Association between blood Aβ and brain Aβ burden (Aβ status determined by PET Aβ)

Technology type	Biomarkers measured	Cohort details	Change in Aβ+ group *vs* Aβ- group	Accuracy to identify increased amyloid pathology (AUC for Aβ+ vs Aβ-)	Correlation with *in vivo* amyloid pathology
IP-MS (MALDI-TOF/MS) *(Kaneko et al., 2014)*	Plasma Aβ40, Aβ42 and APP669–711 (Aβ-3–40)	62 participants: 22 Aβ- and 40 Aβ+	• Plasma Aβ42: ↓ in Aβ+ group • Plasma Aβ42/Aβ40 ratio: ↓ in Aβ+ group • Plasma APP669–711/Aβ42 ratio: ↑ in Aβ+ group	• Plasma Aβ42: 0.808 • Plasma Aβ42/Aβ40 ratio: 0.798 • Plasma APP669–711/Aβ42 ratio: 0.969	SUVR and • Plasma Aβ42: r= **-0.374** • Plasma Aβ42/Aβ40 ratio: r= **-0.316** • Plasma APP669-711/Aβ42 ratio: r= **0.687**
IP-MS (LC-MS/MS) *(Ovod et al., 2017)*	Plasma Aβ38, Aβ40 and Aβ42	41 participants: 23 Aβ- and 18 Aβ+ Note: Aβ status was determined by PET when available and otherwise by CSF Aβ42 concentration	• Plasma Aβ42/Aβ40 ratio: ↓ in Aβ+ group	Plasma Aβ42/Aβ40 ratio: 0.887	
IP-MS (MALDI-TOF/MS) *(Nakamura et al., 2018)*	Plasma Aβ40, Aβ42 and APP669–711 (Aβ-3–40) **Composite biomarker:** average of the normalized values of APP669-711/Ab42 ratio and Aβ40/Aβ42 ratio	• Cohort 1 (NCGG, n=121): 71 Aβ- and 50 Aβ+ • Cohort 2 (AIBL, n= 252): 115 Aβ- and 137 Aβ+ ➢ PiB (n=111; 51 Aβ- and 60 Aβ+) and FLUTE/ FBP (n=141)	Both NCGG and AIBL: • Plasma Aβ42: ↓ in Aβ+ group • Plasma Aβ40/Aβ42 ratio: ↑ in Aβ+ group • Plasma APP669-711/Aβ42 ratio: ↑ in Aβ+ group • Plasma Composite biomarker: ↑ in Aβ+ group	• Plasma Aβ42: ➢ NCGG: 0.872 (0.913)# ➢ AIBL PiB: 0.757 (0.812)# ➢ AIBL Overall: 0.718 (0.797)# ➢ NCGG + AIBL (PiB): 0.835 ➢ NCGG + AIBL (overall): 0.789 • Plasma Aβ40/Aβ42 ratio: ➢ NCGG: 0.967 (0.979)# ➢ AIBL PiB: 0.889 (0.897)# ➢ AIBL Overall: 0.837 (0.851)# ➢ NCGG + AIBL (PiB): 0.935 ➢ NCGG + AIBL (overall): 0.886 • Plasma APP669-711/Aβ42 ratio: ➢ NCGG: 0.923 (0.933)# ➢ AIBL PiB: 0.895 (0.905)# ➢ AIBL Overall: 0.828 (0.854)# ➢ NCGG + AIBL (PiB): 0.907 • NCGG + AIBL (overall): 0.861 • Plasma Composite biomarker: • NCGG: 0.967 (0.974)# • AIBL PiB: 0.941 (0.940)# • AIBL Overall: 0.883 (0.888)# • NCGG + AIBL (PiB): 0.954 ➢ NCGG + AIBL (overall): 0.914 #AUC after adjustment for age, gender, APOE and clinical category	• SUVR and plasma Aβ42 in: ➢ NCGG: r= **-0.601** ➢ AIBL PiB: r= **-0.423** ➢ NCGG + AIBL PiB: r= **-0.529** ➢ Overall (NCGG + AIBL overall): r= **-0.484** • SUVR and plasma Aβ40/Aβ42 ratio in: ➢ NCGG: r= **0.767** ➢ AIBL PiB: r= **0.601** ➢ NCGG + AIBL PiB: r= **0.694** ➢ Overall (NCGG + AIBL overall): r= **0.626** • SUVR and plasma APP669-711/ Aβ42 ratio in: ➢ NCGG: r= **0.715** ➢ AIBL PiB: r= **0.612** ➢ NCGG + AIBL PiB: r= **0.670** ➢ Overall (NCGG + AIBL overall): r= **0.606** • SUVR and plasma composite biomarker in: ➢ NCGG: r= **0.785** ➢ AIBL PiB: r= **0.684** ➢ NCGG + AIBL PiB: r= **0.735** ➢ Overall (NCGG + AIBL overall): r= **0.678**
IP-MS (LC-MS/MS) (Schindler et al., 2019)	Plasma Aβ38, Aβ40 and Aβ42	158 participants: • 115 Aβ-, 43 Aβ+ • PiB (n=117; 88 Aβ- and 29 Aβ+), AV45 (Florbetapir; n=41; 27 Aβ- and 14 Aβ+)	• Plasma Aβ42/Aβ40 ratio: ↓ in Aβ+ group	• Plasma Aβ42/Aβ40 ratio: 0.88 • Plasma Aβ42/Aβ40 ratio, age and APOE: 0.94	• PET centiloid and plasma Aβ42/Aβ40 ratio: r= -0.55 (*p-value not reported; 95% CI= -0.65 to -0.43*)
IP-MS (LC-MS/MS) vs Simoa (Keshavan et al., 2021)	LC-MS: Plasma Aβ38, Aβ40, Aβ42, APP669–711 (Aβ-3–40), Composite biomarker (average of the z-scores of APP669-711/Aβ42 ratio and Aβ40/Ab42 ratio Simoa: Plasma Aβ40 and Aβ42 (also measured p-tau181; refer to supplementary Table 2)	441 participants: • 359 Aβ-, 82 Aβ+ • Florbetapir PET	LC-MS: • Plasma Aβ42: ↓ in Aβ+ group • Plasma Aβ42/Aβ40: ↓ in Aβ+ group • Plasma Composite biomarker: ↑ in Aβ+ group Simoa: • Plasma Aβ42: ↓ in Aβ+ group • Plasma Aβ42/Aβ40: ↓ in Aβ+ group	• All subjects (410 CU, 7 MCI, 24 prior neurological condition): LC-MS: ➢ Plasma Aβ42: 0.736 (0.789)# ➢ Plasma Aβ42/Aβ40: 0.817 (0.841)# ➢ Plasma Composite biomarker: 0.820 (0.843)# Simoa: ➢ Plasma Aβ42: 0.590 (0.705)# ➢ Plasma Aβ42/Aβ40: 0.620 (0.727)#	

				➢ Plasma Aβ42/Aβ40 and p-tau181: 0.696 (0.776)# LC-MS and Simoa: ➢ LC-MS Plasma Aβ42/Aβ40 and Simoa Plasma p-tau181: 0.826 (0.851)# ➢ LC-MS Plasma composite and Simoa Plasma p-tau181: 0.829 (0.850)# • CU (n=410) : LC-MS: ➢ Plasma Aβ42: 0.734 (0.785)# ➢ Plasma Aβ42/Aβ40: 0.817 (0.839)# ➢ Plasma Composite biomarker: 0.823 (0.842)# Simoa: ➢ Plasma Aβ42: 0.570 (0.694)# ➢ Plasma Aβ42/Aβ40: 0.610 (0.720)# # AUC after adjustment for age, gender and APOE	
Simoa *(Janelidze et al., 2016)*	Plasma Aβ40, Aβ42 Measured using "first-generation" singleplex from Quanterix	340 participants: • 125 controls, 103 subjective cognitive decline (SCD), 112 MCI • Flutemetamol PET		• Plasma Aβ42, age and gender: 0.604 • Plasma Aβ42/Aβ40 ratio, age and gender: 0.621	• SUVR and plasma Aβ42 in: ➢ All subjects: r= **-0.162** ➢ Controls: r= 0.005 ➢ SCD: r= -0.189 ➢ MCI: r= **-0.295** • SUVR and plasma Aβ42/Aβ40 ratio in: ➢ All subjects: r= **-0.167** ➢ Controls: r= -0.130 ➢ SCD: r= **-0.205** ➢ MCI: r= -0.154
Simoa *(Verberk et al., 2018)*	Plasma Aβ40, Aβ42 Measured using Neurology 3-plex from Quanterix	69 participants: • 23 Aβ+ and 46 Aβ- • Florbetaben (n=33), Florbetapir (n=20), Flutemetamol (n=6), PiB (n=10)	• Plasma Aβ42: ↓ in Aβ+ group • Plasma Aβ42/ Aβ40 ratio: no significant difference between Aβ+ and Aβ- groups (*p=0.057*)	• Plasma Aβ42: 0.66 • Plasma Aβ42/Aβ40 ratio: 0.68 • Plasma Aβ42/Aβ40 ratio, age and APOE: 0.79	
Simoa and ELISA *(De Meyer et al., 2020)*	Plasma Aβ40, Aβ42 Measured using prototype Simoa Amyblood assay or ELISA (EUROIMMUN) Plasma total-tau (t-tau) was also measured using ELISA.	199 participants: • 161 Aβ- and 38 Aβ+ • 161 CU: 137 Aβ- and 24 Aβ+ (Flutemetamol) • 38 aMCI: 24 Aβ- and 14 Aβ+ (Florbetaben)	• Plasma Aβ42/Aβ40 ratio: For both Simoa and ELISA platforms, the ratio ↓ Aβ+ groups in the total cohort, CU and aMCI subgroups • Plasma Aβ42/t-tau ratio: For both Simoa and ELISA platforms, the ratio ↓ Aβ+ groups in the total cohort, CU and aMCI subgroups	• Plasma Aβ42/Aβ40 ratio: ➢ All: Simoa: 0.79 (0.81)# *vs* ELISA: 0.78 (0.78)# ➢ CU: Simoa: 0.77 (0.76)# *vs* ELISA: 0.79 (0.75)# ➢ aMCI: Simoa: 0.86 (0.92)# *vs* ELISA: 0.81 (0.84)# • Plasma Aβ42/t-tau ratio: ➢ All: Simoa: 0.77 (0.80)# *vs* ELISA: 0.77 (0.79)# ➢ CU: Simoa: 0.74 (0.77)# *vs* ELISA: 0.74 (0.75)# ➢ aMCI: Simoa: 0.86 (0.89)# *vs* ELISA: 0.88 (0.88)# #AUC after adjustment for age and APOE	• PET centiloid and plasma Aβ42/Aβ40 ratio: ➢ All: Simoa: r=**-0.32** *vs* ELISA: r=**-0.32** ➢ CU: Simoa: r=**-0.26** *vs* ELISA: r=**-0.25** ➢ aMCI: Simoa: r=**-0.62** *vs* ELISA: r=**-0.68** • PET centiloid and plasma Aβ42/t-tau ratio: ➢ All: Simoa: r=**-0.29** *vs* ELISA: r=**-0.36** ➢ CU: Simoa: r=**-0.24** *vs* ELISA: r=**-0.31** ➢ aMCI: Simoa: r=**-0.58** *vs* ELISA: r=**-0.59**
Simoa *(Brickman et al. 2021)*	Plasma Aβ40, Aβ42 Measured using Neurology 3-plex from Quanterix	300 participants: Of the 300 participants, 40 participants have amyloid PET scan: 8Aβ+ and 32 Aβ-	• Plasma Aβ42/ Aβ40 ratio: no significant difference between Aβ+ and Aβ- groups		
IMR *(Tzen et al., 2014)*	Plasma Aβ40, Aβ42	45 participants			• SUVR and plasma Aβ42/Aβ40 ratio: β= **0.652** (Regression)
MDS	Plasma oligomeric Aβ	50 participants			• SUVR and plasma MDS RLU: r= 0.430

(Wang et al., 2017)	Measured in MDS relative luminescence unit (RLU): higher MDS RLU = higher Aβ oligomers				(*p-value not reported*)
MSD *(Vogelgsang et al., 2018)*	Plasma Aβ38, Aβ40, Aβ42 Note: in most plasma samples, Aβ38 was below the LLOD and excluded from further analysis	41 participants: Of the 41 participants, 20 have amyloid PET scan: 10 Aβ- and 10 Aβ+	• Plasma Aβ42: no significant difference between Aβ+ and Aβ- groups • Plasma Aβ42/Aβ40 ratio: no significant difference between Aβ+ and Aβ- groups		
APEX and Simoa *(Lim et al., 2019) and (Tanaka et al., 2020)*	Plasma exosome-bound Aβ42 (APEX), Simoa-measured Aβ40 and Aβ42	72 participants (4 subjects were excluded in the study by Tanaka et al.,2020): 45 Aβ- and 23 Aβ+		• Plasma APEX-Aβ42: 0.995 • Plasma Simoa-Aβ42: 0.776 • Plasma Simoa-Aβ42/Aβ40 ratio: 0.816	SUVR and • Plasma APEX-Aβ42: r=**0.949** • Plasma Simoa-Aβ42: r= **-0.342** • plasma Simoa-Aβ42/Aβ40 ratio: r= **-0.351**
Interdigitated microelectrode system *(Kim et al., 2019)*	Plasma Aβ in heterogenous (monomers + oligomers/ aggregates) and monomerized states Measured in self-standard ratio, which is calculated by dividing the concentration of homogenous Aβ monomers (in EPPS-treated plasma) by that of heterogenous Aβ (nontreated)	• Cohort 1 (n=53): ➢ Florbetaben PET • Cohort 2 (n=53): ➢ FC119S PET			SUVR and self –standard ratio in: • Cohort 1: r= **0.551** • Cohort 2: r= **0.414**

Association between blood Aβ and brain Aβ burden (Aβ status determined by CSF Aβ)

Technology type	Biomarkers measured	Cohort details	Change in Aβ+ group *vs* Aβ- group	Accuracy to identify increased amyloid pathology (AUC for Aβ+ vs Aβ-)	Correlation with *in vivo* amyloid pathology
IP-MS (MALDI-TOF/MS; confirmed by LC-MS/MS) *(Pannee et al., 2014)*	Plasma Aβ38, Aβ40 and Aβ42 (absolute quantification by selected reaction monitoring)	19 participants: • 9 AD and 10 controls	• Plasma Aβ38, Aβ40, Aβ42 or Aβ42/40 ratio: no significant difference between AD and controls		CSF Aβ42 and plasma Aβ42 in: • Controls: r= -0.067 • AD: r= -0.11
IP-MS (LC-MS/MS) *(Ovod et al., 2017)*	Plasma Aβ38, Aβ40 and Aβ42	41 participants: • 23 Aβ- and 18 Aβ+ Note: Aβ status was determined by PET when available and otherwise by CSF Aβ42 concentration	• Plasma Aβ42/Aβ40 ratio: ↓ in Aβ+ group	• Plasma Aβ42/Aβ40 ratio: 0.887	• CSF Aβ42/Aβ40 ratio and plasma Aβ42/Aβ40 ratio: r= **0.700**
IP-MS (MALDI-TOF/MS) *(Nakamura et al., 2018)*	Plasma Aβ40, Aβ42 and APP669-711 **Composite biomarker values:** average of the normalized values of APP669-711/Ab42 ratio and Ab40/Ab42 ratio	In cohort 2: 46 participants		In this group of 46 subjects, • If PET Aβ was used as the standard for Aβ+/ Aβ- status: Plasma composite biomarker: 0.838 • If CSF Aβ42 was used as the standard for Aβ+/ Aβ- status: Plasma composite biomarker: 0.876	CSF Aβ42 and • Plasma Aβ42: r= **0.408** • Plasma Aβ40/Aβ42 ratio: r= **-0.534** • Plasma APP669-711/Ab42 ratio: r= **-0.601** • Plasma composite biomarker: r= **-0.660**
IP-MS (LC-MS/MS) (Schindler et al., 2019)	Plasma Aβ40, Aβ42	158 participants		• Plasma Aβ42/Aβ40 ratio: 0.85	CSF Aβ42/Aβ40 ratio and plasma Aβ42/Aβ40 ratio: r=0.66 (95% CI: 0.56, 0.75)
Simoa *(Janelidze et al., 2016)*	Plasma Aβ40, Aβ42 Measured using "first-generation" singleplex from Quanterix	719 participants: • 174 SCD, 214 MCI, 57 AD, 274 CU • 74 Aβ+ CU • 200 Aβ- CU • 60 Aβ+ Subjective cognitive decline (SCD) • 121 Aβ+ MCI • 53 Aβ+ AD	• Plasma Aβ42: ↓ in Aβ+ CU, Aβ+ SCD, Aβ+ MCI, Aβ+ AD *vs* Aβ- CU • Plasma Aβ42/Aβ40 ratio: ↓ in Aβ+ CU, Aβ+ SCD, Aβ+ MCI, Aβ+ AD *vs* Aβ- CU	• Plasma Aβ42, age and gender: 0.655 • Plasma Aβ42/Aβ40 ratio, age and gender: 0.683	• CSF Aβ42 and plasma Aβ42 in: ➢ All subjects: r= **0.274** ➢ CU: r= **0.188** ➢ SCD: r= **0.182** ➢ MCI: r= **0.270** ➢ AD: r= **0.288** • CSF Aβ42/Aβ40 ratio and plasma Aβ42/Aβ40 ratio in: ➢ All subjects: r= **0.215** ➢ CU: r= **0.166** ➢ SCD: r= **0.160** ➢ MCI: r= **0.202** ➢ AD: r= -0.003
Simoa *(Verberk et al., 2018)*	Plasma Aβ40, Aβ42 Measured using Neurology 3-plex from Quanterix	248 participants: • 57 Aβ+ and 191 Aβ-	• Plasma Aβ42: ↓ in Aβ+ group • Plasma Aβ42/Aβ40 ratio: ↓ in Aβ+ group	• Plasma Aβ42: 0.66 • Plasma Aβ42/Aβ40 ratio: 0.77 • Plasma Aβ42/Aβ40 ratio, age and APOE: 0.83	• CSF Aβ42 and plasma Aβ42: r= **0.18** • CSF Aβ42 and plasma Aβ42/Aβ40 ratio: r= **0.38**
Simoa *(Startin et al., 2019)*	Plasma Aβ40, Aβ42	54 participants: • 27 controls and 27 Aβ+ AD (and 31 Down syndrome)	• Plasma Aβ42: No significant difference between Aβ+ AD and controls • Plasma Aβ42/Aβ40 ratio: ↓ in Aβ+ AD *vs* controls		
Simoa *(Thijssen et al., 2018)*	Plasma Aβ40, Aβ42 Measured using prototype or commercially available Quanterix assay	40 participants: • 20 Aβ- SCD and 20 Aβ+ AD		• Prototype assay plasma Aβ42/Aβ40 ratio: 0.953 • Commercial assay plasma Aβ42/Aβ40 ratio: 0.852	CSF Aβ42 and plasma Aβ42/Aβ40 ratio in: • Prototype assay: r= **0.711** • Commercial assay: r= **0.527**
Simoa and ELISA *(De Meyer et al., 2020)*	Plasma Aβ40, Aβ42 Measured using prototype Simoa Amyblood assay or ELISA (EUROIMMUN) Plasma total-tau (t-tau) was also measured using ELISA.	199 participants: • 161 Aβ- and 38 Aβ+ • 161 CU: 137 Aβ- and 24 Aβ+ (Flutemetamol) • 38 aMCI: 24 Aβ- and 14 Aβ+ (Florbetaben)			• CSF Aβ42/t-tau and plasma Aβ42/Aβ40 ratio: ➢ All: Simoa: r= **0.29** *vs* ELISA: r= **0.41** ➢ CU: Simoa: r= 0.25 *vs* ELISA: r= 0.34 ➢ aMCI: Simoa: r= **0.51** *vs* ELISA: r= **0.80**

					• CSF Aβ42/t-tau and plasma Aβ42/t-tau ratio: ➢ All: Simoa: r= **0.41** *vs* ELISA: r= **0.50** ➢ CU: Simoa: r= **0.41** *vs* ELISA: r= **0.50** ➢ aMCI: Simoa: r= **0.61** *vs* ELISA: r= **0.68**
MDS *(Wang et al., 2017)*	Plasma oligomeric Aβ Measured in MDS relative light unit (RLU) → higher MDS RLU = higher Aβ oligomers	50 participants			• CSF Aβ42 and MDS RLU: r= -0.443 (*p-value not reported*)
MSD *(Vogelgsang et al., 2018)*	Plasma Aβ38, Aβ40, Aβ42	41 participants: • Of the 41 participants, 33 subjects have CSF analysis			• CSF Aβ42 and plasma Aβ42: r= 0.017 • CSF Aβ42/Aβ40 ratio and plasma Aβ42/Aβ40 ratio: r= **0.425**
IMR *(Teunissen et al., 2018)*	Plasma Aβ42	• Cohort 1 (n=51) ➢ 33 Aβ+ AD and 18 Aβ- CU (controls) • Cohort 2 (n=55): ➢ 30 AD and 25 SCD (controls)	Combining subjects of the 2 sites: • Plasma Aβ42: ↑ in AD *vs* controls		CSF Aβ42 and plasma Aβ42: • Controls: r=0.186 • AD: r= -0.352 (*p-value not reported*)
Elecsys immunoassay *(Palmqvist et al., 2019)*	Plasma Aβ40, Aβ42	• Cohort 1 (n=842): ➢ 368 Aβ+ and 474 Aβ- ➢ 513 CU: 147 Aβ+, 366 Aβ- ➢ 265 MCI: 157 Aβ+, 108 Aβ- ➢ 64 AD: all Aβ+ • Cohort 2 (n=237): ➢ 34 CU ➢ 109 MCI ➢ 94 AD mild dementia	Cohort 1: ➢ Plasma Aβ42 or Aβ42/Aβ40 ratio: ↓ in Aβ+ group ➢ Plasma Aβ42 or Aβ42/Aβ40 ratio: ↓ in Aβ+ CU, Aβ+ MCI and Aβ+ AD *vs* Aβ- CU and Aβ- MCI	• Cohort 1: Plasma Aβ42 in: ➢ All subjects: 0.71 ➢ CU: 0.71 ➢ MCI+AD: 0.72 Plasma Aβ42/Aβ40 ratio in: ➢ All subjects: 0.77 ➢ CU: 0.78 ➢ MCI+AD: 0.75 Plasma Aβ42 and Aβ40 in: ➢ All subjects: 0.80 ➢ CU: 0.78 ➢ MCI+AD: 0.80 Plasma Aβ42, Aβ40 and APOE in: ➢ All subjects: 0.85 ➢ CU: 0.84 ➢ MCI+AD: 0.84 • Cohort 2: ➢ Plasma Aβ42 and Aβ40: 0.86	Cohort 1: • CSF Aβ42 and plasma Aβ42 in: ➢ All subjects: r= **0.373** ➢ CU: r= **0.284** ➢ MCI: r= **0.368** ➢ AD: r= **0.395** • CSF Aβ42/40 ratio and plasma Aβ42/40 ratio in: ➢ All subjects: r= **0.476** ➢ CU: r= **0.452** ➢ MCI: r= **0.410** ➢ AD: r= -0.047

All studies used PiB-PET for amyloid imaging unless stated otherwise.
Significant correlation coefficients (p<0.05) are in red font.

References

De Meyer, S., Schaeverbeke, J. M., Verberk, I. M. W., Gille, B., De Schaepdryver, M., Luckett, E. S., . . . Poesen, K. (2020). Comparison of ELISA- and SIMOA-based quantification of plasma Aβ ratios for early detection of cerebral amyloidosis. *Alzheimer's Research & Therapy, 12*(1), 162. doi:10.1186/s13195-020-00728-w

Janelidze, S., Stomrud, E., Palmqvist, S., Zetterberg, H., van Westen, D., Jeromin, A., . . . Hansson, O. (2016). Plasma β-amyloid in Alzheimer's disease and vascular disease. *Scientific Reports, 6*, 26801. doi:10.1038/srep26801

https://www.nature.com/articles/srep26801#supplementary-information

Kaneko, N., Nakamura, A., Washimi, Y., Kato, T., Sakurai, T., Arahata, Y., . . . Yanagisawa, K. (2014). Novel plasma biomarker surrogating cerebral amyloid deposition. *Proc Jpn Acad Ser B Phys Biol Sci, 90*(9), 353-364.

Keshavan, A., Pannee, J., Karikari, T. K., Rodriguez, J. L., Ashton, N. J., Nicholas, J. M., . . . Schott, J. M. (2021). Population-based blood screening for preclinical Alzheimer's disease in a British birth cohort at age 70. *Brain, 144*(2), 434-449. doi:10.1093/brain/awaa403 %J Brain

Kim, Y., Yoo, Y. K., Kim, H. Y., Roh, J. H., Kim, J., Baek, S., . . . Hwang, K. S. (2019). Comparative analyses of plasma amyloid-beta levels in heterogeneous and monomerized states by interdigitated microelectrode sensor system. *Sci Adv, 5*(4), eaav1388. doi:10.1126/sciadv.aav1388

Lim, C. Z. J., Zhang, Y., Chen, Y., Zhao, H., Stephenson, M. C., Ho, N. R. Y., . . . Shao, H. (2019). Subtyping of circulating exosome-bound amyloid β reflects brain plaque deposition. *Nature Communications, 10*(1), 1144. doi:10.1038/s41467-019-09030-2

Nakamura, A., Kaneko, N., Villemagne, V. L., Kato, T., Doecke, J., Doré, V., . . . Yanagisawa, K. (2018). High performance plasma amyloid-β biomarkers for Alzheimer's disease. *Nature, 554*, 249. doi:10.1038/nature25456

https://www.nature.com/articles/nature25456#supplementary-information

Ovod, V., Ramsey, K. N., Mawuenyega, K. G., Bollinger, J. G., Hicks, T., Schneider, T., . . . Bateman, R. J. (2017). Amyloid β concentrations and stable isotope labeling kinetics of human plasma specific to central nervous system amyloidosis. *Alzheimers Dement, 13*(8), 841-849. doi:10.1016/j.jalz.2017.06.2266

Palmqvist, S., Janelidze, S., Stomrud, E., Zetterberg, H., Karl, J., Zink, K., . . . Hansson, O. (2019). Performance of Fully Automated Plasma Assays as Screening Tests for Alzheimer Disease-Related β-Amyloid Status. *JAMA Neurology*, e191632. doi:10.1001/jamaneurol.2019.1632

Pannee, J., Törnqvist, U., Westerlund, A., Ingelsson, M., Lannfelt, L., Brinkmalm, G., . . . Portelius, E. (2014). The amyloid-β degradation pattern in plasma--a possible tool for clinical trials in Alzheimer's disease. *Neurosci Lett, 573*, 7-12. doi:10.1016/j.neulet.2014.04.041

Schindler, S. E., Bollinger, J. G., Ovod, V., Mawuenyega, K. G., Li, Y., Gordon, B. A., . . . Bateman, R. J. (2019). High-precision plasma β-amyloid 42/40 predicts current and future brain amyloidosis. *93*(17), e1647-e1659. doi:10.1212/WNL.0000000000008081 %J Neurology

Startin, C. M., Ashton, N. J., Hamburg, S., Hithersay, R., Wiseman, F. K., Mok, K. Y., . . . Strydom, A. (2019). Plasma biomarkers for amyloid, tau, and cytokines in Down syndrome and sporadic Alzheimer's disease. *Alzheimers Res Ther, 11*(1), 26. doi:10.1186/s13195-019-0477-0

Tanaka, T., Ruifen, J. C., Nai, Y. H., Tan, C. H., Lim, C. Z. J., Zhang, Y., . . . Chen, C. (2020). Head-to-head comparison of amplified plasmonic exosome Aβ42 platform and single-molecule array immunoassay in a memory clinic cohort. *Eur J Neurol*. doi:10.1111/ene.14704

Teunissen, C. E., Chiu, M. J., Yang, C. C., Yang, S. Y., Scheltens, P., Zetterberg, H., & Blennow, K. (2018). Plasma Amyloid-beta (Abeta42) Correlates with Cerebrospinal Fluid Abeta42 in Alzheimer's Disease. *J Alzheimers Dis, 62*(4), 1857-1863. doi:10.3233/jad-170784

Thijssen, E., Verberk, I. M. W., Vanderstichele, H. M., Heijst, H., Scheltens, P., Stoops, E., & Teunissen, C. E. (2018). A PROTOTYPE SIMOA ASSAY QUANTIFYING PLASMA AMYLOID BETA 1-42 AND 1-40 ISOFORMS CAN DIFFERENTIATE PARTICIPANTS WITH AD FROM HEALTHY CONTROL SUBJECTS. *Alzheimer's & Dementia: The Journal of the Alzheimer's Association, 14*(7), P1039. doi:10.1016/j.jalz.2018.06.2824

Tzen, K.-Y., Yang, S.-Y., Chen, T.-F., Cheng, T.-W., Horng, H.-E., Wen, H.-P., . . . Chiu, M.-J. (2014). Plasma Aβ but Not Tau is Related to Brain PiB Retention in Early Alzheimer's Disease. *ACS Chemical Neuroscience, 5*(9), 830-836. doi:10.1021/cn500101j

Verberk, I. M. W., Slot, R. E., Verfaillie, S. C. J., Heijst, H., Prins, N. D., van Berckel, B. N. M., . . . van der Flier, W. M. (2018). Plasma Amyloid as Prescreener for the Earliest Alzheimer Pathological Changes. *Ann Neurol, 84*(5), 648-658. doi:10.1002/ana.25334

Vogelgsang, J., Shahpasand-Kroner, H., Vogelgsang, R., Streit, F., Vukovich, R., & Wiltfang, J. (2018). Multiplex immunoassay measurement of amyloid-beta42 to amyloid-beta40 ratio in plasma discriminates between dementia due to Alzheimer's disease and dementia not due to Alzheimer's disease. *Exp Brain Res, 236*(5), 1241-1250. doi:10.1007/s00221-018-5210-x

Wang, M. J., Yi, S., Han, J.-y., Park, S. Y., Jang, J.-W., Chun, I. K., . . . Kim, S. (2017). Oligomeric forms of amyloid-β protein in plasma as a potential blood-based biomarker for Alzheimer's disease. *Alzheimer's Research & Therapy, 9*(1), 98. doi:10.1186/s13195-017-0324-0