EDITORIAL

Stress and COVID-19 epidemic

Stella Allen, Vera Green

Allen S, Green V. Stress and COVID-19 epidemic. J Clin Psychol Cogn Sci. 2022; 6(2):5-7.

ABSTRACT

Stress researchers in psychology and neuroscience have significant problems as a result of the COVID-19 epidemic. Physical social contacts are utilised in well-known experimental paradigms like the Trier Social Stress Test to create stress via social-evaluative threat. Established stress induction models are typically difficult to utilise since lockdowns and contact restrictions hinder in-person interactions. Despite these obstacles, stress research is critical since the pandemic is expected to increase the prevalence of stress-related mental diseases. As a result, we examine the use of new research trends such as virtual reality, pre-recordings, and online adaptations for known stress induction paradigms. Such approaches are not only critical for stress research during COVID-19, but they are also likely to drive the field long after the pandemic has passed. They may make it easier to conduct research in new settings and with homebound or mobility-restricted participants. Furthermore, they enable fresh experimental variants that may advance methods as well as the understanding of stress. While the COVID-19 epidemic will undoubtedly provide hurdles for stress researchers, it may eventually turn into a driving factor for advancement.

Key Words: *Stress research; COVID-19; Corona virus; Social-evaluative threat; Trier Social Stress Test (TSST); Online studies; Ecological field research; Chronic stress.*

INTRODUCTION

We are all familiar with the feeling of being stressed because we meet various difficult circumstances in our daily lives (e.g., having to give an oral presentation at work, acting under time pressure, or facing the next exam). External demands surpass internal resources, causing the organism to activate a neuroendocrine stress response. First, the Sympathetic Nervous System (SNS) is activated, causing the adrenal medulla to secrete catecholamines like adrenaline and noradrenaline and physiological indices like heart rate, blood pressure, and perspiration to rise [1]. The Hypothalamus-Pituitary-Adrenal Cortex (HPA) axis is engaged in the second stage. This sets off a hormonal cascade those results in the release of glucocorticoids like cortisol, which target cells all across the body, memory (Wolf, 2009), and extinction learning and relapse have all been demonstrated to be affected by glucocorticoids [1,2]. The organism is thought to benefit from the induced alterations in order to satisfy current environmental demands. As a result, the acute stress reaction is seen as an adaptive coping strategy. Stress, on the other hand, can be harmful to the body if it is experienced too forcefully or too frequently. Despite significant advances in recent decades, the specific mechanisms and intricate interactions between genetic and environmental risk factors across the lifespan risk factors across the lifespan remain poorly known. The ongoing COVID-19 pandemic can be described as a universal and chronic stressor that affects people from all walks of life around the world. As a result, it has the potential to produce a massive public mental health disaster. Given the ongoing rise in the prevalence of mental health problems, this review article (1) outlines the urgent need to conduct experimental stress research using standardised stressors during the current COVID-19 pandemic; (2) discusses the conceptual and methodological challenges the discipline faces in this peculiar situation; and (3) In order to resolve the dichotomy between the urgent need and the procedural hurdles for stress research during the COVID-19 pandemic and beyond, this paper examines trends, viewpoints and technology improvements [3]. Stress-related mental disorders have been on the rise in recent years causing individual distress as well as financial and social problems for society as a whole. In general, stress has been discussed as a critical factor in etiological diathesis-stress models of mental disorders such as posttraumatic stress disorder and anxiety disorders, depression.

Editorial Office, Journal of Clinical Psychology and Cognitive Science, Windsor, Berkshire, England

Correspondence: Stela Allen, Editorial Office, Journal of Clinical Psychology and Cognitive Science, Windsor, Berkshire, England, E-mail clinicalpsycology@emedicalscience.com

Received: 05-March-2022, Manuscript No. puljcpcs-22-4596; Editor assigned: 07-March-2022, PreQC No. puljcpcs-22-4596(PQ); Reviewed: 17-March-2022, QC No. puljcpcs-22- 4596(Q); Revised: 19-March-2022, Manuscript No. puljcpcs-22-4596(R); Published: 26-March-2022, DOI: 10.37532/puljcpcs.22.6(2).05-07.

Assuming that stress is of significant relevance in the development of these mental diseases, it must be noted that the COVID-19 pandemic may operate as an extra stressor. The fact that the pandemic is novel, unpredictable, and uncontrollable (three major qualities of stresses) supports this argument. Furthermore, the political efforts used to prevent the virus from spreading further may be distressing. Public opinion polls have backed up this assertion. Many people are concerned about COVID-19 infections, disrupted daily routines, and a lot of ambiguity about what will happen in the future. Health care workers and those who have lost their employment or are facing financial difficulties as a result of the pandemic, for example, report higher levels of stress. Similarly, people who are lonely as a result of stringent contact limitations and parents who are dealing with childcare as a result of schools and kindergartens closing were shown to be under a lot of stress. As a result, the COVID-19 pandemic must be considered a one-of-a-kind stressor with serious implications for health and well-being because it is chronic and widespread [4]. Recent research has found that during the continuing epidemic, anxiety and depression are at an all-time high, with specific elements such as social or economic resources influencing stress perception. Holman et al. (2020) employed a probability-based technique in a nationally representative sample to anticipate the progression of mental disorders at an early but important stage of the unfolding pandemic in the United States. Individuals with pre-existing health problems, those exposed to secondary stressors, and those exposed to higher COVID-19-related media coverage are all at risk for worse mental health outcomes, according to the authors. Boyraz and Legros (2020) and Bridgland et al. (2021) both write in this vein. emphasise that the pandemic could be a terrible experience, thus increasing the occurrence of PTSD [4,5].

Events like 9/11, school shootings, or natural disasters like earthquakes or hurricanes have all caused spikes in mental health issues in the past. During the Great Recession of 2007–2009, there was an uptick in mental health difficulties. Previous pandemics, such as the Middle East Respiratory Syndrome or the Severe Acute Respiratory Syndrome, have shown an increase in mental health disorders. Based on these data, one might deduce that the COVID-19 pandemic will have comparable disastrous implications as a global and long-term stressor. Kickbusch et al. even label it "[....] the greatest threat to health and wellbeing, social welfare, and the global economy in living memory," meaning that it is more devastating than any previous crises in recent memory [5,6].

Other theories, on the other hand, propose that stress, or more especially, stress overload, is to blame for an unresolved variation in COVID-19's real distribution. Given the differential distribution of infections across countries and social strata, suggested that stress increased individual susceptibility to COVID-19 infection. In line with this, evidence is mounting that chronic stress impairs immune function, increasing the likelihood of, as well as the severity and length of, infectious infections or other sickness episodes. To summarise, different lines of study show a link between stress and negative health consequences in the context of COVID-19, emphasising the critical need to prioritise the pandemic as a public health priority. Under these unique circumstances, stress research has a high societal value. On the one hand, epidemiological and clinical views are required to comprehend the COVID-19 pandemic's impact.Basic stress research, on the other hand, is especially important in the current epidemic, as will be discussed below [7].

We propose that basic stress research can yield fundamental insights that are critical for clinical applications, in addition to epidemiological and clinical stress research during the pandemic. Basic research has already produced studies on acute laboratory stress that have had direct or indirect therapeutic implications. For example, were the first to show that cortisol response kinetics defined by a lack of habituation to recurrent stress exposure could be a health measure? Buske-Kirschbaum et al and Buske-Kirschbaum et al. (2010) backed up this theory by finding that atopic illnesses reduce cortisol responsiveness. Finally, such observations can be incorporated into McEwen's theoretical views on allostatic load (1998).

Clinical approaches to stress during COVID-19 should be complemented by basic research that integrates pertinent data into a common consensus on underlying causes. Clinical problems usually show up in stages, with the first signs and symptoms being quite minor (Myin-Germeys et al., 2009; van Os et al., 2009). It would be simple to focus just on persons with obvious clinical symptoms, given that psychopathological outcomes connected to COVID-19 are an extreme on a larger continuum. It's more likely that even people who don't satisfy diagnostic criteria have suffered subclinical and latent changes. Stress-related alterations in brain function and structure could be to blame. Previous research has revealed links between chronic stress and reduced volume in prefrontal and limbic regions as well as altered functional connectivity within frontoparietal brain circuits.

Salomon et al. used magnetic resonance imaging to explore stress-related brain plasticity in Israel during an early stage of the epidemic (MRI). The researchers scanned healthy people before and after the pandemic, comparing volumetric changes in the brain to control people who were measured twice under pre-pandemic settings. Salomon et al. (2020) discovered volumetric increases in brain areas linked to stress and anxiety neuronal networks. Importantly, our findings show that the COVID-19 pandemic can cause abnormalities in the brain not just in those who have been infected with the virus. On a behavioural level, pandemic-related changes could show as minor changes in habits. Alcohol consumption, for example, has been demonstrated to have increased in various countries. Such unhealthy coping mechanisms for stress can have a negative impact on one's health and increase the likelihood of catching COVID-19 and/or developing other physical or mental diseases. Basic stress research can supplement epidemiological and clinical techniques by attempting to uncover subtle as well as severe modifications caused by harsh conditions during the COVID-19 pandemic. If stress plays a role in the development of mental disorders, physiological or endocrine mechanisms in the HPA axis or the SNS are likely to be involved. Specific pathways, on the other hand, are rarely examined in large-scale population research since collecting physiological markers like as saliva samples from bigger cohorts is expensive and time-consuming. Because it can examine numerous physiological and neurological stress signals, basic stress research can help close this gap. It has been successful in identifying sources of intra- and interindividual variability using a combination of comprehensive assessment of the stress response and the fundamental strengths of laboratory research (e.g., control of confounding variables, standardisation of experimental procedures). These considerations may help to explain how and why people differ

in their susceptibility to stress-related health problems. It is currently questioned whether chronic levels of increased stress impact acute stress processing in terms of how COVID-19 related fundamental stress research may expand our overall understanding of human stress processing. Chronic or cumulative stress, according to an increasing body of evidence, causes insensitivity to acute stressors, resulting decreased a slowed stress response. However, rigorous study on this topic is still lacking, as exposing people to prolonged stress and putting them at risk for poor mental health outcomes for the sake of research is unethical.

McEwen spent a lot of time talking about chronic stress (1998). In the lab, he discovered evidence for four scenarios in which the adaptive allostatic response is triggered (1) too frequently, (2) too long, (3) not at all, or (4) lacks environmental adaption. If the COVID-19 pandemic causes the organism to exhibit one or more of these maladaptive response patterns, it could be a once-in-a-lifetime opportunity to learn more about chronic stress.

As previously stated, it is their social-evaluative component that mostly prevents the application of known stress induction models in the COVID-19 epidemic. Despite the fact that we stated experimental procedures that do not include this component, it is widely regarded as a necessary component for legitimate and ecological stress induction. As a result, it's worth considering whether stress researchers are forced to choose between (a) conducting (less effective) stress induction without a social-evaluative threat, (b) pausing their studies until the pandemic is over, or (c) whether there are some promising alternatives to psychosocial stress induction. Recent research suggests that deviating from in-person social encounters between participants and panellists can be used to induce social-evaluative threat. Andrews et al. (2007), for example, demonstrated that stress reactivity can be obtained using an inconspicuous TSST panel. By substituting the panel with a video camera, Düsing et al. (2016), among others, were able to cause large increases in cortisol levels. Düsing et al. (2016), for example, regarded their application a milder form of the TSST and expected a less prominent increase in cortisol levels as a result. This is consistent with Dickerson and Kemeny's (2004) meta-analytic evidence that stronger social evaluation leads to larger effect sizes. This shows that variations in mean cortisol release are insufficient because they do not account for the degree of cortisol reactivity. To summarise, HPA axis reactivity may be a result of the social-evaluative component's intensity, which can be manipulated according to particular research proposals (Andrews et al., 2007). In order to achieve substantial cortisol reaction in fundamental stress research, it may be desired to impose strong social-evaluative threat (possible under the given circumstances).

CONCLUSION

To summarise, further epidemiological, clinical, and basic stress research is needed to better understand the consequences of the current COVID-19 epidemic on wellbeing and the increased prevalence of mental disorders over a generation. In terms of experimental manipulations, the COVID-19 pandemic has prompted researchers to rethink social-evaluative components and how they are implemented in existing stress induction paradigms. We conclude that, using a variety of techniques and technologies, different adaptations for experimental protocols such as the TSST can be developed.. These will make it easier to conduct research in different settings and with homebound and mobility-restricted participants. Finally, they open up new possibilities for experimental variants in terms of flexibility in optimising protocols and experimental manipulations for specific research goals.

REFERENCES

1. Achdut N, Refaeli T Unemployment and psychological distress among young people during the COVID-19 pandemic: psychological resources and risk factors Int J Environ Res Public Health. 2020; 17(19): 7163.

2. Adam EK, Kumari M. Assessing salivary cortisol in large-scale, epidemiological research Psychoneuroendocrinology.2009; 34: 1423-1436.

3. Agterberg G, Passchier J. Stress among seamen Psychol Rep. 1998; 83(2): 708-710

4. Aguilera G. HPA axis responsiveness to stress: implications for healthy aging Exp Gerontol. 2011; 46(2-3): 90-95.

5. Allen AP, Kennedy PJ, Cryan JF, et al. Biological and psychological markers of stress in humans: focus on the Trier Social Stress Test. Neurosci Biobehav Rev.2014; 38 , 94-124.

6. Allen AP, Kennedy PJ, Dockray S, et al. The trier social stress test: principles and practice Neurobiol Stress. 2017; 6: 113-126.

7. Amirkhan JH. Stress overload in the spread of coronavirus. Anxiety Stress Coping. 2021; 34(2): 121-129.