THE PSYCHOLOGICAL IMPACT OF SARS-COV-2 QUARANTINE: OBSERVATIONS THROUGH THE LENS OF THE POLYVAGAL THEORY

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Abstract

According to the polyvagal theory, quarantine and social distancing following COVID-19 pandemic may dampen nucleus ambiguous (NA) activity in the brainstem and hinder homeostatic cardiorespiratory functioning, emotional self-regulation and health. In addition, enduring quarantine may foster heightened implicit vigilance for social threat, emotional dysregulation, poor sleep and immune response, potentially increasing the chance of infections. Promoting activities aimed at increasing NA functioning, like self-compassion, may support emotional self-regulation, adequate immune response and health.

Key words: SARS-CoV-2, COVID-19, quarantine, psychological impact, polyvagal theory, vagus nerve, self-compassion

On March 11, 2020, considering the “alarming levels of spread and severity, and by the alarming levels of inaction”, World Health Organization characterized the Severe Acute Respiratory Syndrome - CoronaVirus - 2 (SARS-CoV-2) situation as a pandemic (Bedford et al., 2020). Considering 7,375 laboratory-confirmed cases of SARS-CoV-2 and 366 deaths, on March 9, 2020 Italy decreed a community quarantine (Sjödin, Wilder-Smith, Osman, Farooq, & Rocklöv, 2020). Quarantine is defined as the separation and restriction of movement of people who have potentially been exposed to a contagious disease to ascertain if they become unwell, so reducing the risk of them infecting others (Brooks et al., 2020). Therefore, quarantine has the potential to impact social engagement of humans, though it is a mandatory measure aimed at curbing SARS-CoV-2 outbreak. Together with the psychological consequences of emergency situations, already dealt extensively in the literature, it may be necessary to reflect about the psychological consequences of quarantine, from different perspectives (Dell’Ossò et al., 2011; Dell’Ossò et al., 2011; Carmassi et al., 2016; Carmassi et al., 2017; Carmassi et al., 2018; Di Giuseppe et al., 2020; Di Giuseppe, Gemignani, & Conversano, 2020; Martino, Langher, Cazzato, & Vicario, 2019; Conversano, 2019).

According to the polyvagal theory (Porges, 2007), the structural organization and function of the human autonomic nervous system is hierarchically rooted in its phylogenetic heritage. The social engagement system stems from the myelinated ventral vagal complex (VVC), whose cardioinhibitory fibers originate in the nucleus ambiguous (NA) in the brainstem. VVC is a challenge-response system that is the least homeostatically disruptive, the phylogenetically youngest and the most rapidly acting (due to its myelinated fibers). The sympathetic nervous system (SNS) is phylogenetically older than the VVC, its activation promotes faster heart rate, respiration and mobilization for active threat responses such as escape or confrontational defense. The dorsal vagal complex (DVC), whose unmyelinated cardioinhibitory fibers originate in the dorsal motor nucleus of the vagus (DMNX) in the brainstem, is the phylogenetically oldest of the autonomic subsystems and includes a vestigial immobilization function that first arose in early vertebrates. DVC is involved in both homeostatic and threat reactions and primarily innervates organs below the diaphragm. This complex also disrupts digestive processes and conserves metabolic resources when recruited during threat responses (Porges, 2007). Interestingly, DVC is the system that is primarily involved in post-traumatic responses following psychological trauma (Kolacz & Porges, 2018). When social engagement is available and VVC is active, SNS and DVC are not recruited in threat reactions. When social engagement is not available and the nervous system detects danger or life threat through neuroception (a neural process that, according to the polyvagal theory, detects environmental or interpersonal safety or danger), SNS or DVC defense strategies, respectively, are recruited to manage the threat (Porges, 2007).

Quarantine, though necessary to curb SARS-CoV-2 outbreak, leads to social isolation and strongly limits the spontaneous human behavior aimed at recruiting


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the social engagement system to promote self-regulation and health. Furthermore, social isolation is well known to promote poor health, clinical psychiatric symptoms, and increased mortality in humans (Holt-Lunstad, Smith, Baker, Harris, & Stephenson, 2015; Orrù, Ciacchini, Gemignani, & Conversano, 2020). Animal models research has shown that social isolation induces cardiac and autonomic responses consistent with increased risk of cardiac pathophysiology, and depressive and anxiogenic behaviors in prairie voles (Grippo, Lamb, & Porges, 2007). Four weeks of isolation induced progressive functional cardiac changes, including elevated heart rate (HR) and reduced HR variability, as indexed by reduced respiratory sinus arrhythmia (RSA) amplitude that, according to the polyvagal theory, stems from NA activity and promotes health and self-regulation (Porges, 2007). Social isolation induced behavioral disruptions relevant to affective disorders. Isolated animals spent less time in open sections and more time in closed sections of the elevated plus maze (a behavioral measure of anxiety), with no generalized activity changes, suggesting anxiogenic behaviors. In addition, isolated animals showed decreased responsiveness to sucrose that is a valid and reliable operational index of depression in rodents (Grippo et al., 2007). Furthermore, evidence shows that social isolation causes hypervigilance in the brain state. An upregulation of the neuropeptide tachykinin 2 was evident in the anterior dorsal bed nucleus of the stria terminalis, central nucleus of the amygdala, dorsomedial hypothalamus and the anterior cingulate cortex following two weeks of social isolation in rats. In particular, during the two weeks of social isolation, rats’ acute freezing responses to threats could be converted to persistent ones by the upregulation of tachykinin 2 expression. In fact, tachykinin 2 signaling on enduring responses to threats are not determined simply by the region in which the neuropeptide acts but also by the level of peptide expression and by potentially different thresholds for neuropeptide effects in each area (Zelikowsky et al., 2018). Human research has linked social isolation with suicidal ideation and parasuicide in adulthood. In a sample of 19724 individuals from general population, aged 15 years with frequent feelings of loneliness were more likely to have had suicidal thoughts, and to have attempted suicide (Stravynski & Boyer, 2001). In lonelier adults, sleep is poorer, as well. Studies that rely on self-reported measures have found poor sleep duration, quality and efficiency (Pressman et al., 2005), while a study that recorded eyelid and muscle movement found that lonely young adults exhibited more restless sleep as indicated by the amount of wake time during sleep (Cacioppo et al., 2002). In young adults, eye-tracking (Bangee, Harris, Bridges, Rotenberg, & Qualter, 2014) and fMRI scans (Cacioppo, Norris, Decety, Monteleone, & Nusbaum, 2009) revealed that lonelier individuals appeared hypervigilant for social threat in particular. In addition, circulating cortisol during the first 30 min post-awakening is increased following days with more intense feelings of loneliness (Doane & Adam, 2010) and elevated cortisol levels in lonely individuals were associated with lower natural killer cell activity and poorer T-lymphocyte responses to mitogen stimulation (Kiecolt-Glaser et al., 1984). In accordance with these observations, it has been shown that decreased RSA was associated with an increased immune reactivity (higher tumor necrosis factor-a) in humans (Tonhajzerova, Mokra, & Venclovova, 2013). Thus, heightened implicit vigilance (i.e., increased neuroception, according to the polyvagal theory) for social threat may play a role in impaired sleep quality.

Interestingly, SARS-CoV-2 shows a neuroinvasive potential that may contribute to respiratory failure of COVID-19 patients. Viral antigens have been detected in the brainstem, where the infected regions included the nucleus of the solitary tract (the main input source for both DMNX and NA in the polyvagal theory) and NA (Li, Bai, & Hashikawa, 2020), whose myelinated fibers contribute to VVC, health and social engagement, potentially characterizing COVID-19 as a cardiorespiratory disease. Unfortunately, enduring quarantine may impinge on the same brainstem regions, as well: quarantine may dampen NA function (e.g., reducing RSA amplitude), and promote a shift towards SNS/DVC activation that, in turn, may trigger a re-emergence of traumatic memories (Kolacz & Porges, 2018). In addition, quarantine may foster an upregulation of tachykinin 2 expression and persistent freezing in response to threats (Zelikowsky et al., 2018), that is coherent with DVC-mediated immobilization, coactivated SNS-mediated alertness and a traumatic response (Porges, 2007; Kolacz & Porges, 2018). Enduring quarantine may maintain elevated levels of neuroception and a persistent DVC/SNS coactivation. Long-term social isolation may result in persistent SNS activation that can weaken the immune response (Hawkley & Capitanio, 2015), significantly diminishing antibody production against flu and pneumococcal pneumonia vaccines and increasing the chance of viral infection (D’Acquisto, 2017). Thus, promoting NA activity may ameliorate immunomodulation, emotional self-regulation and contribute to decrease the chance of viral infection preventing a persistent SNS activation. Conversely, the proposed cholinergic anti-inflammatory role of the DMNX has been recently questioned (Benarroch, 2019).

Recent research has shown that participants exhibited greater RSA during compassion induction compared with a neutral control, another positive emotion or a prosocial emotion lacking appraisals of another person’s suffering (Stellar, Cohen, Oveis, & Keltner, 2015). Considering this finding, promoting self-compassion programs (Neff and Germer, 2013; Arch et al., 2014) may be useful to reduce the psychological impact of quarantine, promoting emotional self-regulation and health.

References


