we are uncertain if these markers demonstrate linear or quadratic responses during that time. Because of the complexity in the cytokine network involving bi-directional feedback, pleiotropism makes it complicated to surmise the exact mechanisms of individual markers. Additionally, these cells have been noted to drift between neuroprotection and neuroinflammation, so even within the same marker lies complex answers to questions about how the immune system works (i.e., the role of IL-6 in AD etiology) [39]. Thus, having more frequent assessments of these markers might help to better elucidate their responses to the APMC formula and its ability to modulate immune system function over an extended period of time.

CONCLUSIONS

AD is an escalating burden for patients and their families. In addition, with the global population of aged individuals increasing exponentially, AD represents a significant economic drain on society. The development of an effective approach for the treatment of AD is thus of major importance, as current strategies are limited to agents that attempt to attenuate disease symptomatology without addressing the causes of dis- ease. A considerable need exists for the development of an effective therapy to prevent, or at least delay, the progression of AD.

The APMC formula used in the current study was well-tolerated among all subjects. The product showed a significant improvement in the ADAS-cog cognition score and demonstrated sound immunomodulator activity with noteworthy responses in cytokines and several lymphocyte and monocyte subsets. Several correlations were found between the cognitive assessments and the physiological outcomes at baseline and 12 months follow-up. Our results are consistent with prior work by other investigators using similar oligosaccharide-based formulae, who also demonstrated improvements in various indices of quality of life and functioning in other disease states. At this time, the mechanism by which APMC influences cognitive functioning in AD is unclear. The amelioration of cognitive functioning may be associated with some modulation of host immune activity, but additional immune functioning data are required to understand with more certainty. However, what is clear is that our results compel further study, especially in the investigation of an AD-type neurodegeneration model that may eventually enable elucidation of the mechanism(s) at work. Utilizing an AD-type neurodegeneration model would allow us to gain deeper, if not novel, insights into the pathophysiology of a disease that is the source of much human suffering.

Thus, our study shows that a high-quality, concentrated dietary supplement may offer an alternative option for persons with AD. This APMC formula may not only facilitate cognitive improvement, but may also improve the inflammatory and immune functioning profile as well, thereby enhancing host recovery and improving overall quality of life.

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