and gorilla males will occasionally groom, play with, and/or carry immatures.[73] Males of multiple nonhuman primate species have also been shown to adopt orphaned infants, although such events are exceedingly rare.[102] Overall, this phylogenetic perspective suggests that there might have been sufficient behavioral plasticity among our hominin ancestors to allow for early forms of prosocial male-offspring interactions in

the absence of any adaptive changes resulting from chance mutations.

A shift toward hominin males spending more time within sensory range of their offspring could have been initiated through females' choice of males who maintained such proximity, certain males exploiting opportunities for novel, coalitionary reproductive strategies, and/or increased overall emphasis on cooperative sociality and group cohesion as dietary regimes and ecological circumstances changed, beginning in the early Pleistocene.[5,6,10,78,79,103,104] Through evolutionary time, mutations could have occurred that specifically increased the sensitivity of hominin male physiology (for example, regulation of T) to the presence of their offspring. Alternatively, novel increased sensory or psychoemotional exposure of males to their young could have led some to experience downregulation of T due to physiological plasticity. If fathers' ability to downregulate their T was linked to novel mutations or previously neutral or cryptic genetic variation (genetic accommodation), and if reduced T facilitated male investment with benefits to offspring, selection for these genetic variants could have led to evolutionary change and increased representation of a biobehavioral paternal phenotype characterized by substantial paternal care and diminished T production.[5,105]

The notion that aspects of HPG regulation and T production are related to genetic variation are supported by familial studies showing moderate heritability of T levels.[106] There is evidence that the HPG axis can evolve sensitivity to proximate environmental stimuli such as the well-characterized relationship between external seasonality cues (particularly light exposure) and activation or quiescence of the HPG axis in seasonally breeding vertebrate species.[107] There is similar cross-species evidence that HPG function can be regulated by socially mediated sensory cues (such as olfactory stimulation via pheromones in some nonhuman taxa).[108] Moreover, social stimuli can affect gene expression patterns in the brain (including aspects of the gonadotropin-releasing hormone (GnRH) neuronal network, which, orchestrates T production) in species-specific and life history stage-specific manners. Such observations provide support for the notion that genetic variation related to the control of neurobiological responsivity (for example, genetic influence over connectivity in neurobiological networks or neurotransmitter receptor densities or sensitivities) could undergo selection as social ecologies change and novel, salient stimuli arise.[108]

The hypothesis that male T responsivity to partnering or parenting has some underlying genetic basis could be tested by comparing candidate "responsivity alleles" across hominoids,[109] based on the assumption that the frequencies of such alleles would be reduced or absent in our Great Ape relatives. An additional within-species approach would be to test for gene loci differences across men that covary with T responses to marriage or fatherhood. For both models, candidate gene loci, such as those linked to T production and other functional aspects of HPG physiology (for example, GnRH regulation/production), would need to be identified.[110] Subsequently, additional genetic analyses could be undertaken to assess how recently alleles arose or how intensely they have undergone selection, which could provide insights into the time frame over which males began cooperating more extensively with females to raise young.[111]

This approach perhaps would help to inform a biocultural model on why, for example, human fathers do not ubiquitously meet the high bar of paternal care set by some of our New World monkey relatives . . .

A dichotomous distinction between Old World primates as "paternal care present" versus "paternal care absent" might obfuscate the deep phylogenetic history of cross-primate male behavioral flexibility and its relevance to the evolutionary history of human fatherhood. Particularly, it might be fruitful to apply a "switches and knobs" perspective to human fathers' physiology.[17] A "switch" approach would suggest that novel genetic mutations would necessarily have emerged in the hominin lineage, leading to large-scale neurobiological restructuring or attunement shifts. These shifts would have allowed males to respond biologically to parenthood and offspring cues, leading to binary profiles for "paternal psychobiology" (humans: present; most other Old world primates: absent).

In contrast, rather than a "yes or no" switch dichotomy, a "knob" perspective would frame primate male psychobiological and behavioral plasticity on a continuum ranging from exceedingly rare male-infant interaction (as among common chimpanzees) to prevalent paternal care and psychobiological responsivity (as among humans and siamangs). The emergence of smaller, less disruptive genetic changes could "turn the knob" on primate male neurobiological pathways that might already be responsive to other social cues or sensory stimuli from conspecifics as socioecological shifts emerge through evolutionary time.[17] Although testing for a potentially large number of small knob allele changes will admittedly be challenging, this model, which allows degrees of variation in primate psychobiological responses to affiliative behaviors with young, is more consistent with the limited data we have across Old World primates (olive baboons, gibbons, siamangs, and humans).[22,23,25,74,76,77] From an evolutionary perspective, this nuanced, continuum-oriented, knob approach is also commensurate with the substantive range of variation in human paternal care behaviors, both in degree and type, that men exhibit across and within cultures.[6,9,10] This approach perhaps would help to inform a biocultural model on why, for example, human fathers do not ubiquitously meet the high bar of paternal care set by some of our New

World monkey relatives, such as *Saguinus*, *Callithrix, Aotus, and Callicebus*,[19] in spite of men having seemingly evolved paternal tendencies and accommodating biology.

APPLYING AN EVOLUTIONARY SOCIOENDOCRINE PERSPECTIVE TO COOPERATIVE BREEDING

Given the ongoing conversations in biological anthropology as to the role of cooperative breeding in the emergence of modern human life history, I will discuss whether this evolutionary-grounded socioendocrine model can be fruitfully applied to other caregivers, such as grandmothers and older offspring (siblings). In cooperative breeding avian and mammalian species, there is evidence of biological mechanisms influencing the behavior of nonparental allomothers. A recent review nicely summarized much of the relevant mammalian literature.[112] I argue that examining neurobiological-endocrine measures could provide important insights into the socio-ecological circumstances that interrelate with caregiving differences between grandmothers. However, a socioendocrine approach would be unlikely to inform our understanding of the evolutionary history of post-menopausal grandmaternal care. The strength of selection decreases as individuals age and the indirect fitness benefits accrued by grandmothers might not be sufficient for a "grandmaternal endocrine" phenotype to evolve. More importantly, grandmothers, by definition, have a biological history of successful motherhood. While the very low levels of estrogen in postmenopausal women might affect some relevant socioendocrine pathways, such as OT, grandmothers' cognitive and physiological systems would have "memory" (such as receptor densities or neural connections) of appropriate child care behaviors and attachment processes.[113,114] Decades of animal research have shown that multiparous females require less or no hormonal priming (that is, ovarian steroid exposure during pregnancy) to express maternal care.[20] Put simply, while it may have been critical for hominin male physiology to undergo specific selection for paternal care to emerge evolutionarily, grandmaternal physiology need not have been similarly targeted by selection for them to be invested, effective allomothers.

Older human offspring also cooperate with mothers in household productivity and direct care of younger, highly dependent young. This "helper at the nest" phenomenon was potentially important to the evolutionary emergence of human life history.[1] For most of their childhood and juvenile periods, such helpers would have quiescent HPG axes, limiting the implications of those pathways. However, data from some cooperative breeding mammalian and bird species show that allomothers who remain in their natal groups show elevated PRL.[112,115] Although these findings suggest positive selection for allomothering physiology, it is possible this socioendocrine pattern could have evolved because older, mature offspring who remained in their parents' territory were exposed to nestling sensory stimulation, causing increased PRL production as a result of parenting physiological substrates, which inclined those individuals to become nonbreeding allomothers.[115] If observed, PRL activity in immature human allomothers might simply reflect selection for human parental physiology rather than an adaptive childhood or juvenile socioendocrine profile. Thus, as in the preceding discussion of grandmothers, it is unclear whether applying this framework to humans would inform our understanding of "helpers at the nest" during hominin evolution. Yet there is evidence that PRL endocrine pathways are active in human children.[116] To my knowledge, no research has explored that hormone's potential facilitatory effects on their household productivity or direct care. Moreover, it has long been argued that juvenile allomothers hone their parenting skills before maturation and parenthood.[7] It would be of great value for future research to test whether there are socioendocrine programming effects based on caring for one's siblings during development. (For example, does elevated PRL or OT during childhood or juvenility affect adult psychobiology?)

CONCLUSION

As with many questions of human biology and behavior, what is perhaps most intriguing about these data is the unsettled but seemingly vital role of culture and the breadth of developmental experiences that may influence the extent to which human males show neurobiological-endocrine changes relative to partnering and/or fatherhood. Unlike many other mammalian species that have evolved biparental care, paternal care in humans is not presently obligate, meaning that mothers can raise young without the aid, specifically, of the father.[3,6,19] There is ongoing debate as to whether hominin males played a prominent role in cooperating with mothers in provisioning and caregiving, thus facilitating the emergence of modern human life history. If males were key figures in this regard, there probably was still substantial variability in the type and intensity of male investment in offspring throughout hominin evolution, (a phrase that encompasses an enormous, ecologically variable time frame). This would have depended on many factors, including, for example, foraging strategies, resource availability, group size, natal dispersal and locality, kinship structure, longevity, and age-specific survival rates.[1–5,104] Altogether, these factors could have led to the selection of a plastic male psychobiological profile that retains the ability to facilitate and/or respond to fatherhood and caregiving given the appropriate cues or milieu, but, likewise, is not inevitably canalized to do so.

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REFERENCES

1 Kramer KL. 2010. Cooperative breeding and its significance to the demographic success of humans. Annu Rev Anthropol 39:417–436.

2 Hawkes K, O'Connell JF, Blurton-Jones NG, et al. 1998. Grandmothering, menopause, and the evolution of human life histories. Proc Natl Acad Sci 95:1336–1339.

3 Hrdy SB. 2009. Mothers and others: the evolutionary origins of mutual understanding. Cambridge, MA: Belknap Press of Harvard University Press.

4 Kaplan H, Hill K, Lancaster J, et al. 2000. A theory of human life history evolution: diet, intelligence, and longevity. Evol Anthropol 9: 156–185.

5 Gettler LT. 2010. Direct male care and hominin evolution: why male-child interaction is more than a nice social idea. Am Anthropol 112:7–21.

6 Gray PB, Anderson KG. 2010. Fatherhood: evolution and human paternal behavior. Cambridge, MA: Harvard University Press.

7 Hrdy SB. 1976. Care and exploitation of nonhuman primate infants by conspecifics other than the mother. In: Rosenblatt J, Hinde R, Shaw E, et al., editors. Advances in the study of behavior. New York: Academic Press, p 101–158.

8 Bercovitch FB, Ziegler TE. 2002. Current topics in primate socioendocrinology. Annu Rev Anthropol:45–67.

9 Hewlett BS, editor. 1992. Father-child relations: cultural and biosocial contexts. New York: Aldine De Gruyter.

10 Bribiescas RG, Ellison PT, Gray PB. 2012. Male life history, reproductive effort, and the evolution of the genus *Homo*: new directions and perspectives. Curr Anthropol 53:S424–S435.

11 Ellison PT. 2003. Energetics and reproductive effort. Am J Hum Biol 15:342–351.

12 Smuts BB, Gubernick DJ. 1992. Male-infant relationships in nonhuman primates: paternal investment or mating effort? In: Hewlett BS, editor. Father-child relations: cultural and biosocial contexts. New York: Aldine De Gruyter. p 1–30.

13 van Anders SM. 2013. Beyond masculinity: testosterone, gender/sex, and human social behavior in a comparative context. Front Neuroendocrinol 34:198–210.

14 Ketterson ED, Nolan V. 1992. Hormones and life histories: an integrative approach. Am Nat 140:S33–S62.

15 Young KA, Gobrogge KL, Liu Y, et al. 2011. The neurobiology of pair bonding: insights from a socially monogamous rodent. Front Neuroendocrinol 32:53–69.

16 Wang Z, Young LJ, De Vries Jr G, et al. 1999. Voles and vasopressin: a review of molecular, cellular, and behavioral studies of pair bonding and paternal behaviors. Prog Brain Res 119:483–499.

17 Young LJ, Hammock E. 2007. On switches and knobs, microsatellites and monogamy. Trends Genet 23:209–212.

18 Wingfield JC, Hegner RE, Ball GF, et al. 1990. The "challenge hypothesis": theoretical implications for patterns of testosterone secretion, mating systems, and breeding strategies. Am Nat 136:829–846.

19 Fernandez-Duque E, Valeggia CR, Mendoza SP. 2009. The biology of paternal care in human and nonhuman primates. Annu Rev Anthropol 38:115–130.

20 Numan M, Insel TR. 2003. The neurobiology of parental behavior. New York: Springer.

21 Gettler LT, McDade TW, Feranil AB, et al. 2012. Prolactin, fatherhood, and reproductive behavior in human males. Am J Phys Anthropol 148:362–370.

22 Gettler LT, McDade TW, Feranil AB, et al. 2011. Longitudinal evidence that fatherhood decreases testosterone in human males. Proc Natl Acad Sci 108:16194–16199.

23 Muller MN, Marlowe FW, Bugumba R, et al. 2009. Testosterone and paternal care in East African foragers and pastoralists. Proc R Soc B 276:347–354.

24 Alvergne A, Faurie C, Raymond M. 2009. Variation in testosterone levels and male reproductive effort: insight from a polygynous human population. Horm Behav 56:491–497.

25 Gettler LT, McKenna JJ, Agustin SS, et al. 2012. Does cosleeping contribute to lower testosterone levels in fathers? Evidence from the Philippines. PLoS ONE 7:e41559.

26 Wynne-Edwards KE, Timonin ME. 2007. Paternal care in rodents: weakening support for hormonal regulation of the transition to behavioral fatherhood in rodent animal models of biparental care. Horm Behav 52:114–121.

27 Ziegler TE, Wegner FH, Snowdon CT. 1996. Hormonal responses to parental and nonparental conditions in male cotton-top tamarins, *Saguinus oedipus*, a New World primate. Horm Behav 30:287–297.

28 Schradin C, Anzenberger G. 2004. Development of prolactin levels in marmoset males: from adult son to first-time father. Horm Behav 46:670–677.

29 Dixson AF, George L. 1982. Prolactin and parental behavior in a male New-World primate. Nature 299:551–553.

30 Ziegler TE, Prudom SL, Zahed SR, et al. 2009. Prolactin's mediative role in male parenting in parentally experienced marmosets (*Callithrix jacchus*). Horm Behav 56:436–443.

31 Gray PB, Parkin JC, Samms-Vaughan ME. 2007. Hormonal correlates of human paternal interactions: a hospital-based investigation in urban Jamaica. Horm Behav 52:499–507.

32 Gordon I, Zagoory-Sharon O, Leckman JF, et al. 2010. Prolactin, oxytocin, and the development of paternal behavior across the first six months of fatherhood. Horm Behav 58:513–518.

33 Storey AE, Walsh CJ, Quinton RL, et al. 2000. Hormonal correlates of paternal responsiveness in new and expectant fathers. Evol Human Behav 21:79–95.

34 Storey AE, Noseworthy DE, Delahunty KM, et al. 2011. The effects of social context on the hormonal and behavioral responsiveness of human fathers. Horm Behav 60:353–361.

35 Gettler LT, Agustin SS, McDade TW, et al. 2011. Short term changes in fathers' hormones during father-child play: impacts of paternal attitudes and experience. Horm Behav 60:599–606.

36 Anderson ST, Barclay JL, Fanning KJ, et al. 2006. Mechanisms underlying the diminished sensitivity to prolactin negative feedback during lactation: reduced STAT5 signaling an upregulation of cytokine-inducible SH2 domain-containing protein (CIS) expression in tuberoinfundibular dopaminergic neurons. Endocrinology 147:1195–1202.

37 Odendaal J, Meintjes R. 2003. Neurophysiological correlates of affiliative behaviour between humans and dogs. Veterinary J 165:296–301.

38 Gubernick DJ, Winslow JT, Jensen P, et al. 1995. Oxytocin changes in males over the reproductive cycle in the monogamous, biparental California mouse, *Peromyscus californicus*. Horm Behav 29:59–73.

39 Kenkel WM, Suboc G, Carter SC. 2014. Autonomic, behavioral and neuroendocrine correlates of paternal behavior in male prairie voles. Physiol Behav:epub.

40 Woller MJ, Sosa ME, Chiang Y, et al. 2011. Differential hypothalamic secretion of neurocrines in male common marmosets: parental experience effects. J Neuroendocrinol 24:413–421.

41 Saito A, Nakamura K. 2011. Oxytocin changes primate paternal tolerance to offspring in food transfer. J Comp Physiol A 197: 329–337.

42 Feldman R, Gordon I, Schneiderman I, et al. 2010. Natural variations in maternal and paternal care are associated with systematic changes in oxytocin following parent–infant contact. Psychoneuroendocrinology 35:1133–1141.

43 Gordon I, Zagoory-Sharon O, Leckman JF, et al. 2010. Oxytocin and the development of parenting in humans. Biol Psychiatry 68:377–382.

44 Feldman R, Gordon I, Influs M, et al. 2013. Parental oxytocin and early caregiving jointly shape children's oxytocin response and social reciprocity. Neuropsychopharmacology 38: 1154–1162.

45 Feldman R, Gordon I, Zagoory-Sharon O. 2010. Maternal and paternal plasma, salivary, and urinary oxytocin and parent-infant synchrony: considering stress and affiliation components of human bonding. Dev Sci 14: 752–761.

46 Weisman O, Zagoory-Sharon O, Feldman R. 2012. Oxytocin administration to parent enhances infant physiological and behavioral readiness for social engagement. Biol Psychiatry 72: 982–989.

47 Weisman O, Zagoory-Sharon O, Feldman R. 2013. Oxytocin administration alters HPA reactivity in the context of parent-infant interaction. European Neuropsychopharmacol 23: 1724–1731.

48 Mascaro JS, Hackett PD, Gouzoules H, et al. 2013. Behavioral and genetic correlates of the neural response to infant crying among human fathers. Soc Cogn Affect Neurosci:epub.

49 Bartz JA, Zaki J, Bolger N, et al. 2011. Social effects of oxytocin in humans: context and person matter. Trends Cogn Sci 15:301–309.

50 Schneiderman I, Zagoory-Sharon O, Leckman JF, et al. 2012. Oxytocin during the initial stages of romantic attachment: relations to couples' interactive reciprocity. Psychoneuroendocrinology 37:1277–1285.

51 Lende DH, Downey G, editors. 2012. The encultured brain: an introduction to neuroanthropology. Cambridge, MA: MIT Press.

52 Lederbogen F, Kirsch P, Haddad L, et al. 2011. City living and urban upbringing affect neural social stress processing in humans. Nature 474:498–501.

53 Riem MM, Bakermans-Kranenburg MJ, Pieper S, et al. 2011. Oxytocin modulates amygdala, insula, and inferior frontal gyrus responses to infant crying: a randomized controlled trial. Biol Psychiatry 70:291–297.

54 Bakermans-Kranenburg MJ, van Ijzendoorn MH, Riem MM, et al. 2012. Oxytocin decreases handgrip force in reaction to infant crying in females without harsh parenting experiences. Soc Cogn Affect Neurosci 7:951–957.

55 Goodson JL, Bass AH. 2001. Social behavior functions and related anatomical characteristics of vasotocin/vasopressin systems in vertebrates. Brain Res Rev 35:246–265.

56 Kozorovitskiy Y, Hughes M, Lee K, et al. 2006. Fatherhood affects dendritic spines and vasopressin V1a receptors in the primate prefrontal cortex. Nat Neurosci 9:1094–1095.

57 Atzil S, Hendler T, Zagoory-Sharon O, et al. 2012. Synchrony and specificity in the maternal and the paternal brain: relations to oxytocin and vasopressin. J Am Acad Child Adolesc Psychiatry 51:798–781.

58 Gouin J, Carter CS, Pournajafi-Nazarloo H, et al. 2010. Marital behavior, oxytocin, vasopressin, and wound healing. Psychoneuroendocrinology 35:1082–1090.

59 Apter-Levi Y, Zagoory-Sharon O, Feldman R. 2013. Oxytocin and vasopressin support distinct configurations of social synchrony. Brain Res:epub.

60 Uzefovsky F, Shalev I, Israel S, et al. 2012. Vasopressin selectively impairs emotion recognition in men. Psychoneuroendocrinology 37:576–580.

61 Thompson R, George K, Walton J, et al. 2006. Sex-specific influences of vasopressin on human social communication. Proc Natl Acad Sci USA 103:7889–7894.

62 Zink CF, Stein JL, Kempf L, et al. 2010. Vasopressin modulates medial prefrontal cortex–amygdala circuitry during emotion processing in humans. J Neurosci 30:7017–7022.

63 Guastella AJ, Kenyon AR, Alvares GA, et al. 2010. Intranasal arginine vasopressin enhances the encoding of happy and angry faces in humans. Biol Psychiatry 67:1220–1222.

64 Rilling JK, DeMarco AC, Hackett PD, et al. 2012. Effects of intranasal oxytocin and vasopressin on cooperative behavior and associated brain activity in men. Psychoneuroendocrinology 37:447–461.

65 Shalev I, Israel S, Uzefovsky F, et al. 2011. Vasopressin needs an audience: neuropeptide-elicited stress responses are contingent upon perceived social evaluative threats. Horm Behav 60:121–127.

66 Reed W, Clark M, Parker P, et al. 2006. Physiological effects on demography: a long-term experimental study of testosterone's effects on fitness. Am Nat 167:667–683.

67 Schradin C, Yuen C. 2011. Hormone levels of male African striped mice change as they switch between alternative reproductive tactics. Horm Behav 60:676–680.

68 Trainor BC, Marler CA. 2002. Testosterone promotes paternal behaviour in a monogamous mammal via conversion to oestrogen. Proc R Soc B 269:823–829.

69 Nunes S, Fite JE, Patera KJ, et al. 2001. Interactions among paternal behavior, steroid hormones, and parental experience in male marmosets (*Callithrix kuhlii*). Horm Behav 39: 70–82.

70 Ziegler TE, Jacoris S, Snowdon CT. 2004. Sexual communication between breeding male and female cotton-top tamarins (*Saguinus oedipus*), and its relationship to infant care. Am J Primatol 64:57–69.

71 Gettler LT, McDade TW, Agustin SS, et al. 2013. Do testosterone declines during the transition to marriage and fatherhood relate to men's sexual behavior? Evidence from the Philippines. Horm Behav 64:755–763.

72 Digby LJ, Ferrari SF, Saltzman W. 2006. The role of competition in cooperatively breeding species. In: Campbell C, Fuentes A, MacKinnon K, et al., editors. Primates in perspective. New York: Oxford University Press. p 85–106.

73 Rosenbaum S, Silk J, Stoinski T. 2011. Male-immature relationships in multi-male groups of mountain gorillas (*Gorilla beringei beringei*). Am J Primatol 73:356–365.

74 Shur MD. 2008. Hormones associated with friendship between adult male and lactating female olive baboons. Ph.D. dissertation, Rutgers University.

75 Beehner JC, Gesquiere L, Seyfarth RM, et al. 2009. Testosterone related to age and life-history stages in male baboons and geladas. Horm Behav 56:472–480.

76 Rafacz ML, Margulis S, Santymire RM. 2012. Hormonal correlates of paternal care differences in the hylobatidae. Am J Primatol 74: 247–260.

77 Barelli C, Heistermann M. 2012. Sociodemographic correlates of fecal androgen levels in wild male white-handed gibbons (*Hylobates lar*). Int J Primatol 33:784–798.

78 Scelza BA. 2013. Choosy but not chaste: multiple mating in human females. Evol Anthropol 22:259–269s.

79 Gavrilets S. 2012. Human origins and the transition from promiscuity to pair-bonding. Proc Natl Acad Sci USA 109:9923–9928.

80 van Honk J, Tuiten A, Verbaten R, et al. 1999. Correlations among salivary testosterone, mood, and selective attention to threat in humans. Horm Behav 36:17–24.

81 Derntl B, Windischberger C, Robinson S, et al. 2009. Amygdala activity to fear and anger in healthy young males is associated with testosterone. Psychoneuroendocrinology 34: 687–693.

82 Goetz SM, Tang L, Thomason ME, et al. 2014. Testosterone rapidly increases neural reactivity to threat in healthy men: a novel two-step pharmacological challenge paradigm. Biol Psychiatry:epub.

83 Volman I, Toni I, Verhagen L, et al. 2011. Endogenous testosterone modulates prefrontal-amygdala connectivity during social emotional behavior. Cerebral Cortex 21:2282–2290.

84 Pollet TV, van der Meij L, Cobey KD, et al. 2011. Testosterone levels and their associations with lifetime number of opposite sex partners and remarriage in a large sample of American elderly men and women. Horm Behav 60:72–77.

85 McIntyre M, Gangestad SW, Gray PB, et al. 2006. Romantic involvement often reduces men's testosterone levels - but not always: the moderating role of extrapair sexual interest. J Pers Soc Psychol 91:642–651.

86 van Anders SM, Goldey KL. 2010. Testosterone and partnering are linked via relationship status for women and "relationship orientation" for men. Horm Behav 58:820–826.

87 Mazur A, Michalek J. 1998. Marriage, divorce, and male testosterone. Soc Forces 77: 315–330.

88 Perini T, Ditzen B, Fischbacher S, et al. 2012. Testosterone and relationship quality across the transition to fatherhood. Biol Psychol 90:186–191.

89 Julian T, McKenry PC. 1989. Relationship of testosterone to men's family functioning at midlife: a research note. Aggressive Behav 15: 281–289.

90 Gray PB, Yang CJ, Pope HG. 2006. Fathers have lower salivary testosterone levels than unmarried men and married non-fathers in Beijing, China. Proc R Soc B 273:333–339.

91 Booth A, Johnson DR, Granger DA. 2005. Testosterone, marital quality, and role overload. J Marriage Fam 67:483–498.

92 Alvergne A, Jokela M, Faurie C, et al. 2010. Personality and testosterone in men from a high-fertility population. Pers Indiv Differ 49: 840–844.

93 Ronay R, von Hippel W. 2010. The presence of an attractive woman elevates testosterone and physical risk taking in young men. Soc Psychol Pers Sci 1:57–64.

94 Gray PB, Campbell BC. 2009. Human male testosterone, pair bonding and fatherhood. In: Ellison PT, Gray PB, editors. Endocrinology of social relationships. Cambridge, MA: Harvard University Press, p 270–293.

95 Gray PB. 2003. Marriage, parenting, and testosterone variation among Kenyan Swahili men. Am J Phys Anthropol 122:279–286.

96 Mascaro JS, Hackett PD, Rilling JK. 2013. Testicular volume is inversely correlated with nurturing-related brain activity in human fathers. Proc Natl Acad Sci USA 110:15746–15751.

97 Kuzawa CW, Gettler LT, Muller MN, et al. 2009. Fatherhood, pairbonding, and testosterone in the Philippines. Horm Behav 56:429–435.

98 Fleming AS, Corter C, Stallings J, et al. 2002. Testosterone and prolactin are associated with emotional responses to infant cries in new fathers. Horm Behav 42:399–413.

99 Jasienska G, Jasienski M, Ellison PT. 2012. Testosterone levels correlate with the number of children in human males, but the direction of the relationship depends on paternal education. Evol Hum Behav 33:665–671.

100 Mazur A. 2014. Testosterone of young husbands rises with children in the home. Andrology 2:125–129.

101 Trumble BC, Cummings DK, O'Connor KA, et al. 2013. Age-independent increases in male salivary testosterone during horticultural activity among Tsimane forager-farmers. Evol Hum Behav 34:350–357.

102 Thierry B, Anderson JR. 1986. Adoption in anthropoid primates. Int J Primatol 7:191–216.

103 Geary DC, Bailey DH, Oxford J. 2011. Reflections on the human family. In: Salmon CA, Shakelford Todd K, editors. The Oxford handbook of evolutionary family psychology. Oxford University Press. p 365–385.

104 Fuentes A, Wyczalkowski MA, MacKinnon KC. 2010. Niche construction through cooperation: a nonlinear dynamics contribution to modeling facets of the evolutionary history in the genus homo. Curr Anthropol 51:435–444.

105 West-Eberhard MJ. 2003. Developmental plasticity and evolution. Oxford University Press.

106 Harris JA, Vernon PA, Boomsma DI. 1998. The heritability of testosterone: a study of Dutch adolescent twins and their parents. Behav Genet 28:165–171.

107 Dawson A, King VM, Bentley GE, et al. 2001. Photoperiodic control of seasonality in birds. J Biol Rhythms 16:365–380.

108 Maruska KP, Fernald RD. 2011. Social regulation of gene expression in the hypothalamic-pituitary-gonadal axis. Physiology 26:412–423.

109 Marks J. 2003. What it means to be 98% chimpanzee: apes, people, and their genes. Oakland: University of California Press.

110 Ohlsson C, Wallaschofski H, Lunetta KL, et al. 2011. Genetic determinants of serum testosterone concentrations in men. PLoS Genet 7:e1002313.

111 Mitchell-Olds T, Willis JH, Goldstein DB. 2007. Which evolutionary processes influence natural genetic variation for phenotypic traits? Nat Rev Genet 8:845–856.

112 Solomon NG, Hayes LD. 2012. The biological basis of alloparental behaviour in mammals. In: Bentley GR, Mace R, editors. Substitute parents: biological and social perspective on alloparenting across human societies. New York: Berghahn Books, p 13–49.

113 Sjoeholm A, Bridges RS, Grattan DR, et al. 2011. Region-, neuron-, and signaling-pathway-specific increases in prolactin responsiveness in reproductively experienced female rats. Endocrinology 152:1979–1988.

114 Ivell R, Walther N. 1999. The role of sex steroids in the oxytocin hormone system. Mol Cell Endocrinol 151:95–101.

115 Schoech SJ, Mumme RL, Wingfield JC. 1996. Prolactin and helping behaviour in the cooperatively breeding Florida scrub-jay, *Aphelocoma c. coerulescens*. Anim Behav 52: 445–456.

116 Lozoff B, Felt BT, Nelson EC, et al 1995. Serum prolactin levels and behavior in infants. Biol Psychiatry 37:4–12.

117 Martin J, Kagerbauer SM, Schuster T, et al. 2014. Vasopressin and oxytocin in CSF and plasma of patients with aneurysmal subarachnoid haemorrhage. Neuropeptides:epub.

118 Landgraf R, Neumann ID. 2004. Vasopressin and oxytocin release within the brain: a dynamic concept of multiple and variable modes of neuropeptide communication. Front Neuroendocrinol 25:150–176.

119 Churchland PS, Winkielman P. 2012. Modulating social behavior with oxytocin: how does it work? what does it mean? Horm Behav 61: 392–399.

120 Born J, Lange T, Kern W, et al. 2002. Sniffing neuropeptides: a transnasal approach to the human brain. Nat Neurosci 5:514–516.

121 Chen X, Fawcett J, Rahman Y, et al. 1998. Delivery of nerve growth factor to the brain via the olfactory pathway. J Alzheimer's Dis 1: 35–44.

122 Meyer-Lindenberg A, Domes G, Kirsch P, et al. 2011. Oxytocin and vasopressin in the human brain: social neuropeptides for translational medicine. Nat Rev Neurosci 12:524–538.