Quantification of Higher Order Forms of Amyloid Beta and Total Alpha Peptides in CSF

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Abstract

The hypothesis of Alzheimer's disease (AD) posits that oligomers and/or aggregates, but not monomers or plaques, are causative. The total Aβ assay uses an N-terminal capture antibody (6E10*) and a mid-epitope detection antibody (4G8*) (Figure C). The assay detects the Aβ42, and monomeric Aβ40 concentration (Figure P). Total Aβ was measured in human CSF. The Aβ oligomers were generated from pure Aβ42 (0, 0.5, 2.5, and 10 ng/mL) and (2) a constant level of Aβ40 concentration (38-42, and 0.01-100 ng/mL). This may represent a change from monomeric Aβ and oligomeric forms of Aβ40 and Aβ42 peptides were generated and used to develop the assay. The assay was then characterized in normal human CSF. The assay detects oligomeric Aβ, monomeric Aβ, and total Aβ. The assay also has the unique ability to discern differences in Aβ concentration treated to induce oligomeric forms.

Methods

The Aβ oligomers were generated from pure Aβ42 (0, 0.5, 2.5, and 10 ng/mL) and a constant level of Aβ40 concentration (38-42, and 0.01-100 ng/mL). The assay was then characterized in normal human CSF. The assay detects oligomeric Aβ, monomeric Aβ, and total Aβ. The assay also has the unique ability to discern differences in Aβ concentration treated to induce oligomeric forms.

Total Aβ Assay

The total Aβ assay has the ability to detect both monomeric (ECL Signal 10000000) and a heterogeneous detection antibody (4G8*). The assay detects the Aβ42, and monomeric Aβ40 concentration (Figure P). Total Aβ was measured in human CSF. The Aβ oligomers were generated from pure Aβ42 (0, 0.5, 2.5, and 10 ng/mL) and a constant level of Aβ40 concentration (38-42, and 0.01-100 ng/mL). This may represent a change from monomeric Aβ and oligomeric forms of Aβ40 and Aβ42 peptides were generated and used to develop the assay. The assay was then characterized in normal human CSF. The assay detects oligomeric Aβ, monomeric Aβ, and total Aβ. The assay also has the unique ability to discern differences in Aβ concentration treated to induce oligomeric forms.

Cligomeric Aβ Assay

The oligomeric Aβ assay uses the same N-terminal capture antibody (6E10*) and the anti-detection antibody (4G8*). This assay format was developed by Covance for use in higher order disease state detection. This assay format was developed by Covance for use in higher order disease state detection. This assay format was developed by Covance for use in higher order disease state detection. This assay format was developed by Covance for use in higher order disease state detection. This assay format was developed by Covance for use in higher order disease state detection.

Peptide-Specific Aβ Assay: Aβ40-Specific Capture

The oligomer-specific assay was performed using the 6E10 Capture Antibody (12F4) and a mid-epitope detection antibody (4G8*). The assay detects the Aβ42, and monomeric Aβ40 concentration (Figure P). Total Aβ was measured in human CSF. The Aβ oligomers were generated from pure Aβ42 (0, 0.5, 2.5, and 10 ng/mL) and a constant level of Aβ40 concentration (38-42, and 0.01-100 ng/mL). This may represent a change from monomeric Aβ and oligomeric forms of Aβ40 and Aβ42 peptides were generated and used to develop the assay. The assay was then characterized in normal human CSF. The assay detects oligomeric Aβ, monomeric Aβ, and total Aβ. The assay also has the unique ability to discern differences in Aβ concentration treated to induce oligomeric forms.

Conclusion

The development of oligomer-specific assays for the measurement of total Aβ and oligomers Aβ is crucial to understanding AD progression. The assay design and performance is in line with the necessity to measure oligomers Aβ. The assay was successfully characterized in normal human CSF. The Aβ oligomers were generated from pure Aβ42 (0, 0.5, 2.5, and 10 ng/mL) and a constant level of Aβ40 concentration (38-42, and 0.01-100 ng/mL). This may represent a change from monomeric Aβ and oligomeric forms of Aβ40 and Aβ42 peptides were generated and used to develop the assay. The assay was then characterized in normal human CSF. The assay detects oligomeric Aβ, monomeric Aβ, and total Aβ. The assay also has the unique ability to discern differences in Aβ concentration treated to induce oligomeric forms.

Human CSF Sample Testing

Five normal CSF pools from normal donors and 6 ill-convoluted CSF individual samples from patients with various neurological diseases were used in this study. The assay was characterized in normal human CSF. The Aβ oligomers were generated from pure Aβ42 (0, 0.5, 2.5, and 10 ng/mL) and a constant level of Aβ40 concentration (38-42, and 0.01-100 ng/mL). This may represent a change from monomeric Aβ and oligomeric forms of Aβ40 and Aβ42 peptides were generated and used to develop the assay. The assay was then characterized in normal human CSF. The assay detects oligomeric Aβ, monomeric Aβ, and total Aβ. The assay also has the unique ability to discern differences in Aβ concentration treated to induce oligomeric forms. The assay was characterized in normal human CSF. The Aβ oligomers were generated from pure Aβ42 (0, 0.5, 2.5, and 10 ng/mL) and a constant level of Aβ40 concentration (38-42, and 0.01-100 ng/mL). This may represent a change from monomeric Aβ and oligomeric forms of Aβ40 and Aβ42 peptides were generated and used to develop the assay. The assay was then characterized in normal human CSF. The assay detects oligomeric Aβ, monomeric Aβ, and total Aβ. The assay also has the unique ability to discern differences in Aβ concentration treated to induce oligomeric forms.