Commentary

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A Brief Note on Blood Based Biomarkers

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About the Study

Measuring brain disease biomarkers in the blood presents a variety of challenges that need sensitive and precise assays as well as thorough validation. Because the blood-brain barrier restricts free movement of compounds between the CNS and the blood compartments, brain-derived biomarkers are normally found in low quantities in the blood. Furthermore, several biomarkers linked to Alzheimer's disease pathology are detected in non-cerebral regions, which might make blood testing difficult. Furthermore, heterophilic antibodies (endogenous antibodies that react with the antibodies of the immunochemical test to quantify the biomarker) may be present in the blood, resulting in inaccurately high or low results. In CSF, where antibody levels are substantially lower, these sorts of antibodies are much less. Finally, the analyte of interest may be proteolytically degraded in plasma by a variety of proteases.

Targeted blood-based biomarkers

Plasma A: Matrix effects (mostly other plasma proteins binding Aβ) can alter plasma A results and the analytical sensitivity of the original tests did not allow for reducing such matrix effects. Aβ42 ultrasensitive Single molecule array (Simoa) test was described in 2011. The enhanced analytical sensitivity revealed that the ratio of Aβ42 to A40 in plasma was lowered in amyloid PET-positive individuals in a similar manner to CSF Aβ42/Aβ40, but with less evident separation. A few years ago, accurate immunoprecipitation mass spectrometry (IP-MS) based tests for Aβ40 and Aβ42 were published, with about 90% diagnostic accuracy, demonstrating a reduction in the plasma Aβ42/Aβ40 ratio (similar to the CSF test). These are extremely promising discoveries, prompting the launch of multiple technique comparison and standardization investigations. These novel assay formats do not address the problem of non-cerebral expression of Aβ, such as in blood platelets, which lowers the test's specificity for cerebral A plaques, but they are still a significant step forward.

Plasma tau: Plasma tau concentrations, evaluated by ultrasensitive assays, are raised in the dementia stage of Alzheimer's disease compared to cognitively normal control persons, although not as obviously as in CSF, which is a well-replicated observation. There is limited agreement on how these findings should be applied to intermediary individuals in the Moderate Cognitive Impairment (MCI) stage of the disease, with reports being contradictory. Nonetheless, Mielke and colleagues recently explored the connection of plasma T-tau concentration, as assessed by Simoa, with cognitive impairment in 458 Mayo Clinic Study on Aging individuals and observed that high plasma T-tau was related with quicker clinical disease development. Another prospective cohort study used data from the Framingham Heart Project in the United States, with replication in the Memento project, and revealed that plasma T-tau was strongly associated with incident Alzheimer's disease dementia.

miRNA biomarker panels: The area is also becoming more interested in epigenetics, with gene control by micro RNA (miRNA) being one such target for biomarker development. MiRNAs are thought to be carried within liposomes, HDLs, exosomes and other proteins, where they are protected from destruction. The panel could also distinguish AD patients from those with MCI, multiple sclerosis, Parkinsonism, severe depression, schizophrenia, and bipolar disorder with 74%-78% accuracy. Other research identifying panels of miRNAs have been found to differentiate between Alzheimer's disease patients and controls with accuracies ranging from 75% to 95%.