One does not need an advanced degree to appreciate that humans are highly social. Our prolonged period of immaturity demands constant care and protection in early life, a condition that compels the intense bonds that develop between infants and caregivers (Bowlby, 1969). But our propensity for enduring attachments extends beyond the infant–caregiver relationship; humans form similar bonds with close others throughout the life span (Mikulincer & Shaver, 2007). Group living, more generally, confers many advantages—from sharing resources and labor to fending off predators. Indeed, humans possess a sophisticated skill set to facilitate social life: our ability to identify the thoughts and feelings of others, empathize with and help others in need, and cooperate. Given the necessity of relationships for survival, biological mechanisms have likely evolved to support the initiation and maintenance of social bonds. Although the candidates are numerous—and likely do not operate in isolation—one that has received considerable attention is oxytocin.

Here, I review research on oxytocin and human social cognition and behavior; although other methodological approaches have been used (e.g., measuring peripheral oxytocin and oxytocin-related genes), I focus on work in which oxytocin has been pharmacologically manipulated to probe its functional role and test causality. This work implicates oxytocin in human sociality; however, oxytocin’s effects are neither simple nor straightforward, with oxytocin augmenting prosocial cognition and behavior in some situations or for some individuals, but undermining it in other situations or for other individuals. I argue that such variability is not necessarily random error but may offer clues about underlying processes, the understanding of which is critical for a comprehensive account of human affiliation.

Oxytocin and Affiliation

Oxytocin is a hormone known largely for its role in parturition and lactation; however, oxytocin also acts as a neuromodulator in the brain, where it has widespread distributional effects that impact several behavioral systems, including the complex social processes related to affiliation (see Stoop, 2012). For example, abundant research in nonhuman animals shows that oxytocin is

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Abstract

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Keywords
oxytocin, attachment, social, motivation, individual differences, intranasal, Syntocinon

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Oxytocin and the Pharmacological Dissection of Affiliation

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Abstract

Popularly hailed as the “love hormone,” oxytocin has emerged as a key variable in the regulation of human social cognition and behavior. In particular, research using intranasal oxytocin to pharmacologically manipulate the availability of oxytocin shows that oxytocin augmentation can promote a wide range of affiliative processes; however, evidence also shows null and even antisocial effects. Rather than random error to be eliminated, such variability may offer clues about the mechanisms by which oxytocin modulates human sociality. Three potential mechanisms—anxiety reduction, social salience, and affiliative motivation—are discussed, along with recent work showing how the affiliative-motivation hypothesis can simultaneously account for oxytocin’s pro- and antisocial effects. Appreciating oxytocin’s nuanced social effects is important for advancing our understanding of the neuroscience and psychology of affiliation.

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critical for (a) triggering the onset of maternal behavior in numerous species, including rats (Pedersen, Ascher, Monroe, & Prange, 1982), mice (Takayanagi et al., 2005), and sheep (Kendrick, Keverne, & Baldwin, 1987); (b) facilitating adult–adult pair-bonding in monogamous voles (Williams, Insel, Harbaugh, & Carter, 1994); and (c) supporting social memory (Ferguson et al., 2000)—a prerequisite for attachment, since attachment bonds are formed with specific individuals. (See the Recommended Reading section for comprehensive reviews of the animal literature.)

Compared to the animal work, research investigating the functional role of oxytocin in human sociality has lagged because of methodological challenges manipulating oxytocin in humans. This changed, however, when Born and colleagues (2002) showed that administering peptides like oxytocin via nasal spray can alter central levels (but see Leng & Ludwig, 2015). Following this advance, there was a dramatic increase in publications on this topic with some notable parallels to the animal literature. For example, in an early and influential study, Kosfeld, Heinrichs, Zak, Fischbacher, and Fehr (2005) administered intranasal oxytocin (Syntocinon Nasal Spray) to participants who then played the “trust game,” in which they (the “investors”) were given money and could transfer any amount to their partner (the “trustees”). Because the amount transferred triples in value, investors should, theoretically, transfer the whole allotment—but this assumes trustees will share their earnings, an assumption many people are reluctant to make. Astoundingly, participants receiving oxytocin transferred significantly more money than those receiving placebo, suggesting that oxytocin plays a critical role in human trust. Following this groundbreaking study, subsequent research showed that oxytocin facilitates various prosocial processes including cooperation, generosity, liking, empathic accuracy and emotion sharing, gaze to the eye region, and memory for faces (see Bartz, Zaki, Bolger, & Ochsner, 2011, and the Recommended Reading section for reviews). These and other such studies transformed oxytocin from a hormone known mainly for its labor-inducing properties to the “love hormone”—a panacea for our social deficiencies.

The Social Effects of Oxytocin in Humans Are Nuanced

Studies, however, also emerged showing opposite (anti-social) or moderated effects, prompting a more nuanced view (Bartz, Zaki, et al., 2011). Take the oxytocin–trust finding: Subsequent studies have indicated that oxytocin decreases trust and/or cooperation when people are interacting with complete strangers (Declerck, Boone, & Kiyonari, 2010) or out-group members (De Dreu et al., 2010). Similarly, individual differences in the extent to which others are chronically perceived as trustworthy and reliable attenuates the social effects of oxytocin. My colleagues and I (Bartz, Simeon, et al., 2011) administered oxytocin or placebo to adults with borderline personality disorder (BPD; a psychiatric disorder marked by chronic fears about separation and abandonment). Participants then played the “assurance game” (AG), a variation on the classic “prisoner’s dilemma” (PD), with a partner. Whereas the PD allocates the highest payoff when participants defect and their partners cooperate, the AG allocates the highest payoff when participants and their partners jointly choose the cooperative strategy; by incentivizing mutual cooperation in this way, the AG makes salient questions regarding interpersonal trust and, for this reason, was chosen as the dependent variable in this study (of note, the AG was also used in Declerck et al.’s 2010 study). Contrary to the love-hormone hypothesis, oxytocin significantly decreased trusting expectations and the likelihood of hypothetical cooperation in individuals with BPD (a finding replicated by Ebert et al., 2013).

In another study (Bartz et al., 2010), we found that even normal variation in attachment anxiety moderates the effects of oxytocin. Here, we were interested in whether oxytocin supports attachment representations in humans, given the animal work on oxytocin and social memory; to this end, we administered intranasal oxytocin and placebo (within-subjects) and measured memories of maternal care (questionnaire items included, e.g., “was affectionate to me,” “spoke to me in a warm and friendly voice,” and “frequently smiled at me”) and closeness—two features of the attachment bond—in childhood. Consistent with the popular view, securely attached participants remembered their mother as more caring and closer to them in childhood when they received oxytocin compared with when they received placebo. Conversely, anxiously attached participants remembered their mother as less caring and less close to them in childhood when they received oxytocin (vs. placebo). These data suggest that oxytocin does not cast a rose-colored hue for all. Rather, for people who are generally secure, oxytocin brings to mind instances of when mother met their needs for felt security, but for people who are anxiously attached, oxytocin appears to summon memories of when mother’s care and closeness were lacking. To our knowledge, this is the first study to show that such a phasic manipulation can alter people’s childhood memories of their mother; this work also speaks to factors that may influence the stability of attachment (see, e.g., Fraley & Shaver, 2000).

Work in mice shed additional light on these findings. Guzman et al. (2013) placed mice in the cage of an aggressive resident—a “social defeat” experience that
stimulates oxytocin release. Subsequent testing showed that oxytocin had no effect on the initial fear experience; rather, oxytocin potentiated fear conditioning by enhancing the memory of social defeat. Interestingly, another report by Guzman et al. (2014) showed that oxytocin’s effects on social memory were not specific to negative interactions—oxytocin both enhanced fear after negative social interactions and reduced fear after positive social interactions. Consistent with our data on maternal recollection, oxytocin intensified the emotional salience of social memories, be they positive or negative. As Guzman et al. (2013) noted, such bidirectional effects are adaptive. Indeed, a child finding him- or herself in a dangerous situation would not do well to misremember mother as consistently caring and reliable when she is not; such biased memories would likely preclude the attachment-seeking behavior meant to secure the inconsistently responsive caregiver’s attention, leaving the child at greater risk.

**Potential Mechanisms Underlying Oxytocin’s Social Effects**

The findings reviewed here, and other data, suggest that the way oxytocin modulates affiliative behavior in humans is less straightforward than initially thought. In an earlier review (Bartz, Zaki, et al., 2011), my colleagues and I found that just under half of the published findings on oxytocin and prosociality showed no main effect of oxytocin, and 60% showed that oxytocin’s prosocial effects were moderated by person or context; moreover, approximately 20% of the findings showed antisocial effects like those described above. We argued that this variance is not necessarily random error. On the contrary, oxytocin’s context and person dependency may be meaningful and may provide clues about the more basic processes at play.

In particular, we outlined three mechanisms (derived from existing research) by which oxytocin may modulate sociality in humans. First, oxytocin may reduce anxiety and alleviate the inhibition inherent to many social situations (e.g., Heinrichs & Domes, 2008; McCarthy, 1995). Although this hypothesis explains some of findings, the observation that oxytocin can be anxiogenic (Guzman et al., 2013) dampens enthusiasm for the universality of this hypothesis. Second, oxytocin may augment the salience of social cues. This hypothesis is supported by animal work on oxytocin and social recognition/memory (e.g., Kendrick, Levy, & Keverne, 1992) and recent human work showing that oxytocin biases attention to social cues such as eye gaze. Third, oxytocin may enhance the desire or goal to affiliate. This hypothesis is based on the considerable work on oxytocin and maternal and pair-bonding behavior in animals and, particularly, vole research linking between- and within-species differences in affiliation with the density of oxytocin receptors in brain regions involved in reward and reinforcement (see Ross & Young, 2009). To date, little work has investigated whether any of these mechanisms account for oxytocin’s social effects in humans (see Bartz, Zaki, et al., 2011, for detailed discussion). Although the jury is still out, my colleagues and I have recently been focusing on the affiliative-motivation hypothesis.

**Oxytocin Induces a Motivational State to Affiliate**

As we have noted (Bartz, Lydon, et al., 2015), a careful review indicates that the person-dependent effects of oxytocin are somewhat systematic: Augmenting oxytocin appears to increase prosocial behavior and cognition, particularly among those who are less socially attuned and/or motivated (e.g., avoidantly attached individuals, those on the autism spectrum), but exacerbate interpersonal insecurities in those who are preoccupied with and anxious about interpersonal closeness (e.g., anxiously attached individuals, those with BPD). One way to approach this puzzle is from an interactionist perspective. People differ in how they encode and construe situations, their chronic expectancies and affective responses, their goals and values, and the strategies they possess for affecting outcomes; according to Mischel and colleagues (e.g., Mischel & Shoda, 1995), these factors combine to produce unique if-then contingencies that guide people’s responses to specific situations. The individual differences in response to oxytocin could thus be due to the way people’s if-then contingencies interact with a state of heightened affiliation.

Along these lines, we theorized that if oxytocin acts in a normative way to heighten affiliative strivings (and the subjective value of social connection), then those who are least socially motivated at baseline, such as the avoidantly attached, should show the most pronounced increases in affiliation from oxytocin. However, oxytocin should not be especially helpful (socially) to those who are sufficiently affiliative/communal; moreover, oxytocin might be detrimental to those individuals for whom the pursuit of affiliation and agentic goals are at odds, such as the anxiously attached. Indeed, prior work indicates that when affiliative strivings are activated contextually, anxiously attached individuals become more anxious and uncertain and less self-confident—that is, less agentic (Bartz & Lydon, 2006). Based on this, we theorized that anything that enhances affiliative strivings—such as oxytocin—could threaten the fulfillment of agentic goals in the anxious.

Having first established in an independent sample (N = 534) that, at baseline, attachment avoidance is
negatively associated with affiliation/communion, whereas attachment anxiety is positively (albeit modestly) associated with communion and negatively associated with agency, we then investigated whether oxytocin differentially affects communion and agency for avoidant and anxiously attached individuals. Results showed that oxytocin produced a slight increase in communion for the average participant, but, as predicted, and consistent with an interactionist perspective, avoidant individuals (who are typically low in communion) showed the most pronounced increase, describing themselves as significantly more “gentle,” “kind,” “warm,” and so on following oxytocin administration. Additionally—and aside from the effects on affiliation—we found that oxytocin selectively decreased agency in anxiously attached participants, who also described themselves as less “self-confident” and more “inferior” following oxytocin administration (Bartz, Lydon, et al., 2015).

We argue that this weakening of agency may account for some of the antisocial effects of oxytocin. Threats to agency (self-efficacy, personal control) typically result in a host of negative outcomes including depression, alienation, emotion dysregulation, and maladaptive coping (Bandura, 1982; Ryan & Deci, 2000). Moreover, individuals with low self-esteemagency often prioritize self-protection over prosociality (Murray, Holmes, & Collins, 2006). Thus, if oxytocin threatens agency in anxious-secure individuals, they may react to such feelings of vulnerability by defensively protecting the self and/or lashing out at others, as was observed in our BPD study. This perspective also sheds light on data showing that oxytocin increases inclinations toward interpersonal violence among high trait aggressive individuals (DeWall et al., 2014), since such inclinations may be part of their unique interpersonal if-then contingency. In sum, these data suggest that affiliative motivation (along with its paradoxical effects in some interpersonally vulnerable individuals) may be one mechanism by which oxytocin modulates sociality in humans. Although, to date, direct tests of this hypothesis in humans are limited, the idea is consistent with much of the human data (e.g., Kosfeld et al., 2005) and with the large body of work in nonhuman animals on oxytocin and maternal behavior and bonding.

A Model for the Social Effects of Oxytocin

The data reviewed indicate that the love-hormone view of oxytocin is overly simplistic and that oxytocin’s social effects depend critically on the situation and person; a model incorporating these influences is depicted in Figure 1. Consistent with the popular view, oxytocin augments affiliative goals and their subjective value and prompts behaviors such as trust (Kosfeld et al., 2005) to facilitate social connection. However, oxytocin’s effects on such prosocial behaviors will depend on whether the environment is affiliation-goal supportive; if there are significant barriers to closeness—for example, if one is interacting with a stranger or an out-group member—oxytocin will not increase trust and may even undermine it (Declerck et al., 2010; De Dreu et al., 2010). Additionally, oxytocin’s prosocial effects will depend on person characteristics; for example, as described, oxytocin can decrease trust during a novel social interaction in those who are chronically concerned about whether they are worthy of affection and/or whether others are reliable (Bartz, Simeon, et al., 2011).

Of note, there are several routes by which person characteristics may influence oxytocin’s prosocial effects. First, negative expectancies based on prior interpersonal experiences could influence whether the situation is perceived as supportive—even if the context generally bodes well for social connection, negative expectancies could attenuate predictions of whether one’s social overtures, for example, will be reciprocated and temper prosocial behavior accordingly. Another source of influence is habitual tendencies and/or associated goals. We know from research on social cognition that our goals and the “action plans” we habitually use to achieve those goals are cognitively linked; when a goal is activated, so too is the associated action plan (Aarts & Dijksterhuis, 2000; Bargh, 1990; Custers & Aarts, 2010). Thus, if one typically pursues closeness by being submissive, we would expect oxytocin to produce submissive tendencies in such individuals, even in novel situations. Similarly, goals can activate related goals. For example, affiliation and self-protection goals might be closely linked for those who have experienced significant interpersonal harms. If the goal to affiliate is activated, self-protection goals could be activated even if self-protection is unwarranted in the new context. Finally, other potential influences to consider in future work include self-regulatory capacity, emotion regulation styles, and social skills (knowledge about, e.g., effective support provision).

Outstanding Questions

This is a relatively new research area; below I discuss some outstanding questions pertaining to the work reviewed here. First, I focused on the affiliative-motivation hypothesis, but this does not disqualify the social-salience or anxiety-reduction hypotheses. Indeed, these mechanisms may be interrelated. In particular, increasing affiliative motivation should bias attention to cues relevant to pursuing this goal (i.e., social information). Conversely, increasing the salience of social cues could elicit affiliative goal-directed behavior. Indeed, Marlin, Mitre, D’Amour, Chao, and Froemke (2015) recently showed that oxytocin enables maternal behavior (pup retrieval)
in virgin female mice by modulating neural circuits to enhance the acoustic salience of pup cries—essentially, prompting affiliation by transforming an irrelevant stimulus into one that has personal significance.

Second, notwithstanding Born et al. (2002), questions remain about whether intranasal oxytocin crosses the blood-brain barrier (BBB; e.g., Leng & Ludwig, 2015). Studies addressing this have shown inconsistent results, but, if oxytocin is not (consistently) crossing the BBB, this could explain why “main effect” findings have been sporadic (cf. Bartz, Zaki, et al., 2011). It is less obvious, however, how this hypothesis explains oxytocin’s divergent (antisocial) effects or the aforementioned pattern of person-dependent effects.

Third, the human data supporting the affiliative-motivation hypothesis are largely based on males (e.g., Kosfeld et al., 2005). Given that some (but not all) research suggests that oxytocin’s effects can be sexually dimorphic (MacDonald, 2013), might the effects of oxytocin on affiliative motivation be specific to men? This seems unlikely, in view of the extensive work on oxytocin and maternal behavior. That said, gender differences on measures of affiliation could preclude the detection of an effect in one group or render one group more sensitive to change, thus giving the impression of sexual dimorphism.

Finally, other than illuminating neurobiology, does (or can) this research agenda extend our knowledge of human attachment and affiliation? The future will reveal the full impact of this work; however, knowing that distinct psychological processes (e.g., infant–caregiver attachment and in-group/out-group behavior) share a common neurobiology invites hypotheses about other areas of overlap that could broaden our understanding of these psychological processes in unexpected ways. Additionally, I would argue, this work offers a tool to prime affiliation in a way that circumvents much of the variance associated with psychological approaches. Psychological priming typically uses words or pictures, but the associated meanings can vary; such procedures are also vulnerable to defensive processes. Manipulating oxytocin pharmacologically to prime affiliation, however, potentially offers a more direct route to activating this goal state and thus allows for a richer understanding of human attachment and affiliation. Relatedly, as the title of this article suggests, we also have a tool to “pharmacologically dissect” (e.g., Soloff, 2000, p. 172) the complex construct of human attachment. Although attachment is recognized to be multifaceted, efforts to parse individual differences in attachment insecurity have largely focused on expectancies. That oxytocin
modulates social motivation/reward and is selectively beneficial for avoidant (but not anxious) individuals suggests it is not only expectancies that distinguish avoidants but also the value they attach to closeness (Bartz, Baldwin, & Lydon, 2015). Disentangling the complexities of attachment has been difficult to do empirically, and understanding the neurobiology may be a way to tackle this issue.

Concluding Comments

Humans are distinguished by a propensity to affiliate—to form and maintain close bonds with others. Research in nonhuman animals highlights the neurohormone oxytocin in regulating this basic drive. Recent human work using intranasal oxytocin as a pharmacological probe to mimic endogenous release supports the view that oxytocin has activational effects, prompting individuals to assume roles and engage in behaviors to facilitate social connection. Critically, however, the specific roles and behaviors adopted will depend on features of the situation and individuals’ unique if-then contingencies for affiliation. Appreciating this nuance is important, as it can help gauge the suitability of oxytocin pharmacotherapy (a worthwhile objective given recent interest—e.g., https://clinicaltrials.gov/ct2/show/NCT01944046) and provide greater conceptual clarity and predictive power in our efforts to chart the neuroscience and psychology of affiliation.

Recommended Reading


Ross, H. E., & Young, L. J. (2009). (See References). A clearly written and relatively comprehensive review for readers who wish to expand their knowledge on oxytocin and affiliation in nonhuman animals.

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References


