DOWNLOAD PDF PROGRESS IN DEMENTIA RESEARCH

Chapter 1 : Keeping pace: Progress in dementia research capacity | Alzheimer's Research UK

Research & Progress This is a time of unprecedented promise in the quest to end Alzheimer's. Today, we are growing philanthropic support for Alzheimer's research, fostering a dynamic community of Alzheimer's scientists and securing increased federal funding for research - all of which are instrumental to finding new treatments to stop, slow and prevent Alzheimer's disease.

And we are closing in on a cure. Over the past year, we made great progress, as our top five advances demonstrate. These results were disappointing, but many promising drugs are still advancing. And in , we will ensure that even more diverse treatments reach clinical trials, because the ADDF is committed to getting effective drugs to patients. As we age, our inflammatory response increases. Our funded researchers are developing new drugs to combat neuroinflammation and repurposing approved drugs that target systemic inflammation in other diseases. Several have already reached clinical trials, including etanercept, being tested by Dr. And in Boston, Dr. Phillip Haydon and his colleagues at the biotech Gliacure completed a successful phase 1 trial of GC In , we expect other drugs targeting inflammation to enter clinical trials, including an exciting program from Cleveland Clinic spin-off NeuroTherapia. Think of it like a dimmer switch: In , the first epigenetic treatment for any neurodegenerative disease entered clinical trials. Developed by Oryzon Genomics in Barcelona, the drug called ORY successfully completed a phase 1 trial and will enter phase 2 in Breakthroughs are happening in diagnostics, too. Jacob Hooker at Massachusetts General Hospital allowed us to see epigenetic changes in the brain for the first time. Thanks to the progress made in , epigenetic treatments are on track to become a reality in our lifetime. And others are developing MRI contrast agents to better visualize beta-amyloid plaques and vascular cognitive impairment. Lower-cost tools are also in the works, such as blood tests and mass spectrometry of cerebrospinal fluid. These advances will have huge impacts. Accurately diagnosing patients will result in earlier and better treatment and ensure that clinical trials enroll the right patients. New Evidence for Prevention Perhaps the most exciting findings in were in dementia prevention. We learned that the prevalence of dementia is declining, thanks in part to better management of risk factors such as diabetes and hypertension and higher educational attainment. And new findings added to the evidence that diets rich in DHA can improve brain health. Overall, the findings suggest that we can delay dementia and slow cognitive aging, which is good news. On the site, our neuroscientists review all of the available research and provide clear, unbiased ratings on options to improve brain health and potentially prevent dementia.

Chapter 2 : Pressing for Progress: Women in Dementia Research | Alzheimer's Research UK Blog

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Women in Dementia Research Pressing for Progress: Women in Dementia Research By Katy Stubbs from Science news on 9 October 2 Today is Ada Lovelace Day, a global day celebrating the achievements of women in science, technology, engineering and maths. We spoke with some of the incredible researchers we work with about dementia research, potential barriers and how we can support women in science. Diversity of ideas drives innovation in research Understanding why senior research positions are still predominantly occupied by men is important, because we know that having diversity in a field boosts productivity and innovation. For example, authors of a recent study analysed 1. This can only be a good thing. It drives discussion and it drives novel thinking and novel ideas. While at PhD level, there were slightly more women than men, male professors outnumbered female ones. When you look across all science disciplines and career stages, women make up just The majority of my colleagues at early stages of their careers, PhD students and research assistants are women. Reversing this is no easy task. Both sexes must fight against biases in thinking which can favour male candidates or male progression towards the top of the field. You can work at home quite a lot of the time as well. You can have a life and children as well as a scientific career. The representation of women in the early career stages of my field is fantastic. However, this drops off as you raise through the ranks of academia with far fewer women professors than men. Whether this is by choice needs to be determined. It is not always easy at first glance to understand what the inequality is due to. Having high ranking women to look up to, whether in science or elsewhere, is pivotal for progress being made around gender equality. I was lucky to learn from the more senior researchers who went before me. Not only are these women providing inspiration to others, they are forging a path for others to follow. She was the first woman Chief of Neurology in the US and a fantastic, outspoken role model for women in science. She was awarded the Nobel Prize in Physiology or Medicine jointly with Stanley Cohen for the discovery of nerve growth factor. Prof Levi-Montalcini was discouraged from going to college as it was deemed that this would be deleterious to her being a wife and a mother. Clearly, she took no notice! When we looked at the people applying to our grant schemes, we saw that while more men than women apply to us for research funding, the success rates are on par between men and women. I think widening participation activities and STEM activities in school age children is the way forward. Tweet us ARUKnews and use adalovelaceday and pressforprogress to join the conversation.

Chapter 3 : Progress and Challenges in Frontotemporal Dementia Research: A Year Review - IOS Press

Pressing for Progress: Women in Dementia Research By Katy Stubbs from Science news on 9 October 2 Today is Ada Lovelace Day, a global day celebrating the achievements of women in science, technology, engineering and maths. #adalovelaceday That includes the incredible female researchers helping to bring about the first life-changing.

Knowledge of the clinical phenomenology, cognition, neuroimaging, genetics, pathology of the different subtypes of FTD, and their relations to other neurodegenerative conditions, has increased rapidly, due in part, to the growing interests into these neurodegenerative brain conditions. This article reviews the major advances in the field of FTD over the past 20 years, focusing primarily on the work of Frontier, the frontotemporal dementia clinical research group, based in Sydney, Australia. Topics covered include clinical presentations cognition, behavior, neuroimaging , pathology, genetics, and disease progression, as well as interventions and carer directed research. This review demonstrates the improvement in diagnostic accuracy and capacity to provide advice on genetic risks, prognosis, and outcome. The next major challenge will be to capitalize on these research findings to develop effective disease modifying drugs, which are currently lacking. It is an almost impossible task: Looking at some of the papers on FTD from the s produces the same effect. Semantic dementia SD was just getting recognized but the distinction from the non-fluent forms of progressive aphasia was being worked out and these were not generally considered under the rubric of FTD. Very little was known about the pathology of FTD: The discovery of TDP did not occur until the mid s. Similarly, in the area of genetics, the high rate of familial transmission was evident but only one of the gene mutations responsible for the disease, MAPT, had been identified. The overall prevalence of FTD was unknown, information on prognosis was limited, and we did not have clear implementable diagnostic criteria. It was recognized that occasionally patients with FTD develop motor neuron disease MND and vice versa, but the extent of the clinical overlap was not realized, nor was the pathology and genetics overlaps between FTD and MND. Most studies at that time involved small numbers of cases and it was difficult to draw firm conclusions. While we have learned a lot over the past 20 years, sadly effective therapies still elude us. The best we can do is counsel, support, and help the families of patients with this devastating collection of diseases. Since , over 6, papers have been published on FTD, which is twenty times the number in the previous decade. It is impossible to cover the whole topic and this review unashamedly focuses on our own work. A renaissance of interest in the focal dementias began in the s [1] and accelerated in thes. Workers from Lund, Sweden [2] reported on a large series of patients with dementia and found that a high proportion had evidence of frontal lobe degeneration. At approximately the same time, the Manchester group [3] began a series of important clinico-pathological studies of patients with pre-senile dementia. Gradually, the label of FTD [6] was applied to such cases, but later, after the realization that patients with progressive aphasia share clinical and pathological features, the term bvFTD was adopted [7, 8]. Epidemiological studies have established that collectively FTD is the second most common cause of dementia under age 65 with a prevalence that approximates that of AD [9, 10]. Much of our early research focused on the cognitive features of bvFTD in an attempt to understand the clinical manifestations with the view to improve diagnosis. A range of research over the past 15 years has therefore focused on ways to measure other clinical features, in particular the alterations in social cognition which are core to the syndrome. Social cognition is an umbrella term that refers to a set of complex abilities necessary for successfully engaging in social interactions. Numerous tests have been developed in recent years that attempt to unpack these different aspects of social cognition; some of which include the Empathy for Pain task [12], or the Awareness of Social Inference Test which uses video vignettes [13, 14]. This is a key deficit that almost certainly contributes to their impaired empathy, a source of considerable carer distress [16]. One way of assessing ToM is via the use of cartoons which may or may not have a ToM element. A recent study using cartoons showed an associated between ToM abilities and the right anterior temporal lobe in bvFTD as well as in patients with SD involving right sided structures [17]. In keeping with this, an exploration of cognitive and affective aspects of empathy highlighted the role of the frontoinsular cortex in empathy [20]. At around the same time as the ToM study, we explored the hypothesis

that bvFTD patients may also have deficit in emotion recognition. This hypothesis was confirmed by us [21] and others [22] and led the way to a series of studies charting the extent of the emotion processing deficits and their relation to pathology of the orbito-frontal, anterior insular cortices, and amygdala [23] regions that have been shown to be affected early in the course of bvFTD and that contain a unique population of von Economo cells [24, 25]. For example, patients may be presented with a facial emotional expression and asked to match the expression with the emotional label, to decide whether two faces portray the same emotion, or be asked to point to a face from an array, which matches a specific expression [26]. It is now clear that bvFTD patients demonstrate generalized deficits in facial emotion recognition [26, 27], in part mediated by face recognition deficits [28, 29]. This pattern is observed regardless of the specific task demands and has been interpreted as evidence of a primary emotion processing impairment a hypothesis that is supported by the finding that bvFTD patients are also poor at recognizing emotions from non-face stimuli such as laughter, retching, and crying [21]. Furthermore, we have shown that even recognition of emotion portrayed in music is impaired [30, 31]. At the behavioral level, it seems that in bvFTD, recognition of all negative emotions is impaired, with no clear evidence of disproportionate impairment of any one emotion over another. Interestingly, however, we have demonstrated using voxel-based morphometry neuroimaging techniques that recognition of specific basic emotions does indeed appear to be linked with the degree of atrophy to specific brain regions [23]. One recent proposal that explains this breakdown in social cognition has been to see these deficits as reflecting a failure to correctly process and incorporate contextual information that will help determine the relevant social cues for an appropriate response [32]. This process is supported in part by the anterior insula, a brain region which acts as a hub where information from various sources perceptual, cognitive, interoceptive is integrated toward an adaptive response. Importantly, the insula is one of the regions most vulnerable to the pathological processes in bvFTD. Patients with bvFTD demonstrate a reduced capacity to integrate these various sources of information in a meaningful and relevant ways, with marked deficits in apportioning the appropriate weight to these bits of information toward a decision. Alteration in sensitivity to social rewards also appears to pay a role the social cognitive deficits in bvFTD and is an active area of current research likely to generate important information in coming years [34, 35]. Although tests of social cognition have told us a great deal about the underlying cognitive deficits in bvFTD, a challenge for the next few years will be to develop tests that are clinically applicable. Our work also examined other behavioral changes in bvFTD. For example, work that began by quantifying the disturbance in eating behavior observed in bvFTD has since blossomed to reveal a range of metabolic changes [36]. Increased food consumption with a craving for sweet food is one the characteristic and discriminating features of bvFTD [37]. We have shown that this reflects early involvement of the hypothalamus [38] as well as alterations in a complex network cingulate and orbitofrontal cortices and cerebellum that controls food intake [39]. These behavioral changes are accompanied by alterations in cholesterol, insulin, neuroendocrine levels, and metabolic rate which appear to have significant effect on survival [40]. This work indicates a re-think on how FTD should be regarded: It also opens the way for potential interventions and patient management by targeting these behaviors. It was long believed that impaired episodic memory was the early hallmark of AD whereas memory was preserved in bvFTD, with relative sparing of episodic memory in the context of impaired executive function being one of the diagnostic criteria for the disease. A series of landmark studies by our group showed this simplistic dichotomy to be false. On formal tests of episodic memory, patients with bvFTD score in the same range as those with AD [41, 42]. Prior findings most likely reflected the admixture of true bvFTD patients and phenocopy cases in clinical cohorts. The neural basis of the memory impairment in bvFTD has been explored with evidence of both frontal and hippocampal contributions to the deficit [43, 44]. Additional work also uncovered deficits in other aspects of memory, such as personal autobiographical memory, future thinking, and imagination [20, 45], some of which again being as severe as those found in AD. Early work suggests that tests of spatial memory are helpful in helping discriminate between these two syndromes [46]. This is an area ripe for further exploration. The features of SD were clarified in our seminal paper of [48]. Semantic memory is the term applied to the component of long-term memory that represents our knowledge about things in the world and their inter-relationships, facts, and concepts as well as words and their meaning [48,

49]. The syndrome of SD has been particularly important from a theoretical perspective because, in contrast to AD, patients have relatively good day-to-day episodic memory and recent autobiographical memory [45, 50], intact immediate or working memory at least as assessed by digit span , and good visually based problem solving and visuo-perceptual abilities [48, 51]. This relative selectivity of the semantic memory impairment in SD makes these patients ideal subjects for the study of the effects of semantic dissolution uncontaminated by other cognitive deficits. Over the past decade, it has become clear that as well as a unique cognitive syndrome, SD is associated with a highly characteristic pattern of asymmetric brain atrophy involving the perirhinal cortex and anterior temporal pole, a region that acts as a hub in the semantic network linking information held in other cortical regions and with a typically asymmetric distribution with greater left than right temporal lobe involvement [52â€”55]. VH was unable to identify from face or name even very famous people e. In parallel with this literature, the group led by Bruce Miller in San Francisco drew attention to the bizarre behaviors including irritability, impulsiveness, alterations in dress, limited and fixed ideas, and decreased facial expression exhibited by patients with predominantly right temporal lobe atrophy [57, 58]. We have shown that deficits in face emotion processing and face detection are also seen in such patients similar to those seen in bvFTD [28, 60]. A recent study using modern longitudinal structural brain imaging methods has showed that the patterns of progression in these two variants of SD also differ in ways that the atrophy spreads through the brain over time [61] suggesting that the two variants are not simple mirror images of each other. Moreover, the pathology is distinct. In contrast to SD, PNFA is a much more heterogeneous disorder in that patients share the characteristic of halting speech with pauses and distortion but this may or may not be accompanied by distortion of speech, phonological errors, or syntactic deficits. Based upon landmark clinical and imaging work from the UCSF group, clarity emerged with a splitting of the nonfluent cases into two groups [67]. The other progressive nonfluent aphasic syndrome, referred to as logopenic progressive aphasia LPA , is characterized by anomia and phonological errors accompanied by marked reduction in verbal span [68â€”70]. The atrophy in such cases centers on the angular gyrus and superior temporal lobe [67, 71]. The tripartite classification of the progressive aphasia has been enshrined in International consensus criteria [72] which proposed clear criteria for the diagnosis of each variant. We were among the first to substantiate this classification using amyloid-based Pittsburgh compound B imaging to show that LPA was invariably associated with AD pathology, which was present in a minority of those with the other two forms of progressive aphasia. We also proposed a simplified classification system based features of which could be derived from our clinical language assessment [73]. To aid further with the diagnosis of the progressive aphasias, we have developed a multimodal language test, the SYDBAT, which is gaining wide usage [74]. Despite this work, the nonfluent progressive aphasic syndromes remain problematic as differentiation depends on expert assessment and subjective opinion on the nature of the language features and patients often present at an advanced stage when clinical features become blurred. Importantly, it is now evident that changes in social cognition are not limited to bvFTD and are also found in the language presentations of FTD. Indeed, early on, changes in emotion recognition and emotion processing, as well as ToM were reported in the early stages of SD. Recent investigations have demonstrated that patients presenting with nonfluent progressive aphasia also exhibit subtle social cognition deficits. Importantly, this work indicates that deficits in facial emotion recognition can be remediated to some degree in these patients, when making the relevant information more salient, indicating that part of this deficits is caused by an attentional deficit, rather than a primary emotion processing deficit perse [75]. The addition of social cognition investigations may be relevant in improving diagnosis accuracy in these language presentations. This lack of clinical diagnostic specificity has important repercussions, given that patients with underlying AD pathology may potentially benefit from acetylcholinesterase inhibitors whereas those with underlying FTLD will not. In a recent study [76], we showed that the combination of reduced episodic memory with preserved social cognition was indicative of AD pathology whereas the reverse pattern was suggestive of FTLD pathology. Clinically, integrity or otherwise of the motor system may provide further useful cues and help differentiate between the two nonfluent progressive aphasia syndromes, where extrapyramidal features tend to be more common in PNFA than in LPA [77, 78]. Such features have a significant impact on caregiver burden and

present considerable management problems [82]. A mixed behavioral and language syndrome is typical with disproportionate impairment of verb, compared with noun, knowledge [87, 88], and marked impairment of grammatical processing [89]. Patients with clinically diagnosed AD, whether young or old, familial or sporadic, tend to have very similar pathological changes intraneuronal tangles and extracellular amyloid plaques. In contrast, the pathological changes found in FTD are heterogeneous. Twenty years ago, cases were classified as those with and without tau positive inclusions. We now know that a range of inclusion pathologies are found in FTD [90]. Three major patterns are currently recognized. The first group includes cases with tau-positive inclusion pathology. This group, in turn, encompasses a number of subforms: This group has been further subdivided in 5 subforms A to E each with distinctive morphological appearance depending on the cellular distribution of TDP and the appearance of the inclusion pathology [92, 93]. The third group includes cases with fused in sarcoma protein FUS protein pathology. Such patients are rare, typically sporadic and associated with young onset and a high rate of neuropsychiatric symptoms.

Chapter 4 : Progress in dementia research (Book,) [racedaydvl.com]

SEQ spoke with Dr Matthew Norton, Director of Policy and Strategy for Alzheimer's Research UK, about progress made since , where dementia research stands today - including the development of drug discovery programmes - and what the future holds.

This is crucial if we are to develop diagnostics, treatments and preventions to help people with dementia. Here are just some of the amazing progress that has been made thanks to your generous support. Causes and risk factors Our scientists are uncovering crucial biological changes happening in the brain in dementia. This includes funding Prof Michel Goedert and Prof Maria Grazia Spillantini at the University of Cambridge â€" world-leading experts in tau â€" and through a Research Fellowship to Dr Amy Pooler who discovered vital clues to how the culprit tau protein spreads from cell to cell. Thanks to these landmark discoveries, researchers now believe they understand the genetic changes responsible for over half of our genetic risk of the disease. By understanding what these genes do, researchers have now linked new biological processes to the disease. You can read on our blog how the discovery of a gene linked to inflammation has sparked an important new avenue of investigation in the search for new treatments. Researchers at the University of Manchester have formed an international consortium of over 30 research groups across the world and we are funding the largest and most comprehensive genetic study of people with FTD ever undertaken. Research we funded at the University of Cambridge revealed that over 65s who had a better education, a more complex job in mid-life and greater social engagement later in life had a lower risk of cognitive decline. These discoveries inspired the launch of our Stem Cell Research Centre , which will now build on this progress to screen potential new dementia drugs. In , we supported a clinical trial that highlighted the dangers of long-term antipsychotic use in people with dementia. This finding kick-started a national campaign to reduce their use and ensure they are prescribed appropriately. Their landmark research paper in Lancet showed that the protein was cleared from the brain despite showing no benefit to patients, provoking major discussions in the field and helping to refine current approaches to drug development. Their findings have been used to urge the need for current clinical trials to be tested in people at a very early stage of the disease. You can read more about this on our blog. In , we launched our Global Clinical Trials Fund to support early-stage clinical trials to test new dementia treatments in people â€" a vital step towards putting new treatments into the hands of those who need them. Diagnosis As part of his Clinical Research Fellowship, Dr George Pengas helped to design a new memory test to aid diagnosis of dementia. Many clinicians are now comparing the TYM test to current memory and thinking tests, to investigate its potential for use in the clinic. Our support has helped researchers at University College London and the University of Manchester better characterise the changes that affect someone with frontotemporal dementia FTD. Their research has led to the characterisation of language-specific forms of the disease and helped to develop more sensitive diagnostic tests to assess symptoms such as lack of empathy. By uncovering how key changes in the brain in this disease correlate with clinical symptoms, people with FTD can now understand more about their condition and be given the chance to get involved in research at an earlier stage. Their work is helping to develop more sensitive ways to detect the disease as well as developing visual aids to help people live with their symptoms. Be the first to know the latest developments. Join the conversation online.

Chapter 5 : Alzheimerâ€™s Society Annual Conference Making progress in dementia research - Neuro Ce

Our report highlights the progress being made in dementia research capacity. Tweet Share Email New analysis from Alzheimer's Research UK reveals that there are nearly double the amount of dementia researchers and scientific publications, compared to six years ago.

These occur in strokes and other brain injuries. The disease more commonly leads to problems with movement and motor control, but it also can cause dementia in some people. Frontotemporal dementia Frontotemporal dementia refers to a group of dementias that often cause changes in personality and behavior. It can also cause language difficulty. Mixed dementia Mixed dementia is dementia in which multiple types of dementia-causing brain abnormalities are present. No single test can determine whether you have dementia. Diagnosis is based on a range of medical tests and your medical history. If you exhibit symptoms of dementia your doctor will perform: Some common tests used to diagnose dementia include: The MMSE uses a point scale and includes questions that test memory, language use and comprehension, and motor skills, among other things. A score of 24 or higher indicates normal cognitive function. While scores 23 and below indicate that you have some degree of cognitive impairment. Mini-Cog test This is a short test for helping your doctor diagnose dementia. It involves these three steps: This score is based on your performance in these and other tests, as well as your medical history. The scores are as follows: A score of 0 is normal. A score of 0. A score of 1 is mild dementia. A score of 2 is moderate dementia. A score of 3 is severe dementia. Dementia progresses differently in everyone. MCI is characterized by losing things often, forgetfulness, and having trouble coming up with words. Mild dementia People may still be able to function independently in mild dementia. Common symptoms of mild dementia include: It becomes harder to perform regular daily activities and self-care as dementia progresses. Common symptoms during this stage include: Severe dementia often can cause: People with dementia will progress through these stages at different speeds and with differing symptoms. If you suspect you may be experiencing early symptoms of dementia, talk to your doctor. Early diagnosis also allows people to participate in clinical trials. This helps researchers develop new treatments and eventually find a cure. Medically reviewed by Timothy J.

Chapter 6 : Capacity of research | Dementia Statistics Hub

Progress in Dementia Research Allan Levey, MD., Ph.D., is a professor and chair of the Department of Neurology at Emory University. His lecture, Life of the Mind and Life of the Failing Mind: Progress in Dementia Research, was given as part of Emorys Life of the Mind lecture series.

With the mobile game Sea Hero Quest, Deutsche Telekom is facilitating important progress in global dementia research. Standardized data on spatial orientation was gathered with the aid of a mobile game, Sea Hero Quest, which has been downloaded over four million times worldwide since its release in May To compile the study, the scientists drew on data from more than half a million people from 57 countries. They analyzed anonymized data from players from countries with at least participants who voluntarily provided their age, gender, and nationality information. This baseline study yielded the following important findings: Men performed better than women on average, but the gender gap narrowed in countries with greater gender equality. Spatial orientation begins to deteriorate from early adulthood and continues to do so throughout our lives. Currently used tests for dementia are not effective at detecting the earliest symptoms of spatial disorientation. As such, the scientists plan to publish an adapted version of Sea Hero Quest as a diagnostic screening instrument. They also believe the game could be useful for monitoring the progression of the disease in individuals and serve as a useful tool for comparing the results of clinical trials. Deutsche Telekom is now making the anonymized data available to other scientists for research purposes. With this step, the company hopes to aid further discoveries beyond dementia, across the wider field of neuroscience research. With this step, we hope to advance research in some of the most pressing healthcare issues of our time. The data collected through the Sea Hero Quest app is helping scientists better understand the links between aging and dementia, a key prerequisite for developing procedures for diagnostics and prevention. Sea Hero Quest was designed by the English game developer Glitchers. The players take on the role of sailors who have to constantly adjust their orientation in a variety of scenarios. People who have a good sense of direction can solve the given tasks more quickly. Classifying the results The scientists believe that the correlation between per capita gross domestic product GDP and performance could be due to associations with education standards, health, and ability to travel. They focused on GDP for this analysis as it was a standard metric available for every country, but they will follow up with further comparisons of other factors. The scientists have proposed one possible explanation for the better performance of people in some countries: In less densely populated countries like Finland, Denmark, and Norway or New Zealand, Canada and the United States people tend to travel long distances. To do so god spatial navigation capabilities are required. As the scientists see it, the findings suggest that gender differences in cognitive capabilities are not fixed. Instead, they are influenced by cultural environments, such as the role of women in society.

Chapter 7 : Progress in Dementia Research | Woodruff Health Sciences Center | Emory University

Recent research has also demonstrated that the type, and stage of dementia also impacts on the burden experienced by carers. Not surprisingly, burden of care tends to increase with disease progression, regardless of the type of dementia, although this is not universal [ ].

Chapter 8 : Our progress | Alzheimer's Research UK

The Alzheimer's Society (London, UK) is now funding more research than ever before, supporting over research projects. With no new treatments for dementia in over 15 years this is an important focus for the charity.

Chapter 9 : UK Dementia Research Institute - About us - Medical Research Council

Research article Open access The Alzheimer's Prevention Initiative Autosomal-Dominant Alzheimer's Disease Trial: A

study of crenezumab versus placebo in preclinical PSEN1 EA mutation carriers to evaluate efficacy and safety in the treatment of autosomal-dominant Alzheimer's disease, including a placebo-treated noncarrier cohort.