EVIDENCE-BASED DEMENTIA RESEARCH

A CURE AROUND THE CORNER IN A ROUND WORLD

Mario D. Garrett PhD
Professor of Gerontology

DAILY EXPRESS

10p

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PENSIONS BOOST AS SHARES SOAR TO 5 YEAR HIGH

3P BLOOD PRESSURE PILL BEATS DEMENTIA

Cheap drug slows memory loss

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FRIDAY 13TH SHOULD WE BE SCARED?

DRUG TO BEAT ALZHEIMER'S

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CURE AROUND THE CORNER

5p CHEAPER THAN THE DAILY MAIL AND TEN TIMES BETTER!

DAILY EXPRESS

THE WORLD'S GREATEST NEWSPAPER express.co.uk WEATHER: SHOWERS TUESDAY AUGUST 14, 2012 50p

MURDERED TIA WRAPPED IN A BLACK BED SHEET AND PUT INSIDE A BAG

SEE PAGE 7

60MPH STORMS TO LASH BRITAIN

SEE PAGE 4

CHOCOLATE CAN HALT DEMENTIA

By **Jo Willey** Health Correspondent

A DAILY dose of cocoa could be the secret to halting Alzheimer's disease, researchers claim.

Scientists have found that the potent ingredient in chocolate can dramatically improve cognitive impairment.

Experts say pensioners should have some cocoa every day in a bid to keep dementia and Alzheimer's at bay.

The key lies in health-boosting flavanols which are abundant in the sweet treat.

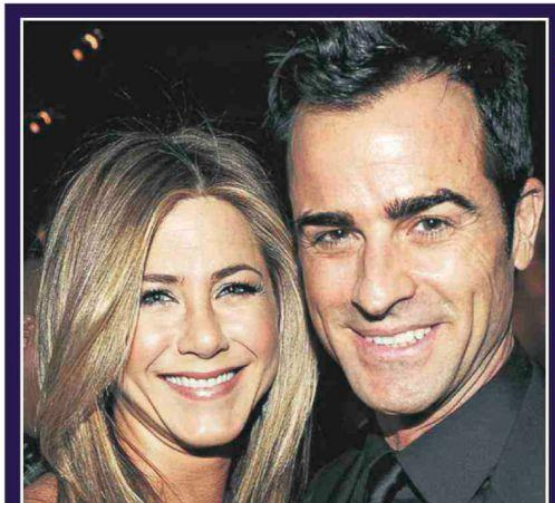
Flavanols are an antioxidant also found naturally in fruit and vegetables such as grapes, berries and apples, drinks including tea and red wine, and other plant-based foods.

Dr Giovambattista Desideri, who headed the research team, said: "This study provides encouraging evidence that consuming cocoa flavanols as part of a calorie-controlled and nutritionally balanced diet could improve cognitive function.

"Given the global rise in cognitive disorders, which have a true impact on an individual's quality of life, the role of cocoa flavanols in preventing or slowing the progression of mild cognitive impairment to dementia warrants

TURN TO PAGE 4

ARLENE PHILLIPS



JENNIFER ANISTON ENGAGED

...just a few days before ex-hubby Brad Pitt marries

CURE AROUND THE CORNER

THE TIMES

Max 22C min 7C | Thursday July 28 2016 | thetimes.co.uk | No 71974 | Only 80p to subscribers £1.40

The rise of Power Hair

Do's and don'ts of fringe politics

INSIDE TIMES2

Idiot's guide to shellfish

The Table

When in Rome Aside from the serious business of Brexit, Theresa May enjoyed a lighter moment with her Italian counterpart, Matteo Renzi, in Italy yesterday. Page 6

Scientists create the first drug to halt Alzheimer's

Islamic hate books found in prisons despite ban

Andrew Norfolk
Chief Investigative Reporter

Extremist Islamist books promoting antisemitism and preaching hatred toward non-Muslims were distributed by imams in prisons for months despite an order that they should be removed, *The Times* has learnt.

Among the prohibited titles are a tract described as the *Mein Kampf* of Islamist terrorism, a pamphlet extolling the virtues of violent jihad and a book urging Muslims to fight and subjugate unbelievers.

One of the books, by the jihadist ideologue Sayyid Qutb, blames Jews for "materialism, animal sexuality, the destruction of the family and the dissolution of society".

Another focuses on "the sexual deviation known as homosexuality". It states: "The spread of this depraved practice in a society disrupts its natural life pattern and makes those who practise it slaves to their lusts, depriving them of decent taste, decent morals and a decent manner of living."

A third states that jihad is an obligation from Allah on every Muslim.

Copies of one or more of the publications were found in chaplaincy rooms at nine of 11 prisons that were inspected during a review of radicalisation behind bars. Among them were high-security category A establishments.

When prison imams were challenged about the widespread distribution of antisemitic, misogynistic, sectarian and homophobic texts, some are understood to have claimed that they were too busy to assess the suitability of

CURE AROUND THE CORNER

DELHI, MONDAY, APRIL 25, 2016

THE HINDU

INDIA'S NATIONAL NEWSPAPER SINCE 1878

www.thehindu.in • Regd. DL(ND)-11/6110/2006-07-08 • RNI No. TNENG/2012/49940 • ISSN 0971 - 751X • Vol. 6 • No. 98 • CITY EDITION • 28 Pages • Rs. 8.00

THE HINDU | MONDAY | APRIL 25, 2016

Science & Technology
Indian researchers show cancer drugs can stop the progression of Alzheimer's, Parkinson's
PAGE 2

Youth
Team work is not just about working with like-minded persons. Find out the key ingredient that facilitates synergy
PAGE 3

Goalpost
Sinjini finds the University of Southampton, with its state-of-the-art labs, the best place for the study of vertebrae fossils
PAGE 4

Cancer drugs can stop progression of Alzheimer's, Parkinson's

R. PRASAD

In a breakthrough, researchers from the University of Delhi found that the progression of Alzheimer's and Parkinson's diseases can be suppressed or stopped by down-regulating the expression level of d-myc in Drosophila (fruit fly). Since d-myc in an evolutionarily conserved human homolog of c-myc proto-oncogene, the findings in fruit fly may be applicable to humans as well.

Results of a study were published on March 21 in the journal *Molecular Neurobiology*.

Alzheimer's and Parkinson's diseases occur due to accumulation of abnormal clumps of proteins in neuronal cells. The abnormal clumps are formed when a mutation to tau protein acts as a trigger and causes the addition of more phosphate group to the tau protein.

The addition of more phosphate group (phosphorylation) to the tau protein causes it to fold into the wrong shape and stick together to form fibre-like structure, which eventually bundle up to form clumps (neurofibrillary tangles) of proteins in the affected brain cells.

BREAKTHROUGH | At the lab, the focus was on finding the right gene. PHOTO: SPECIAL ARRANGEMENT

The c-myc proto-oncogene is a drug target for cancer, and Alzheimer's and Parkinson's diseases.

diseases by reducing or preventing protein clumps from being formed in the brain cells," said Dr. Surajit Sarkar from the Department of Genetics, University of Delhi. "We screened over 1,000 genes in Drosophila and found d-myc as a candidate gene that was restricting the pathogenesis of Alzheimer's more. Dr. Sarkar's team has successfully zeroed in on d-myc in fruit flies (c-myc proto-oncogene in humans) as a possible target gene.

The c-myc proto-oncogene in humans is a global regulator of gene expression. When this gene is down regulated, it prevents other genes from enabling more phosphate expression, the use of such drugs in people with early stages of Alzheimer's and Parkinson's disease can help in stopping the progression of the disease.

"Our study demonstrates for the first time that a common drug target c-myc proto-oncogene can be used to treat two most devastating human and Parkinson's appeared just as his team concluded the experimental work. "These scientists used anti-cancer drugs without knowing how and why the drugs were effective. Now, our study shows why the drugs helped in treating Alzheimer's and Parkinson's diseases," he said.

Cellular and molecular investigations were carried out in fruit fly as undertaking such studies on human subjects is unethical. Also, brain tissues are completely degenerated in people with Alzheimer's who have died.

The fruit fly was genetically programmed to develop symptoms of Alzheimer's and Parkinson's disease. Interestingly, expression of human's

CURE AROUND THE CORNER

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THE TIMES

The new thinking woman's crumpet

Are you man enough for the summer food craze?

Abbot of Ampleforth questioned over child sex abuse claims

Alzheimer's drug is huge leap forward for sufferers

Game-changing jab could be offered by NHS

May pledges to restrict EU migrants after Brexit

FINANCIAL TIMES

WORLD BUSINESS NEWSPAPER

Why drive? The idiocy of owning a car in the city — BUSINESS LIFE, PAGE 14

Beyond the scandal. Toshiba's deception is an opportunity for investors — JOHN GAPPER, PAGE 13

Don't panic. How to reassure your staff at a time of disruption — PAGE 14

Alzheimer's breakthrough hopes add to promise of pharma revival

• Eli Lilly clinical trial yields positive signs • Research advances end innovation drought

Briefing

Cameron's aviation committee tips the scales towards third Heathrow runway

Corbyn fever rattles Labour's moderates

World Markets

FORTUNE

HOW THE DOLLAR-STORE WAR WAS WON BY SHAWN TULLY

SMART GUNS ARE READY. Are We? BY ROGER PARLOFF

ISSUE 5.1.15

ALZHEIMER'S THE RACE TO A CURE

IN THE EPIC CONTEST TO DEFEAT THE TOUGHEST DISEASE IN THE WORLD, BIOGEN PULLS AHEAD. CAN IT WIN THE ULTIMATE PRIZE?

BY ERIKA FRY

CURE AROUND THE CORNER

Sing for a Cure

a lip sync competition

Brookes, G., Harvey, K., Chadborn, N., & Dening, T. (2018). “Our biggest killer”: multimodal discourse representations of dementia in the British press. *Social Semiotics*, *28*(3), 371-395.

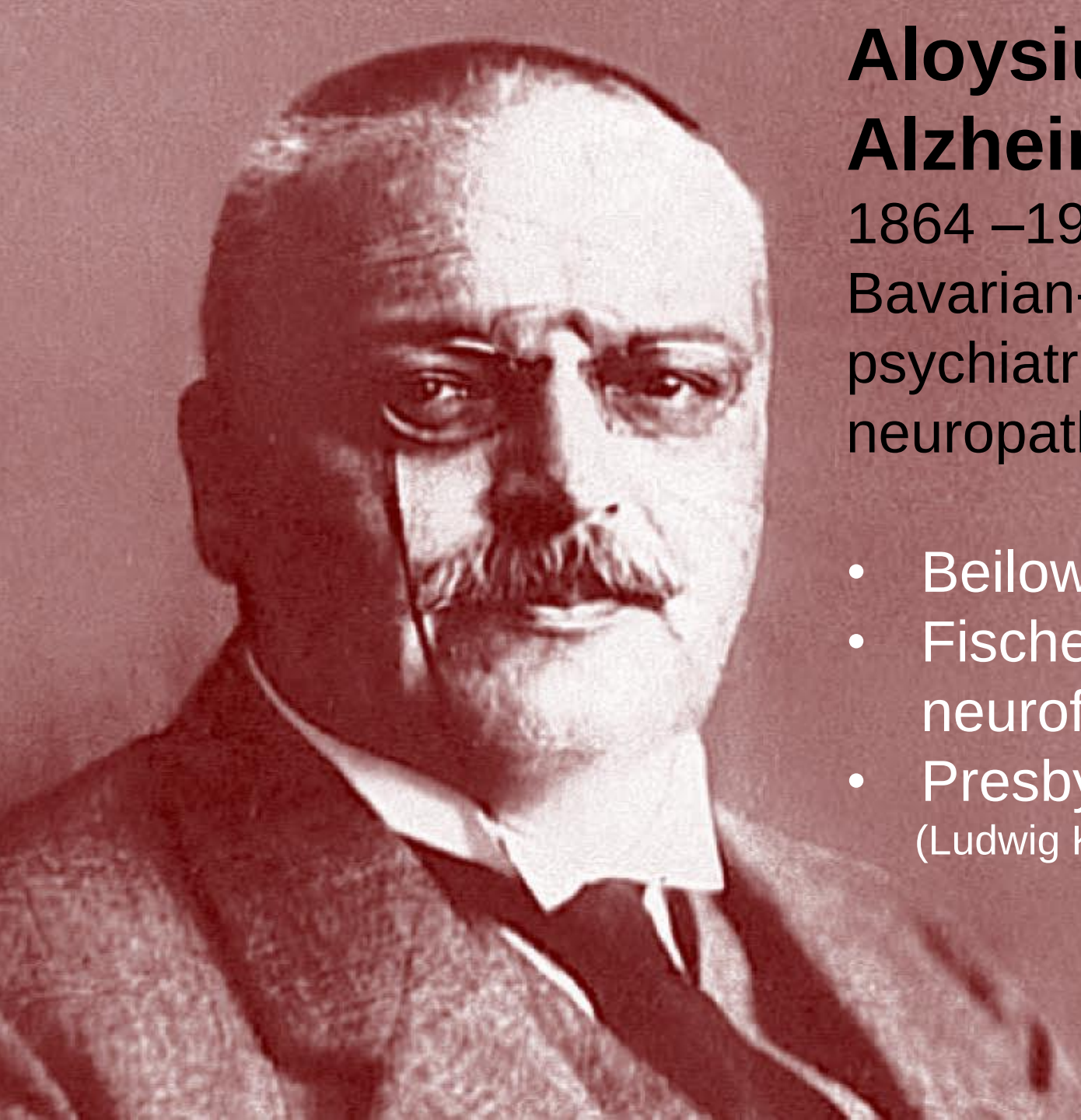
Gavin Brookes

This intensely lurid type of representation not only fails to address the ageist misinformation and common misunderstandings that all too commonly surround dementia, but is also likely to exacerbate the stress and depression frequently experienced by people with dementia and their families.

A TRAGEDY IN FOUR ACTS

Act 1

Search for Biology

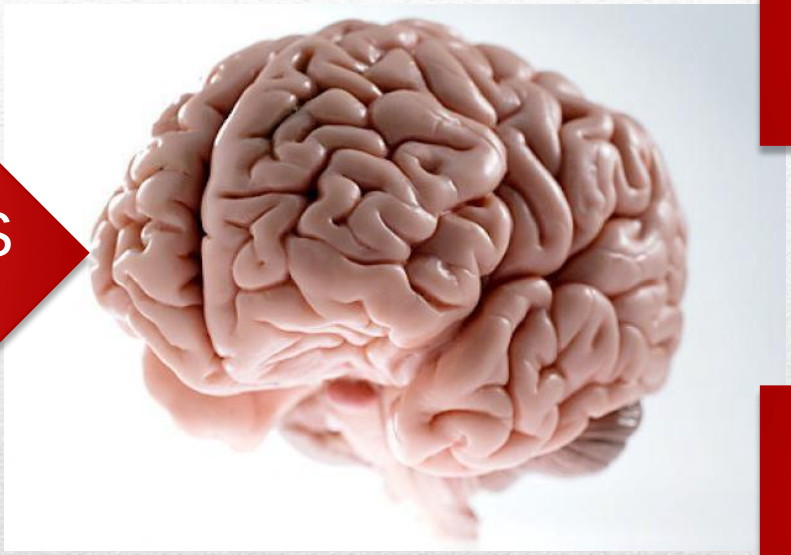


Aloysius "Alois" Alzheimer MD

1864 –1915
Bavarian-born psychiatrist and neuropathologist

- Beilowsky technique
- Fischer Plaques/ neurofibrils
- Presbyophrenia (Ludwig Kahlbaum in 1838)

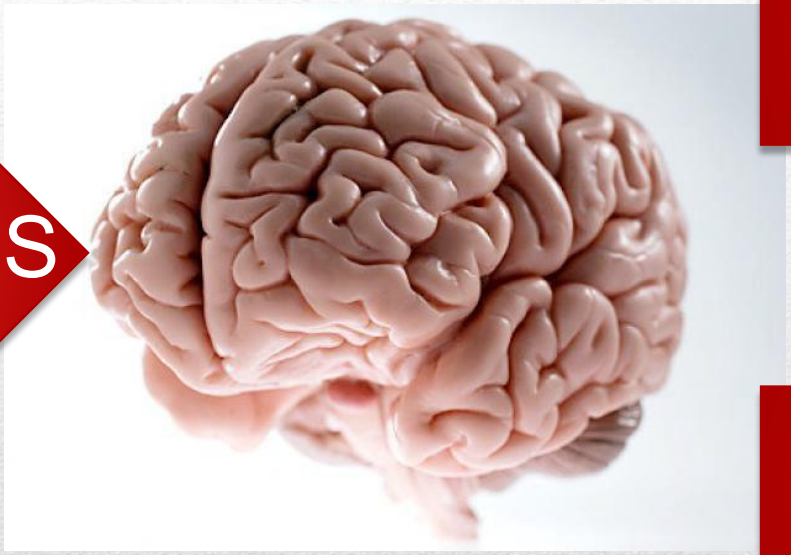
NEUROSYPHILIS



PLAQUES

TANGLES

ALZHEIMER'S



PLAQUES

TANGLES

Alzheimer's Disease

"….because senile dementia was out of the question since the patient was only 56 years of age" and then again "Senile dementia was never considered because of the onset at the age of 54 …"

(Alzheimer, 1912).

BIOLOGICAL DETERMINISM

The intellectual fathers of modern psychiatry,
Emil Kraepelin and Eugen Bleuler were both hardcore eugenicist

Eugenics

Act 2

Politics

USA FUNDING OF RESEARCH

1973 President Nixon Oct. 30 vetoed HR 14424 to establish the NATIONAL INSTITUE ON AGING

1974 President Nixon signed into law the NATIONAL INSTITUE ON AGING

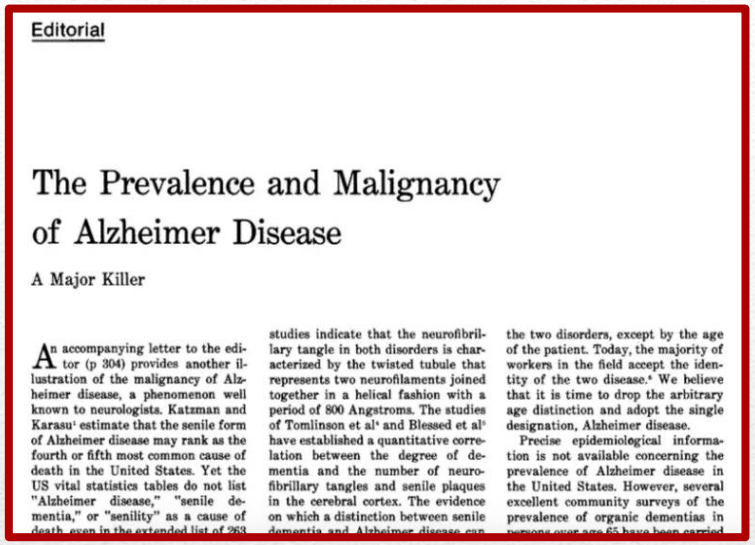
1976 Bob Butler appointed first NIA director.

Robert Katzman

In 1976 Katzman published a landmark editorial, ***The Prevalence and Malignancy of Alzheimer's Disease, a Major Killer*** in Archives of Neurology.

- Founding director of the NIA-funded Shiley-Marcos Alzheimer's Disease Research Center at UCSD
- Original member of the NIA's National Advisory Council on Aging
- Founder of the Alzheimer's Association.

USA FUNDING OF RESEARCH



Editorial

The Prevalence and Malignancy of Alzheimer Disease

A Major Killer

An accompanying letter to the editor (p 304) provides another illustration of the malignancy of Alzheimer disease, a phenomenon well known to neurologists. Katzman and Karasu[1] estimate that the senile form of Alzheimer disease may rank as the fourth or fifth most common cause of death in the United States. Yet the US vital statistics tables do not list "Alzheimer disease," "senile dementia," or "senility" as a cause of

studies indicate that the neurofibrillary tangle in both disorders is characterized by the twisted tubule that represents two neurofilaments joined together in a helical fashion with a period of 800 Angstroms. The studies of Tomlinson et al[4] and Blessed et al[5] have established a quantitative correlation between the degree of dementia and the number of neurofibrillary tangles and senile plaques in the cerebral cortex. The evidence on which a distinction between senile

the two disorders, except by the age of the patient. Today, the majority of workers in the field accept the identity of the two disease.[6] We believe that it is time to drop the arbitrary age distinction and adopt the single designation, Alzheimer disease.

Precise epidemiological information is not available concerning the prevalence of Alzheimer disease in the United States. However, several excellent community surveys of the prevalence of organic dementias in

Senile Dementia

AD

Overnight sensation

- **World's 4/5th killer**
- **Neurobiological**
- **Ageism**
- **Cure**

Katzman, R. (1976). The prevalence and malignancy of Alzheimer disease: a major killer. *Archives of neurology*, *33*(4), 217-218.

1984 criteria Alzheimer's disease:

1. Insidious onset and progressive impairment of memory/cognitive functions.
2. No motor, sensory, or coordination deficits early in the disease.
3. Diagnosis cannot be determined by laboratory tests.
4. Other causes must be excluded beforehand.
5. Diagnosis of probable, possible, and definite Alzheimer's disease

McKhann, G, Drachman, D., Folstein, M., Katzman, R., Price, D, & Stadlan EM
Report of the NINCDS-ADRDA Work Group* under the auspices of Department of Health and Human Services Task Force on Alzheimer's Disease

1984 DEFINITION OF ALZHEIMER'S DISEASE

Introduction

Published in final edited form as:
Alzheimers Dement. 2011 May ; 7(3): 257–262. doi:10.1016/j.jalz.2011.03.004.

Introduction to Revised Criteria for the Diagnosis of Alzheimer's Disease: National Institute on Aging and the Alzheimer Association Workgroups

Clifford R. Jack Jr, **Marilyn Albert**, **David S. Knopman**, **Guy M. McKhann**, **Reisa A. Sperling**, **Maria Carillo**, **William Thies**, and **Creighton H. Phelps**

Introduction

Criteria for the clinical diagnosis of Alzheimer's Disease (AD) were established by a National Institute of Neurological and Communicative Disorders and Stroke (NINCDS) and the Alzheimer's Disease and Related Disorders Association (ADRDA) workgroup in 1984 [1]. These criteria were universally adopted, have been extremely useful, and have survived intact without modification for over a quarter of a century. In the intervening 27 years, however, important advances in our understanding of AD, in our ability to detect the pathophysiological process of AD, and changes in conceptualization regarding the clinical spectrum of the disease have occurred.

By 2009 broad consensus existed throughout academia and industry that the criteria should be revised to incorporate scientific advances in the field. In response to this imperative the National Institute on Aging (NIA) and the Alzheimer's Association (AA) sponsored a series of advisory round table meetings in 2009 whose purpose was to establish a process for

ELSEVIER

preclinical

Alzheimer's & Dementia

Alzheimer's & Dementia 7 (2011) 280–292

NIA/AA Alzheimer's disease Guidelines

Toward defining the preclinical stages of Alzheimer's disease: Recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease

Reisa A. Sperling[a,*], Paul S. Aisen[b], Laurel A. Beckett[c], David A. Bennett[d], Suzanne Craft[e], Anne M. Fagan[f], Takeshi Iwatsubo[g], Clifford R. Jack, Jr.[h], Jeffrey Kaye[i], Thomas J. Montine[j], Denise C. Park[k], Eric M. Reiman[l], Christopher C. Rowe[m], Eric Siemers[n], Yaakov Stern[o], Kristine Yaffe[p], Maria C. Carrillo[q], Bill Thies[q], Marcelle Morrison-Bogorad[r], Molly V. Wagster[r], Creighton H. Phelps[r]

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MCI

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Alzheimer's & Dementia 7 (2011) 270–279

Alzheimer's & Dementia

NIA/AA Alzheimer's disease Guidelines

The diagnosis of mild cognitive impairment due to Alzheimer's disease: Recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease

Marilyn S. Albert[a,*], Steven T. DeKosky[b,c], Dennis Dickson[d], Bruno Dubois[e], Howard H. Feldman[f], Nick C. Fox[g], Anthony Gamst[h], David M. Holtzman[i,j], William J. Jagust[k], Ronald C. Petersen[l], Peter J. Snyder[m,n], Maria C. Carrillo[o], Bill Thies[o], Creighton H. Phelps[o]

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Alzheimer's

Alzheimer's & Dementia 7 (2011) 263–269

Alzheimer's & Dementia

NIA/AA Alzheimer's disease Guidelines

The diagnosis of dementia due to Alzheimer's disease: Recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease

Guy M. McKhann[a,b,*], David S. Knopman[c], Howard Chertkow[d,e], Bradley T. Hyman[f], Clifford R. Jack, Jr.[g], Claudia H. Kawas[h,i,j], William E. Klunk[k], Walter J. Koroshetz[l], Jennifer J. Manly[m,n,o], Richard Mayeux[m,n,o], Richard C. Mohs[p], John C. Morris[q], Martin N. Rossor[r], Philip Scheltens[s], Maria C. Carrillo[t], Bill Thies[t], Sandra Weintraub[u,v], Creighton H. Phelps[w]

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Autopsies

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Alzheimer's & Dementia 8 (2012) 1–13

Alzheimer's & Dementia

NIA/AA Alzheimer's disease Guidelines

Featured Articles

National Institute on Aging–Alzheimer's Association guidelines for the neuropathologic assessment of Alzheimer's disease

Bradley T. Hyman[a], Creighton H. Phelps[b], Thomas G. Beach[c], Eileen H. Bigio[d], Nigel J. Cairns[e,f], Maria C. Carrillo[g], Dennis W. Dickson[h], Charles Duyckaerts[i], Matthew P. Frosch[j], Eliezer Masliah[k,l], Suzanne S. Mirra[m], Peter T. Nelson[n], Julie A. Schneider[o,p,q], Dietmar Rudolf Thal[r], Bill Thies[g], John Q. Trojanowski[s], Harry V. Vinters[t,u], Thomas J. Montine[v,*]

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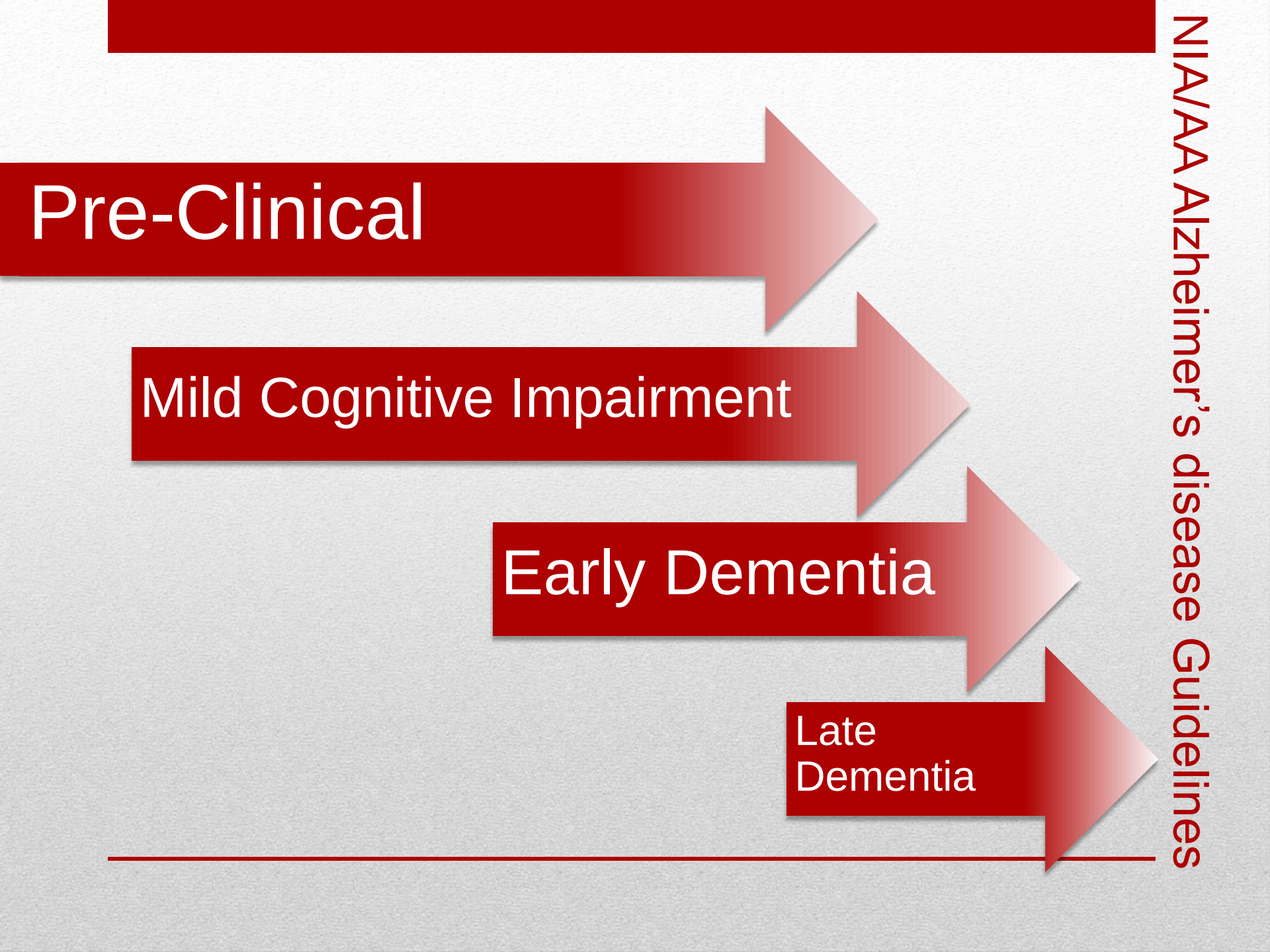
[m]*Department of Pathology, SUNY Downstate Medical Center, Brooklyn, NY, USA*


Pre-Clinical
Mild Cognitive Impairment
Early Dementia
Late Dementia
NIA/AA Alzheimer's disease Guidelines

Act 3

Confusion

Research *cul de sac* we still do not know what Alzheimer's disease is

http://ijh.sciedupress.com International Journal of Healthcare 2016, Vol. 2, No. 2

REVIEWS

A century of confusion in researching Alzheimer's disease

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[2] Department of Psychology, Boğaziçi University, Istanbul, Turkey

Received: February 19, 2016 **Accepted:** April 5, 2016 **Online Published:** April 14, 2016
DOI: 10.5430/ijh.v2n2p13 **URL:** http://dx.doi.org/10.5430/ijh.v2n2p13

ABSTRACT

More than a century ago Alois Alzheimer published a case study that later evolved into the Amyloid Cascade hypothesis—which assumes that increasing proliferation of plaques and tangles in the brain cause dementia. However, studies involving the removal of plaques—amyloid-β—in patients' brains resulted in worse cognitive performance, suggesting that plaques cannot solely be the disease. The search then focused on tau misfolded protein. But the evidence is uncertain. This paper suggests a critical history approach to understanding this confusion in Alzheimer's disease research. Confusion is related to variability in expression of the disease, inaccuracy of clinical diagnostic tools, the relationship to other diseases, and the increasing neurological variance among older adults. The final verdict is that there is an unclear relationship between the biology and the expression of the

Amyloid Cascade hypothesis— accumulation of the amyloid-β peptide (Plaques) and Tau-т protein (Tangles) in the brain is a central event in Alzheimer's disease pathology.

However this hypothesis has not been supported by the data on humans (Iqbal, Liu & Gong, 2014).

Hardy, J.A. and Higgins, G.A. (1992). Alzheimer's disease, the amyloid cascade hypothesis.Science.286: 184–185

"To date most of the treatments tested in human clinical trials are drugs that remove amyloid-β. The success of these drugs in removing the plaques from the brains of AD patients was offset by their worse performance on cognitive testing."

(Gilman et al, 2005; Boche et al, 2010).

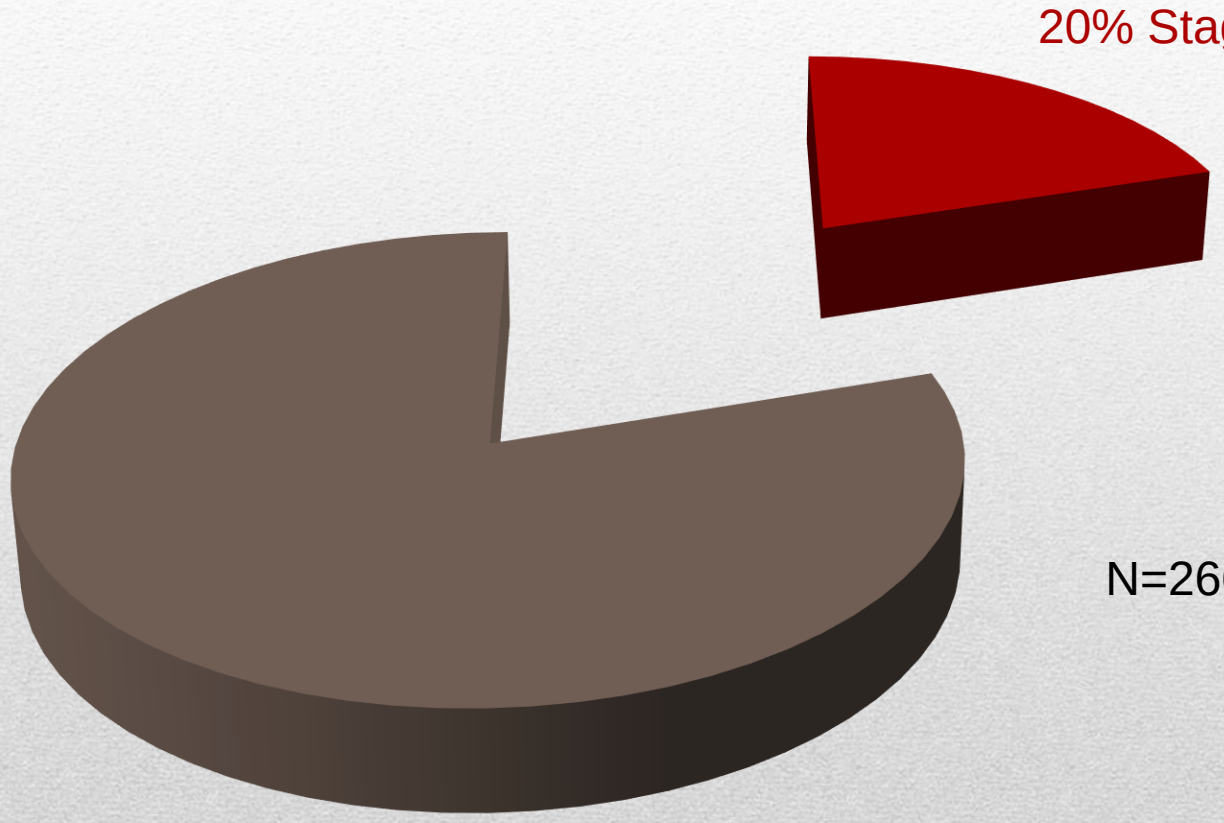
Removal of Plaques

Aβ as Protective

Aβ is a natural antibiotic that protects the brain from infection. Most surprisingly, Aβ aggregates trap and imprison bacterial pathogens. It remains unclear whether Aβ is fighting a real or falsely perceived infection in AD.

Kumar, D. K. V., Choi, S. H., Washicosky, K. J., Eimer, W. A., Tucker, S., Ghofrani, J., ... & Moir, R. D. (2016). Amyloid-β peptide protects against microbial infection in mouse and worm models of Alzheimer's disease. *Science translational medicine*, *8*(340), 340ra72-340ra72.

26-30 year-olds during autopsies



Braak H, Braak E. Frequency of stages of Alzheimer-related lesions in different age categories. Neurobiol Aging 1997; 18:351-357.

Dementia Errors

From literature review:

- 19%-67% have neuropathology but no dementia

James A. Mortimer, Amy R. Borenstein, Karen M. Gosche, David A. Snowdon. Very early detection of Alzheimer neuropathology and the role of brain reserve in modifying its clinical expression. Journal of Geriatric Psychiatry & Neurology. Dec 2005

All of us have plaques and tangles and brain plasticity controls it on a daily basis.

What stops us maintaining this plasticity?

CONFUSION

- Physicians and mental health professionals are still struggling to diagnose Alzheimer's disease correctly (Homer, 1988; McDaniel, Lukovits & McDaniel, 1993).
- Confused with other neurological diseases such as Creutzfeldt-Jakob disease (Robinson, 2011), Lewy Body dementia (Mok el al, 2004; Rokovets, 2012) and Vascular dementia, which causes the highest incidence of misdiagnosis (Wetterling, Kanitz & Borgis, 1996).
- As well as social/behavioral problems such as anxiety (Guziak & Smith, 2014), low education, cultural variability and—the main cause of misdiagnoses—depression (Prince et al, 2003; Gaugler et al 2013).
- It is rare for AD to occur in isolation from depression (Wagner et al, 2011) and anxiety (Guziak & Smith, 2014) and the diagnostic tools are too crude to differentiate these confounds.

CONFOUNDS

CONFUSION

1) Alzheimer's Disease Diagnostic and Treatment Centers (ADDTC), 1992;
2) Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-V), 2014;
3) International Classification of Diseases, 11th revision (ICD-11), 1994-2017;
4) National Institute of Neurological Disorders and Stroke–Association Internationale pour la Recherche et l'Enseignement en Neurosciences (NINDS-AIREN), 1993.

- Wetterling, Kanitz & Borgis (1996) evaluated different existing criteria on a **sample of 167** older adult patients who were admitted to a hospital with probable dementia and found that the concordance was very poor since only **5 cases** met the criteria for Vascular Dementia of all diagnostic guidelines
- Erkinjuntti, et al (1997) agreement in only 20 out of 1,879 dementia cases
- Pohjasvaara, et al (2000) concurred in 31 out of 107 patients.

VALIDITY QUESTIONS

CONFUSION

- Correlation between Alzheimer's disease neuropathology and its clinical expression **declines with age** (Savva et al, 2009).
- Increasing prevalence of cerebral pathologies in older patients (Giannakopoulos et al, 2007).

- **Half** of clinically diagnosed demented oldest-old have **insufficient neuropathology** findings to account for their dementia, while approximately half of individuals without dementia meet the neuropathological criteria for Alzheimer's disease (Crystal et al, 1988; Polvikoski et al, 2001).

- Between 9 and 13 percent of residents in nursing homes and assisted living facilities have **idiopathic normal-pressure hydrocephalus** are likely mis-diagnosed with Alzheimer's disease and can be cured. (Marmarou, Young & Aygok, 2007)

CLINCALLY UNRELIABLE

Mario D Garrett Ph.D.
iAge

Is Research Entering Into a New Eugenics Period?

Alzheimer's disease neurological spin helped Research and Domain Criteria RDoC

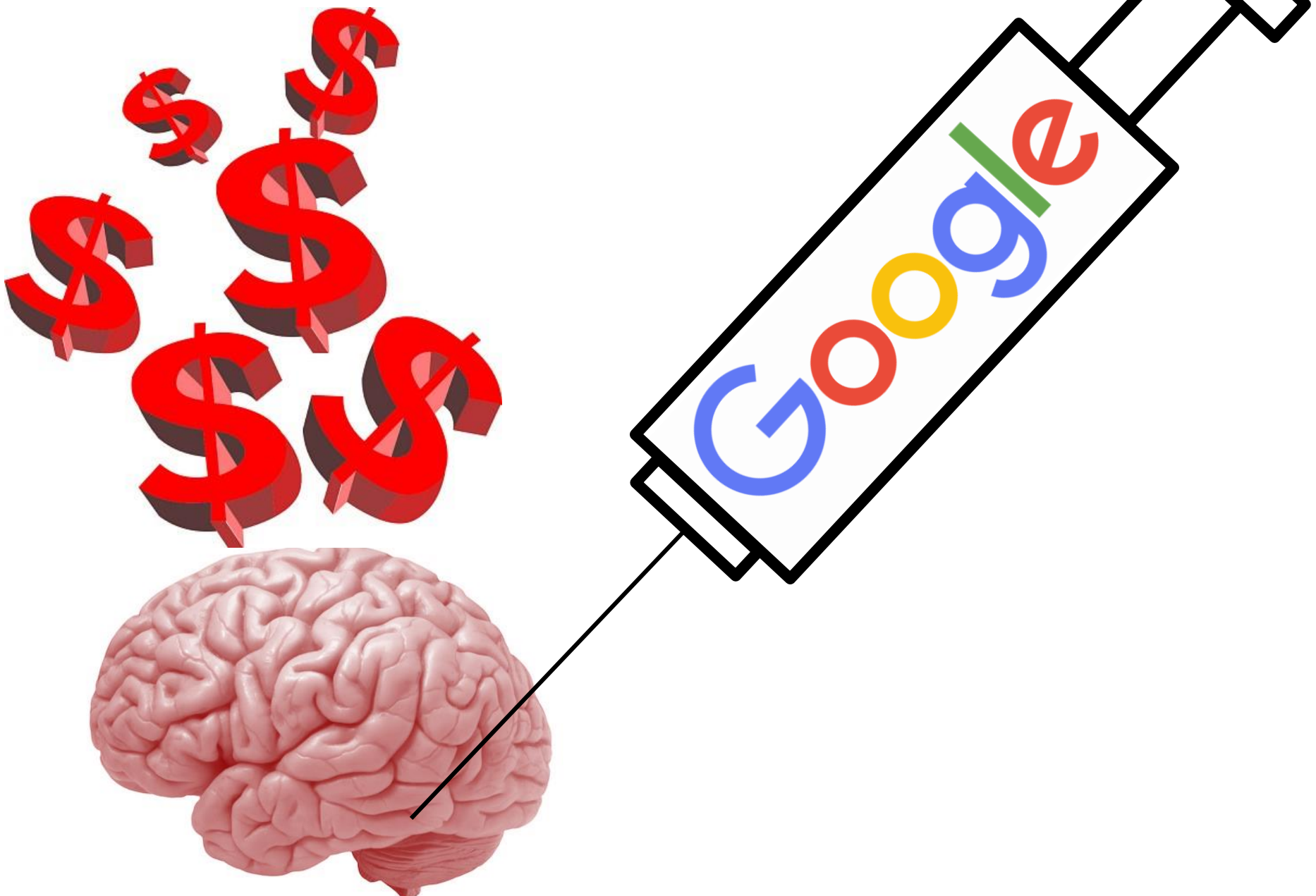
Posted Dec 10, 2015

Research Domain Criteria (RDoC) is a new classification of diseases initiated by the U.S. National Institute of Mental Health

https://www.psychologytoday.com/blog/iage/201512/is-research-entering-new-eugenics-period

Research Domain Criteria (RDoC) introduced by U.S. National Institute of Mental Health (NIMH) director Thomas Insel in 2008. Now works for Google Life Sciences as Verily, a for profit health company.

Thomas Insel


Google

Repositioning of drugs requires theoretical backing. Dementia is a safe bet as most patients are terminal and caregivers are desperate for a cure.

Just Biomarkers no Clinical Indicators

"A, T, N System" (Amyloid, Tau, and Neurodegeneration)

ELSEVIER

CrossMark

Alzheimer's & Dementia 14 (2018) 535-562

Alzheimer's & Dementia

2018 National Institute on Aging—Alzheimer's Association (NIA-AA) Research Framework

NIA-AA Research Framework: Toward a biological definition of Alzheimer's disease

Clifford R. Jack, Jr.,[a,*], David A. Bennett[b], Kaj Blennow[c], Maria C. Carrillo[d], Billy Dunn[e], Samantha Budd Haeberlein[f], David M. Holtzman[g], William Jagust[h], Frank Jessen[i], Jason Karlawish[j], Enchi Liu[k], Jose Luis Molinuevo[l], Thomas Montine[m], Creighton Phelps[n], Katherine P. Rankin[o], Christopher C. Rowe[p], Philip Scheltens[q], Eric Siemers[r], Heather M. Snyder[d], Reisa Sperling[s]

Contributors[†]: Cerise Elliott, Eliezer Masliah, Laurie Ryan, and Nina Silverberg

The diagnosis is not based on the clinical consequences of the disease (i.e., symptoms/signs) in this research framework, which shifts the definition of AD in living people from a syndromal to a biological construct. The research framework focuses on the diagnosis of AD with biomarkers in living persons. Biomarkers are grouped into those of b amyloid deposition, pathologic tau, and neurodegeneration [AT(N)].

Curr Neurobiol 2018; 9(1): 49-58 *ISSN 0975-9042*

A Critique of the 2018 National Institute on Aging's. Research Framework: Toward a biological definition of Alzheimer's disease

Mario D Garrett

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Abstract

Shame on the National Institute on Aging (NIA) for sponsoring a new way of defining Alzheimer's disease based on biomarkers (plaques and tangles). Heiko Braak in 2011 after dissecting 2,332 brains ranging in age from 1 to 100 found that only 10 cases had complete absence of Alzheimer's disease related biology. Every person over 25 years of age had Alzheimer's disease biomarkers. The new framework sponsored by the NIA makes every older person liable for a diagnosis of Alzheimer's disease. The legal implications were not even considered. The pharmaceutical connections of most researchers involved brings into question the intent of this framework. This article details the scientific argument against using biology as the only indicator of the disease while ignoring the clinical aspects. The conclusion advocates for a careful reassessment of an emerging eugenics movement where biological markers are becoming more readily relied on when the science supporting these indicators remains incomplete.

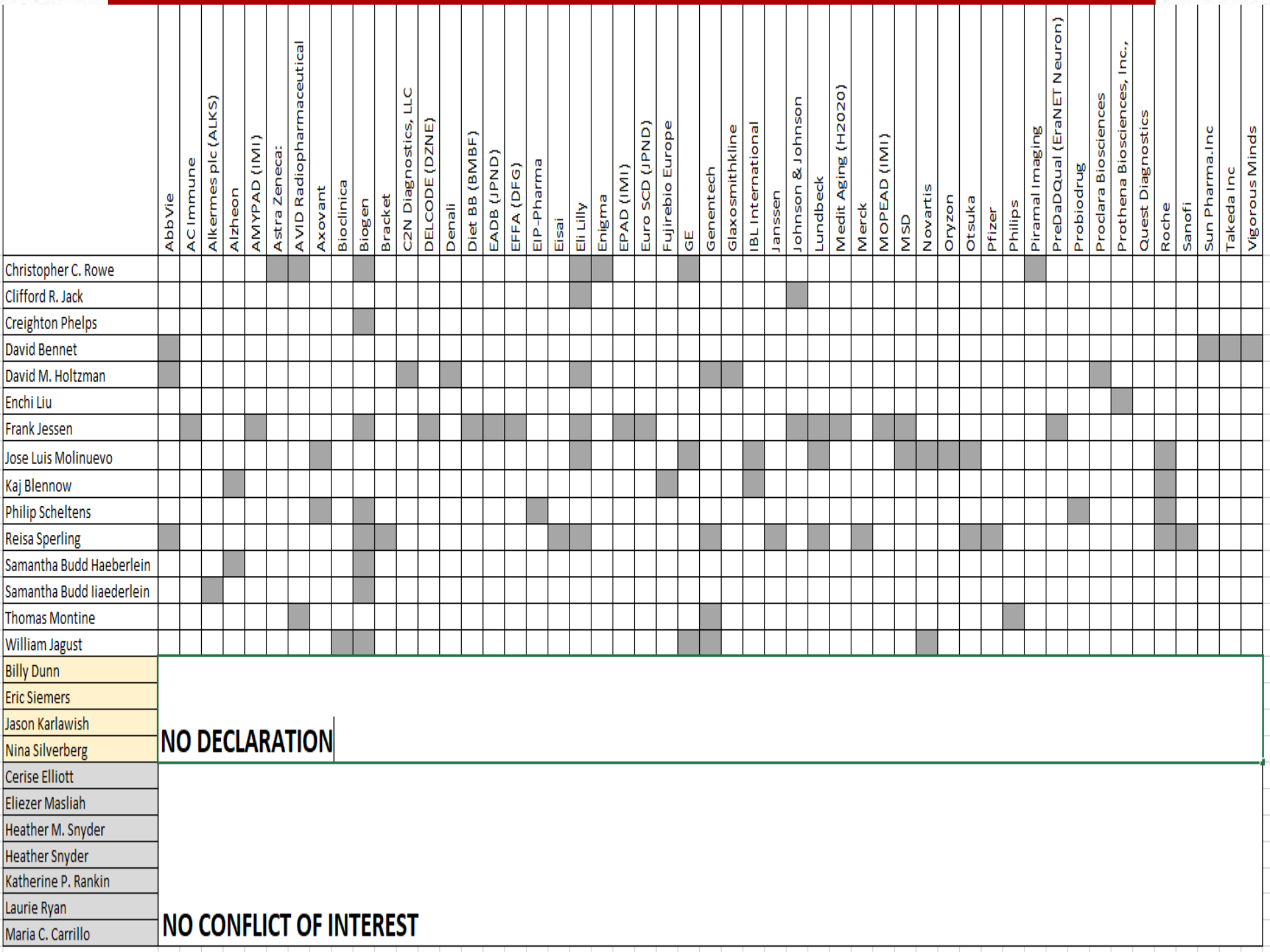
Introduction

After more than a century of research the National Institute on Aging and the Alzheimer's Association (NIA-AA) are yet again reverting to the original definition of Alzheimer's disease. A definition which Emil Kraepelin—Alois Alzheimer's supervisor—hastily formalized as a "new disease" in 1911. The recycled definition, published in 2018, is the NIA-AA's Research Framework: Toward a biological definition of Alzheimer's disease and was headed by Clifford Jack (referred to from now on as the Framework) [1].

The Framework relies on the plaques and tangles as the signature of Alzheimer's disease, while overall neurological damage defines the severity of Alzheimer's disease. This time around, in contrast to the 2011 Guidelines [2] the Framework ignores the clinical features of the disease. This is important because for the first time the clinical aspect of the disease—what we think of as Alzheimer's disease, which is how it is expressed through loss of memory, changes in mental capacities and even

definition of AD [Alzheimer's disease] is a logical step toward greater understanding of the mechanisms underlying its clinical expression." (p.536)]1]. That Alzheimer's disease can only be diagnosed through these biological markers (biomarkers) while by ignoring the real disease which is its clinical expression they lose their reference outcome. , they defined the disease but not what is not-pathological. The authors argue that the clinical and neuropathological features of the disease are "…two very different entities…" (p.536) [1] and that "…cognitive symptoms are not an ideal way to define AD [Alzheimer's disease]" (p538) [1]. As a vehicle for scientific exploration, understanding and ultimately cure of Alzheimer's disease, the Framework ignores science, obfuscates methodology, and fudges outcomes in order to drive through an agenda based on pharmaceutical (in contrast to scientific) considerations. There are serious repercussions from this approach but it is a lack of scientific rigor that will eventually expose this approach for what it is, a sham. This paper exposes the lack of scientific methodology utilized by the NIA-AA in reaching their conclusion.

Shame on the National Institute on Aging (NIA) for sponsoring a new way of defining Alzheimer's disease based on biomarkers (plaques and tangles). Heiko Braak in 2011 after dissecting 2,332 brains ranging in age from 1 to 100 found that only 10 cases had complete absence of Alzheimer's disease related biology. Every person over 25 years of age had Alzheimer's disease biomarkers. The new framework sponsored by the NIA makes every older person liable for a diagnosis of Alzheimer's disease. The legal implications were not even considered. The pharmaceutical connections of most researchers involved brings into question the intent of this framework. This article details the scientific argument against using biology as the only indicator of the disease while ignoring the clinical aspects. The conclusion advocates for a careful reassessment of an emerging eugenics movement where biological markers are becoming more readily relied on when the science supporting these indicators remains incomplete.


AbbVie
AC Immune
Alkermes plc (ALKS)
Alzheon
AMYPAD (IMI)
Astra Zeneca:
AVID Radiopharmaceutical
Axovant
Bioclinica
Biogen
Bracket
C2N Diagnostics, LLC
DELCODE (DZNE)
Denali
Diet BB (BMBF)
EADB (JPND)
EFFA (DFG)
EIP-Pharma
Eisai
Eli Lilly
Enigma
EPAD (IMI)
Euro SCD (JPND)
Fujirebio Europe
GE
Genentech
Glaxosmithkline
IBL International
Janssen
Johnson & Johnson
Lundbeck
Medit Aging (H2020)
Merck
MOPEAD (IMI)
MSD
Novartis
Oryzon
Otsuka
Pfizer
Philips
Piramal Imaging
PreDaDQual (EraNET Neuron)
Probiodrug
Proclara Biosciences
Prothena Biosciences, Inc.,
Quest Diagnostics
Roche
Sanofi
Sun Pharma.Inc
Takeda Inc
Vigorous Minds
Christopher C. Rowe
Clifford R. Jack
Creighton Phelps
David Bennet
David M. Holtzman
Enchi Liu
Frank Jessen
Jose Luis Molinuevo
Kaj Blennow
Philip Scheltens
Reisa Sperling
Samantha Budd Haeberlein
Samantha Budd Iiaederlein
Thomas Montine
William Jagust
Billy Dunn
Eric Siemers
Jason Karlawish
Nina Silverberg
NO DECLARATION
Cerise Elliott
Eliezer Masliah
Heather M. Snyder
Heather Snyder
Katherine P. Rankin
Laurie Ryan
Maria C. Carrillo
NO CONFLICT OF INTEREST

Several co-authors were employees of pharmaceutical companies including Biogen, Roche, Genentech, Lilly, Pfizer, Lundbeck, and Janssen. Three researchers have patents that benefit from the Framework.

CONFLICTOF INTEREST

In 2001 the highest French administrative court (Conseil d'Etat) requested the immediate **withdrawal** of guidelines on dementia elaborated by the French National Health Authority (Haute Autorité de Santé) owing to undisclosed serious **CONFLICT OF INTEREST** for panel members.

Lenzer J (2011) French guidelines are withdrawn after court finds potential bias among authors. BMJ 342:d4007

" Because most people who take an Alzheimer's medication will receive no meaningful benefit, together with the relatively high price tag and the risk of rare but important safety concerns, we are unable to choose any of these drugs as a Best Buy."

This report was updated in May 2012.

https://www.consumerreports.org/health/resources/pdf/best-buy-drugs/AlzheimersFINAL.pdf

Biological Determinism

nature biotechnology

Rong Chen, R., Shi, L., Hakenberg, J….Friend SH (2016). Analysis of 589,206 genomes identifies individuals resilient to severe Mendelian childhood diseases. nature biotechnology ADVANCE ONLINE PUBLICATION doi:10.1038/nbt.3514

Analysis of 589,306 genomes identifies individuals resilient to severe Mendelian childhood diseases

Rong Chen[1,2,12], Lisong Shi[1,2,12], Jörg Hakenberg[1,2], Brian Naughton[3,11], Pamela Sklar[1,2,4], Jianguo Zhang[5], Hanlin Zhou[5], Lifeng Tian[6], Om Prakash[7], Mathieu Lemire[8], Patrick Sleiman[6], Wei-yi Cheng[1,2], Wanting Chen[5], Hardik Shah[1,2], Yulan Shen[5], Menachem Fromer[1,2,4], Larsson Omberg[9], Matthew A Deardorff[6], Elaine Zackai[6], Jason R Bobe[1,2], Elissa Levin[1,2], Thomas J Hudson[8], Leif Groop[7], Jun Wang[10], Hakon Hakonarson[6], Anne Wojcicki[3], George A Diaz[1,2], Lisa Edelmann[1,2], Eric E Schadt[1,2] & Stephen H Friend[1,2,9]

Genetic studies of human disease have traditionally focused on the detection of disease-causing mutations in afflicted individuals. Here we describe a complementary approach that seeks to identify healthy individuals resilient to highly penetrant forms of genetic childhood disorders. A comprehensive screen of 874 genes in 589,306 genomes led to the identification of 13 adults harboring mutations for 8 severe Mendelian conditions, with no reported clinical manifestation of the indicated disease. Our findings demonstrate the promise of broadening genetic studies to systematically search for well individuals who are buffering the effects of rare, highly penetrant, deleterious mutations. They also indicate that incomplete penetrance for Mendelian diseases is likely more common than previously believed. The identification of resilient individuals may provide a first step toward uncovering protective genetic variants that could help elucidate the mechanisms of Mendelian diseases and new therapeutic strategies.

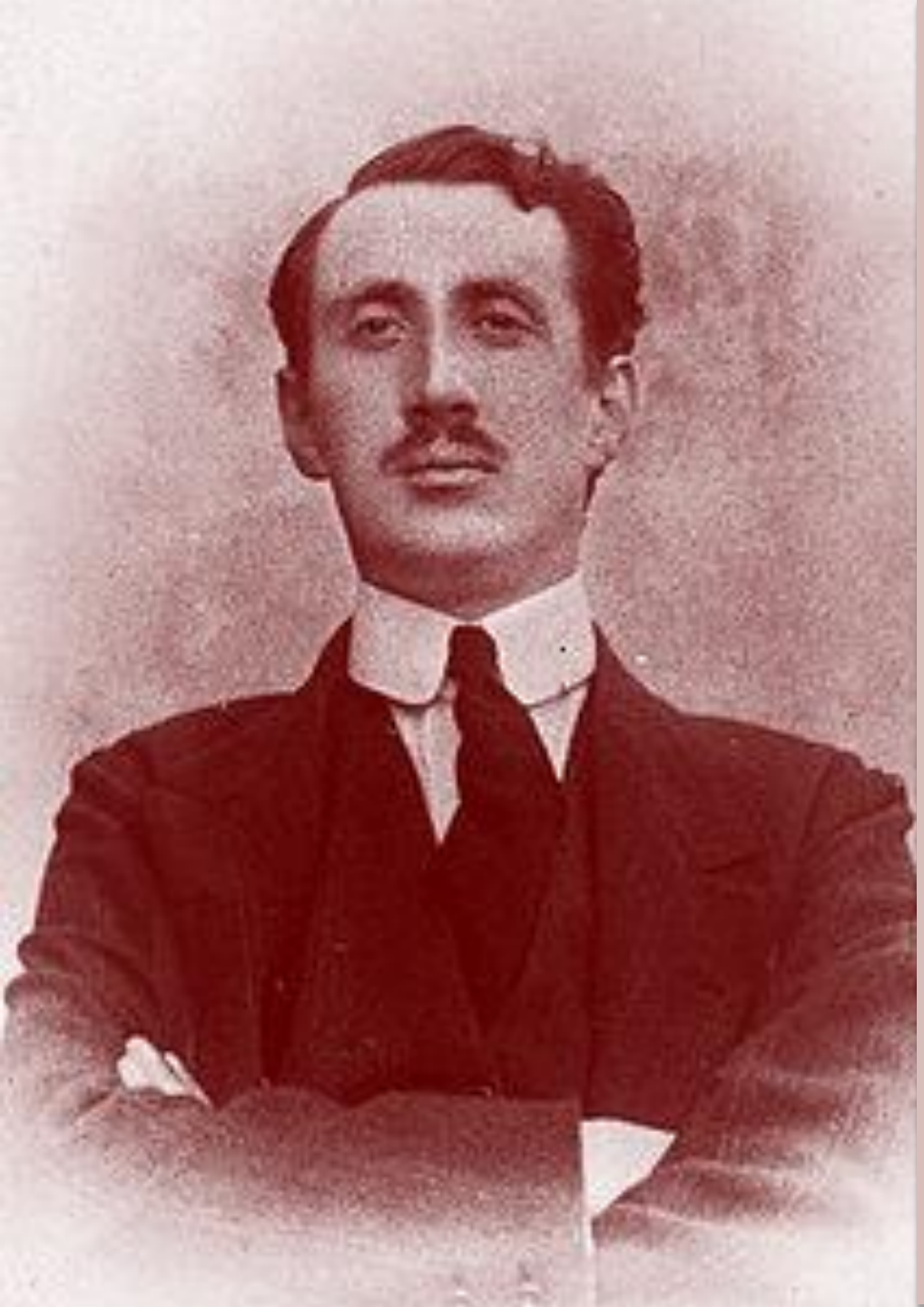
Biological Determinism

A comprehensive screen of 874 genes in 589,306 genomes led to the identification of 13 adults harboring mutations for 8 severe Mendelian conditions, with no reported clinical manifestation of the indicated disease

Biological Determinism

The final 13 candidates all harbored homozygous (autosomal recessive disease) or heterozygous (autosomal dominant disease) mutations to one of eight different severe Mendelian childhood disorders that would normally be expected to cause severe disease before the age of 18 years: cystic fibrosis, Smith-Lemli-Opitz syndrome, familial dysautonomia, epidermolysis bullosa simplex, Pfeiffer syndrome, autoimmune polyendocrinopathy syndrome, acampomelic campomelic dysplasia and atelosteogenesis

BUT THEY HAD NO EXPRESSION OF THE DISEASE



Gaetano Perusini MD

(1879–1915)

Italian pupil and colleague of Alois Alzheimer

Wounded by a grenade in1915 died that same year at the young age of 36

“of course, as usually happens when anatomo-pathological datum offers easy enticement, there will be more than one person who, on the basis of these findings will make the most useless and fanciful anatomo-psychic guesses, and those who amuse themselves with anatomically localizing the location of conscience, the will and related matters, would **find a good playground**, in which the tangles, for instance, might offer the most clear-cut explanation for the disorientation observed in the senile demented patient…”
Perusini (1911, p144)

A GOOD PLAYGROUND

Act 4

Complex Brain

COMPLEX BRAIN

- 86 billion neurons
- Each neuron 1,000 - 10,000 synapses
- 125 trillion synapses in the cerebral cortex alone.
- 1,000 times the number of stars in our galaxy.
- Stephen Smith Stanford University one synapse = 1,000 molecular-scale switches.

The total number of synaptic switches per brain thus approaches 10^20 [Sextillion]…(equal to) the total number of transistor “switches” in all of the computer chips on earth today, put together…one human brain (compares) to the entire global internet, rather than to any single computer.

Stephen J Smith, Ph.D., Prof. of Molecular and Cellular Physiology at the Stanford University School of Medicine.

Breakdown of complex systems

- Changes in resilience, reduction in internal body temperature, hormonal changes (especially for women) all affect the blood-brain-barrier that protects the brain from infections.
- Allows an onslaught of bacterial, fungal, viral, metal, and other invasions.
- The plaques and the tangles in this scenario are responses to this attack

Symbiosis

www.symbiosisonline.org
www.symbiosisonlinepublishing.com

Research Article | SOJ Neurology | Open Access

A New Public Health Paradigm for Alzheimer's Disease Research

Mario D Garrett* and Ramon Valle

San Diego State University, California, USA

Received: July 21, 2015; Accepted: November 18, 2015; Published: December 19, 2015

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Abstract

In industrialized countries Alzheimer's disease is becoming a pandemic. Over the next few decades one in six people are predicted to have Alzheimer's disease. This will evolve into a public health tragedy. Unfortunately there is a problem with dementia research. After more than a century of research we cannot answer basic questions about the disease, whether the biomarkers are truly the disease or whether these biomarkers are symptoms of another yet unknown disease. This paper summarizes the literature to show that there are other correlates—and possible causes—of Alzheimer's disease that have not received attention, but if the disease is approached from a public health perspective, then the disease can be organized under the four potential integrated processes of Trauma, Penumbra, Perfusion and Plasticity. Through this re-framing of the disease as a public health problem an opportunity emerges that can expand and reinvigorate research in Alzheimer's disease. Such new insights can elucidate a better and fuller understanding of the disease and provide some real hope to defining a road map to a cure.

Keywords: Alzheimer's disease; public health; trauma, penumbra, perfusion; plasticity

left out of the National Institute on Aging-Alzheimer's Association (NIA-AA) 2011 research agenda for Alzheimer's disease [2].

The Amyloid Cascade hypothesis is the dominant theory in Alzheimer's disease [3]. This theory postulates that the deposition of the amyloid-β peptide (plaques) in the brain is a central event in Alzheimer's disease pathology, followed by tau protein deposits (tangles) that clog up the brain causing impaired cognition leading to dementia. As a result of this theory, research on Alzheimer's disease has exclusively focused on neurobiology and biochemistry. This exclusivity was reinforced when the Amyloid Cascade hypothesis was adopted by the 2011 NIA-AA guidelines that concentrate on identifying biomarkers in the pre-clinical stage of the disease [4].

But research continues to expose anomalies that cannot be easily explained by these guidelines [5,6]. Additionally, there is now a valid and persuasive criticism of the current research methodologies in Alzheimer's disease: the lack of evidence that biomarkers cause the disease; that the disease behaves more as

Alzheimer's disease Theory

Trauma

- Genes
- Viral
- Bacteria
- Behavior
- Environmental
- Vascular
- Psychosocial
- Other…

Penumbra

Plasticity

- Education
- Dancing
- Music
- Brain Exercises
- Other…

Perfusion

- Temperature
- Activity
- Anesthetic
- Vascular
- Other…

Alzheimer's

Trauma

Genes less than 25% of all dementia, most are sporadic and have unknown causes (Bird, 2014).

- **viral** (HIV/AIDS, herpes simplex virus type I, Varicella zoster virus, cytomegalovirus, Epstein-Barr virus),
- **bacteria** (syphilis and Lyme-disease/ borrelia),
- **parasites** (toxoplasmosis, cryptococcosis and neurocysticercosis),
- **behavior** (Alcohol, cigarette smoking, recreational drugs, concussion/mild/severe brain trauma)
- **environmental** (possibly aluminum),
- **infections** (prions as in Cretchfeldt-Jakobs disease), and
- **vascular** (stroke, multiple-infarct dementia hydrocephalus, injury and brain tumors).

Perfusion

Temperature: 98.6° F --Baltimore Longitudinal Study of Aging, men with a core body temperature below the median lived significantly longer (Roth et al, 2002).

Activity: Rogers, Meyer & Mortel, (1990) reported that retirees who elected to become physically active maintained more constant perfusion level scoring better on cognitive testing after a four-year follow-up.

General Anesthetic: In a population of healthy elderly patients, undergoing non-vascular abdominal surgery Casati, et al (2007) reported that cerebral desaturation—and indication of hypoperfusion—can occur in up to one in every four patients. Those patients have higher incidence of early postoperative cognitive decline and longer hospital stay.

Vascular: Alzheimer's disease and vascular dementia are the two most common forms of dementia, sharing many common pathological, symptomatic and neurochemical features (Kalaria, 2002). At autopsy, 60–90% of patients with Alzheimer’s disease exhibit variable cerebrovascular pathology and almost 30% show evidence of cerebral infarction (Olichney et al 1996; Premkumar et al, 1996).

Plasticity

William James (1890) The Principles of Psychology
Ernesto Lugaro in the early 1906

Education: Nuns' Study, David Snowdon et al (1997) found that 8% of the nuns who behaved and acted free from had the most severe neuropathology. Education found to be an important moderating factor dose-response relation (Ott, et al 1995).

Dancing/Music: (Verghese et al, 2003) Frequency of participation in leisure activities and physical-activity after follow-up period of 5.1 years, and reported that only reading, playing board games, playing musical instruments, and dancing were associated with a reduced risk of Alzheimer's disease and vascular dementia.

Brain Exercises: ACTIVE and IMPACT (Ball et al., 2002; Willis et al., 2006). Early to moderate Alzheimer's disease using computer exercises had better performance on cognitive tests and report improvement in symptoms related to depression (Wolinsk et al, 2009). Related to growth in the hippocampus (Erickson et al, 2011).

Penumbra

‘Sometimes there are also rod-like cell elements of obvious glial origin lying in the halo. At both poles of the core lies a sack filled with greenish granules. Sometimes brighter streaks with straight or jagged bent edges extend through the halo and end at the core of the plaque.’
(Alzheimer, 1912, 80)

Similarity

Journal of Parkinson's Disease 7 (2017) S11–S22
DOI 10.3233/JPD-179006
IOS Press

Review

The MPTP Story

J. William Langston*
Parkinson's Institute, Sunnyvale, CA, USA

Dr. Langston is the Scientific Director, Chief Scientific Officer and Founder of the Parkinson's Institute in Sunnyvale, California. A graduate of the University of Missouri School of Medicine, he served as faculty at Stanford University Medical School and Chairman of Neurology at Santa Clara Valley Medical Center in San Jose, California before founding the Parkinson's Institute. He gained international recognition in 1980s for the discovery of the link between MPTP and Parkinson's disease, which triggered a renaissance of the basic and clinical research in Parkinson's disease. He authored nearly 400 publications, most of which are on Parkinson's disease and is a founding member of the Scientific Advisory Board for the Michael J Fox Foundation for Parkinson's Research.

https://www.researchgate.net/publication/314301010_The_MPTP_story

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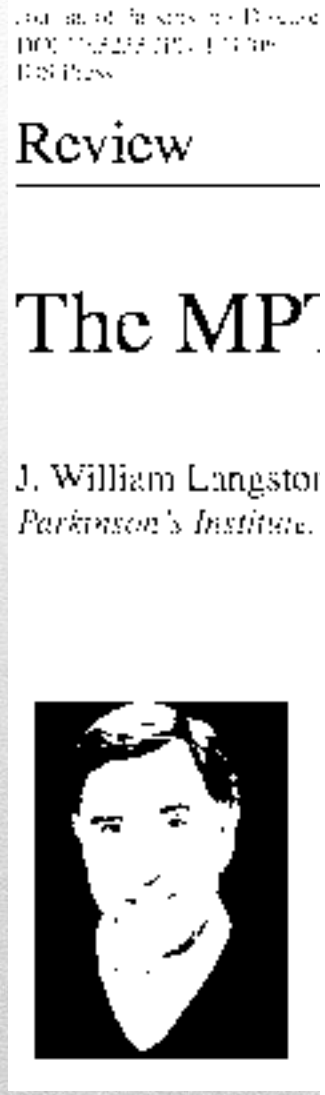
Review

The MPT

J. William Langston
Parkinson's Institute

“Finally, I would like to conclude with some closing thoughts: If there is an overarching lesson from this story for clinicians, it is to never forget the power of clinical observation.”

https://www.researchgate.net/publication/314301010_The_MPTP_story

CARE NOW CURE LATER

1. Protected Citizenship

From “patient” Tom Kitwood we came to see a “person” and an “embodied self.” Now, with legal rights we see people with dementia as “citizens.”

2. Nothing about us, without us

Involve the person living with dementia in their own care and policy as much as possible

3. Wellbeing First

Review medications, reduce agitation, explore options, look after yourself

1. Garrett (2019, forthcoming). Developing a Modern Mythology for Dementia and Alzheimer’s disease. Dementia:.
2. Bryden, C. (2015). Nothing about us, without us!: 20 years of dementia advocacy. Jessica Kingsley Publishers.

FINAL FRONTIER

Sing for care

a lip sync competition

THANK YOU


Curr Neurobiol 2018; 9(1): 49-58
ISSN 0975-9042
A Critique of the 2018 National Institute on Aging's. Research Framework: Toward a biological definition of Alzheimer's disease
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Abstract


Symbiosis
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Research Article
SOJ Neurology
Open Access
A New Public Health Paradigm for Alzheimer's Disease Research
Mario D Garrett* and Ramon Valle
San Diego State University, California, USA


Short Communication
Published: 30 Apr, 2019
Journal of Neurology Forecast
Short Communication
Mario D. Garrett*
Department of Social Work, San Diego State University, California, USA


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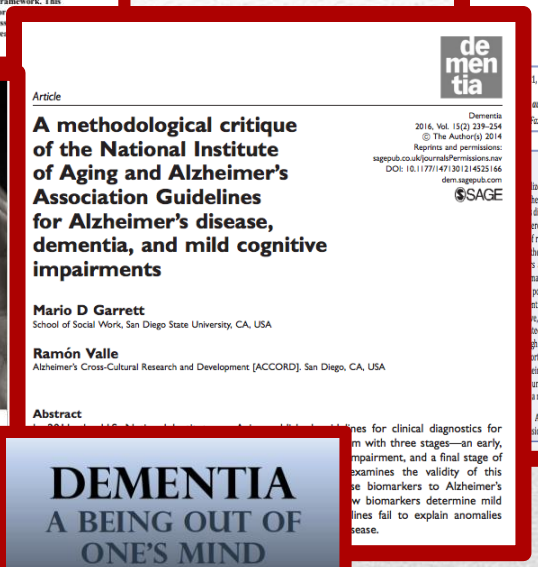
dementia
Article
A methodological critique of the National Institute of Aging and Alzheimer's Association Guidelines for Alzheimer's disease, dementia, and mild cognitive impairments
Mario D Garrett
School of Social Work, San Diego State University, CA, USA
Ramón Valle
Alzheimer's Cross-Cultural Research and Development [ACCORD]. San Diego, CA, USA
Abstract


Reclaiming Senile
Mario Garrett
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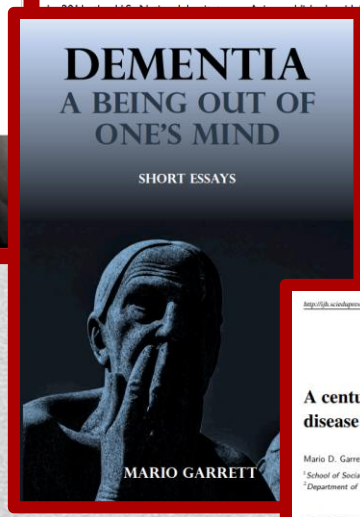
DEMENTIA
A BEING OUT OF ONE'S MIND
SHORT ESSAYS
MARIO GARRETT


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POLITICS OF ANGUISH
How Alzheimer's disease became the malady of the 21st century
Mario Garrett
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REVIEWS
A century of confusion in researching Alzheimer's disease
Mario D. Garrett*[1,2], Ramón Valle[1]
[1]School of Social Work, San Diego State University, California, USA
[2]Department of Psychology, Boğaziçi University, Istanbul, Turkey
ABSTRACT