Table 1
Appropriate Use Criteria in Demented and Non-Demented

	Dementia (n=95)				MCI (n=25)		CN (n=50)
	AD (n=68)		Non-AD (n=27)				
	APUC+ (n=45)	APUC- (n=23)	APUC+ (n=14)	APUC- (n=13)	APUC+ (n=18)	APUC- (n=7)	APUC- (n=50)
Age (SD)	65 (±7)	66 (±7)	65 (±10)	67 (±7)	65 (±7)	66 (±7)	61 (7%)
Males (%)	25 (56%)	9 (39%)	13 (93%)	9 (69%)	12 (67%)	6 (86%)	29 (58%)
MMSE (SD)	22 (±4)	21 (±5)	24 (±3)	23 (±6)	27 (±2)	27 (±2)	28 (±3)
[18F]FBB+ (%)	30 (67%)	21 (91%)	7 (50%)	1 (8%)	10 (56%)	5 (71%)	13 (26%)
Change in syndrome diagnosis (%)	2 (4%)	1 (4%)	0 (0%)	0 (0%)	1 (6%)	1 (14%)	3 (6%)
Change in suspected etiology (%)	15 (33%)	1 (4%)	4 (29%)	0 (0%)	5 (28%)	2 (29%)	14 (28%)

etiologically mixed presentation, or [C] that were young (≤65) with dementia. **Results:** Patients who participated (n=170) did not differ in age and sex compared to non-participants (n=273), but had slightly higher MMSE (25±5 vs. 23±6; p0.02). Of participating patients, 95(56%) were demented, 25(15%) had MCI and 50(29%) were cognitively normal. After disclosure of PET results, syndrome diagnosis changed in 8(5%) patients. In demented patients, suspected etiology changed in 16/68(24%) patients with pre-PET suspected AD etiology, and in 4/27(15%) with suspected non-AD etiology. In non-demented patients, suspected etiology changed in 6/18(33%) with pre-PET suspected AD etiology, and in 15/57(26%) with suspected non-AD etiology. When we applied APUC (Table 1), 59/95 dementia and 18/25 MCI patients met the criteria. By definition, cognitively normals were APUC-. In dementia, suspected etiology changed in 19/59(32%) for APUC+, compared to 1/26(4%) in APUC-. In non-demented patients, suspected etiology changed irrespective of APUC status (APUC+28%; APUC-28%). **Conclusions:** In an unselected sample of memory clinic patients, amyloid PET has diagnostic value as it contributes to the ideas regarding suspected underlying etiology in a substantial number of patients. APUC are helpful to identify patients with dementia who may benefit from amyloid imaging, but do not adequately identify non-demented patients benefitting from amyloid PET.

IC-P-012 AMYLOID PET IN CLINICAL PRACTICE

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Background: Amyloid PET imaging has been considered as a major advance in the clinical assessment of patients with cognitive impairment; however there is lack of agreement regarding the use of Amyloid PET scanning in the routine clinical evaluation of dementia. The Alzheimer's Association and the Society of Nuclear Medicine and Molecular Imaging formulated appropriate use criteria (AUC) for amyloid PET imaging in clinical settings especially when knowledge of the presence or absence of amyloid-beta pathology is expected to increase diagnostic certainty and alter the clinical management of the patients. Aims; To analyse the evidence for the use of Amyloid PET scanning in the clinical context and to understand whether any baseline clinical variables reliably differentiated negative from positive Amyloid -PET scan patients. **Methods:** In this observational cohort study we analysed the clinical data for 35 patients meeting AUC who had F-18 florbetapir Amyloid PET scans. **Results:** 42% (15/35) Amyloid positivity was observed in the cohort. The positivity in the subgroup of Amnestic Mild Cognitive Impairment (aMCI) was 75% followed by Probable Alzheimer's Disease (AD) 66.6%, Possible AD 43% and complex patients with diagnostic uncertainty 30%. **Conclusions:** The data confirms that the presence/absence of amyloid pathology in the brain on amyloid PET scans, can serve as important diagnostic confirmatory tool even when the diagnosis is made by expert clinicians. Also it shows that in clinically challenging presentations of dementia syndrome with a heterogeneous aetiology, confirmation of the absence of amyloid brain pathology can aid in excluding AD. Other significant observations in the study include increased amyloid positivity in the aMCI group with stronger correlation with reduced perfusion to temporo-parietal region on a blood flow SPECT scan and the presence of family history of AD.

IC-P-013 PET STAGING OF AMYLOIDOSIS: EVIDENCE THAT AMYLOID OCCURS FIRST IN NEOCORTEX AND LATER IN STRIATUM

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Background: Autopsy studies in sporadic Alzheimer's disease (AD) support the hypothesis that fibrillary amyloidosis (Aβ) occurs first in neocortex and later in striatum. We tested whether Aβ-PET could identify this sequence *in-vivo*, and potentially provide a staging measure for Aβ, in the Alzheimer's Disease Neuroimaging Initiative (ADNI) and the Harvard Aging Brain Study (HABS). **Methods:** We included 1087 ADNI (367 cognitively normal (CN), 523 mild cognitive impairment (MCI), and 197AD) and 336 HABS (275CN, 46MCI, and 15AD) subjects with Aβ-PET (ADNI: [18F]-Florbetapir/AV45, ADNI composite reference; HABS:

Table 1
Distribution of subjects by Aβ-stages, defined by neocortex (CX) and striatum (STR) baseline PET

Aβ Stages	Stage 0	Stage 1	Stage 2	Indeterminate
Aβ Signal in CX/STR	Low CX Low STR	High CX Low STR	High CX High STR	Low CX High STR
AD (ADNI, n=197)	12%	19%	68%	<1%
MCI/AD (HABS, n=61)	36%	10%	54%	0%
MCI (ADNI, n=523)	40%	29%	31%	<1%
CN (ADNI, n=367)	61%	30%	8%	<1%
CN (HABS, n=275)	72%	14%	14%	0%

MCI and AD subjects in HABS have been grouped for power issues

[C11]-PiB, cerebellar gray reference). Subjects were classified as high or low Aβ in neocortical and striatal (caudate and putamen) aggregates, using Gaussian mixture models. We evaluated changes in Aβ classification between baseline and follow-up Aβ-PET (0.9-5.1 years), and the association between Aβ classification at baseline and longitudinal memory performance, longitudinal hippocampal volume, and cross-sectional tau-PET ([18F]-T807/AV1451). **Results:** Both cortical and striatal AV45/PiB have bimodal distribution (Lilliefors KS-test>0.1, p<0.001), allowing categorization. Striatal Aβ is elevated only if cortical Aβ is elevated, classifying subjects in three Aβ-stages: stage 0: low-Aβ in both regions, stage 1: high-cortical but low-striatal Aβ, stage 2: high-Aβ in both regions. Less than 1% of subjects have an indeterminate stage: high-striatal but low-cortical Aβ (Table 1, Figure 1). Subjects with Aβ-stage 0 at baseline are more likely to transition to high cortical than to high striatal Aβ after follow-up (ADNI: χ^2=26.2, p<0.001; HABs: χ^2=4.9, p=0.026). Aβ-stage 1 subjects are more likely to transition to high striatal Aβ than Aβ-stage 0 subjects (ADNI: χ^2=19.6, p<0.001; HABs: χ^2=33.5, p<0.001, Figure 2). Subjects with high-striatal Aβ at baseline are more at-risk for longitudinal memory decline, longitudinal hippocampal atrophy, and tau deposition than subjects with low-striatal Aβ, even among subjects with high-cortical Aβ (Table 2). **Conclusions:** Both datasets provide evidence that striatal Aβ accumulation occurs after neocortical Aβ, at later disease stages. Striatal Aβ is a risk factor for memory decline, hippocampal atrophy, and tau deposition, making it a potentially useful biomarker for tracking disease progression. Further work is ongoing to determine the potential added value of striatal Aβ for predicting cognitive decline in preclinical stages.

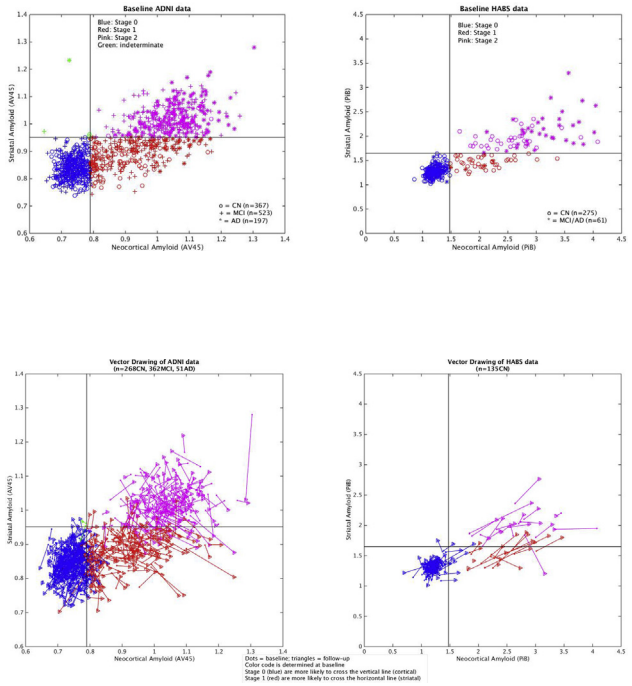

Table 2
Association between Aβ-stages and: (1) longitudinal memory decline (2) longitudinal hippocampal volume and (3) cross-sectional tau (entorhinal and inferior temporal aggregate, cerebellar gray reference)

Comparison	Stage 1 vs 0	Stage 2 vs 0	Stage 2 vs 1
Memory, ADNI (All: n=1087)	T=-4.9, p=e-5	T=-16.6, p=e-60	T=-10.8, p=e-27
Memory, ADNI (CN: n=367)	T=-3.1, p=0.002	T=-3.3, p=0.001	T=-0.9, p=0.352
Memory, HABS (All: n=332)	T=-2.7, p=0.006	T=-6.6, p=e-11	T=-2.5, p=0.011
Memory, HABS (CN: n=275)	T=-2.9, p=0.004	T=-4.9, p=e-5	T=-1.3, p=0.193
Hippocampus, HABS (CN: n=141)	T=+0.4, p=0.707	T=-2.0, p=0.046	T=-1.8, p=0.077
Tau, HABS (CN: n=121)	T=+1.9, p=0.057	T=+5.4, p=e-7	T=+2.6, p=0.010

Linear mixed models with random intercept and slopes, adjusted for age and sex (and education for memory)

IC-P-014

COMBINATION OF VERBAL DELAYED RECALL, STROOP COLOR-WORD, AND MRI CAN BE UTILIZED FOR THE DISCRIMINATION OF AMYLOID DEPOSITION STATUS IN MCI INDIVIDUALS

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Background: It is important to know whether a mild cognitive impairment (MCI) patient has significant amyloid deposition to characterize the brain condition and predict the prognosis. We tried to compare the ability of various clinical, neuropsychological, structural imaging information, and their combinations for the discrimination of amyloid deposition positivity in the brain of MCI individuals. **Methods:** Thirty amnestic MCI subjects were