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Cognitive Improvements in Patients with Mild Cognitive Impairment and Alzheimer's Disease through a Personalized Mito Food Plan Diet and Cell Repair Therapy



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Mild cognitive impairment (MCI) causes a slight but noticeable and measurable decline in cognitive abilities, including memory and thinking skills. People a ected by MCI have a four- fold increased risk of developing dementia or Alzheimer's disease (AD) compared to cognitively healthy individuals [1]. Although it is unknown if AD is the leading cause of MCI, recent evidence has shown that MCI oien occurs due to the same type of brain damage found in Alzheimer's disease and other forms of dementia [2]. Here are currently no accepted treatments for MCI, nor are there any treatments that have demonstrated prevention of progression to AD, which is the leading cause of dementia worldwide. AD is considered the most prevalent neurodegenerative disorder, progressing from inconsiderable memory loss to eventually death, and has become the most expensive disease in America [3]. At the microscopic and imaging level, the core hallmarks of AD are characterized by hyper phosphorylated and misfolded tau proteins and accumulated beta amyloid (Aβ). For the last two decades, beta amyloid has been the primary paradigm of AD research; however, since 2003, over 250 compounds in more than 400 clinical trials (Phases 1-3) of potential new AD treatments have been studied, with only one (meantime) leading to FDA approval [4]. His 99.6% clinical trial failure rate is the highest of all diseases including cancer (81% failure rate) [5], suggesting that beta amyloid may not be the cause of the disease, and specific etiologies that have been suggested to be prominent in the pathogenesis of AD need to be more thoroughly studied.

The Mito-AD-01 study (clinical trial NCT03630419) was conducted in hopes of determining that oxidative stress and inflammation could be measured and ameliorated through specific, individualized treatment; therefore, improving all around cognition and mental clarity in patients with MCI and AD. Since diet has been thought to play a role in decreasing cognitive decline, a specific Mito food plan geared to decrease inflammation and work on mitochondria was created for each subject based on individual physiological and metabolic results. Cellular Repair therapy was also implemented as an adjunctive therapy to incorporate an all-encompassing non-drug, quickly implemented treatment that could slow disease progression and even improve cognition in those with MCI and AD.

Eligible patients, 50-90 years of age who met criteria for mild to moderate probable AD according to the National Institute of Neurologic and Communicative Disorders and Stroke- Alzheimer's Disease and Related Disorders Association criteria, were enrolled. Eligible MCI patients had to be clinically diagnosed with amnestic MCI as defined and documented by their neurologist. All patients were required to have a Mini Mental State Examination (MMSE) score greater than 17 and score greater than a 10 on the Montreal Orientation Cognitive Assessment (MoCA) at screening to meet study eligibility. Additional inclusion criteria included scoring a 4 or greater on the Constructional Praxis exam portion of the Alzheimer's Disease Assessment Scale-Cognitive (ADAS-Cog) test. Study participants were also required to provide informed consent and (a caregiver/significant other) keep track of their daily food intake (Mito food plan diary). In addition to meeting eligibility criteria, study subjects had to avoid high-intensity activity 24 h prior to day of comprehensive body assessment and had to avoid all physical exercise for at least 3 h prior. Treatment with approved or o label use of AD medications or antiinflammatory supplements were permitted as long as they were on a stable dose for at least 3 months prior to screening. All study procedures were approved by the Western Institutional Review Board (WIRB) and were conducted with the understanding and consent of all subjects. Informed consents were obtained from all individuals in accordance with institutional review board requirements prior to the start of any study-related procedures at screening.

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Prior to enrollment, potential subjects were evaluated, screened, and consented. Medical history was captured, and a physical and neurological exam was performed at screening. A Mini-Mental Status Exam (MMSE), Montreal Cognitive Assessment (MoCA), Alzheimer's disease Assessment in Cognition (ADAS-Cog) constructional praxis, and Quality of Life in AD (QOL-AD) questionnaires were completed for eligibility purposes and for baseline measures. All subjects who met study eligibility underwent cellular and physiological comprehensive body assessments at Cerulean Advanced Wellness and Fitness. Hese comprehensive assessments included a series of non-invasive Physiological and Cellular Health tests that were able to measure body fat, lean muscle, total body water, cellular health index, inflammation, and body hydration through Bioelectrical Impedance Analysis (BIA), manufactured by the Body Stat Quad Scan 4000. An electrocardiograph (ECG) and hemodynamic diagnostics such as heart rate (HR), blood pressure (BP) and arterial oxygen saturation (SpO2) were also recorded for safety measures. Once these assessments were completed and analyzed, each study subject was given a specific Mito food plan. He Mito food plan was created based on individual subject assessments and results. Caloric intake was based on Resting Metabolic Rate (RMR), Respiratory Quotient (RQ), and Metabolic (ᵬciency (ME) as measured through RMR testing. Daily recommended servings of specific macronutrients varied for each subject. Since the macronutrient recommendations yielded from the RMR and Body Stat results, the Mito food plan was individualized to each subject's physiological body results. All subjects were required to fill out a daily diet dairy which was reviewed at each visit. Four of the five subjects opted to undergo adjunctive Cellular Repair Herapy for 30 minutes 3 times a week for a total of 12 weeks.

Resting Metabolic Rate (RMR) was tested for an objective measurement of metabolism through Ultima™ CardiO2® gas exchange analysis system. His machine measures resting energy metabolism through the measurement of oxygen (O2) consumption and carbon dioxide (CO2) production. Each subject was required to wear a flow sensor mask for 20 minutes. From the measurement of VO2 and VCO2 , the resting energy expenditure (REE) was calculated to assess energy fuel utilization and energy expenditure. In the body, the calories from food are burned in the presence of oxygen, and because the process of oxidation in the body is well understood, the amount of total heat or energy produced by the body at rest (RMR) can be measured. Energy produced by the body at rest is measured from the measured amounts of oxygen consumed and the carbon dioxide produced (exhaled). Resting metabolism occurs in a continual process 24 h a day and remains relatively constant over time. Resting metabolism is the largest component (typically 60% to 70%) of "calories out" in the energy equation. Performing an RMR assessment allowed for an individualized predictable approach to how many calories and macros each subject should consume during the course of the study.

Biography:

Dr. Nicole C Hank is currently working in Department of Neurology, Perseverance Research Center, LLC, 11000 North Scottsdale Road, 110, Scottsdale, USA. She is a clinical research professional with over 19 years of research experience. Her areas of research include genetic and molecular biology in neuro-oncology, clinical work in epilepsy, amyotrophic lateral sclerosis, inclusion body myositis, neuropathy, alzheimer's disease, stroke, traumatic brain injury, autism and multiple sclerosis. She has coordinated and conducted over 65 clinical trials and many of her research findings have been published in various medical journals. Finding a cure or a therapy that can ameliorate the lives of individuals who suffer from neurological disorders is Nicole's truest passion. She has led several support groups for patients and caregivers over the years and will continue to do so at Perseverance Research Center. She is dedicated to improving quality of life through natural alternatives, creating NeuroVitality, a company that has developed a vitamin supplement to address the pain associated with neuropathy and other nerve disorders. Aside from her Masters in Health Sector Management with an emphasis in Epidemiology from the WP Carey School of Business at Arizona State University, she has obtained her Masters in Clinical Research and a PhD in Clinical Research from Texila American University.

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About Company:

Perseverance Research Center was started by three clinical research professionals with over 40 years of combined clinical research experience. After working in a variety of settings including large academic institutions, hospitals, and clinical research organizations, they decided to create their own Research Center focusing on a clinical research and education in a variety of therapeutic areas.

References:

1. De Bruijn RF, Akoudad S, Cremers LG, Hofman A, Niessen WJ, et al. (2014) Determinants, MRI correlates, and prognosis of mild cognitive impairment: the Rotterdam Study. J Alzheimers Dis 3: 239-249.
2. Mitchell AJ, Shiri-Feshki M (2009) Rate of progression of mild cognitive impairment to dementia – meta-analyssis of 41 robust inception cohort studies. Acta Psychiatr Scand 119: 252-265.
3. Apostolova LG (2016) Alzheimer Disease. Continuum (Minneap Minn) 22: 419-434.
4. Cummings J, Lee G, Mortsdorf T, Ritter A, Zhong K (2017) Alzheimer's disease drug development pipeline: 2017. Alzheimers Dement (N Y) 3: 367-384.
5. Zhong K, Cummings J (2016) Healthybrains.org: From registry to randomization. J Prev Alzheimers Dis 3: 123-126.
6. Van J Eldika, MC Carrillo, PE Cole, D Feuerbach, Greenberg BD, et al. (2016) Interventions He roles of inflammation and immune mechanisms in Alzheimer's disease, Alzheimer's & Dementia: Translational Research & Clinical Interventions 2: 99-109.
7. Cai H, Cong WN, Ji S, Rothman S, Maudsley S, et al. (2012) Metabolic Dysfunction in Alzheimer's Disease and Related Neurodegenerative Disorders. Curr Alzheimer Res 9: 5-17.
8. Guo C, Sun L, Chen X, Zhang D (2013) Oxidative stress, mitochondrial damage and neurodegenerative diseases. Neural Regen Res 8: 2003-2014.
9. Roberts RO, Roberts LA, Geda YE, Cha RH, Pankratz VS, et al. (2012) Relative intake of macronutrients impacts risk of mild cognitive impairment or dementia. J Alzheimers Dis 32: 329-339.
10. Cederholm T, Barazzoni R, Austin P, Ballmer P, Biolo G, et al. (2017) ESPEN guidelines on definitions and terminology of clinical nutrition. Clin Nutr 36: 49-64.
11. Rinninella E, Cintoni M, Addolorato G, Triarico S, Ruggiero A, et al (2018). Phase angle and impedance ratio: Two specular ways to analyze body composition. Ann Clin Nutr 1: 1003.
12. Crichton GE, Bryan J, Murphy KJ (2013) Dietary antioxidants, cognitive function and dementia-a systematic review. Plant Foods Hum Nutr 68: 279-292.
13. Lobo V, Patil A, Phatak A, Chandra N (2010) Free radicals, antioxidants and functional foods: Impact on human health. Pharmacogn Rev 4: 118-126.
14. He Mito Food Comprehensive Guide (2016). Institutional of Functional Medicine.
15. Davalli P, Mitic T, Caporali A, Lauriola A, D'Arca D (2016) ROS, Cell Senescence, and Novel Molecular Mechanisms in Aging and Age-Related Diseases. Oxid Med Cell Longev 7: 12-14.
16. Wei YH, Ma YS, Lee HC, Lee CF, Lu C (2001) Mitochondrial theory of aging matures—roles of mtDNA mutation and oxidative stress in human aging. Zhonghua Yi Xue Za Zhi (Taipei) 64: 259-270.
17. Schmidt M, Homsen M, Schmidt U (2012) Suitability of ivy extract for the treatment of paediatric cough. Phytotherapy Res 12: 1942-1947.
18. Tanuja N, Bhagwat B, Jasmin J, Narenda B, Deepa C (2004) Clinical validation of eتcDcy and safety of herbal cough formula: study of herbal cough syrup. J Herb Pharmacother 4: 1-12.
19. Buechi S, Vogelin Roger, Von ELوMM, Ramos M, Melzer J (2005) Open trial to assess aspects of safety and eتcDcy of a combined herbal cough syrup with ivy and thyme. Forsch Komplementarmed Klass Naturheilkd 12: 328-332.
20. Michelle S, Jennie J, Daniel R, Gordon G, Mark L (2012) Zinc for the treatment of the common cold: A systematic review and meta-analysis of randomized controlled trials. CMAJ 184: E551-E561.
21. Hemila H, Elizabeth C (2015) He eوectLveness of high dose zinc acetate lozenges on various common cold symptoms: A meta-analysis. BMC Fam Pract 16: 1-11.
22. George A, Eby III (2010) Zinc lozenges as cure for the common cold: A Review and hypothesis. Med hypotheses 74: 482-492.
23. Ali C, Smail A, Mohamed R, Amal B, Mohamed E, et al. (2017) Polyphenols content and evaluation of antioxidant activity of Anacyclus pyrethrum (L.) Lag. from timahdite a moroccan middle atlas region. Int J Adv Res 5: 569-577.
24. Sarieh S, Javad S, Farzaneh M, Mohammad R, Mohammad R (2014) Eو ects of aqueous root extracts of anacyclus pyrethrum on gonadotropins and testosterone serum in adult male rats. Am J Phytomed Clin Her 2: 767-772.
25. Irwin RS (2006) Complications of cough. Chest 129: 54S-58S.
26. Somro A, Akram M, khan MI, Asif H, Sami A, et al. (2011) Pharyngitis and sore throat: A review. Afr J Biotechnol 10: 6190-6197.

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