UNDERGRADUATE POSTER FINALIST

The Role of Beta Amyloid Following Traumatic Brain Injury: A Critically Appraised Topic
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Focused Clinical Question: Is beta amyloid (Aβ) deposition increased following traumatic brain injury (TBI)?

Data Sources: Relevant articles were searched using MEDLINE, and SportDiscus databases, limited to those published after 2013. The search terms used were traumatic brain injury (TBI), chronic traumatic encephalopathy (CTE), biomarker*, beta amyloid, and neurodegen*.

Study Selection: In vivo and cadaveric human studies were included. Systematic reviews with meta-analysis of animal studies were included if the focus of the study centered on linking Aβ to TBI.

Data Extraction: The results of Aβ comparisons across time for animal or human studies were included in the analysis. Results were measured using PET scan imaging or immunoblotting of cerebral tissue biomarkers.

Summary Measures: Based on the available evidence, changes from baseline to follow-up for the differences between healthy and injured subjects were considered. Standardized effect sizes were used for comparing the level of Aβ deposition in those exposed to TBI compared to non-exposed subjects. For studies that reported frequency counts of increased Aβ in TBI subjects compared to healthy subjects, odds ratios were considered. For differences studies, Hedge's g bias-corrected effect size (g) was used.

Evidence Appraisal: Each study was appraised using the following criteria: 1) human, animal, or cadaveric, 2) prospective or retrospective, and 3) individual study or systematic review. Based on the availability of the evidence for Aβ, all types of research were included. The Strength of Reporting Taxonomy (SORT) was used for classifying the type of evidence.

Search Results: Twenty-six studies were identified and 3 were selected for review after meeting the criteria above. One article was a meta-analysis of animal studies, 1 article was an in vivo case-control imaging study of TBI survivors, and 1 article was a case-control study of deceased athletes and military veterans (cadaveric study).

Data Synthesis: To report on the effects of traumatic brain injury on Aβ deposition, a qualitative review of the evidence was conducted. Where applicable, odds ratios between those with and without TBI were considered. In a study of cadaveric brain specimens from deceased athletes and military veterans, those diagnosed with CTE secondary to TBI had Aβ deposition that occurred at an accelerated rate and with altered dynamics compared to healthy aging (OR=3.8, p<0.001). In a meta-analysis of animal studies in which TBI was induced, there were consistent acute increases in cerebral Aβ in animals 24h to 1 month following TBI (OR=2.97 ±0.40, p 0.001). Regardless of the mechanism for inducing TBI, the overall trend was that Aβ deposition increases in the presence of TBI. In a case control study of 9 TBI patients, 9 healthy age-matched controls, and 10 Alzheimer’s’ Disease (AD) patients, those with a history of TBI demonstrated increased Aβ deposition in the cerebellum compared to both the control and AD patients with a large effect size for the between-group comparisons. (TBI-Control: g=1.96±1.12, TBI-AD: g=7.12±2.43).

Evidence Quality: The quality of evidence from the studies provided was a level 3 (disease-oriented evidence) from bench research or a small retrospective human study. Based on the level of evidence, while suggestive of a connection between TBI and Aβ deposition, no causal link can be drawn yet for generalizability to humans suffering TBI.

Conclusions: While the current prospective evidence is confined to bench research, trends suggest Aβ deposition is a sequelae of TBI. As technology advances and our ability to clinically recognize the long-term consequences of TBI improves, athletic trainers should become more aware of Aβ and its role in the long-term neurological outcome of patients they treat for TBI. They should also be prepared to educate the population on the protracted health risks of TBI. Word Count: 598