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The social brain

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Social species create stable structures beyond the individual. Social neuroscience is defined as the study of the neural, hormonal, cellular, and genomic mechanisms underlying superorganismal structures and processes, and the goal of social neuroscience is to identify these biological mechanisms and to specify the transduction pathways between social and neural structures and processes. To investigate the role of beneficial social connections on brain and biology, I adopted the subtractive methods commonly used in the neurosciences. In this method, the effects of the presence of some element (e.g., gene, brain nucleus) in an organism are contrasted with the effects of the absence (or graded absence) of that element. For instance, to investigate the function of a particular gene, you might compare the differences in outcomes on various measures between mice who have their full genetic complement and mice who have been genetically altered to be genetically identical to the normal mice except that they are missing the one specific gene whose function is of interest. Any differences observed between the normal mice and the genetically altered mice potentially reveals the functional role played by this target gene. To study the effects of social connections on brain and biology, we investigated differences observed between individuals who have social relationships and those who are socially isolated. Social interactions can range from hospitable to hostile, trustworthy to exploitive, and safe to threatening, and the nature of the interactions can change across time. Therefore, the brain is the key organ for evaluating, forming, monitoring, maintaining, repairing, and replacing salutatory connections with others as well as regulating physiological processes relevant to morbidity and mortality. Perceived social isolation is termed "loneliness," so I began our studies of biological effects of loneliness more than two decades ago. The association between indices of objective social isolation and loneliness are surprisingly weak (r ~ .19; see meta-analysis by Holt-Lunstad et al., 2015), and we found loneliness, net objective isolation, uniquely predict morbidity and mortality. Indeed, a recent meta-analysis of 70 prospective studies involving more than 3 million participants who were followed for an average of 7 years found that, even after controlling for objective social isolation and potential confounding variables, loneliness increased the odds of mortality by 26% (Holt-Lunstad et al., 2015).

According to our evolutionary model (J. Cacioppo, Cacioppo, & Boomsma, 2014; Cacioppo et al., 2006), the agony of perceived social isolation, or loneliness, motivates an individual to attend to, repair, or replace damaged social connections to reinstate a healthy social body, just as the agony of pain motivates an individual to protect the body from tissue damage. For instance, prior research has shown that loneliness is positively correlated with incidental social memory and heightened attention to faces and voices, as predicted by the social monitoring hypothesis, and animal studies have

demonstrated that a socially isolated mouse, compared to a group housed mouse, responds initially with approach behaviors when exposed to a safe social target (i.e., a novel juvenile mouse). In many contexts across human history, however, a chief threat to human reproductive success and survival has come from other humans. The unfettered motivation to form trusting relationships with others in such contexts may prove fatal. Our evolutionary model of loneliness, therefore, also posits that loneliness promotes an emphasis on short-term self-preservation, including increases in implicit vigilance for social threats, depressive symptomatology, self-centeredness, and social withdrawal, and decreases in appetitive responses to pleasant social stimuli, executive functioning, and sleep salubrity. More importantly, this model, and the neurobiological model underlying it, has led to the identification of a number of pathways through which loneliness alters neural, hormonal, cellular, and genomic mechanisms (e.g., gene expression).

The presentation covers this work and outlines mechanistic explanations for the putative effects of loneliness on morbidity and mortality. Specifically, the early effects of loneliness on social attention and cognition are posited to increase the likelihood that lonely individuals engage in behavioral confirmation processes, contributing to more negative social interactions and producing evidence that they are unworthy and/or others are potential threats. These dispositions and the focus on self-preservation in what is perceived to be a deficient social environment, in turn, alter the nature and likelihood of social engagement and activate neurobiological mechanisms that alter hemodynamic aspects of cardiovascular functioning, activation of the hypothalamic pituitary adrenal (HPA) axis, organismal inflammatory processes and decreases viral immunity, and sleep fragmentation. Repeated or chronic activation of threat surveillance in a social context, coupled with diminished anabolic processes, contribute to dysregulated brain and physiological systems and an elevated risk for broad based morbidity and mortality. According to our evolutionary model, the longterm costs of loneliness increasing the emphasis on self-preservation have been offset across our evolutionary history by the increased likelihood of short-term survival. However, in contemporary society, especially with the recent extensions of the average human lifespan, the long-term deleterious effects of loneliness on mental and physical health have become costly to the point that they represent a significant health risk.

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