

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

Technology type	Biomarkers measured	Cohort details	Change in A β + group vs A β - group	Accuracy to identify increased amyloid pathology (AUC for A β + vs A β -)	Correlation with <i>in vivo</i> amyloid pathology
IP-MS (MALDI-TOF/MS) (Kaneko et al., 2014)	Plasma A β 40, A β 42 and APP669–711 (A β -3–40)	62 participants: 22 A β - and 40 A β +	<ul style="list-style-type: none"> Plasma Aβ42: ↓ in Aβ+ group Plasma Aβ42/Aβ40 ratio: ↓ in Aβ+ group Plasma APP669–711/Aβ42 ratio: ↑ in Aβ+ group 	<ul style="list-style-type: none"> Plasma Aβ42: 0.808 Plasma Aβ42/Aβ40 ratio: 0.798 Plasma APP669–711/Aβ42 ratio: 0.969 	SUVR and <ul style="list-style-type: none"> 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 A β 38, A β 40 and A β 42	41 participants: 23 A β - and 18 A β + Note: A β status was determined by PET when available and otherwise by CSF A β 42 concentration	<ul style="list-style-type: none"> Plasma Aβ42/Aβ40 ratio: ↓ in Aβ+ group 	Plasma A β 42/A β 40 ratio: 0.887	
IP-MS (MALDI-TOF/MS) (Nakamura et al., 2018)	Plasma A β 40, A β 42 and APP669–711 (A β -3–40) Composite biomarker: average of the normalized values of APP669-711/Ab42 ratio and A β 40/A β 42 ratio	<ul style="list-style-type: none"> Cohort 1 (NCGG, n=121): 71 Aβ- and 50 Aβ+ Cohort 2 (AIBL, n= 252): 115 Aβ- and 137 Aβ+ <ul style="list-style-type: none"> PIB (n=111: 51 Aβ- and 60 Aβ+) and FLUTE/ FBP (n=141) 	Both NCGG and AIBL: <ul style="list-style-type: none"> 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 	<ul style="list-style-type: none"> Plasma Aβ42: <ul style="list-style-type: none"> 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: <ul style="list-style-type: none"> 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: <ul style="list-style-type: none"> 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: <ul style="list-style-type: none"> 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 <p>[#]AUC after adjustment for age, gender, APOE and clinical category</p>	<ul style="list-style-type: none"> SUVR and plasma Aβ42 in: <ul style="list-style-type: none"> 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: <ul style="list-style-type: none"> 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: <ul style="list-style-type: none"> 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: <ul style="list-style-type: none"> 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 A β 38, A β 40 and A β 42	158 participants: <ul style="list-style-type: none"> 115 Aβ-, 43 Aβ+ PIB (n=117: 88 Aβ- and 29 Aβ+), AV45 (Florbetapir; n=41: 27 Aβ- and 14 Aβ+)) 	<ul style="list-style-type: none"> Plasma Aβ42/Aβ40 ratio: ↓ in Aβ+ group 	<ul style="list-style-type: none"> Plasma Aβ42/Aβ40 ratio: 0.88 Plasma Aβ42/Aβ40 ratio, age and APOE: 0.94 	<ul style="list-style-type: none"> 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 A β 38, A β 40, A β 42, APP669–711 (A β -3–40), Composite biomarker (average of the z-scores of APP669-711/A β 42 ratio and A β 40/Ab42 ratio) Simoa: Plasma A β 40 and A β 42 (also measured p-tau181; refer to supplementary Table 2)	441 participants: <ul style="list-style-type: none"> 359 Aβ-, 82 Aβ+ Florbetapir PET 	LC-MS: <ul style="list-style-type: none"> Plasma Aβ42: ↓ in Aβ+ group Plasma Aβ42/Aβ40: ↓ in Aβ+ group Plasma Composite biomarker: ↑ in Aβ+ group <p>Simoa:</p> <ul style="list-style-type: none"> Plasma Aβ42: ↓ in Aβ+ group Plasma Aβ42/Aβ40: ↓ in Aβ+ group 	<ul style="list-style-type: none"> All subjects (410 CU, 7 MCI, 24 prior neurological condition): LC-MS: <ul style="list-style-type: none"> Plasma Aβ42: 0.736 (0.789)[#] Plasma Aβ42/Aβ40: 0.817 (0.841)[#] Plasma Composite biomarker: 0.820 (0.843)[#] Simoa: <ul style="list-style-type: none"> Plasma Aβ42: 0.590 (0.705)[#] Plasma Aβ42/Aβ40: 0.620 (0.727)[#] 	

				<ul style="list-style-type: none"> ➢ Plasma Aβ42/Aβ40 and p-tau181: 0.696 (0.776)[#] <p>LC-MS and Simoa:</p> <ul style="list-style-type: none"> ➢ 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)[#] <p>• CU (n=410) :</p> <p>LC-MS:</p> <ul style="list-style-type: none"> ➢ Plasma Aβ42: 0.734 (0.785)[#] ➢ Plasma Aβ42/Aβ40: 0.817 (0.839)[#] ➢ Plasma Composite biomarker: 0.823 (0.842)[#] <p>Simoa:</p> <ul style="list-style-type: none"> ➢ Plasma Aβ42: 0.570 (0.694)[#] ➢ Plasma Aβ42/Aβ40: 0.610 (0.720)[#] <p>[#] AUC after adjustment for age, gender and APOE</p>	
Simoa (Janellidze 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		<ul style="list-style-type: none"> • Plasma Aβ42, age and gender: 0.604 • Plasma Aβ42/Aβ40 ratio, age and gender: 0.621 	<ul style="list-style-type: none"> • SUVR and plasma Aβ42 in: ➢ All subjects: r= -0.162 ➢ Controls: r= 0.005 ➢ SCD: r= -0.189 ➢ MCI: r= -0.295 <ul style="list-style-type: none"> • 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)	<ul style="list-style-type: none"> • Plasma Aβ42: ↓ in Aβ+ group • Plasma Aβ42/ Aβ40 ratio: no significant difference between Aβ+ and Aβ- groups (p=0.057) 	<ul style="list-style-type: none"> • 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)	<ul style="list-style-type: none"> • 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 	<ul style="list-style-type: none"> • 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)[#] <p>[#]AUC after adjustment for age and APOE</p>	<ul style="list-style-type: none"> • 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-	<ul style="list-style-type: none"> • Plasma Aβ42/ Aβ40 ratio: no significant difference between Aβ+ and Aβ- groups 		
IMR (Tzen et al., 2014)	Plasma Aβ40, Aβ42	45 participants			<ul style="list-style-type: none"> • SUVR and plasma Aβ42/Aβ40 ratio: β= 0.652 (Regression)
MDS	Plasma oligomeric Aβ	50 participants			<ul style="list-style-type: none"> • SUVR and plasma MDS RLU: r= 0.430

(Wang et al., 2017)	Measured in MDS relative luminescence unit (RLU): higher MDS RLU = higher A β oligomers				(p-value not reported)
MSD (Vogelgsang et al., 2018)	Plasma A β 38, A β 40, A β 42 Note: in most plasma samples, A β 38 was below the LLOD and excluded from further analysis	41 participants: Of the 41 participants, 20 have amyloid PET scan: 10 A β - and 10 A β +	<ul style="list-style-type: none"> 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 A β 42 (APEX), Simoa-measured A β 40 and A β 42	72 participants (4 subjects were excluded in the study by Tanaka et al., 2020): 45 A β - and 23 A β +		<ul style="list-style-type: none"> Plasma APEX-Aβ42: 0.995 Plasma Simoa-Aβ42: 0.776 Plasma Simoa-Aβ42/Aβ40 ratio: 0.816 	SUVR and <ul style="list-style-type: none"> 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 A β in heterogenous (monomers + oligomers/ aggregates) and monomerized states Measured in self-standard ratio, which is calculated by dividing the concentration of homogenous A β monomers (in EPPS-treated plasma) by that of heterogenous A β (nontreated)	<ul style="list-style-type: none"> Cohort 1 (n=53): ➢ Florbetaben PET Cohort 2 (n=53): ➢ FC119S PET 			SUVR and self –standard ratio in: <ul style="list-style-type: none"> Cohort 1: r= 0.551 Cohort 2: r= 0.414

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

Technology type	Biomarkers measured	Cohort details	Change in A β + group vs A β - group	Accuracy to identify increased amyloid pathology (AUC for A β + vs A β -)	Correlation with <i>in vivo</i> amyloid pathology
IP-MS (MALDI-TOF/MS; confirmed by LC-MS/MS) (Pannee et al., 2014)	Plasma A β 38, A β 40 and A β 42 (absolute quantification by selected reaction monitoring)	19 participants: • 9 AD and 10 controls	• Plasma A β 38, A β 40, A β 42 or A β 42/40 ratio: no significant difference between AD and controls		CSF A β 42 and plasma A β 42 in: • Controls: $r = -0.067$ • AD: $r = -0.11$
IP-MS (LC-MS/MS) (Ovod et al., 2017)	Plasma A β 38, A β 40 and A β 42	41 participants: • 23 A β - and 18 A β + Note: A β status was determined by PET when available and otherwise by CSF A β 42 concentration	• Plasma A β 42/A β 40 ratio: \downarrow in A β + group	• Plasma A β 42/A β 40 ratio: 0.887	• CSF A β 42/A β 40 ratio and plasma A β 42/A β 40 ratio: $r = 0.700$
IP-MS (MALDI-TOF/MS) (Nakamura et al., 2018)	Plasma A β 40, A β 42 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 A β was used as the standard for A β + / A β - status: Plasma composite biomarker: 0.838 • If CSF A β 42 was used as the standard for A β + / A β - status: Plasma composite biomarker: 0.876	CSF A β 42 and • Plasma A β 42: $r = 0.408$ • Plasma A β 40/A β 42 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 A β 40, A β 42	158 participants		• Plasma A β 42/A β 40 ratio: 0.85	CSF A β 42/A β 40 ratio and plasma A β 42/A β 40 ratio: $r = 0.66$ (95% CI: 0.56, 0.75)
Simoa (Janellidze et al., 2016)	Plasma A β 40, A β 42 Measured using "first-generation" singleplex from Quanterix	719 participants: • 174 SCD, 214 MCI, 57 AD, 274 CU • 74 A β + CU • 200 A β - CU • 60 A β + Subjective cognitive decline (SCD) • 121 A β + MCI • 53 A β + AD	• Plasma A β 42: \downarrow in A β + CU, A β + SCD, A β + MCI, A β + AD vs A β - CU • Plasma A β 42/A β 40 ratio: \downarrow in A β + CU, A β + SCD, A β + MCI, A β + AD vs A β - CU	• Plasma A β 42, age and gender: 0.655 • Plasma A β 42/A β 40 ratio, age and gender: 0.683	• CSF A β 42 and plasma A β 42 in: > All subjects: $r = 0.274$ > CU: $r = 0.188$ > SCD: $r = 0.182$ > MCI: $r = 0.270$ > AD: $r = 0.288$ • CSF A β 42/A β 40 ratio and plasma A β 42/A β 40 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 A β 40, A β 42 Measured using Neurology 3-plex from Quanterix	248 participants: • 57 A β + and 191 A β -	• Plasma A β 42: \downarrow in A β + group • Plasma A β 42/A β 40 ratio: \downarrow in A β + group	• Plasma A β 42: 0.66 • Plasma A β 42/A β 40 ratio: 0.77 • Plasma A β 42/A β 40 ratio, age and APOE: 0.83	• CSF A β 42 and plasma A β 42: $r = 0.18$ • CSF A β 42 and plasma A β 42/A β 40 ratio: $r = 0.38$
Simoa (Startin et al., 2019)	Plasma A β 40, A β 42	54 participants: • 27 controls and 27 A β + AD (and 31 Down syndrome)	• Plasma A β 42: No significant difference between A β + AD and controls • Plasma A β 42/A β 40 ratio: \downarrow in A β + AD vs controls		
Simoa (Thijssen et al., 2018)	Plasma A β 40, A β 42 Measured using prototype or commercially available Quanterix assay	40 participants: • 20 A β - SCD and 20 A β + AD		• Prototype assay plasma A β 42/A β 40 ratio: 0.953 • Commercial assay plasma A β 42/A β 40 ratio: 0.852	CSF A β 42 and plasma A β 42/A β 40 ratio in: • Prototype assay: $r = 0.711$ • Commercial assay: $r = 0.527$
Simoa and ELISA (De Meyer et al., 2020)	Plasma A β 40, A β 42 Measured using prototype Simoa Amyblood assay or ELISA (EUROIMMUN) Plasma total-tau (t-tau) was also measured using ELISA.	199 participants: • 161 A β - and 38 A β + • 161 CU: 137 A β - and 24 A β + (Flutemetamol) • 38 aMCI: 24 A β - and 14 A β + (Florbetaben)			• CSF A β 42/t-tau and plasma A β 42/A β 40 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: $r = 0.80$

					<ul style="list-style-type: none"> CSF Aβ42/t-tau and plasma Aβ42/t-tau ratio: <ul style="list-style-type: none"> > 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 A β Measured in MDS relative light unit (RLU) → higher MDS RLU = higher A β oligomers	50 participants			<ul style="list-style-type: none"> CSF Aβ42 and MDS RLU: r= -0.443 (p-value not reported)
MSD (Vogelgsang et al., 2018)	Plasma A β 38, A β 40, A β 42	41 participants: <ul style="list-style-type: none"> Of the 41 participants, 33 subjects have CSF analysis 			<ul style="list-style-type: none"> 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 A β 42	<ul style="list-style-type: none"> Cohort 1 (n=51) <ul style="list-style-type: none"> 33 Aβ+ AD and 18 Aβ- CU (controls) Cohort 2 (n=55): <ul style="list-style-type: none"> 30 AD and 25 SCD (controls) 	Combining subjects of the 2 sites: <ul style="list-style-type: none"> Plasma Aβ42: ↑ in AD vs controls 		<ul style="list-style-type: none"> CSF Aβ42 and plasma Aβ42: <ul style="list-style-type: none"> Controls: r=0.186 AD: r= -0.352 (p-value not reported)
Elecsys immunoassay (Palmqvist et al., 2019)	Plasma A β 40, A β 42	<ul style="list-style-type: none"> Cohort 1 (n=842): <ul style="list-style-type: none"> 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): <ul style="list-style-type: none"> 34 CU 109 MCI 94 AD mild dementia 	Cohort 1: <ul style="list-style-type: none"> 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 	<ul style="list-style-type: none"> Cohort 1: <ul style="list-style-type: none"> Plasma Aβ42 in: <ul style="list-style-type: none"> All subjects: 0.71 CU: 0.71 MCI+AD: 0.72 Plasma Aβ42/Aβ40 ratio in: <ul style="list-style-type: none"> All subjects: 0.77 CU: 0.78 MCI+AD: 0.75 Plasma Aβ42 and Aβ40 in: <ul style="list-style-type: none"> All subjects: 0.80 CU: 0.78 MCI+AD: 0.80 Plasma Aβ42, Aβ40 and APOE in: <ul style="list-style-type: none"> All subjects: 0.85 CU: 0.84 MCI+AD: 0.84 Cohort 2: <ul style="list-style-type: none"> Plasma Aβ42 and Aβ40: 0.86 	<ul style="list-style-type: none"> Cohort 1: <ul style="list-style-type: none"> CSF Aβ42 and plasma Aβ42 in: <ul style="list-style-type: none"> 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: <ul style="list-style-type: none"> 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., Schaeveerbeke, 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 A β 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
- 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., Vilmagne, 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/jamaneuro.2019.1632
- Panee, 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. *Neurology*, *93*(17), e1647-e1659. doi:10.1212/WNL.0000000000008081
- 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 A β 42 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 (A β 42) Correlates with Cerebrospinal Fluid A β 42 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 A β 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