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The Relationship Between Alzheimer’s And Genetics

This paper is about the relationship between AD (Alzheimer’s disease) and genetics. In this paper, the reader will read about the different risk factors relating to AD and they will understand what the different risk factors are and if those certain risk factors pertain to them. This paper will also include the difference between early-onset and late-onset and what each of those terms mean. The reader will understand the two different components of AD and what each of those components is. Also, the reader will know the signs and symptoms of AD and what to look out for. The reader will know who founded and discovered the disease. The reader will know what chromosomes or cells carry that trait and how it can be passed down from generation to generation. This paper will explain and define clearly what AD is and how it is treated and how it is passed down throughout generations.

THE RELATIONSHIP BETWEEN ALZHEIMER’S AND GENETICS

“Alzheimer’s disease was first discovered by a German man named Alois Alzheimer. It is the most common cause of dementia and currently there are five million people around the world that have Alzheimer’s disease (Kennedy 2013).” In this paper I will discuss the causes of Alzheimer’s disease and the different risk factors. This paper will also discuss the difference between early-onset and late-onset Alzheimer’s is. Lastly, this paper will discuss the treatments and effects on the brain this deadly disease has. This paper will elaborate on what the different genes and chromosomes are that participate in Alzheimer’s disease.

DEFINITION OF ALZHEIMER’S DISEASE

“AD is an incurable degenerative disease of the brain first described in 1906 by the German neuropathologist Alois Alzheimer (b. June 14, 1864; d. Dec. 19, 1915). It is the most common cause of dementia in adults and is estimated to affect more than 5 million men and women over the age of 65 in the U.S. (Kennedy 2013).” Alzheimer’s disease (AD) is the most “common cause of dementia, and is a progressive illness (Kennedy 2013).”

CAUSES OF AD AND RISKS

The brains of people with AD have been found to have two specific types of abnormal structures. These structures are the physical evidence of AD. But it is still not known whether they are the primary cause of damage to the brain. Most experts agree that it is probably a complex set of genetic and non-genetic factors, rather than a single cause, that triggers the disease process (Kennedy 2013). “The cause of AD is unknown, but there are a number of risk factors for developing the disease; advanced age is the most important. Although the risk of developing AD is less than 1% before 50 years of age, it increases steeply with each successive decade of life to reach 30% or more by the age of 90. Genetic factors are also important. Mutations in several genes have been associated with patients who have rare familial types of AD . . . several risk factors are associated with AD. For example, there are the genetic mutations associated with early-onset AD. Other risk factors include increasing age, a family history of the disease, and one gene that increase that risk of late-onset AD. Risk factors for heart disease may also increase the risk for AD. One such risk factor is high blood pressure. There is also evidence that remaining mentally active may be associated with a lower risk.” Many tests were performed to find a cure or cause to this disease . . . methods of accurate diagnosis will be most useful only when effective treatments are available. Until the exact cause of AD is determined, a cure will remain elusive. There are no treatments that reverse the primary cognitive impairments or that retard the course of illness. Some drug clinical trials have sought to improve memory symptoms by treating the deficits in acetylcholine neurotransmission. Three such drugs have been approved for use, although their effectiveness is limited. Other potential treatments include vitamin E; nonsteroidal anti-inflammatory agents; and a drug called memantine, which in clinical trials has been found to slow the cognitive decline of mid- and later-stage AD patients. In mid-1999 researchers working with plaque-prone mice developed a vaccine that stimulates the body to produce antibodies to beta amyloid, the key element in plaque. The antibodies were able to prevent the development of plaque in younger mice and clear away the plaques in older mice. Whether this approach will work in humans remains unclear, since it is not known if the immune response is sustainable over long periods of time; moreover, AD researchers are not sure whether the plaques or the tangles do more damage to the brains of patients. (Kennedy 2013).

Although the precise cause of Alzheimer's disease remains a mystery, research has increasingly focused on a genetic basis for the condition. Scientists now suggest that genes on at least three different chromosomes may be responsible for Alzheimer's disease. Chromosome 19 may control the common form, which appears after age 65, while two other, relatively rare forms of the condition, occurring at an earlier age, may be linked to genetic material on chromosomes 14 and 21. If these three chromosomes do play a developmental role, however, they may do so in combination with other factors. Some researchers have suggested that an infectious agent similar to a virus, or perhaps concentrations of aluminum in the brain, may be involved (Whitehouse 2012).

SYMPTOMS

Symptoms are divided into two categories-cognitive (intellectual), or psychiatric. The importance of the differences so they don’t mess up the meds. Cognitive symptoms are amnesia, aphasia, apraxia, and agnosia (four A’s of alzheimer’s). Major psychiatric symptoms include personality changes, depression, hallucinations, and delusions (Alzheimer’s Foundation of America 2012).

Some symptoms of AD are personality and behavior changes, decline in memory, reduced awareness of one’s surroundings and recent events, repetitive behavior, increased restlessness, and agitation in late afternoon and evening; wandering. Also, bowel and bladder incontinence and suspicion. There is no current way to stop the progression of the disease. Medications can help symptoms but not cure them.” Whitehouse (2012) states “since the earliest symptoms of Alzheimer's disease develop gradually, the exact onset of the condition may be difficult to identify. Often the earliest sign is forgetfulness, which may be accompanied by subtle changes in personality. As the disease progresses, other impairments develop, including difficulties with language, perception, and complex motor skills. The patient gradually declines over a period of five to ten years and may reach a point of losing control of body functions and becoming unresponsive to those around him or her. Death often results from complications, such as pneumonia, related to the patient's lack of mobility (Badasch et al. 2011)

EFFECTS ON THE BRAIN

Alzheimer’s disease is defined by specific changes in the brain. The disease involves shrink age of the brain together with loss of nerve cell in several areas of the brain thought to be important for intellectual activity. Under the microscope two abnormalities are usually seen. Clusters of neuritic plaques, composed of degenerating nerve cell elements surrounding a core of the brain protein B-amyloid, are widespread. In addition, fibrous structures called neurofibrillary tangles are found in diseased neurons and may interfere with normal cell function. Associated with these nerve cell changes is a reduction in the brain’s neurotransmitters, chemical messengers used for communication between nerve cells. Understanding the changes in these neurotransmitter levels in particularly important since many of the drug treatments for neurological and psychiatric diseased work by way of neurotransmitter systems. A major neurotransmitter affected in AD is acetylcholine (which plays a role in memory), because the condition destroys a portion of the brain’s acetylcholine-producing nerve cells. While other neurotransmitters are also affected in Alzheimer’s disease, the alterations in acetylcholine are the most severe and the most consistently found (Whitehouse 2012).

DIAGNOSIS

Specific tests for Alzheimer's disease are virtually nonexistent. To make a diagnosis, a physician must generally rule out all other possible causes of dementia-like symptoms and assess whether the patient's symptoms are consistent with Alzheimer's. Although Alzheimer's disease is the most common cause of dementia, many other diseases can result in similar changes in behavior and intellect. In about 10% of patients, dementia has a potentially reversible cause such as stroke, brain tumor, head injury, liver or kidney disease, thyroid or other endocrine problems, or brain infection. In addition, some psychiatric disorders, such as depression, can be mistaken for dementia. A thorough evaluation of the patient with dementia is therefore essential before the diagnosis of Alzheimer's is accepted. Definitive diagnosis, however, is possible only through a brain biopsy of a living patient (which is rare) or through an autopsy of the brain after death. Encouraging progress in the diagnosis of Alzheimer's was announced in 2007, when a blood test was developed that successfully distinguished the blood of people with Alzheimer's from the blood of people without Alzheimer's with 90% accuracy. Experts caution that the test is still preliminary (Whitehouse 2012).

THERAPY

Methods of accurate diagnosis will be most useful when effective treatments are available. No such treatment has yet been found that consistently ameliorates the symptoms of Alzheimer's disease or reverses the disease process. Nevertheless, a variety of therapies can be helpful for the individual affected by this disease. Research indicates that the medication known as tacrine can bring about some mental improvement, including the ability to remember and reason, in a portion of Alzheimer's patients. The drug inhibits the enzyme acetylcholinesterase, which breaks down acetylcholine.

Specific medications can also be used to treat the depression and other psychological problems that may accompany the disease and increase the symptoms of dementia. Also, psychotherapy can help both patient and family deal with the illness, and support groups such as those organized in the United States by the Alzheimer's Association offer substantial help.” Another issue is what all of the stress has done to the families . . . alzheimer's disease causes tremendous psychological strain on both patient and family. Legal and financial issues often become complicated when an individual is no longer competent to manage his or her own affairs. Often the most difficult decision the family faces in the later stages of the disease is whether or when to place the patient in an extended-care facility (Whitehouse 2012).

RESEARCH

Considerable research is being directed toward the development of therapies for Alzheimer's disease. One potential treatment involves the implantation of fetal tissue into the brain in order to improve neurological function. Other promising therapies may result from the contributions of a number of medical disciplines. By examining the demographic characteristics of Alzheimer's patients, for example, epidemiologists may be able to identify factors that place certain people at high risk for the disease. In addition, neurologists, psychiatrists, and psychologists are studying individual patients in the hope of better defining the nature of the intellectual impairments associated with Alzheimer's; neuropathologists are working to understand why only some nerve cells are affected by the disease; and neurochemists are analyzing the neurotransmitter changes linked to the condition. With this combined effort, it is hoped that understanding of Alzheimer's disease will substantially increase (Whitehouse 2012).

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