ORIGINAL RESEARCH PAPER
Volume - 13 | Issue - 06 | June - 2024 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

SOCIAL DETERMINANTS OF HEALTH RELATED TO RACIAL AND ETHNIC DISPARITIES: FROM POSTMORTEM MOLECULAR STUDIES

Psychiatry

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ABSTRACT

One of the top goals for our discipline should be to comprehend and address racial and ethnic inequities in mental health, well-being, and psychiatric care. In keeping with this priority, this issue of the Journal begins with an overview by Dr. Margarita Alegría of Harvard Medical School and her co-authors (1) on the social determinants of health as they relate to disparities in mental health and psychiatric care and how they contribute to such race and ethnic-related disparities. This article reviews the literature on the relationship between social determinants of health and mental illness and offers suggestions for planning research and putting treatments in place that target the socioeconomic determinants of health that contribute to racial and ethnic inequities.

This issue also includes papers that are pertinent to dementia and schizophrenia. Two investigations broaden our knowledge of the molecular changes and biomarkers associated with schizophrenia. In the first, dorsolateral prefrontal cortical interneurons in the brains of schizophrenic individuals are used to identify changes that may be mechanistically related to the cognitive symptoms of schizophrenia. The second letter is a priority data letter that describes how anticholinergic medicines affect brain electrophysiological biomarkers that accompany anticholinergic related cognitive abnormalities. This information has direct implications for treating schizophrenia. This issue also includes a study that establishes the relationship between small vessel cerebro vascular disease and the development of dementia in later life by longitudinally assessing a large cohort of individuals using recurrent neuroimaging.

KEYWORDS

Critical factors linked to differences in mental health among older Black and Hispanic/Latinx adults are identified by Jester and colleagues (2). In this article, the researchers define variations in depression symptoms, cognitive measures, and self-rated health between Black, Hispanic/Latinx, and White adults using data from over 11,000 individuals (aged 51–89) from the Health and Retirement Study. In line with earlier research, those who identify as Black or Hispanic/Latinx performed worse on tests of health and cognition as well as depression symptoms when compared to White people. The researchers then evaluated the degree to which these racial and ethnic inequalities were related to the social determinants of health.

The social determinants of health were assessed using the following metrics: years of education, parents' educational attainment, years of employment, marital status, veteran status, residence in different US regions, national or foreign birthplace, household income, employer-sponsored health insurance, Medicare, and Medicaid. Social determinants of health explained 39% of the differences in cognitive scores, 37% of the differences in self-assessments of health, and 51% of the differences in depressive symptoms between Black and White persons. These impacts outweighed those associated with other health-related variables, including sex, age, health-related behaviors, and use of medical services. Comparing Latinx and Hispanic people to.

In white individuals, socioeconomic determinants of health explained 76% of the variation in cognition and 75% of the variation in self-reported health, but only 28% of the variability in depression symptoms. Drs. Carlos Jaramillo from the University of Antioquia and Javier Escobar and William Vega from Florida International University provide a comprehensive overview of issues pertinent to research focused on health care disparities related to race and ethnicity, and they discuss the significance and implications of these findings in their editorial (3).

Schizophrenia-Related GABA Neuronal Alterations in the Dorsolateral Prefrontal Cortex

The dorsolateral prefrontal cortex (dl PFC) of people with schizophrenia has been shown to have functional, anatomical, and molecular changes in a number of neuroimaging and postmortem investigations. Comprehending the molecular changes that contribute to modified dorsolateral PFC function in individuals with schizophrenia is a crucial step in developing novel treatments targeting symptoms linked to impaired cognitive and executive functioning. Changes in prefrontal cortical GABA interneurons, which serve to supply inhibitory input to other cortical neurons, particularly excitatory glutamatergic neurons, have been linked to postmortem findings that are particularly intriguing. GABA interneurons come in a variety of subtypes, and two subtypes associated with schizophrenia can be distinguished by the presence of mRNAs encoding the peptide somatostatin (SST) or the calcium-binding protein parv albumin (PV). In the brains of people with schizophrenia, decreases in the mRNAs for these molecules have been observed in the prefrontal cortex in both situations. Whether the reductions in PV and SST mRNAs are caused by a decline in the number of neurons that express these molecules or rather by lower synthesis of PV and SST within a normal number of neurons is a significant question with therapeutic implications that has not yet been satisfactorily resolved. In order to address this query, Dienel et al. (4) present results from an approach that simultaneously and independently measures the density of PV and SST GABA neurons in layers two and four of the dlPFC as well as the quantity of PV and SST mRNA within these neurons. tissue from 30 dorsolateral prefrontal cortex.

It is significant to remember that low levels of PV and SST mRNA in dlPFC neurons were previously shown in the schizophrenia brains chosen for investigation. The results from this subgroup of schizophrenia patients showed that, rather than a reduction in the density of PV or SST GABA neurons, the observed drop in PV and SST mRNA levels is caused by lower gene expression in a normal number of neurons. In addition, the authors examined other variables linked to schizophrenia, such as suicide death, drug abuse problem at the time of death, and usage of psychopharmacologic medications at the time of death. They discovered that these analyses had no impact on the results.

Small Vessel Disease In The Brain And The Development Of Dementia

While cerebrovascular disease has been associated with dementia, it has been difficult to causally link small vessel cerebrovascular disease to the later development of dementia. In this regard, Jacob and colleagues (6) followed a cohort of individuals with cerebral small vessel disease (SVD) to characterize cerebrovascular risk factors associated with the later onset of dementia. The participants in the study with sporadic SVD and not dementia (N5503) were recruited from a neurology outpatient clinic in the Netherlands and followed for 14 years. MRIs were performed to characterize SVD, which included assessment of white matter hyperintensities, lacunes, and evidence of cerebral microbleeds as well as the integrity of white matter. Of the 498 participants in whom dementia was assessed, 21.5% or 108 individuals developed dementia (of these: 35% Alzheimer's, 32%, vascular, and 24% mixed). Results demonstrated that the degree of SVD at initial evaluation, and the magnitude of the progression of SVD characterized in the364individualsinwhich at least two MRIs were acquired, predicted the development of dementia. The SVD markers tended to be more associated with the development of vascular dementia, whereas decreased hippo campal volume, less white matter hyper intensities, and less affected white matter tracts were associated with the development of Alzheimer's disease. This study is distinguished by its

long period of follow-up, which the authors argue allows them to infer a causal relationship between the severity of SVD and the later development of dementia. Additionally, the authors suggest that intervention strategies aimed at reducing the progression of SVD could protect vulnerable individuals from developing dementia. In his editorial, Dr. David Steffens (7) from the University of Connecticut provides a general discussion related to cerebrovascular dementias and highlights the strengths of the Jacob et al. study, including the methods used and the importance of the long-term follow-up period.

Anticholinergic Burden Is Associated with Schizophrenia-Related Alterations in Neurophysiological Biomarker

Psychotropic medications with anticholinergic effects, most notably antipsychotic and some antidepressant drugs, are commonly used to treat psychiatric patients. The peripheral effects associated with the anticholinergic action of these drugs (e.g., dry mouth, constipation, urinary retention, and blurred vision) are well known and generally obvious to patients and their providers. Anticholinergics also have central effects and depending on the dose and age of the patient can result in sedation and cognitive impairment. Joshi and colleagues (8) previously demonstrated that an individual's overall anticholinergic burden (ACB) score, calculated by estimating ones' total amount of anticholinergic exposure from all medications, was associated with cognitive impairment in patients with schizophrenia. This is an important observation that helps to explain the complex factors that contribute to cognitive deficits in patients with schizophrenia and has direct treatment implications. In the current issue of the Journal, Joshi and colleagues extend this work by examining whether ACB scores are also related to alterations in EEG biomarkers of early information processing that occur in individuals with schizophrenia (9). The specific EEG biomarkers used are sequential components of auditory eventrelated potentials elicited by unexpected background stimuli, i.e., mismatch negativity and P3a. The neurophysiological processes reflected by these markers are fundamental to normal cognitive processing. The data reported in this study was collected from 555 patients and was compared to 1,062 controls. Most of the patients were diagnosed with schizophrenia, with a small number diagnosed with schizoaffective disorder, depressed type. Consistent with the previously reported relation between increased ACB scores and cognitive impairment, the current results demonstrated a negative relation between an individual's ACB score and the magnitude of their mismatch negativity and P3a EEG responses. When performing regression analyses to include the effects of other factors such as functional and neurocognitive measures, ACB was still found to be associated with the mismatch negativity response, whereas this was not the case for the P3a response. Taken together, these findings point to the importance of accounting for ACB, both clinically and in research studies, when assessing factors related to cognitive impairments in individuals with schizophrenia. In his editorial, Dr. Steven Siegel from the University of Southern California (10) describes in more depth what evoked response potentials are and more specifically discusses the meaning of the findings linking ACB to alterations in the mismatch negativity and the P3a responses.

CONCLUSION

In order to fully comprehend the complex interactions among risk and resiliency factors that lead to illness and affect treatment outcomes, it is important to conduct research at various levels of analysis (e.g., molecular, cellular, circuit, individual, and societal). The studies in this issue of the Journal demonstrate the breadth of our field. I would especially want to draw attention to the studies conducted by Alegría et al., Jester et al., and Escobar et al. These studies aimed to describe the relationship between differences in mental health and well-being and racial/ethnic inequalities in the social determinants of health. These contributions are particularly crucial for our readership to comprehend the causes of the differences in mental health care that people of color encounter and to serve as a foundation for creating techniques to alter and lessen the causes of these biases.

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