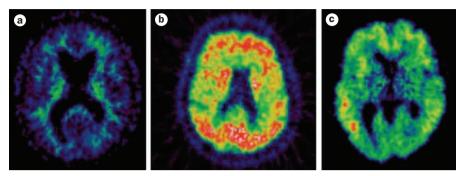
## RESEARCH HIGHLIGHTS

## **DEMENTIA**

## Visualizing the progress of dementia

PET scanning using the thioflavin-based radiotracer Pittsburg compound B (11C-PIB PET) could provide a noninvasive method to reveal the extent of amyloid deposition in brains of patients with mild cognitive impairment (MCI) or Alzheimer disease (AD). David Brooks' group at Hammersmith Hospital, London, UK found that 82% of people with MCI who had raised baseline levels of amyloid- $\beta$  (A $\beta$ ), as detected with <sup>11</sup>C-PIB PET (Figure 1), converted to AD over 3 years, compared with only 7% of amyloid-negative cases. "Using this technique to measure amyloid load in patients with isolated recall difficulties would appear to be of significant prognostic value," observes Brooks.

The capacity of <sup>11</sup>C-PIB PET to produce longitudinal outcome data offers great advantages over autopsy studies. The sensitivity of the technique could allow accurate predictions of which patients with MCI will develop AD, thereby aiding implementation of effective therapeutic strategies. "Some studies suggest that <sup>11</sup>C-PIB PET may be more sensitive



**Figure 1** | Brain amyloid load measured by  $^{11}$ C-PIB PET scanning. **a** | Normal control (MMSE score 28). **b** | Patient with Alzheimer disease (MMSE score 20). **c** | Patient with amnestic mild cognitive impairment (MMSE score 27). Abbreviation: MMSE, Mini-Mental State Examination. Image provided by Prof. David Brooks.

than cerebrospinal fluid  $A\beta$  and tau measurements for detecting an early AD process in MCI," explains Brooks.

Patients with MCI who converted to AD showed high levels of PIB retention in brain areas associated with cognition. The individuals with MCI are currently being followed up with serial <sup>11</sup>C-PIB PET scans, "[enabling] us to follow the kinetics of amyloid deposition in these patients *in vivo*, and to determine whether such

changes correlate with *APOE* ε4 gene carriage," notes Brooks. The researchers are also measuring neuroinflammatory changes in MCI to determine whether cortical inflammation in AD correlates with rates of amyloid deposition.

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NATURE REVIEWS | NEUROLOGY VOLUME 5 | SEPTEMBER 2009 | 465