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The Moderating Role of Oxytocin in the Relationship between Intergroup Bias and Disgust

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Existing literature suggests that the human disgust response is susceptible to intergroup bias effects; that is, a disgusting stimulus evoked by an out-group member might cause greater perceived disgust than that from an in-group member. The following theoretical paper expands upon an idea explored previously by Kavaliers and Choleris (2013) and proposes that the effects of intergroup bias on the disgust response is moderated by the neuropeptide oxytocin, which has been shown to be associated with social recognition activities such as in-group favoritism/out-group derogation. Achieving a better understanding of the role of oxytocin in moderating effects of intergroup bias may reveal interesting consequences of interpersonal relations on physiological well-being and its behavioral determinants.

Keywords: oxytocin, intergroup bias, disgust, social recognition, disease avoidance

Des études récentes suggèrent que chez l'humain, la réponse de dégout est influencée par les effets des biais endogroupe et exogroupe. En d'autres mots, un stimulus dégoutant produit par un membre d'un exogroupe pourrait être perçu comme étant plus dégoutant que s'il avait été produit par un membre de l'endogroupe. Ce présent article développe une idée étudiée par Kavaliers et Choleris (2013) et suggère que l'effet du biais intergroupe sur la réponse de dégout est modérée par l'oxytocine, un neuropeptide qui a été associé à des activités de reconnaissance sociale tel le biais pro endogroupe et la dérogation de l'exogroupe. Une meilleure compréhension du rôle modérateur de l'oxytocine dans les effets du biais intergroupe pourrait révéler les effets des relations interpersonnelles sur le bien-être physiologique et ses déterminants comportementaux.

Mots-clés : ocytocine, biais intergroupe, dégoût, reconnaissance sociale, évitement des maladies

Research has demonstrated that intergroup bias affects the human disgust response. It has been found that people perceive a greater degree of disgust towards individuals whom they consider to be members of an out-group than they perceive towards individuals whom they consider to be members of an in-group (Oaten, Stevenson, & Case, 2009). Empirical data from the late 1990s found that disgust is heightened by bias towards an out-group, as Schiefenhövel (1997) discovered that ethnic out-groups often instigated disgust reactions from people (Oaten et al., 2009). Furthermore, social behaviors associated with out-group bias, such as ethnocentrism and prejudice, are associated with greater levels of disgust (Kavaliers & Choleris, 2011; Schaller & Murray, 2008). On the other hand, researchers have found that a disgusting stimulus is rated as less disgusting when invoked by an in-group member (Oaten et al., 2009).

Following attempts to unravel the relationship between intergroup bias (a social influence) and disgust (a physiological response), the mammalian neuropeptide oxytocin has emerged as a molecule that

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links the two variables. Past experiments conducted by Martin Kavaliers and Elena Choleris (2013), experts on the neuropeptides oxytocin and its structural analogue arginine vasopressin, has revealed that oxytocin plays an important role in the processing of intergroup bias. The studies both conducted and compiled by Kavaliers and Choleris (2013) underscore significant evidential support for the idea that oxytocin plays a very important role in social recognition. These two experts have traced the ability to identify social cues to the recognition and processing of odors; additionally, they have elaborated on the importance of being conscious of odor cues for distinguishing normal from diseased mates in sexual selection processes. Furthermore, they have conducted experiments that support the role of oxytocin as a molecule that functions in processing social stimuli in the context of disease transmission, in which disgust may manifest.

While social neuroscientists have ascertained intergroup bias as a source of influence on the human disgust response, this model possesses significant limits: The mechanism by which the strength of the relationship between intergroup bias and disgust may be altered remains unknown. Past work on oxytocin has insinuated that oxytocin mediates the effects of intergroup bias on disgust because of its importance in processing social cues; in functioning as a mediator, oxytocin would serve to justify the presence of the disgust response that result from varying levels of intergroup bias. However, the following theoretical article proposes that oxytocin serves as a moderator in the relationship between disgust and intergroup bias. As a moderator, oxytocin accentuates the effects of intergroup bias on disgust, rendering the relationship between these two variables stronger. Oxytocin possibly serves as a moderator via a central neuroendocrine mechanism that merits further empirical exploration. In the present article, the rationale underlying oxytocin's moderating role will be addressed. There will also be description of the central neuroendocrine mechanism by which oxytocin may moderate the effects of intergroup bias on disgust, which includes oxytocin signaling and genetic predisposition, both of which may yield either a stronger or weaker level of perceived disgust towards a social stimulus. Finally, experiments that would further validate the proposed role of oxytocin as a moderator between intergroup bias and disgust will be suggested.

Disgust as a Universal Emotion that Arises from Disease Avoidance

Disgust is defined as "a feeling of revulsion, sometimes accompanied by nausea, along with a strong desire to withdraw from the eliciting stimulus" (Oaten et al., 2009, p. 303) and may refer to distaste in various domains. In the present article, the disgust to be addressed arises from the possibility of interacting with a negative health consequence; this form of disgust is classified as "pathogen disgust" (Tybur, Lieberman, & Griskevicius, 2009). Other forms of disgust (such as sexual disgust or moral disgust) would be those that are evoked by aberrations of typical and socially acceptable expectations; neither of these forms of disgust will be discussed. Pathogen disgust, hereby referred to as "disgust," may be generated from unhygienic situations such as inappropriate preparation of food, uncleanliness in public domains (such as feces remaining from the previous occupant in a public toilet), infected open wounds, and airborne transmission of microbes (Curtis, 2007).

The disgust response is distinctly identifiable; the first action characterizing disgust includes strong withdrawal from negative stimuli. Following this strong initial response, humans engage in other characterizable actions as part of the disgust response mechanism, both behavioral and physiological in nature (Oaten et al., 2009). The behavioral responses employed by humans include visibility of the tongue as it emerges from the mouth, narrowing of the brows, a wrinkled nose, and a curled upper lip (Rozin, Lowery, & Ebert, 1994). The physiological responses employed by humans (generated by the autonomic nervous system) include reduced blood pressure, among other cardiac measures, as well as decreased skin conductance (Stark, Walter, Schienle, & Vaitl, 2005). The human disgust response varies among individuals with regard to actions that can be controlled (e.g., facial expression) but appears consistent across physiological measures outside of one's control. Moreover, since the disgust response is considerednormative in individuals, it may be generated experimentally from specific images organized by the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 2008) that are intended to convey pathogen disgust. Such images include insects on food, an eye tumor, and mutilation of faces (Lang et al., 2008). The human disgust response's frequency, recognizable nature, and concurrence with the aformentioned behavioral and

physiological symptoms have rendered disgust a subject of extensive study.

Disgust responses to stimuli vary depending on the relationship between the stimulus or individual eliciting the stimulus and the individual perceiving this stimulus and generating a response. It is believed that the disgust response depends upon group bias, with individuals demonstrating varying levels of disgust for members of an in-group (a group with which one identifies) and an out-group (a group with which one does not identify; Oaten et al., 2009).

Variation in the Disgust Response Caused by Intergroup Biases

Experiments studying the disgust response have demonstrated that the amount of disgust experienced by an individual depends of the relationship that exists between the individual and the disgusting stimulus perceived. Specifically, these studies have provided evidence that a disgusting stimulus evoked by an in-group member yields less perceived disgust as compared to an out-group member eliciting the same disgusting stimulus. For example, an experiment conducted by Case, Repacholi, and Stevenson (2006) found that mothers exposed to their own baby's soiled diaper rated it as less disgusting as compared to the soiled diaper of somebody else's baby during blind trials, in which the mother was asked to sample diapers without knowing which diaper belonged to her child. Additionally, mothers rated the smell of their own baby's soiled diaper less disgusting as compared to the soiled diaper of another child (Case et al., 2006). These findings demonstrate that, even when mothers were not made aware that they had smelled feces from their own child, they experienced less disgust when confronted with a disgusting stimulus from their own child than when confronted with a disgusting stimulus from the child of a stranger or an out-group member.

Other studies looking at the disgust response found that negative affect increased in individuals when a body odor (in the form of feces, sweat, flatulence, etc.) derived from a stranger rather than from oneself (Stevenson & Repacholi, 2005) and that individuals considered body fluids more disgusting when emitted by strangers than when emitted by a close relative (Curtis, Aunger, & Rabie, 2004). Curtis et al. (2004) asked participants to identify the individuals with whom they would feel the greatest disgust from sharing their toothbrush. This study found that the amount of disgust perceived by sharing a toothbrush (and hence spreading germs) decreased as the relationship between the individuals sharing the toothbrush transitioned from out-group members to in-group members. While 59% of individuals identified their post officer as an individual with whom they would refrain most from sharing their toothbrush, only 24.7% of individuals identified their "boss at work." The likelihood of feeling disgust decreased when individuals were considered members of their in-group, such as family and friends, with only 3.3% of participants indicating "a sibling," 1.9% of participants indicating "a best friend," and 1.8% of participants indicating "a partner/spouse" as someone with whom they would dislike sharing a toothbrush (Curtis et al., 2004; Oaten et al., 2009). Based on these results, research on disgust responses has found that in -group favoritism predicts the extent to which individuals perceive a stimulus as disgusting.

The important role played by intergroup bias derives from ancestral environments; in their discussion of disgust, Oaten et al. (2009) pointed out that interacting with an in-group member would inherently pose less risk of infection because outsiders may introduce infectious agents to a population lacking immunity, whereas an in-group member would not. Thus, the evolutionary roots of out-group bias exist alongside those of disease avoidance; based on these behavioral tendencies that carry over from ancestral environments, individuals avoid communicable diseases through disgust (Faulkner, Schaller, Park, & Duncan, 2004) because disgust allows individuals to physically separate themselves from others (Rozin, Haidt, & McCauley, 2000). Therefore, social behavior may have been birthed from wariness of disease transmission. To supplement this disease avoidance rationale for intergroup bias, Rozin and colleagues (2000) point out that any form of contact with out-group members rendered unwelcome can be categorized as a form of interpersonal contamination from a social standpoint. This historically founded belief stemming from avoidance of disease transmission possibly fuels the presence of intergroup bias among modern societies today.

The effects of intergroup bias on disgust have also been studied in ethnic contexts, with individuals believing that increased contact with ethnic out-group members increases the likelihood of disease transmission. Members of foreign out-groups have been compared to animals associated with disease

transmission; such creatures include cockroaches, rats, maggots, lice, and flies (Suedfeld & Schaller, 2002). Ethnic out-group members have also been faulted for disease outbreaks; consequently, genocide perpetrators engage in "ethnic cleansing" that follows a disease model, as indicated by the usage of terms like "Jewish vermin" or "Tutsi cockroaches" (Navarette & Fessler, 2007). Faulkner et al. (2004) demonstrated that chronically elevated levels of concern about disease transmission caused negative reactions toward individuals of a different nationality, which resulted in xenophobia, or a fear of foreigners.

Because of the importance of ethnic contexts in analyzing perceived disgust, various experiments examining the effects of in-group and out-group biases have sought to study participants' reactions to stimuli associated with a variety of races. Overall, researchers have used the term "source effect" to describe their findings that disgust is evoked to a greater extent by strangers (out-group members) than by family, friends, or oneself (in-group members; Case et al., 2006; Stevenson & Repacholi, 2005). Oaten et al. (2009) have provided support for the source effect in their detailed account of disgust as a disease-avoidance mechanism. To explain the link that exists between the external social cue of intergroup bias and the normative reaction of the human disgust response, existing social neuroscience literature has targeted the neuropeptide oxytocin.

Oxytocin, Intergroup Bias, and Disgust

Oxytocin is a mammalian neuropeptide hormone secreted by the posterior pituitary gland to perform neuromodulatory functions; it is known for physiologically inducing uterine contractions, which are important for dilation of the cervix prior to birth (Wiqvist, Norström, & Wiqvist, 1984). Oxytocin carries an interesting history as it was, up until recently, colloquially regarded as a "love hormone" because it was thought to promote intimacy and bonding (Magon & Kalra, 2011). However, recent research now reveals that oxytocin functions in social recognition outside of merely prosocial tendencies. Oxytocin is a neuropeptide that is best known for its social role in non-human primates and other mammals; in rats, oxytocin plays a role in social recognition, maternal behaviors, pair bonding, and affiliation (Donaldson & Young, 2008). However, oxytocin has also been shown in experiments to play a role in-group favoritism in humans (Schaller & Murray, 2008; De Dreu, Greer, Van Kleef, Shalvi, & Handgraaf, 2011). Increases in oxytocin have been directly linked to in-group favoritism as evidenced by self-report measures before and after the administration of intranasal oxytocin (De Dreu et al., 2011). Moreover, Kavaliers and Choleris (2011) found that intranasal administration of oxytocin facilitates recognition of words pertaining to relationships in humans (Unkelbach, Guastella, & Forgas, 2008), illustrating the role of oxytocin in social recognition and ostensibly in processing in-group and out-group cues. Additionally, intranasal oxytocin has increased perceived trustworthiness in faces for social stimuli but not for non-social stimuli (Theodoridou, Rowe, Penton-Voak, & Rogers, 2009), enhanced cooperation in financial games involving a partner (Kosfeld, Heinrichs, Zak, Fischbacher, & Fehr, 2005), and induced feeling of familiarity in affiliative social stimuli such as images of people smiling (Guastella, Mitchell, & Mathews, 2008). These findings indicate that oxytocin boosts feelings of trust and familiarity in the context of interpersonal behaviors.

Overall, the numerous social behaviors associated with oxytocin that allow for intergroup bias to manifest may be classified in various categories, including political attitudes, assortative behaviors (defined as behaviors involving the desire to mingle with others who are similar in genotype and/or phenotype, such as mating), and introversion/ extraversion. In some psychology experiments, oxytocin caused prejudice (preconceived notions of others that are not founded in experience) and ethnocentrism (the tendency to view one's in-group as superior to others), both of which serve as manifestations of in-group favoritism (De Dreu et al., 2011). High oxytocin levels occur in mothers during their first trimester of pregnancy, a period that marks the time during which they remain most susceptible to infection; additionally, these women simultaneously demonstrate accentuated levels of ethnocentrism (Navarette & Fessler, 2007). While oxytocin enhances in-group favoritism, to a lesser extent, it has also been shown by studies to promote out-group derogation (De Dreu et al., 2011).

Based on these studies, researchers previously believed that oxytocin linked the effect of intergroup bias on the disgust response. In experiments with rodents, the oxytocin receptor gene has been knocked out (which signifies removing the receptor), and studies following this methodology have demonstrated that rodents lacking the oxytocin receptor cannot recognize familiar individuals, or in-group members

(Kavaliers & Choleris, 2011). In this way, these researchers classified oxytocin as a mediator of the relationship between intergroup bias and disgust, that is, a variable that serves to explain the link between the two variables. Kavaliers and Choleris (2013) have proposed that oxytocin enables social recognition, which has allowed for oxytocin to emerge as a candidate that explains the direct link between the effects of intergroup bias on perceived disgust. However, the mediation model does not clearly delineate oxytocin's role in mediating the specific relationship between intergroup bias and disgust. While these researchers' work conclusively identifies oxytocin as an important molecule in social recognition, such studies have only examined the role of oxytocin in disease avoidance. These experiments have not yielded definitive evidence to suggest that the effects of oxytocin impact the disgust response itself, especially because this research is limited to animal models such as rats. For example, rodent studies revealed that female rodents tended to avoid infected males based on the effects of oxytocin; however, these studies did not directly study disgust responses and were limited to sexual interactions in rats as opposed to situations of intergroup bias.

Furthermore, while oxytocin has been shown to play a role in social recognition, its role in affecting the strength of the relationship between intergroup bias and disgust has not yet been addressed in research efforts. This article proposes that oxytocin moderates the effects of intergroup bias on disgust; in other words, an increase in oxytocin levels and oxytocin signaling would strengthen the relationship between intergroup bias and disgust. Oxytocin exerts its effects on the body via various mechanisms, and its biological variability within the human body could explain how a single disgusting stimulus may be interpreted differently among various types of relationships.

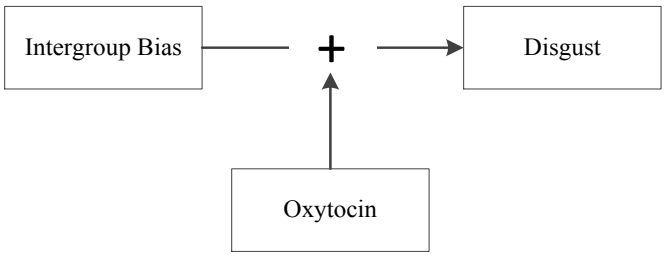

Figure 1. Oxytocin as a moderator of intergroup bias on disgust.

Oxytocin as the Proposed Moderator Between Intergroup Bias and Disgust

While past research has noted the relationship between intergroup bias and disgust, this model is limited because it is unclear how this relationship may be made stronger or weaker. The present article proposes that oxytocin serves as a moderator between intergroup bias and disgust (see Figure 1). Whereas the mediation model described by past researchers would suggest that oxytocin merely serves to account for the influence of intergroup bias (an external social phenomenon) on the disgust response (a physiological reaction), the moderation model proposed here suggests that oxytocin influences the strength of the relationship between intergroup bias and disgust. More specifically, intergroup bias is tied to perceived disgust, strongly in the presence of higher levels of oxytocin or oxytocin signaling. It is believed that oxytocin serves as a moderator of the relationship between intergroup bias and disgust because its effects may yield a wide range of disgust responses for a particular stimulus. This may be due to many reasons, including variations in oxytocin signaling, an individual's genetically predetermined oxytocin receptor type, and the presence of dense populations of oxytocin receptors in areas of the brain.

It is plausible that oxytocin moderates the relationship between intergroup bias and disgust: As oxytocin signaling occurs in numerous different ways, the magnitude of oxytocin's effects may directly influence the degree to which intergroup bias affects disgust. The exact mechanism by which oxytocin moderates the effects of intergroup bias on disgust remains unclear; however, the effects of oxytocin on moderating the human disgust response occur through various processes within the body, and one such process is cell signaling via a central neuroendocrine pathway.

It has been suggested by Kavaliers and Choleris (2013) that oxytocin's function within the aforementioned central neuroendocrine pathway may largely relate to odor cues. Odor-based social recognition in non-human mammalian models has been associated with other molecules expressed within the body: the major histocompatibility complex (MHC), defined as "a large cluster of polymorphic genes coding for the molecules involved in the adaptive (as opposed to the innate) immune response" (Kavaliers & Choleris, 2013, p. 258; see also Milinski, 2006) and major urinary proteins

(MUPs), which function in social and individual recognition and are often found in the salivary and lachrymal glands (Hurst & Beynon, 2004; Kavaliers & Choleris, 2013).

Relationships functioning endogenously (within the body) at a molecular level frequently involve numerous signaling cascades that are initiated or perpetuated by the binding of ligands to receptors. The central neuroendocrine mechanism supported by Kavaliers and Choleris (2013) would involve a signaling cascade with broad effects throughout the body, which emphasizes oxytocin's endogenous role as a systemically circulating molecule and may explain the versatility of oxytocin in social recognition and behavior. As with other neuropeptides and hormones in the body, there is a direct relationship between increased oxytocin signaling and oxytocin function. Enhanced release of oxytocin leads to larger amounts of oxytocin binding to oxytocin receptors, which leads to oxytocin exerting its effects on the body to a greater extent. Based on this direct relationship, it is reasonable that enhanced oxytocin signaling serves to strengthen the processing of social stimuli and thereby strengthen the relationship between intergroup bias and disgust.

Furthermore, the molecular basis of oxytocin's endogenous function in social recognition supports oxytocin's role as a moderating variable, as it sheds light upon oxytocin signaling as a critical point for interaction between genetic makeup and social influence. While oxytocin has played a role in social interactions through its administration exogenously in laboratory experiments, oxytocin affects in-group favoritism, sociality, and immunity endogenously via oxytocin signaling. The oxytocin receptor maintains three different single nucleotide polymorphisms (DNA sequence variations between humans) on rs53576 in intron 3: A/G, A/A, or G/G (Rodrigues, Saslow, Garcia, John, & Keltner, 2009).

The oxytocin receptor for the first two polymorphism types is considered "asocial" because individuals containing an adenine base for this polymorphism demonstrate lower levels of sociality. Conversely, the oxytocin receptor for the third polymorphism type (G/G homozygosity) is considered "prosocial" because individuals containing two guanine bases demonstrate higher levels of sociality (Rodrigues et al., 2009). Genetic polymorphisms for the oxytocin receptor directly indicate variations in the magnitude of the disgust response's range of expression; people who possess the asocial oxytocin receptor type are thought to exhibit disgust responses that are less susceptible to social influence as compared to people who possess the social receptor type. Therefore, individuals possessing the social oxytocin receptor type tend to exhibit larger variations in their disgust response as they are more susceptible to social influence on the whole.

Another mechanism by which oxytocin may moderate the effects of social influence on the human disgust response is at the level of specific brain regions. Work conducted by Norman and colleagues (2010) suggests that oxytocin selectively influences processing of threatening stimuli through a pathway that involves the amygdala. Norman and colleagues (2010) describe oxytocin as "rapidly processed through a subcortical pathway that allows for immediate emotional responses that tend to promote defensive behaviors" (p. 1317). If the threat of disease transmission were to be processed as a threatening social stimulus, then oxytocin might also be functioning through the mesolimbic reward pathway that promotes the reward associated with social behavior (Norman et al., 2010; Ross, et al., 2009; Insel & Young, 2001). Consequently, oxytocin may be acting upon the central amygdala, which is densely populated with oxytocin receptors (Kirsch, et al., 2005). This subcortical mechanism strengthens oxytocin's candidacy as the moderator of the relationship between intergroup bias and disgust, as it pinpoints oxytocin's influence on a region of the brain known for being used in social processing. The idea that oxytocin exerts its effects via a receptor mechanism indicates that a greater degree of signaling will lead to a strengthening of the role played by the molecule; as oxytocin has been shown to function in processing social relationships, then it would strengthen the relationship between intergroup bias and disgust.

The proposed role of oxytocin as a moderator stems from the idea that it should be able to strengthen the relationship between intergroup bias and disgust. As oxytocin signaling may occur via various routes, from throughout the body to within specific regions of the brain, the intensity of oxytocin's effects may directly affect the extent to which intergroup bias affects the human disgust response. While empirical data does not yet exist to support the moderating role of oxytocin in the relationship between intergroup bias and disgust, studies may be performed to demonstrate the strengthening of the relationship between

intergroup bias and the disgust response in the presence of heightened oxytocin signaling.

For example, researchers may seek to conduct studies in which they present participants with disgusting stimuli associated with an in-group or out-group label to evoke effects of intergroup bias. These researchers could then collect self-report measures of disgust from individuals confronted with the disgusting stimuli after administration of intranasal oxytocin as compared to individuals confronted with disgusting stimuli in the absence of intranasal oxytocin administration. This study may reveal that intranasal oxytocin strengthens the relationship between intergroup bias and disgust, with participants perceiving more disgust towards out-group members and less disgust towards in-group members in the presence of oxytocin than in its absence. Additionally, research endeavors may benefit from comparing the levels of perceived disgust of individuals possessing the social oxytocin receptor type to the levels of perceived disgust of individuals possessing the asocial oxytocin receptor type; these differences in oxytocin signaling may provide information about oxytocin's role in strengthening the effects of intergroup bias on disgust. Again, in such experiments, the disgusting stimuli would be associated with a form of in-group or out-group label to evoke effects of intergroup bias in the experimental setting.

Concluding Remarks

Empirical studies have supported the role of oxytocin in social recognition, especially in the context of disease avoidance. However, until this point, oxytocin has not yet been clearly identified as a moderator between intergroup bias and disgust. The well-characterized and normative disgust response that ensues from disease avoidance has been shown to be susceptible to bias effects. Oxytocin emerges as a likely moderator of these effects as it is a neuropeptide associated with a wide gamut of social recognition behaviors and has also been shown to exhibit tremendous variability in its diverse mechanisms of action. The signaling pathways of oxytocin, as well as the context of the relationship between the disgusted individual and the elicitor of disgust, allow for oxytocin to alter the strength of the relationship existing between intergroup bias and the human disgust response. Hopefully, future research in this field will continue to elucidate the possible mechanisms underlying the effects of in-group and out-group bias on the disgust response and yield empirical data to support oxytocin's moderating role.

A better understanding of the relationship between intergroup bias and disgust will contribute significantly to concerns of disease transmission that affect society today. This body of knowledge is especially pertinent to individuals who possess certain high-risk diseases. For example, both older and more recent surveys of attitudes relating to HIV/AIDS reported that people explicitly reported disgust when thinking about HIV/AIDS; some individuals even stated that people with AIDS should live separately from the general population (Oaten et al., 2009). The extent to which bias is felt towards individuals afflicted with diseases may not relate solely to the disease status of affected people but also to levels of oxytocin. Further study of socially relevant variables that affect the human disgust response may allow people to either overcome inhibitions that they may otherwise feel towards others or, at the very least, be better informed about the effects of social influence.

Additionally, recent research has revealed that oxytocin possesses various functions in social behavior, and further experimentation with oxytocin will reveal both greater depth of understanding as to its versatility as well as information about the use of oxytocin as a potential therapeutic agent. Very recently, oxytocin has been shown to enhance brain function in children with autism spectrum disorder (Gordon et al., 2013). Autism has been characterized as a developmental disorder marked with impaired social interaction and communication; the efficacy of oxytocin in improving brain function, as evidenced by experimenters using functional MRI, supports the role of oxytocin in processing socially salient stimuli. Oxytocin is a fascinating neuropeptide, and the full range of its social implication has yet to be discovered.

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