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Social Inclusion Predicts Lower Blood Glucose and Low-Density Lipoproteins in Healthy Adults

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ABSTRACT

Loneliness has been shown to have direct effects on one's personal well-being. Specifically, a greater feeling of loneliness is associated with negative mental health outcomes, negative health behaviors, and an increased likelihood of premature mortality. Using the neuroendocrine hypothesis, we expected social inclusion to predict decreases in both blood glucose levels and low-density lipoproteins (LDLs) and increases in high-density lipoproteins (HDLs). Fifty-two healthy adults provided self-report data for social inclusion and blood samples for hematological tests. Results indicated that higher social inclusion predicted lower levels of blood glucose and LDL, but had no effect on HDL. Implications for theory and practice are discussed.

Among humans, the need for social connection is substantial. Humans have what Baumeister & Leary (1995) called a *need to belong*—a pervasive and adaptive motivation to maintain meaningful social relationships. Unsurprisingly, many problems—including deficits in physical health—are associated with a failure to maintain adequate social contact (Cacioppo & Patrick, 2008). We propose in the present study that social inclusion predicts benefits at the metabolic level, in the form of better lipid control and lower circulating glucose. Below, we review research and theory linking social inclusion to health, and then draw specific hypotheses regarding glucose and lipid levels.

Social Inclusion and Well-Being

Social inclusion is the perception that one has an adequate network of strong social relationships and sufficient opportunity for communication and social interaction (see Abrams, Hogg, & Marques, 2005). Its antonym, social exclusion, indexes a perception that one lacks meaningful social ties, and is associated with experiences such as rejection, ostracism, and loneliness.

A robust literature links social inclusion—primarily via its opposing forces, social isolation, and loneliness—to health-related issues (Heinrich & Gullone, 2006). For instance, loneliness is associated with poorer immunocompetence (Kiecolt-Glaser et al., 1984) and compromised cardiovascular health and sleep quality (Cacioppo et al., 2002), as well as an elevated risk of obesity (Schumaker, Krejei, Small, & Sargent, 1985) and chronic stress (Cacioppo et al., 2000). Considering these associations, it is unsurprising that isolation is associated with premature mortality; in a review of 20 years of longitudinal research, Berkman (1995) concluded that premature mortality increases with social isolation.

Importantly, it is one's *perception* of social inclusion—rather than an objective measure—that appears most strongly connected to health. In a meta-analysis, Uchino, Cacioppo, & Kiecolt-Glaser (1996) reported that health indicators such as greater immunocompetence, lower autonomic activity, and lower baseline levels of stress hormones were more strongly associated with perceived social connectedness than with objective measures of social connectedness. Thus, increasing actual social contact may be less effective at ameliorating the problems of loneliness than increasing the perceived quality or quantity of social relationships.

The robust associations between social inclusion and health outcomes beg the question of which mechanism or mechanisms are operative. As we detail subsequently, the *neuroendocrine hypothesis* offers one possibility.

The Neuroendocrine Hypothesis of Social Isolation

Cacioppo, Cacioppo, Capitano, & Cole (2015) articulated the neuroendocrine hypothesis of social isolation, which proposes that social exclusion impairs the body's ability to manage and recover from stress. Stress has multiple physiological effects, many of which are coordinated by a neuroendocrine system known as the hypothalamic-pituitary-adrenal (HPA) axis. This system is responsible (among other activities) for initiating hormonal responses to stressors, so elevated baseline activity of the HPA axis is indicative of a high stress load. Loneliness manifests in the form of elevated HPA activity; in one investigation, lonely inpatients excreted higher levels of urinary cortisol—an adrenal steroid hormone that increases blood glucose, suppresses the immune response, and promotes metabolism to help prepare the body for fight or flight—as compared to non-lonely patients (Kiecolt-Glaser

et al., 1984). Similarly, Cacioppo et al. (2000) found that lonely young adults had higher morning levels of adrenocorticotropic hormone (ACTH)—a pituitary hormone that prompts cortisol release—than did non-lonely adults.

No single outcome indexes stress or wellness fully, but two wellness markers that show associations with stress are blood glucose and blood lipids. On the basis of Cacioppo et al.'s (2015) hypothesis, we argue that both outcomes may covary with perceived social inclusion, as described subsequently.

Blood Glucose

Glucose, also known as dextrose, is a form of sugar that circulates in human blood to provide cellular energy. Glucose level is regulated by catabolic hormones such as glucagon and cortisol (which increase glucose) and the anabolic hormone insulin (which decreases glucose). Normal values vary over the course of the day and as a function of food intake and metabolic demand. American Heart Association guidelines provide that, for healthy (non-diabetic) adults, fasting glucose levels should be between 70 and 130 mg/dL (American Heart Association, 2015). Stress can also elevate glucose levels. During periods of physical or emotional distress, the adrenal stress hormone cortisol and the neurotransmitter epinephrine elevate blood glucose to fuel the body's fight-or-flight response (Netterström, Danborg, & Olesen, 1988). Based on the ability of affectionate behavior to ameliorate stress, Floyd, Hesse, & Haynes (2007) predicted and found a strong inverse relationship ($\beta = -0.85$) between expressed affection and glycohemoglobin (an index of average blood glucose level over a 12-week period), after controlling for the effects of received affection.

Insofar as blood glucose is elevated by stress, the neuroendocrine hypothesis would also support a link between social ties and healthier blood glucose via the stress-ameliorating emotional effects of strong social connections. We therefore propose that, among healthy adults, higher social inclusion predicts lower levels of blood glucose (H1).

Lipids

Lipids are water-insoluble organic compounds present in the cell membranes of all body tissues that perform a number of essential physiological functions (Shier, Butler, & Lewis, 2004). Two constituent lipids are high-density lipoproteins (HDLs) and low-density lipoproteins (LDLs). HDL is colloquially referred to as "good cholesterol" owing to its ability to transport fat molecules out of arterial walls, reduce the accumulation of white blood cells known as macrophages, and therefore reduce the risk of atherosclerosis, cardiovascular disease, and stroke. In contrast, LDL is known as "bad cholesterol" due to its ability to transport fat molecules into arterial walls and attract macrophages, elevating the risk of atherosclerosis. American Heart Association guidelines provide that, for healthy adults, HDL should be above 40 mg/dL for men and above 50 mg/dL for women; and LDL should be less than 100 mg/dL (American Heart Association, 2015).

Multiple studies have demonstrated that stress is associated with unhealthy lipid levels (e.g., Bacon, Ring, Lip, & Carroll,

2004). The specific mechanisms through which stress influences lipids are unknown, although some speculation suggests that stress-induced increases in energy initiate ancillary processes that elevate levels of LDL in the bloodstream (see Steptoe & Brydon, 2005). Other speculation points to activation of the sympathetic nervous system and the rapid release of catecholamines (such as epinephrine and norepinephrine) and glucocorticoids (such as cortisol).

To the extent that stress exacerbates lipid levels (by increasing LDL and/or decreasing HDL), and to the extent that social inclusion ameliorates stress (or a lack of social inclusion exacerbates stress), social inclusion may therefore predict higher levels of HDL and lower levels of LDL in healthy adults. We therefore predict that, among healthy adults, higher social inclusion predicts higher levels of HDLs (H2) and lower levels of LDLs (H3).

Method

Participants

Participants ($N = 52$) were equal numbers of healthy male and female adults. Ages ranged from 19 to 67 years, with an average of 28.63 years ($SD = 8.36$). Most participants (78.4%) were Caucasian, whereas 11.8% were Asian/Pacific Islander, 7.8% were Hispanic, 2.0% were African-American, 2.0% were Native American, and 3.9% were of other ethnic origins.

Procedures

Prescreening Procedures

Some aspects of the method are also reported in Floyd et al. (2009). Participants were recruited from among the staff, undergraduate student, and graduate student populations at a large university in the southwestern United States. Prospective participants were directed to an online prescreening instrument to determine their eligibility for the study. To be considered eligible, prospective participants had to meet multiple inclusion criteria (reported in Floyd et al., 2009). A total of 188 prospective participants filled out and submitted the online prescreening questionnaire; of that number, 127 (67.6%) met all of the qualifications.

Laboratory Procedures

Qualified participants who consented to take part in the study completed an online questionnaire and then visited the researchers' laboratory. Participants were instructed to be fasting when they reported to the laboratory, having had nothing to eat or drink besides water for at least 8 hours. Due to the fasting requirement, all sessions were scheduled between 7 a.m. and 10 a.m. When they reported for their laboratory visit, participants completed informed consent forms and were asked about their compliance with the fasting instructions (all participants reported compliance). A researcher (one of the junior authors) then used a fingerstick procedure to aspirate 80 μ l of capillary blood into two glass tubes coated with lithium heparin, an anticoagulant. After the puncture site was bandaged, the participant was offered juice and a cookie, was paid \$15, and was excused.

Measures

Social inclusion was measured with the unidimensional six-item Social Activity Index published in Floyd (2002). Using a 7-point Likert-type scale (7 = *strongly agree* and 1 = *strongly disagree*), participants indicated their level of agreement with items including “I take part in quite a few social activities” and “I am always spending time with friends or taking part in social events.” Coefficient alpha was 0.85. *Blood glucose*, *HDL*, and *LDL* were assessed in milligrams per deciliter (mg/dL) with the Cholestech LDX, a Clinical Laboratory Improvement Amendments (CLIA)-waived in vitro diagnostic monitor manufactured by Cholestech (Hayward, CA). We also assessed participants’ body mass index (BMI) and exercise frequency as potential control variables.

Results

Aggregate levels of glucose, HDL, and LDL are all within normal fasting ranges for healthy adults, according to American Heart Association (2015) guidelines. Descriptive statistics and inter-correlations for all study variables are available upon request.

Hypotheses

Glucose (H1). The first hypothesis proposed that higher social inclusion predicts lower levels of blood glucose. Glucose was unrelated to the potential control variables of sex, ethnicity, age, exercise frequency, and BMI, so we examined its association with social inclusion using a one-tailed Pearson correlation, which identified the hypothesized inverse relationship, $r(49) = -0.27$, $p = 0.03$. The first hypothesis is supported.

HDL (H2). The second hypothesis indicated that higher social inclusion predicts higher levels of HDL. Because HDL showed significant associations with sex and with BMI, we tested H2 in a hierarchical multiple regression with sex and BMI entered in the first step and social inclusion entered in the second step. Net of the effects of sex and BMI, social inclusion showed a nonsignificant relationship with HDL, $\beta = -0.12$, $p = 0.33$. The second hypothesis is not supported.

LDL (H3). The final hypothesis offered that higher social inclusion predicts lower levels of LDL. LDL showed a significant association with BMI, so we tested H3 in a hierarchical multiple regression with BMI entered in the first step and social inclusion entered in the second step. Net of the effect of BMI, social inclusion demonstrated the hypothesized inverse relationship with LDL, $\beta = -0.27$, $p = 0.04$. Full regression results appear in Table 1. The third hypothesis is supported.

Discussion

A robust literature links social inclusion—or the lack thereof—to a wide range of health indices. The present study found that social inclusion predicted lower levels of blood glucose and lower levels of LDL, although no significant results were found for HDL levels. These relationships are notable insofar as elevated blood glucose and/or LDL are associated with health problems—such as diabetes mellitus and coronary artery disease—for millions of children, adolescents, and adults (Hagman et al., 2014). A lack of adequate social inclusion is similarly widespread, especially among adolescents and among adults of all ages (Cacioppo & Patrick, 2008).

To the extent that social inclusion predicted LDL, it is perhaps surprising that it did not also predict HDL. Although the two outcomes are closely related, they are not automatically reciprocal. In clinical practice, in fact, it is not uncommon for treatments to improve one parameter while having no effect on—or even worsening—the other (J. Short, MD, personal communication, 2 April 2009).

These results add to a rapidly growing body of empirical evidence supporting connections between social inclusion and various markers of mental and physical wellness. As articulated, social inclusion—and its obverse state, loneliness—have been linked to a wide range of mental and physical health issues, including hypertension, obesity, cardiovascular disease, poor sleep quality, and immunosuppression. Unsurprisingly, the lack of adequate social inclusion is also a major risk factor for premature mortality. The present study is the first, to our knowledge, to link social inclusion directly with the hematological outcomes of blood glucose and lipids, both of which are highly consequential for physical health and wellness. Considered alongside previous findings, the current results increase empirical justification for intervention efforts aimed at curbing social isolation and loneliness, such as those aimed at vulnerable populations like the elderly (Kocken, 2001).

Similarly, in clinical practice, the present findings may suggest the utility of including a brief social and relational screening among routine patient questions.

Due to the prescreening process and the imposition of multiple inclusion and exclusion criteria, the current sample was probably healthier than a comparably sized sample drawn at random from the same population would be. This was indicated by average glucose and lipid values that were within normal test ranges, according to American Diabetes Association (2015) and American Heart Association (2015) guidelines. One consequence is that the associations with social inclusion—although statistically significant—may have marginal clinical significance for a normoglycemic and normolipidemic population.

The sample size was small relative to those typically seen in mainstream interpersonal communication research. It was, however, within the norm for psychophysiological studies (e.g., Marazziti & Canale, 2004), including those conducted within the field of interpersonal communication (e.g., Tardy, Thompson, & Allen, 1989). The relative inability of participants to introduce error variance (at least, in their hematological outcomes) via social desirability or memory biases, and

Table 1. Multiple regression predicting ldl from social inclusion.

Predictor	Zero-order r	B	SE B	β	ΔR^2
Step 1: BMI	−0.22	3.45	0.82	0.53	0.29*
Step 2: Social inclusion	−0.37	−5.81	2.67	−0.27	0.07*

Notes. $R^2 = 0.36$, adjusted $R^2 = 0.33$, $F(2, 43) = 11.88$, $p < 0.001$. * $p < 0.05$; ** $p < 0.01$.

the emergence of statistically significant effects, both argue for the adequacy of the sample size.

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