production of Aβ," says Dr. Wellington.[3]

Despite her progress she emphasizes that our ability to treat or prevent the illness is nascent and that we must not overlook the influence of other factors that do affect overall risk. Intriguingly, what is good for the heart is good for the brain. She explains that "the biggest piece of advice I always give to the general public is never stop exercising; that's probably one of the best things that you can do to promote healthy aging from the cardiovascular, metabolic, and neurologic perspectives."[3]

REFERENCES

1. Fauci A, Braunwald E, Kasper D, Hauser S, Longo D, Jameson J, et al., editors. Harrison's Principles of Internal Medicine, 17th Ed, USA, The McGraw-Hill Companies, Inc., 2008.
2. Ropper A, Samuels M. Adams & Victors Principles of Neurology, 9th Ed. The McGraw-Hill Companies, Inc., USA, 2009.
3. Fan J, Donkin J, Wellington C. Greasing the wheels of Abeta clearance in Alzheimer's disease: the role of lipids and apolipoprotein E. Biofactors. 2009 May-Jun; 35(3):239–48.

University of British Columbia Conference on Dementia 2011

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On January 29, 2011, The University of British Columbia partnered with the Clinic for Alzheimer's Disease and Related Disorders at UBC Hospital (CARD-UBCH) to host Dementia 2011. Speakers included Jean Blake, CEO of Azheimer Society of BC, and Dr. Lynn Beattie, medical director at CARD-UBCH. The objective of this conference was to inform the public on Alzheimer's Disease (AD) diagnostic techniques, current treatments, and future treatments. AD affects 25–45 % of persons over the age of 85, and this percentage is projected to increase as the geriatric population grows. One of the event's focuses was in AD diagnostics, which is currently undergoing substantial review. AD can only be diagnosed upon post-mortem examination, leading to frustration and uncertainty over the appropriate course of treatment for patients.

Current AD diagnostic techniques primarily include assessment of medical history, cognitive examination, and neuroimaging. The Mini Mental Status Examination (MMSE) is an especially important cognitive exam to conduct; the MMSE tests a patient's cognitive capacity through a series of basic questions and tasks. It has high sensitivity but only moderate specificity, occasionally resulting in false positive diagnoses. Part of the difficulty in using the MMSE is that it is a highly subjective test and depends on the age and educational level of the patient. Magnetic resonance imaging (MRI) has also been a useful diagnostic tool, especially when considering the decrease in hippocampal volume. However, a decrease in hippocampal volume is common in many forms of dementia other than AD and therefore does not provide a definitive diagnosis. This is problematic because many AD treatments are most effective when implemented at early stages of the disease. Furthermore, a compounding problem is that the most advanced diagnostic tools are restricted to urban areas resulting in inaccessibility for rural populations.

A relatively newer diagnostic tool used in clinical trials measures the ratio of beta amyloid 42 (Aβ-42) to phosphrylated tau (p-tau) protein as biomarkers in the cerebrospinal fluid (CSF). Biomarkers are measurable biological substances which indicate the presence or absence of a particular disease state. Studies have indicated that Aβ-42 concentration diminishes as AD progresses while p-tau concentration increases with disease severity. Therefore, when the biomarkers are used together, clinicians can accurately assess both disease severity and stage.

The most promising solution is to develop a non-invasive, accessible, and accurate technique such as blood testing which would detect AD at an early stage. Although this field of research is still in its infancy, recent reports have found possible blood biomarkers which could lead to definitive AD diagnosis. AD diagnostics has progressed significantly in the past decade. Looking forward, physicians will increasingly rely on biomarkers found in the CSF and blood plasma to diagnose AD and hopefully to provide patients and their families with some peace of mind.

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REFERENCES

1. Bird TD. Genetic aspects of Alzheimer disease. Genet Med. 2008;10(4):231–9.
2. Barber RC. Biomarkers for Early Detection of Alzheimer Disease. J Am Osteopath Assoc. 2010;110(9 suppl 8):S10–S15.
3. Wahlund LO, Blennow K. Cerebrospinal fluid biomarkers for disease stage and intensity in cognitively impaired patients. Neuroscience Letters. 2003;339(2):99–102.
4. Reddy MM, Wilson R, Wilson J, Connell S, Gocke A, Hynan L, et al. Identification of Candidate IgG Biomarkers for Alzheimer's Disease via Combinatorial Library Screening. Cell. 2011;144, 132–42.