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LETTER TO THE EDITOR

Epigenetic analysis of heat shock activator complex in the peripheral blood of Parkinson's disease patients and its clinical significance: Correspondence

Hinpetch Daungsupawong[1], **Viroj Wiwanitkit**[2]

[1]Private Academic and Editorial Consultant, Phonhong, Laos
[2]Department of Pharmaceutical Sciences, University Centre for Research & Development Chandigarh University Gharuan, Mohali, Punjab, India

The article "Epigenetic analysis of heat shock activator complex in the peripheral blood of Parkinson's disease (PD) patients and its clinical significance" is the topic of discussion in this letter.[1] The purpose of this study was to examine the clinical significance of methylation alterations in genes linked to PD in peripheral blood. Comparing drug-naive patients with PD to controls, the study discovered significant methylation alterations in the genes heat shock proteins 70 (HSP70), (HSP90), heat shock factor 1 (HSF1), heat shock RNA-1 (HSR1), eukaryotic translation elongation factor 1α (eEF1α). The findings demonstrated that eEF1α was considerably more hypermethylated in individuals with PD who had higher Mini-Mental State Examination (MMSE) scores, whereas those with lower MMSE scores had hypomethylated versions of the other genes. Additionally, the study discovered associations between depression and MMSE scores and methylation changes in HSF1 and eEF1α, suggesting a possible function for these genes in the advancement of PD and cognitive decline.

The study's small sample size of 45 participants presents a possible limitation that could restrict the findings' generalizability. Furthermore, the study only examined methylation changes in peripheral blood; however, to better understand how these genes function in the pathophysiology of PD, future research may look into methylation changes in other tissues, such as brain tissue. Additionally, the study did not evaluate additional confounding variables that can affect methylation changes in patients with PD, such as age, sex, or other comorbidities. Moreover, the functional significance of the methylation alterations in these genes and their potential effects on gene expression and the pathogenesis of PD were not investigated in this study.

The authors raised the question of whether they intended to broaden the scope of their investigation to include a more varied and sizable sample population to corroborate their results and possibly pinpoint patient subgroups with unique methylation profiles. It would also be interesting to investigate the connection between methylation changes in these genes and clinical traits of PD, such as the severity of the disease, motor symptoms, and response to treatment. Investigating the possible involvement of these genes in the course of PD and cognitive loss may yield important information about new targets for treatment or biomarkers for PD that may be used for early detection and monitoring.

Research in the future may focus on how methylation alterations in these genes are affected by environmental influences or genetic polymorphisms and how these changes affect the development and susceptibility to PD. Furthermore, examining the relationship among gene methylation,

Correspondence: Hinpetch Daungsupawong. Private Academic and Editorial Consultant, Phonhong, Laos.
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gene expression, and protein function in the pathophysiology of PD may offer a more thorough comprehension of the molecular mechanisms behind this intricate neurodegenerative illness and open up new therapeutic directions. Overall, this work sheds information on the function of methylation modifications in genes linked to PD and emphasizes the need for more investigation to fully understand their clinical relevance and therapeutic potential.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

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