





13th World congress on

Alzheimer's and Dementia

December 06-07, 2018 | Amsterdam, Netherlands

Alzheimer's and Dementia 2018



13th World congress on Alzheimer's and Dementia

December 06-07, 2018 Amsterdam, Netherlands

Michael A Cole et al., J Neurol Neurosci 2018, Volume: 2 DOI: 10.21767/2471-8548-C1-001

ON THE HORIZON: THE VALUE AND PROMISE OF THE Global late stage pipeline of Alzheimer's Disease therapeutics

Michael A Cole^{1,2} and Guy R Seabrook³

¹Clinical Science Program, University of California Berkeley, USA ²Global Neurohealth Ventures, USA ³Johnson & Johnson Innovation, USA

string of recent phase 3 Alzheimer's disease (AD) trial failures targeting Apprimarily amyloid beta $(A-\beta)$ have challenged hopes for finding an effective disease-modifying therapeutic. Despite some recent advances, this has resulted in some skepticism regarding the current value of the AD pipeline and its potentially over-weighted focus on therapeutics targeting A-B. To investigate these concerns, we have compiled a database of all current phase 2 and 3 AD therapeutics that has disease-modifying targets through a query of the National Institutes of Health's ClinicalTrials.gov. We then assessed the potential therapeutic success as well as financial value of the current AD pipeline. Financial modeling utilized risk-adjusted net present value (rNPV) measurements. Results indicate that the preponderance of current phase 3 trials is indeed targeting A-B with only 15% of the therapeutics addressing other targets. However, the current pipeline of phase 2 trials consists of a rich diversity of targets, with A- β based therapeutics representing <30% of those in development. Modeling data with commercial assumptions built on the experiences of adjacent fields such as cardiovascular disease, the estimated total risk adjusted net present value of current phase 2 and 3 therapeutics combined is \$182 billion over 10 years. This figure increases to a theoretical cumulative value of \$422 billion when also treating asymptomatic individuals at high risk for developing AD. This value requires the global availability of rapid and easy to use diagnostic biomarker(s) of AD risk. Results from sensitivity analyses of financial model assumptions and different drug development strategic approaches will be reported. The promise of the current AD therapeutic pipeline will be discussed in addition to the complex financial ecosystem necessary to maintain a healthy AD pipeline. Diagnostic biomarkers of AD risk will be critical to reach the full global potential of treating individuals in need.



Biography

Michael A Cole received his MA in Behavioural Neuroscience from University of Colorado, MBA from UC Berkeley; PhD in Neuropsychology from the University of Florida and completed his Internship and Residency in Clinical Neuropsychology at the UCLA School of Medicine. He is an Assistant Clinical Professor in the UC Berkeley Clinical Science Program and Founding Partner for Global Neurohealth Ventures. He has published 20+ peer-reviewed journal articles and continues active practice as a Clinical Neuropsychologist.

michaelcole@berkeley.edu



13th World congress on Alzheimer's and Dementia

December 06-07, 2018 Amsterdam, Netherlands

Girish J Kotwal, J Neurol Neurosci 2018, Volume: 2 DOI: 10.21767/2471-8548-C1-001

STRATEGY FOR CONTROLLING BRAIN INFLAMMATION DUE TO AGING, HEAD TRAUMA AND GENETIC Predisposition of Alzheimer's Disease

Girish J Kotwal

School of Medicine, University of Massachusetts, USA InFlaMed Inc, USA Kotwal Bioconsulting LLC, USA Noveratech LLC, USA

A lzheimer's disease (AD) is a major cause of dementia. Major risk factors that Can predispose a person to AD include aging, traumatic brain injury (TBI) and the presence of ApoE4 allele. All risk factors involve progression to memory loss due to continuous neurodegeneration causing inflammation. Aging, a major risk factor for AD can result in hypertension, vascular dementia, diabetic complications, coronary *heart* disease etc., which can all contribute to AD. TBI can set in motion to continuous inflammation in the brain. The presence of ApoE4 allele can be detected by isolating genomic DNA from saliva and determining single nucleotide polymorphism in ApoE gene. If ApoE4 allele is found there can be a three-four fold higher predisposition to AD compared to ApoE2 or ApoE3. One of the harmful effects of the ApoE4 allele is the harmful neurodegeneration due to inflammation. Vaccinia virus complement control protein has been shown to control compliment levels in the brain and could contribute to slowing the progression of AD along with life style and nutritional changes that could result in weight, glucose and lipid control.



Biography

Girish Kotwal has completed his PhD from McMaster University, Canada and Postdoctoral Studies from National Institutes of Health, Bethesda, USA. He is an Adjunct Professor of Medicine at UMass Medical School, President at InFlaMed and Kotwal bioconsulting, LLC and Senior Scientist at Noveratech LLC. He has published more than 100 PubMed listed papers in reputed journals and has been serving as an Editorial Board Member of repute. Over a 30-year period beginning in 1988 with a report in the journal *Nature*, he began research on a complement control protein and showed that regulating complement activity in several CNS conditions could be potentially beneficial.

GJKOTW01@gmail.com



13th World congress on Alzheimer's and Dementia

December 06-07, 2018 Amsterdam, Netherlands

Jie Bai et al., J Neurol Neurosci 2018, Volume: 2 DOI: 10.21767/2471-8548-C1-001

THIOREDOXIN-1 IMPROVES THE COGNITIVE FUNCTION OF APP/PS1 MICE

Jie Bai, Liping Bai, Ye Li, Xianwen Zhang, Xiaoshuang Zhou and Cheng Yan

Kunming University of Science and Technology, China

Izheimer's disease (AD) is the most common neurodegenerative disorder Awhich is characterized by impairment of cognitive function. Thioredoxin-1 (Trx-1) is a redox regulating protein, has antioxidant and protecting neuron effects. Our previous study found that Trx-1 improved the cognitive function of Parkinson's disease (PD) mice. However, whether Trx-1 improves the cognitive function of AD is still unknown. In present study, APP/PS1 transgenic mice model were used. Trx-1 overexpressed transgenic mice were hybridizing with APP/PS1 transgenic mice. Our results showed that the escape latency in APP/PS1 transgenic mice was longer when compared with the control group, which was reduced in Trx-1/ APP/PS transgenic mice significantly by using the navigational experiments in Morris water maze. In the spatial probe test, the total number of crossings and the percentage of time spent in the target quadrant were decreased in APP/ PS1 transgenic mice, which were significantly increased in Trx-1/APP/PS mice. The mRNA levels of APP and PS1 were decreased in Trx-1/APP/PS1 mice when compared to APP/PS1 mice. These results suggest that Trx-1 improves cognitive function of APP/PS1 mice. Trx-1 may be a potential therapeutic target for the clinical management of AD.



Biography

Jie Bai has completed her PhD from Kyoto University and Postdoctoral Studies from Health Center, University of Virginia. She is the Professor of Kunming University of Science and Technology, China. She has published more than 50 papers in reputed journals.

jiebai662001@126.com