

4-2024

Analysis of ABCA7 in Alzheimer

Elaine Vanterpool

Oakwood University, evanterpool@oakwood.edu

Johnathan Daly

Oakwood University

Follow this and additional works at: <https://ouscholars.oakwood.edu/student-posters>

Recommended Citation

Vanterpool, Elaine and Daly, Johnathan, "Analysis of ABCA7 in Alzheimer" (2024). *Student Posters*. 39.
<https://ouscholars.oakwood.edu/student-posters/39>

This Poster is brought to you for free and open access by the Student Creative Works at OUScholars. It has been accepted for inclusion in Student Posters by an authorized administrator of OUScholars.



Analysis of ABCA7 in Alzheimer

Johnathan Daly and Elaine Vanterpool, PhD
Oakwood University
Department of Biological Sciences
Huntsville AL, 35896



ABSTRACT

Alzheimer's disease (AD) is a progressive neurological condition that presents a significant global health concern, impacting millions of people worldwide. The causes of AD are intricate and involve a mix of genetic and environmental factors. One noteworthy genetic factor is the ATP-binding cassette transporter A7 (ABCA7) gene, which plays a crucial role in the disease's onset and advancement. This analysis offers a thorough review of the ABCA7 gene's involvement in AD, focusing on its influence on lipid metabolism, immune response modulation, and amyloid-beta processing. Furthermore, it delves into the effects of specific mutations in the ABCA7 gene and their potential role in disease progression. The study also examines the expression patterns of the ABCA7 gene in various tissues, highlighting its diverse functions in human health. While our understanding grows, further research is necessary to fully grasp how the ABCA7 gene contributes to Alzheimer's disease at the molecular level, with the aim of developing innovative treatments targeting this gene and its pathways. Results indicate that the ABCA7 protein features conserved domains, including two nucleotide-binding domains (NBDs) and two transmembrane domains (TMDs), which are vital for its function.

INTRODUCTION

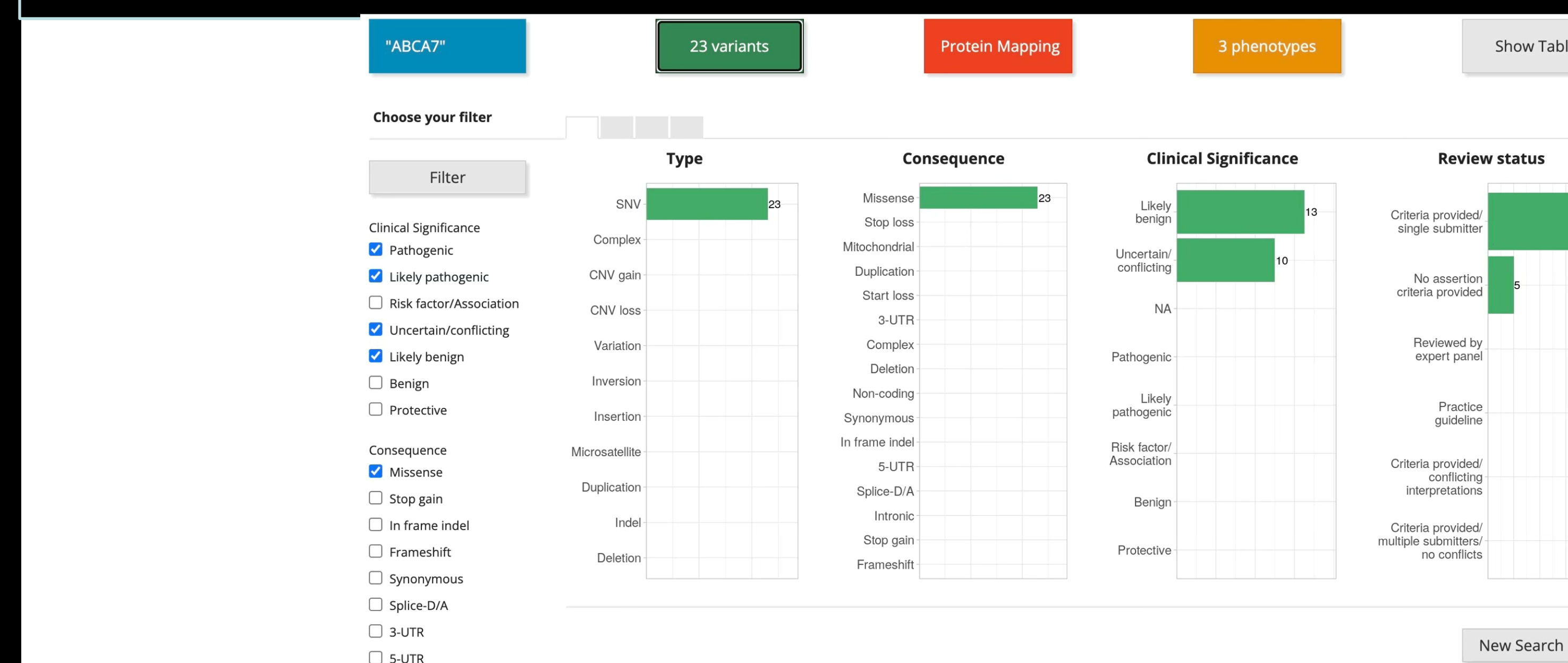
Alzheimer's disease (AD) is a complex neurodegenerative disorder influenced by genetic and environmental factors, leading to cognitive decline and affecting millions globally. While progress has been made in understanding AD's molecular pathways, effective therapies are still lacking. Genetic risk factors like APOE ε4, PSEN1, PSEN2, APP, and ABCA7 offer insights into AD's mechanisms. This review examines AD's genetic, molecular, and cellular processes and potential therapeutic strategies, emphasizing the need for a comprehensive approach to treatment.

METHODS

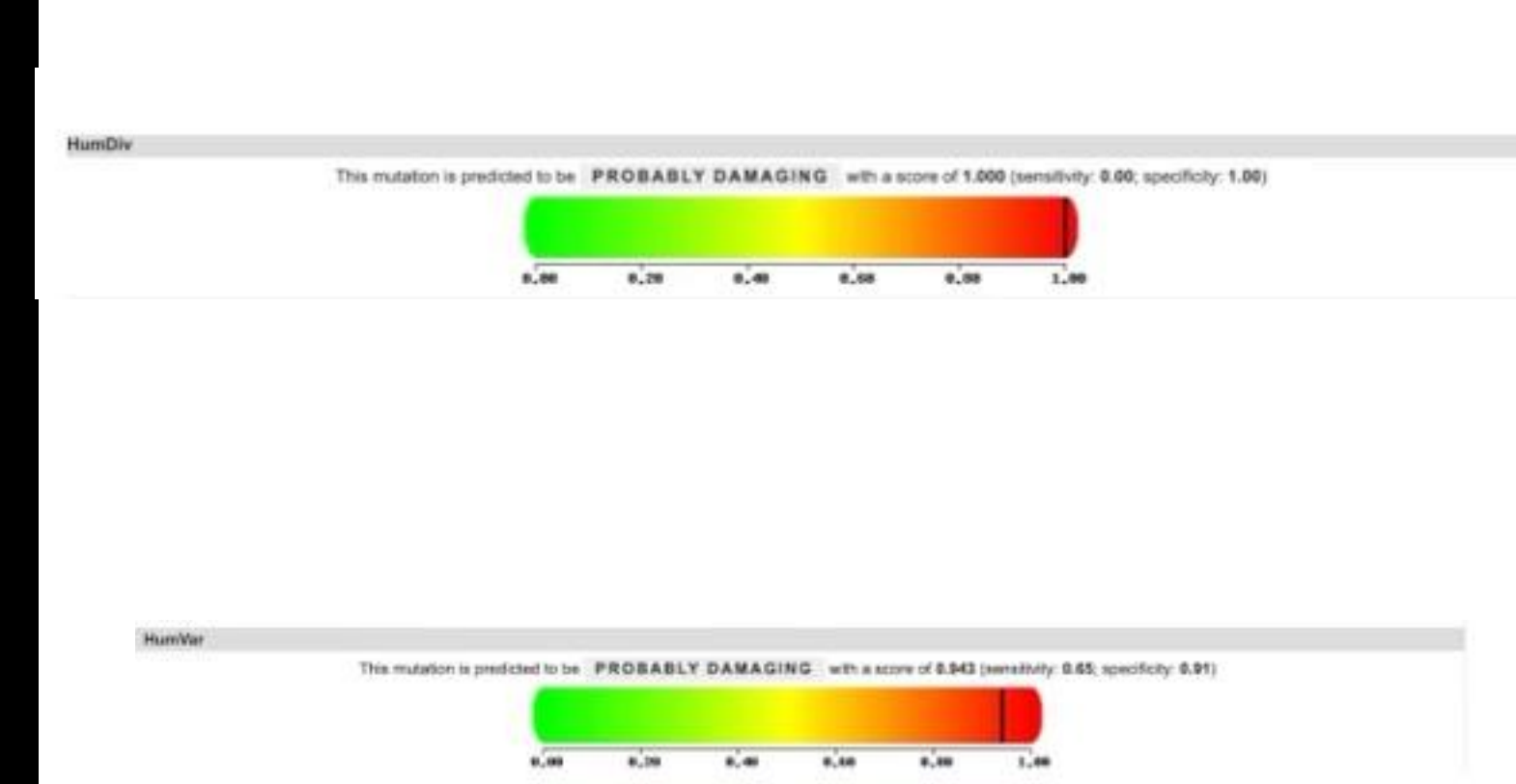
Simple ClinVar Identified genes
SIFT and PolyPhen 2 were utilized to analyze variants
Swiss Moswl was used to illustrate the protein structures

RESULTS

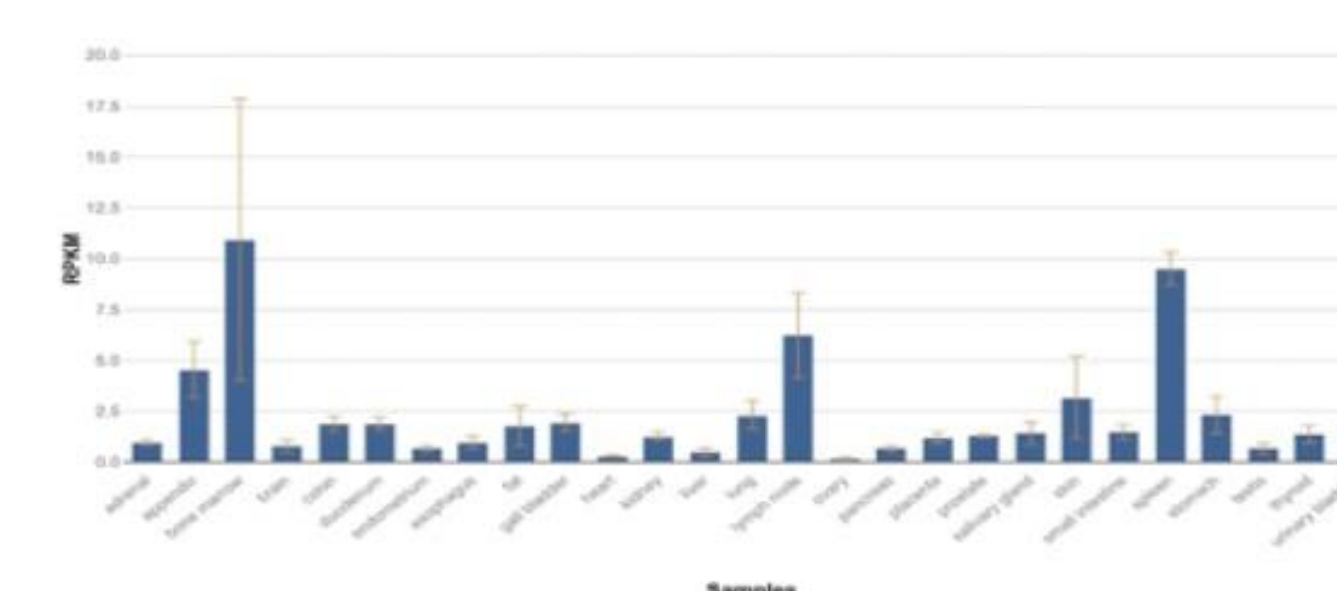
PROTEIN MAPPING



Polyphen 2



Gene Expression



SWISS Pro Model Conserved Domain



DISCUSSION AND CONCLUSION

Conclusion The study's results indicate that the ABCA7 gene plays a pivotal role in the progression of Alzheimer's disease by affecting lipid metabolism and regulating immune responses. Mutations in the ABCA7 gene, particularly missense mutations, have the potential to disrupt its function and contribute to disease development. The gene's expression patterns across different organs suggest that it may have diverse functions, necessitating further exploration. Moreover, the ABCA7 gene's involvement in other diseases like age-related macular degeneration and cardiovascular diseases suggests broader implications for human health. More research is required to uncover the precise molecular mechanisms by which the ABCA7 gene influences Alzheimer's disease and related conditions, which could lead to the development of innovative treatment approaches. Despite advances in understanding the molecular underpinnings of AD and identifying genetic risk factors, its exact causes remain elusive, and effective disease-modifying therapies are still lacking. A comprehensive approach targeting various aspects of AD, including Aβ production, neuro-inflammation, oxidative stress, and impaired energy metabolism, is necessary. Developing novel therapeutic strategies, combined with personalized medicine approaches, is essential for revolutionizing AD management and enhancing the quality of life for patients and their families.

REFERENCES

<https://www.ncbi.nlm.nih.gov/clinvar/variation/4012/>
<https://pubmed.ncbi.nlm.nih.gov/38255788/>

ACKNOWLEDGEMENTS

This project was supported by Oakwood's TIP HBCU UP program. Specially thanks to Dr. Vanterpool, PhD

