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The effects of loneliness and social isolation on cognitive functioning in older adults: a need for nuanced assessments

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In recent years, there has been increased public and research attention directed towards the international “epidemic of loneliness” among older adults, as well as in the broader general population. Overall prevalence varies somewhat by method of assessment and country or region of sampling, but the bulk of studies suggest a general prevalence of approximately 20%–25%. The relationship of aging to the risk and nature of loneliness is complex, with inconsistent findings across studies, and may be affected by a number of specific factors, such as age-related loss of spouse/partners, changes in one’s broader social network, other environmental changes, as well as a range of positive and negative psychological factors. In older and younger adults, loneliness is not only an aversive experience, it has a deleterious impact on cognitive and physical health, and is associated with a substantial increase in all-cause mortality (Cacioppo *et al.*, 2015).

Of the multiple adverse effects of loneliness on health and well-being, one of those most relevant to the maintenance of independent functioning among older adults is the increased risk of cognitive dysfunction and dementia (Cacioppo *et al.*, 2015). Elucidation of the mechanisms underlying these associations is needed to develop better prevention and intervention strategies. One relevant component in that effort is clarifying the nature, evolution, and effects of objective social isolation in combination with loneliness. Prior research has shown that social isolation and loneliness each affect cognitive functioning, health, and mortality, but the two constructs are not synonymous. “Social isolation” refers to deficits in a person’s objective social network and support. In contrast, loneliness represents a subjective internal state resulting from a self-perceived discrepancy between one’s actual social relationships versus one’s desired social relationships. Social isolation can lead to loneliness, and persistent loneliness can exacerbate objective social isolation, but the two constructs are only modestly correlated ($r \sim 0.25$ – 0.28). The distinction is in itself seemingly self-evident as a true introvert may be very isolated but feel no loneliness, while an extrovert may still feel “alone in the crowd.”

Although exploratory in nature, the report from Fung *et al.* (2018) nicely illustrates an approach toward elucidating the potential interactive effects of loneliness and social isolation on cognitive functioning. They employed a cross-sectional design, using in-person

interviews with 497 “cognitively healthy” older adults (ages 60 years) residing in Hong Kong. A key aspect of their study was that social network size was measured in reference to two dimensions: (1) “confiding” versus “non-confiding” network, and (2) “close-ties” (i.e., number of family) versus “non-close” ties (i.e., number of friends). Confiding versus non-confiding network sizes were defined as the numbers of persons whom the respondents felt they could share versus could not share their inner feelings. The focus of analyses was on the interaction of overall loneliness (total score on the Chinese version of the de Jong Gierveld Loneliness Scale) with the above dimensions of social network size, as statistical predictors of the concurrent level of cognitive functioning (a composite of delayed recall from a word list, category fluency, and total score on the Chinese version of the Mini-Mental State Examination). After controlling for various potential confounding factors, they found a significant interaction of loneliness and non-confiding network size in the statistical prediction of concurrent cognitive composite scores. In contrast, there were no significant interactions of loneliness and confiding network size, or total network size, as statistical predictors of cognitive functioning. Further post-hoc analyses suggested that the interaction effects of non-confiding network size were present only for the number of close-ties (i.e., number of family members), not for number of friends in the non-confiding network.

The exploratory nature of Fung *et al.*'s study makes definitive conclusions difficult. There is no clear conceptual model that would have led to an *a priori* hypothesis predicting the above pattern of findings. Thus, the authors are appropriately very cautious in terms of avoiding definitive interpretation of their results. However, the investigators suggest that the findings raise the possibility that interventions focused on expanding social network size may be ineffective in countering the deleterious effects of loneliness. They also noted that “while our results only served to provide a legitimate basis for hypothesis generation, it remains interesting to explore if the unsatisfactory feeling of loneliness would come from some unresolved conflicts or alienation in family relationships.” The nature of intimate and family relationships, not just in terms of presence or number, but quality, tone, and history of the relationships almost certainly can affect the level and nature of loneliness among older adults. Of note, Fung *et al.*'s sample was primarily comprised of long-term residents of Hong Kong—a city with a unique culture and history over the lifespan of the respondents. The potential impact of cultural factors and generational cohort effects on the prevalence and nature of loneliness among older adults across cultures clearly warrants further research attention.

The above interpretive caveats noted, Fung *et al.*'s study serves as a solid example of innovation in moving beyond the now established fact that loneliness and social isolation are each associated with risks for cognitive and physical health impairments, reduced well-being, and early mortality, toward more nuanced examination of the specific nature or forms of loneliness, specific aspects of social isolation, and their interactions in affecting cognition and other outcomes. This type of specific information, when combined with a cogent and testable conceptual model, could have substantial utility in identifying specific actionable changes for personalized care to reduce loneliness and/or to reduce its deleterious effects.

One example of a widely cited conceptual model linking loneliness, social isolation, and health outcomes is “social control” theory. Per this model, self-perceived obligations to, and

the objective influence of people in a person's social network discourage negative health behaviors (e.g., smoking and excessive alcohol use), and promote positive health behaviors (e.g. exercise and good nutrition) (Umberson *et al.*, 2010). A strong social support network may also be associated with health promoting environmental resources (e.g., access to transportation, nutritious food, and health care).

Beyond the indirect association of loneliness with potential social effects on health behaviors and outcomes, persistent loneliness may have a direct deleterious effect on physiologic functioning and subsequent cognitive and health outcomes. For example, Cacioppo *et al.* (2014) proposed a social neuroscience model of loneliness. Their model is complex and has multiple components related to the initiation and maintenance of loneliness and its social and biological effects. However, a key foundation of their model is the assumption that acute loneliness in reaction to social isolation likely evolved as an adaptive social-motivational response. That is, acute loneliness may motivate pro-social behaviors that foster social connections in ways that increase the probability of survival and reproduction. However, they also posited that persistent/chronic loneliness becomes maladaptive and directly deleterious to physiologic functioning and health. More specifically, persistent loneliness may result in chronic social safety concerns, such as a sustained feeling of social threat. This, in turn, may lead to sustained physiologic hyperarousal, including dysregulation of inflammatory and immune functions, which then may have negative biological effects on cognition and physical health (Cacioppo *et al.*, 2014). Although the relevant empirical literature is still relatively small and not fully consistent, there is some emerging evidence of an association between loneliness and physiological dysfunction, such as HPA-axis response, inflammation, hypertension, as well as hyperlipidemia (Cacioppo *et al.*, 2015). Note that many of these same factors are associated with aging itself, and, thus, the combined physiologic effects of aging and loneliness may be even more complex.

There is both a need and potential for further empirical examination of how specific aspects or pattern of loneliness may interact with specific social and physiologic factors in deleteriously affecting cognitive and physical health among older adults. One dimension of loneliness suggested by Cacioppo and colleagues' social neuroscience model is distinguishing between various time-linked patterns of loneliness, such as acute, fluctuating, and persistent loneliness. One plausible hypothesis from their model would be that the deleterious cognitive and health effects of loneliness should be limited to persistent loneliness (not acute or fluctuating loneliness). Unfortunately, the items from the most widely used measures in loneliness research, while psychometrically strong and well-validated, are primarily framed without reference to persistence or fluctuations. Although there have been a few studies employing daily diary logs, the overall degree and impact of normative daily fluctuations is largely unknown. There have been a few studies using standard loneliness scales administered multiple times over successive years. For example, Gum *et al.* (2017) reported a study with time-lagged analyses showing loneliness among older adults does not predict subsequent feelings of hopelessness, but feelings of hopelessness do predict subsequent loneliness. But overall, there remains a need for studies with finer resolution in terms of fluctuations over time to determine what duration of persistence, if any, is adaptive versus maladaptive and harmful to health.

Another example of a potentially important distinction is that between “emotional loneliness” (focused on close/intimate partner relationships) versus “social loneliness” (focused on having a larger social network). There is preliminary evidence that these two types of loneliness may be differentially salient at different points in the adult life-span and may have different predictors/risk factors, although both are deleterious to health and mortality (Dahlberg and McKee, 2014; Julsing *et al.*, 2016).

In addition to attending to the dimensions of loneliness and social isolation, it would also be helpful to consider the types of strategies older adults spontaneously employ to prevent or cope with loneliness and the effectiveness of those methods. For example, through a recent review synthesis of qualitative studies with older adults, Kharicha *et al.* (2018) developed a two-dimensional frame-work for characterizing such strategies, including the degree to which a strategy is focused on prevention/direct action versus endurance/acceptance, and the degree to which it involves coping alone versus those strategies that reference others. The type of specificity and clarity provided by such a framework can facilitate identification of actionable factors, and thereby guide personalized therapeutic loneliness interventions with older adults.

There is also a need for empirical data on the interactive effects of loneliness, aging, and comorbid serious mental illness in terms of social isolation, positive and negative health behaviors, physiologic functioning, and health outcomes. For example, there is a significantly higher rate of loneliness among people with schizophrenia (Eglit *et al.*, 2018), but if and how loneliness in the context of an adult lifetime of schizophrenia affects physiology and outcomes is not yet clear. Other than studies of the association and distinction between loneliness and depressive symptoms, there is also a present dearth of published studies on the cognitive and health effects of loneliness in the context of forms of serious mental illness, including bipolar disorder and among people with post-traumatic stress disorder. These are all important distinctions to consider in comprehensive personalized treatment planning.

In conclusion, as Fung *et al.* (2018) readily acknowledge in their report, their findings are limited in the degree to which they permit definitive conclusions about the interactive effects of non-confiding family network size and loneliness on cognitive health. However, in examining social network size in this multidimensional form and the interaction with loneliness in concurrent prediction of cognitive function, the authors provide a solid example of the viability of researching these constructs in terms of potentially important/relevant nuances. Through further research, building appropriate and testable conceptual models, this line of work is likely to provide clinicians and public health officials with clearer information on viable actionable points of intervention to prevent and reduce both loneliness and its deleterious downstream effects among older adults. Given the high prevalence of loneliness in this population such information could have substantial public health significance.

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