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Helping hands, healthy body? Oxytocin receptor gene and prosocial behavior interact to buffer the association between stress and physical health

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ABSTRACT

Providing help or support to others buffers the associations between stress and physical health. We examined the function of the neurohormone oxytocin as a biological mechanism for this stress-buffering phenomenon. Participants in a longitudinal study completed a measure of charitable behavior, and over the next two years provided assessments of stressful life events and physician-diagnosed physical ailments. Results indicated that charitable behavior buffered the associations between stressful events and new-onset ailments among individuals with the AA/AG genotypes of oxytocin receptor gene (*OXTR*) variant rs53576, but not among those with the GG genotype. These results suggest that oxytocin function may significantly affect health and may help explain the associations between prosocial behavior and health. More broadly, these findings are consistent with a role for the caregiving behavioral system in health and well-being.

Introduction

Providing help or support to others, while often depicted as burdensome, is robustly associated with better health and well-being. Indeed, volunteering or providing care for close others predicts *reduced* morbidity and mortality (e.g., Brown et al., 2003; Konrath et al., 2012; O'Reilly et al., 2008), and *increased* psychological well being (e.g., Brown et al., 2008; Poulin et al., 2010; see Post, 2007, for an overview).

The benefits of engaging in prosocial behavior may be due to stress-buffering features of helping. Helping or supporting others predicts reduced associations between stress and mortality (Krause, 2006; Okun et al., 2010; Poulin et al., in press) as well as depression (Brown et al., 2008). Laboratory research indicates that people respond to acute stress by increasing their prosocial behavior (von Dawans et al., in press), and that helping behavior reduces physiological response to stress (e.g., Floyd et al., 2007), raising the possibility that prosociality may serve as a coping strategy.

We test the prediction that the stress-buffering effects of prosocial behavior are a function of the caregiving behavioral system, and specifically of the neurohormone oxytocin. To do so, we examine the joint role of charitable behavior, stressful life events, and oxytocin receptor (*OXTR*) genotype in predicting new-onset physical ailments.

Prosocial behavior, caregiving, and oxytocin

Engaging in prosocial behavior may reduce stress in several ways, such as increasing positive affect or perceived control (Krause, 2006). Theoretical models of the caregiving behavioral system further suggest that prosocial behavior may also buffer stress more directly (Brown and Brown, 2006; Goetz et al., 2010). The caregiving system, which evolved to facilitate parental behavior, motivates efforts to reduce the suffering and/or increase the well-being of any individual thought to be in need (Bell and Richard, 2000; Collins et al., 2010; Shaver et al., 2010). Recent models of this system suggest it may do so by facilitating social approach behaviors, at least in part via anxiety reduction (Brown and Brown, 2006; Goetz et al., 2010).

Prior research indirectly links the caregiving system with health by showing that prosocial behavior only promotes health and well-being when accompanied by other-focused motivations (Gillath et al., 2005; Konrath et al., 2012) or when extended towards valued others (Poulin et al., 2010). However, a more direct way to study the role of caregiving in health is to examine a key biological mechanism that supports caregiving: the function of the neurohormone oxytocin. In both humans and

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non-human mammals, oxytocin plays a prominent role in motivating parental care (Campbell, 2010; Carter, 1998; Feldman, 2012). Oxytocin also has stress-buffering effects, moderating neural and behavioral responses to negative stimuli (Campbell 2010; Kirsch et al., 2005; Poulin et al., 2012) as well as modulating hypothalamic-pituitary-adrenal (HPA) axis and cardiovascular stress reactivity (Chen et al., 2011; Norman et al., 2012; Rodrigues et al., 2009). Higher blood levels of oxytocin have even been found to predict faster healing time of lab-induced wounds (Gouin et al., 2010). In humans, oxytocin promotes positive interactions with close others beyond offspring (Ditzen et al., 2008; Feldman, 2012) and facilitates prosociality more generally, including charitable giving, empathy, and compassion (e.g., Kogan et al., 2011; Poulin et al., 2012; for a review, see Campbell, 2010).

Together, oxytocin's strong links to caregiving and its known stress-buffering effects make it a plausible mechanism for explaining the stress-buffering effects of prosocial behavior. However, no prior research has linked oxytocin's stress-buffering effects to physical health per se, nor assessed its function as a potential moderator of the stress-buffering effects of prosocial behavior on health. To some degree, this may be because the central functions of oxytocin are difficult to observe in humans. That is, oxytocin is both a peripheral hormone and central nervous system neurotransmitter, with central effects on the amygdala that are believed to motivate prosocial behavior by way of threat reduction (Baumgartner et al., 2008; Campbell, 2010). The exact nature and extent of oxytocin's central effects remain unclear, but it is possible to broadly examine variability in oxytocin function by exploring individual differences in oxytocin receptor gene (*OXTR*) polymorphisms.

Within the *OXTR* gene, the single nucleotide polymorphism (SNP) rs53576 has been shown to predict both stress-buffering and prosocial behavior, with the "GG" genotype, in particular, appearing to indicate greater inclination for engaging in beneficial psychological and social behaviors associated with oxytocin (Bakermans-Kranenburg and van Ijzendoorn, 2008; Rodrigues et al., 2009; Tost et al., 2010). Moreover, although the exact role of rs53576 "G" and "A" alleles remains unclear, the risk allele (A) has been associated with risk allele-load dependent decreases in hypothalamic size and amygdalar activation (Tost et al., 2010), suggesting that risk for psychosocial dysfunction increases in the presence of an "A" allele. If this is the case, and oxytocin accounts for the stress-buffering effects of prosocial behavior on health, we would expect to see a significant interaction between prosocial behavior and the GG genotype that explains the stress-health relationship. This interaction effect could take at least one of two forms. First, it is possible that prosocial behavior promotes oxytocin function to compensate for the lower sensitivity of the non-GG genotype—i.e., a compensatory stress-buffering effect specifically among those with the AA/AG genotype. Second prosocial behavior may combine with the GG genotype to maximally buffer stress—i.e., a synergistic stress-buffering effect specifically among those with the GG genotype. By contrast, the absence of an interaction between prosocial behavior and the GG genotype would cast doubt on the notion that oxytocin helps to explain the stress-buffering effects of prosocial behavior.

The present study

The present study tests whether oxytocin function, as indicated by *OXTR* rs53576 genotype, moderates the stress-buffering effects of prosocial behavior in predicting subsequent longitudinal assessments of health status. We predicted that, adjusting for baseline health status and other relevant covariates, greater charitable involvement would predict a lessened association between stress and new-onset ailments (i.e., a stress-buffering pattern). In addition, we predicted that this stress-buffering effect would be qualified by an interaction (either compensatory or synergistic) between *OXTR* genotype and charitable involvement.

Method

Participants and procedure

Data were collected through Internet-based surveys of a large, nationally representative sample ($N = 2729$; Silver et al., 2006) recruited using stratified random-digit-dial telephone sampling by Knowledge Networks Inc. (KN). KN panel members are compensated with Internet access (if needed), points used to obtain merchandise, and cash incentives for completing surveys. Data used for this investigation were collected at several time points, described below.

Life event and health surveys

The participants completed a survey on mental/physical health and lifetime and ongoing stress by September, 2002 (Wave 1; $N = 1916$). They were subsequently invited to participate in two similar follow-up surveys 12 months (Wave 2; $N = 1571$, 82% of the Wave 1 sample) and 24 months after Wave 1 (Wave 3; $N = 1771$, 92% of the Wave 1 sample).

Social and political survey

A subset of those who completed the Wave 1 survey had completed a KN-administered survey on social attitudes and involvement, including charitable behavior, between April, 2000 and March, 2003. This sample ($n = 924$) was the base sample for all analyses involving charitable behavior.

DNA collection

In 2008, KN re-contacted available participants ($N = 1296$) from the larger longitudinal study to request their participation in the genetic study. Most participants (54.7%; $N = 711$) agreed, and provided saliva for genotyping using OraGene test kits (http://www.dnagenotek.com/) mailed to their homes. Kits were sent to The Centre for Applied Genomics (TCAG; http://www.tcag.ca/) for genotyping. There were 704 participants for whom *OXTR* rs53576 was successfully genotyped (99% call rate), and of these, 631 had completed the Wave 1 survey. This sample ($n = 631$) was the base sample for all analyses involving *OXTR* genotype.

Final sample

Because not all participants provided data on either charitable behavior or genetics, analyses in the present study are not based on a single sample size, but on the largest, most representative sample providing data for all variables in each analysis. For analyses involving just charitable behavior and health, $n = 924$; for analyses involving just *OXTR* genotype and health, $n = 631$; and for analyses involving both charitable behavior and *OXTR* genotype, $n = 365$. Across these separate analyses, data from a total of $N = 1195$ people are examined.

Measures

OXTR

The *OXTR* variant of interest, rs53576, consists of a locus at which either the nucleobase adenine or guanine (denoted by "A" or "G") can occur. The rs53576 SNP genotyping identified 361 "GG", 284 "AG", and 59 "AA" individuals. It was in Hardy–Weinberg equilibrium ($X^2[1] = 0.1$, $p > .05$) with no genotype-by-sex differences ($X^2[2] = 1.32$, $p = .52$) or genotype-by-ethnicity differences ($X^2[6] = 7.22$, $p = .30$). We followed the same grouping strategy used in prior studies addressing genotype-related psychological and social phenotypes—GG respondents were compared to individuals carrying a high risk A allele (GG = 1, AA/AG = 0) (e.g., Poulin et al., 2012; Rodrigues et al., 2009; Tost et al., 2010). This approach maintains consistency with prior studies and ensures sufficient statistical power to detect differences related to the high risk A allele.