

Political and Nursing Considerations for Alzheimer's Disease

By Brandon Dominguez, DNP, MSN, BSN, RN, CHEP

Abstract

Alzheimer's Disease is a progressive mental deterioration that can occur in middle or old age, due to generalized degeneration of the brain. It is the most common cause of premature senility. Alzheimer's disease is the result of one, two, or more different genes that together can produce or predispose to a serious defect, often in concert with environmental factors. As the genetics of neurodegenerative disease, like Alzheimer's Disease (AD), become better understood, opportunities for genetic susceptibility testing for at-risk individuals will increase. As AD is known to be a multifactorial, or complex inheritance disease, there are a few methods of determining risk through genetic patterns. However, there are political and resource implications that must be understood to improve the quality of care for clients diagnosed with AD. The burden of financial resources is minimized when care is provided and planned with a preventative focus, rather than reactive. The Affordable Care Act, Mental Health Parity, and Addiction Equity Act greatly expand the coverage of behavioral health care services for Americans. Nurses play an essential role for improving care. In efforts to reduce the development of amyloid plaques seen in AD, there are a few nutritional recommendations for use with treatment planning in prevention, screening, diagnostics, prognostics, and treatment evaluation. Early detection of AD can benefit patients by increasing chance of benefitting from treatment, decreasing anxiety, increase chance of trial participation, and an opportunity to participate in decisions about care, transportation, living options, financial and legal matters.

Goal Statement

The goal of this course is to provide information about the political policies, resources, and nursing considerations for clients diagnosed with Alzheimer's Disease.

Course Objectives

- a. *Interpret* how a clinician might utilize a genetic analysis
- b. *Discuss* the importance of primary, secondary, and tertiary prevention interventions for clients diagnosed with Alzheimer's Disease.
- c. *Support* the concept of preventative vs. reactive care for Alzheimer's Disease.

Introduction

This introduction is a work of fiction. Names, characters, businesses, places, events, locales, and incidents are either the products of the author's imagination or used in a fictitious manner. Any resemblance to actual persons, living or dead, or actual events is purely coincidental.

Greg is a Registered Nurse working at an urgent care clinic in a metropolitan area. He walks into the first client's room and is met by an adult woman and an older gentleman. The woman states, "Finally someone shows up. We have been waiting forever. My Dad is getting worse and I think he needs some medication like an anti-psychotic or something." Greg looks at the older gentleman and asks, "How are you doing today Mr. Johnson?" The older gentleman slowly turns his head towards Greg, pauses for a few seconds and mumbles, "hmmm fine...fine I suppose...I suppose I am fine." Greg works through his physical assessment and determines that there are no clinical abnormalities. The woman states, "Is everything ok? My Dad is acting like how my Grandfather did. I don't remember my Grandfather ever getting help but I know this is definitely not normal." Upon completing a mental status exam, Greg discovers that Mr. Johnson is having cognitive impairments similar to dementia, including Alzheimer's Disease. With a look of determination, Greg shifts his attention towards the woman and asks, "Tell me more about how long these symptoms have been occurring for."

Evidence-Based Information

FDA Pharmaceutical Guidelines and Regulations

The Food and Drug Administration (FDA) is a government body that is responsible for public

health by overseeing the security, safety, and efficacy of veterinary and human biological products, drugs, and medical devices (Office of the Commissioner, 2018). Within the FDA, there is a center called the Center for Drug Evaluation and Research (CDER) that is responsible for the safety and effectiveness for drugs marketed in the United States. A company that is wishing to market a new drug must submit evidence to CDER that supports the safe use and effectiveness of the new drug. While a drug is in development, companies follow documented best practice guidelines published by CDER labeled, Good Review Practice (GRP). The GRP document details how companies should present their chemical, manufacturing, control, and biometric data for approval review. Details within the GRP are extracted from the Code of Federal Regulations Title 21 (CFR Title 21). The CFR Title 21 is a database that include a codification of the general and permanent rules published in the Federal Register by the Executive departments and agencies of the Federal Government for the rules of the FDA (US Food & Drug, 2018). A study by Caroline Saunders (2017) detail how the FDA should replace the current guidelines with a specialized three-tiered designation system that categorizes wearables (medical devices) according to their risk. This kind of a dynamic approach (Saunders, 2017) will allow the FDA to balance regulation with innovation because the wearables-specific, three-tiered system will provide a regulatory foundation that sets forth clear production standards, ensures safety and effectiveness, and supports the advancement of wearable technology over the longer term.

Financial Resources for Health Care

The concept of capitalism for health care management has strengths and weaknesses. Zivotofsky & Zivotofsky (2014) studied how capitalism can be beneficial for health care systems. The study (Zivotofsky, 2014) argues that advertisements from private medical companies serve as a primary source of information for consumers. For example, how else would a consumer be aware of nearby hospitals or drugs available to treat illnesses aside from a visit to their primary care provider? However, the act of advertising within the concept of capitalism may cause issues within healthcare. For example, Archontaki (2009) discovered a significant increase in rhinitis medicamentosa (RM) in a region of Greece to an intensive media advertising campaign for nasal topical decongestants. Upon further analysis (Archontaki, 2009), 80% of patients who reported RM did not consult with their primary care provider and had made their purchasing decision solely on the basis of the information supplied by the drug advertisement. The success of capitalism in the

healthcare industry relies on a certain degree of free choice, knowledge, and intelligence of the average citizen.

Family Roles in Health Care Decisions

Providing quality patient centered care includes the consideration of external support and motivation. Family and friends of a patient may provide accountability in treatment plan adherence. If possible, clinicians should include patient identified support, such as family and friends, during treatment planning, especially for discharge planning. An article published by Smalley, Ismalley, Kenney, Denboba, & Strickland (2014), studied the family perceptions of shared decision-making (SDM) with health care providers. Approximately 70% of surveyed (Smalley, 2014) families perceived themselves as shared decision-makers in their child's care when having a medical home. A medical home is team of health care providers that provide comprehensive and continuous medical care with a goal to obtain maximal health outcomes (American College of Physicians, 2012). Another article in support of family decision making was published by Aarnio, Kulmala, & Olsson (2018). Aarnio (2018) focused on the husband's role in handling pregnancy complications and explains how there needs to be an increased focus on the community level of male involvement. The study (Aarnio, 2018) concluded that husbands have limited access to knowledge of maternal health, which can compromise their decisions about seeking healthcare, and calls for an increase in ease of accessibility and amount of maternal health information to increase the participation of husbands into maternal health care decisions.

Alzheimer's Disease

Alzheimer's disease is a progressive mental deterioration that can occur in middle or old age, due to generalized degeneration of the brain. It is the most common cause of premature senility (Costa, Whitfield, & Stewart, 1989). Alzheimer's disease is the result of one, two, or more different genes that together can produce or predispose to a serious defect, often in concert with environmental factors (Nussbaum, McInnes, Willard, & Hamosh, 2007). This type of disease is genetically classified as a multifactorial or complex inheritance pattern.

Traits of complex inheritance health issues can be divided into two major categories; qualitative and quantitative. Familial aggregation is a qualitative trait that family members may develop by chance alone. Aside from just genes, families share similar diet, environmental exposures, and behaviors. Disease concordance less than 100% in MZ twins is strong evidence that nongenetic factors play a role in the disease. Such factors could include environmental

influences, such as exposure to infection or diet, as well as other effects, such as somatic mutation, effects of aging, and differences in X inactivation in one female twin compared with the other (Nussbaum, McInnes, Willard, & Hamosh, 2007).

Quantitative traits of genetic diseases include body mass index, blood pressure, and serum cholesterol. For example, late onset Alzheimer's disease is associated with apolipoprotein E, which is a protein component of the low-density lipoprotein particle. However, for Alzheimer's Disease, there appears to be additional genes as well as environmental effects involved but remain to be identified.

Laboratory Testing

Currently, the only way to definitely diagnose Alzheimer's Disease is through a post mortem examination. Brain tissue is microscopically examined for the presence of plaques and tangles associated with the disease. There have been completed studies that explore methods of diagnosing Alzheimer's disease before death. Schoonenboom, Kalisvaart, & Akarriou (2017) studied the use of a PET scan using amyloid markers to assist with the recognition of early signs and symptoms of Alzheimer's disease. Magnetic Resonance Imaging techniques are currently being studied to detect plaques associated with Alzheimer's disease for a definitive diagnosis on a living patient (Rastogi, Tyagi, Singh, Hemanth, Singh, Ghosh, & Roy, 2017). Clinicians must rely on screening tools, such as the Memory Impairment Screen, to identify early factors and plan appropriate interventions to delay the development of plaques and tangles (Fowler, Perkins, Gao, Sachs, Uebelhor, & Boustani, 2018).

As the genetics of neurodegenerative disease, like Alzheimer's disease, become better understood, opportunities for genetic susceptibility testing for at-risk individuals will increase. Such testing raises important ethical and practice issues related to test access, informed consent, risk estimation and communication, return of results, and policies to prevent genetic discrimination (Roberts & Uhlmann, 2013). Direct-to-consumer genetic testing may have unforeseen behavioral and psychological impacts on research and health insurance. Results received from direct-to-consumer tests are often received outside of a clinical environment and without the aid of a clinical geneticist, psychotherapist, and/or genetic counselor.

Chromosomal Analysis

Alzheimer's Disease is subdivided into two types. The first type of AD is called Early-onset. Early-onset is defined by the occurrence of neurocognitive symptoms before the age of 65. This particular type of AD is considered rare, only

occurring in 5% of patients who are diagnosed with AD (Takahashi, 1999). A unique symptom that patients experience who have been diagnosed with Early-onset Alzheimer's is the occurrence of myoclonus. However, Early-onset AD has been discovered to have a unique genetic origin. A study by Wiseman et al. (2015), details how a particular mutation on chromosome 14 is linked to Early-onset AD. This mutation, presenilin 1 (E280A), had been sequenced from individuals in Colombia and was discovered that this mutation has a western European geographic origin (Lalli, 2014).

The second type, and most common type of AD, is Late-onset. Late-onset is defined by the occurrence of neurocognitive symptoms in patients of 65 years of age and older. However, there has yet to be a gene identified for the occurrence of late-onset AD. A study by Misra, Chakrabarti, & Gambhir (2018), utilized large-scale genome-wide association studies to identify single-nucleotide polymorphisms in multiple genes that are associated with AD. Tentative mechanisms have also been elaborated in various studies by which the proteins coded by these genes may exert a role in AD pathogenesis have also been elaborated (Misra, Chakrabarti, & Gambhir, 2018).

Etiology of Alzheimer's Disease

AD is characterized by the occurrence of neurocognitive symptoms such as impairments in memory, mild confusion, personality changes, and difficulties completing activities of daily living. These symptoms are believed to be caused by neuronal death, synaptic deterioration, intracellular neurofibrillary tangles, and amyloid plaques. Ballard, Gauthier, Corbett, Brayne, Aarsland, & Jones (2014) hypothesize that an "amyloid cascade" interferes with synaptic activity and initiates a series of downstream effects that cause increasing amounts of neuronal dysfunction and eventually, cell death. Other signs and symptoms include amyloid deposits in blood vessels and granulovacuolar degeneration in the hippocampus.

Amyloid plaques are composed of various forms of β -amyloid proteins ($A\beta$). $A\beta$ is a product of various cellular protein productions by PS1, PS2, APP, APoE, and SorL1 (Ballard et al., 2014). Although excess amyloid plaques are characteristic of AD, the buildup of $A\beta$ into plaques are not believed to be the cause of AD. Increased amounts of $A\beta$ does not indicate levels of cognitive impairment, can be found cognitively normal adults, and has been associated with cognitive improvement in some AD mouse models (Castillo, 2014).

Many of the neurocognitive symptoms are also believed to originate from neuronal death. The loss of neurons and degradation of synapses lead to a

deficit in acetylcholine (ACh) (Ballard et al., 2014). As neuron death in AD progresses to the brainstem, production of the neurotransmitters norepinephrine and serotonin are then affected. Abnormal levels of these neurotransmitters may contribute to insomnia and dysphoria commonly seen in AD.

Considerations for Alzheimer's Disease Education

As AD is known to be a multifactorial, or complex inheritance disease, there are a few methods of determining risk through genetic patterns. There are two methods that can be used to determine family health histories. The first method is called linkage analysis. Linkage Analysis is a family-based approach that utilizes family pedigrees to trace the inheritance of a disease. Inheritance patterns can be identified by reviewing a few generations occurrence of a disease. The next method for determining family health history through gene identification is called association analysis. Association analysis is a population-based method that tracks the occurrence of a particular allele, or set of alleles, by looking for an increase or decrease of affected individuals from a population. For example, a study by Ghosh & Fardo (2018) used association analysis to track the genetic inheritance of factors that may affect measure of triglycerides and high-density lipoprotein levels. In contrast, a study by Kunkle et al. (2016), utilized linkage analysis to track familial late-onset Alzheimer's disease of non-Hispanic white families. In efforts of attempting to determine disease risk within a certain family, linkage analysis would provide a higher level of detail of inheritance patterns and risk factors for inheriting that particular disease. However, association analysis may provide better insights when searching for external factors that contribute to the occurrence of a disease within a population, such as average temperature, air quality, or UV index.

These methods and considerations are useful topics for patient education. Clinicians may provide referrals to genetic counseling to investigate and mitigate risk factors associated with diseases like AD. Some of the known risk factors for AD include first degree relative that have been diagnosed with AD and advanced age.

Alzheimer's Disease Mutations

AD may have complex and unknown origination factors, ultimately, AD is a genetic disease of inheritance. There have been many advances in treatment and knowledge since the definition and discovery of AD, particularly in the discovery of genetic mutations. Giau, Pyun, Bagyinszky, An, & Kim (2018) discovered a genetic mutation possibly associated with the phenotype of AD. Patients were selected (Giau, 2018) based on neuropsychological tests and neuroimaging and then

genetic tests were performed using next-generation sequencing and found a pathogenic mutation in the presenilin 2 (PSEN2) gene associated with early-onset AD. In another study by Thordardottir et al. (2018), two brothers carrying a mutation H163Y of the PSEN1 gene for Early-onset AD were followed over the course of 22 years. The study (Thordardottir et al., 2018) found that the average age of symptom onset for PSEN1 H163Y is 51 ± 7 years according to previous studies. One of the brothers had a biomarker-verified reduction in penetrance in the mutation and was still symptom-free at the age of 65. This suggests that other genetic, epigenetic, and/or environmental factors modify the onset age (Thordardottir et al., 2018). These studies support the idea that AD is a complex inheritance disease and that mutations may occur not just from chance, but from external environmental factors like diet and behaviors.

Genetics and Policies

Nutrigenomics is the study of food and how they interact with genes (Ballke & Meisterernst, 2012). There is information from multiple scientific disciplines that contribute to the field of nutrigenomics such as molecular biology, pathology, bioinformatics, nutrition, ethics, and genetics. The science behind nutrigenomics is very new which leaves multiple unexplored methods of implementing this information into clinical practice.

There have been many industries already implementing claims supported by nutrigenomics like food and laboratory testing. However, the results and validity of certain claims and genetic testing has raised concerns about ethical issues. There is a need for legal consideration with diagnostic tools and products that develop from nutrigenomics. For example, the European Parliament has created provisions in law that protects consumers by stating that personal data must be processed fairly and lawfully and must be collected for specific, explicit, and legitimate purposes (EUR-Lex, 1995). However, this law can be bypassed by receiving explicit consent from an individual to process data that would reveal ethnic and health information.

Another important legal aspect that should be considered is the products that are created from nutrition and genetic information. The European Parliament places a prohibition of statements that may attribute to any foodstuff the property of preventing, treating or curing a human disease, or refer to such properties (EUR-Lex, 1995). This is important in protecting consumers from treating a particular illness with a product that is not based on factual evidence. However, producers of such content may apply for an exception review if their product has been shown and supported by scientific evidence.

Claims about genetic testing and food products will require intense scrutiny as the field of nutrigenomics develops. Policies should continue to reform to protect consumers health and privacy as producers influence laws that favor bypassing evidenced-based practices (Welch, Fusi, Louafi, & Siciliano, 2017). Consumers should be aware of the implications of forfeiting privacy to certain genetic information. Producers may initiate aggressive campaigns for products that make health claims supported by nutrigenomics and it is important for consumers to be well informed of the science behind the claims.

Nutritional Implications

Lipoproteins are soluble and insoluble complexes that contain lipids and proteins. The two types of lipoproteins that are responsible for transporting cholesterol, which is insoluble in blood, are high-density lipoproteins (HDLs) and low-density lipoproteins (LDLs). Cholesterol that is bound to LDL is associated with atherosclerosis caused by the accumulation of fats on the blood vessel walls (Waldman, Vogt, Crispin, Altenhofer, Riks, & Parhofer, 2017). Having high levels of LDLs is associated with heart disease and Alzheimer's Disease (AD). The increase in the amount of amyloid plaques seen in AD is associated with higher blood serum levels of LDL (Hui, Chen, & Geiger, 2012).

There are multiple risk factors that can influence blood serum levels of LDL. One non-modifiable risk factor that influences LDL is genetics. Mutations of the GPIHBP1 gene are associated with severe hypertriglyceridemia (Buonuomo et al., 2015). Another disease of lipoproteins affected by genetics is Tangier disease. Tangier disease is characterized by abnormally low levels of lipoproteins and is associated with mutations in the ABCA1 gene (Negi et al., 2013).

The development of AD can be affected by the modifiable risk factor of nutrition and diet. Regularly consuming foods that are high in cholesterol decreases cellular sensitivity to utilize cholesterol which causes a surplus of circulating LDL (Waldman, Vogt, Crispin, Altenhofer, Riks, & Parhofer, 2017). Foods with high cholesterol include eggs, fried foods, processed meats, and cheese (Morlan, Roux, & Wing, 2006). These types of foods have a negative impact for the development of AD.

Engaging in regular exercise and eating a balanced diet have positive implications for the development of AD. The American Heart Association (2018) recommends engaging in at least 150 minutes per week of moderate-intensity aerobic activity or 75 minutes per week of vigorous aerobic activity, or a combination of both, preferably spread throughout the week. In conjunction with exercise,

the American Heart Association (2018) recommends that you consume no more than 300 mg of dietary cholesterol per day, 200 mg if you are at risk for developing heart disease.

Treatment Planning & Nutrition

In efforts to reduce the development of amyloid plaques seen in AD, there are a few nutritional recommendations for use with treatment planning in prevention, screening, diagnostics, prognostics, and treatment evaluation. Primary prevention interventions include education about what types of foods are high in cholesterol. Patients who are at risk for developing AD should be aware of what foods are associated with high cholesterol so that diet changes can be made to reduce risk. Nutritional information pamphlets provide a great visualization of foods to avoid and recommendations to consume instead.

A lipid profile blood test is an excellent screening tool that gives clinicians great insight to the dietary history of a patient. LDL, HDL, Triglycerides, and total cholesterol are all components of the lipid profile. Dietary consumptions can be compared and contrasted against LDL and HDL values. A diet high in cholesterol would normally result in a lipid profile containing high triglycerides, higher LDL, and low HDL (Mohammad et al., 2018). If a patients diet does not match the expected results from lipid profile, genetic mutations may be the result of hypercholesterolaemia and hypocholesterolaemia (Waldman, Vogt, Crispin, Altenhofer, Riks, & Parhofer, 2017).

Although AD is a progressive neurodegenerative disease, there are nutritional changes that patients can make to improve prognosis by delaying the progression of AD. Patients can reduce the intake of processed cheeses, such as American cheese, mozzarella sticks, and aerosolized cheese products to minimize the formation of amyloid plaques (Gu Y et al., 2010). Microwave popcorn contains diazetyl, a chemical that may increase amyloid plaques in the brain (Gu Y et al., 2010). High-intensity exercise and lower LDL levels are significantly associated (Albarrati, Alghamdi, Nazer, Alkorashy, Alshowier, & Gale, 2018). Non-pharmacological interventions include changes to diet and exercise. Pharmacological interventions to lower LDL levels include the use of statin medications like atorvastatin and lovastatin (Lalić et al., 2018).

Ethical Considerations

A study by Konigorski, Khorasani, & Lipper (2018) focused on providing personalized treatment for Alzheimers Disease (AD) by identifying predictive markers of the disease. Magnetic resonance imaging (MRI) details the status of AD.

However, definitive diagnosis is made post-mortem via brain tissue biopsy. The study (Konigorski, Khorasani, & Lipper, 2018) combined MRI with genomic sequencing to identify rare genetic markers associated with quantitative traits predicted from convolutional neural networks (CNNs), a type of artificial intelligence or machine learning. The results (Konigorski, Khorasani, & Lipper, 2018) indicate that CNNs provide a fast, scalable, and precise tool to derive quantitative AD traits.

In the United States, healthcare services are paid for by private and public entities. New healthcare laws like the Affordable Healthcare Act include coverage for preventative health services. However, some diseases, like AD, are not curable and cannot be prevented. Although risk factors for AD can be minimized through medicine and behavior changes, the disease progression is inevitable. Considering the new type of technology presented in the Konigorski study (2018), public and private healthcare insurances must not let coverage for healthcare services depend on genetic predisposition to diseases like AD. There needs to be an increase in conversation about the possibility of genetic discrimination. For example, denying insurance eligibility due to the identification of AD genetic markers.

Genomic Impact on Financial Resources

Genetics and genomics are essential for shifting healthcare from reactive to preventative focused treatment. Although many chronic illnesses may not be curable, symptoms can be minimized which will in turn improve the quality of life for the patient and reduce overall health care expenditures. The implementation of this concept is supported by genetics and genomics.

Alzheimer's Disease (AD) is a chronic illness characterized by neuronal death as evidenced by deterioration of cognitive functioning. There are two subdivisions of AD, early and late-onset. Early-onset AD (EOAD) is identified by screening for mutations in the amyloid protein precursor (APP), presenilin-1 (PSEN1), and presenilin-2 (PSEN2) genes (Lanoiselee et al., 2017). Risk factors for Late-onset AD (LOAD) are identified through a family pedigree, advanced age, and history of traumatic brain injury. However, genome-wide association studies have identified single-nucleotide polymorphisms in multiple genes which are associated with LOAD (Misra, Chakrabarti, & Gambhir, 2018).

The burden of financial resources is minimized when care is provided and planned with a preventative focus, rather than reactive. The Affordable Care Act, Mental Health Parity, and Addiction Equity Act greatly expand the coverage of

behavioral health care services for Americans (Beonio, Glied, & Frank, 2014). These newer laws allow mental health services to be on parity with general medical benefits. Many more people will now have access to preventative services like mental status exams that are capable of identifying illnesses at an early stage. An economic evaluation (Knapp, Lemmi, & Romeo, 2013) of dementia care, like AD, that focused on prevention, care, and treatment strategies revealed that early pharmacotherapy, cognitive stimulation therapy, and psychosocial interventions for care givers are more cost effective than care as usual, or reactive care.

Evidence-Based Practice

There is no testing method for a genetic predisposition or pharmacotherapy treatment to late-onset Alzheimer's Disease (LOAD). However, recent genomic research and evidence support the possibility of identifying LOAD predisposition. A genome-wide association study has demonstrated that kidney and brain expressed protein (KIBRA) play an important role in memory impairment, a typical clinical presentation of LOAD (Ling, Huan, Zhang, Wei, & Cheng, 2018). KIBRA polymorphism has a significant association with LOAD risk, especially among Asians, Caucasians, and the advanced age population (Ling, Huan, Zhang, Wei, & Cheng, 2018). Another evidence-based study (Elmegeed, Ahmed, Hashash, Abd-Elhalim, & El-kady, 2015) focused on a synthesis of a novel steroidal curcumin derivative as an anti-Alzheimer's disease candidate. The various tested compounds demonstrated anti-oxidant, anti-cholinesterase, and anti-apoptotic activity that have the potential to manage LOAD much better than single monofunctional targeted drugs (Elmegeed, Ahmed, Hashash, Abd-Elhalim, & El-kady, 2015).

The ability to identify and treat LOAD will have a significant impact on the quality of life for patients and decrease the need for financial resources to manage the population with either type of AD. Early identification through genomic research and testing methods will allow treatment of AD to focus on preventative care, rather than reactive. Primary care providers should implement new practices of genetic screening and pharmacotherapy disease management. A study by Radhika et al., (2018), detailed that there is a higher comorbidity burden and higher healthcare-related costs for patients 6 months prior to a diagnosis of AD.

However, Canevelli, Bruno, Vanacore, De Lena, & Cesari (2016), raise an important issue about implementing evidence-based practice for AD. A 10-year history of random controlled trials for AD was reviewed and it was determined that there was a lack of detailed descriptions of the patient population and

medical comorbidities that make it difficult to implement new practices based on the results.

Nursing Care for Alzheimer's Disease

Care planning for AD focuses on the concepts of primary, secondary, and tertiary prevention interventions. Primary prevention interventions include the use and dissemination of informative brochures and public announcements. Brochures provide information about AD that includes disease definition, signs and symptoms, treatment options, and community clinical resources. Depending on financial resources, public announcements include volunteer supported booths at free community health events, social media postings, and national media coverage. A study by Norton, Matthews, Barnes, Yaffe, and Brayne (2014), indicates that general education about various risk factors could reduce the prevalence of AD in 2050 by 8.3% worldwide.

Secondary prevention interventions include the utilization of AD screening tools like the General Practitioner Assessment of Cognition (GPCOG). Tools like the GPCOG can help identify early stages

of AD. According to the Alzheimer's Association (2018), early detection of AD can benefit patients by increasing chance of benefitting from treatment, decreasing anxiety, increase chance of trial participation, and an opportunity to participate in decisions about care, transportation, living options, financial and legal matters. Patients who may have financial concerns related to screenings can utilize Medicare benefits as part of the Annual Wellness Visit (Cognitive Assessment, 2018).

Tertiary prevention interventions include community support groups and regular provider appointments. The National Alliance on Mental Illness provides users with a national database on support groups for various mental health illnesses. For example, a clinician can search for AD support groups by patient zip code and provide the patient with group topics and meeting times. Patient who regularly attend support groups are less likely to relapse and cope with stress with a healthy method (Mohr, Burns, Schuller, Clark, & Klinkman 2014). A study by Vickers et al., (2013), determined that the integration of mental health resources in a primary care setting leads to increased provider satisfaction and patient access.

Test

- In a capitalistic society, where do most consumers of medical services receive their information about health and available treatments?
 - Primary Care Providers
 - University
 - Media Advertisements
 - Word-of Mouth
- In efforts to increase treatment plan adherence, a clinician would include which of the following in discharge planning?
 - Family Members
 - Disorder Pamphlets
 - Quality of Care Surveys
 - Medication Education
- A clinician would demonstrate an understanding of Alzheimer's Disease when they state the following
 - "Early intense treatment will assist in resolving symptoms."
 - "Medication will restore the clients ability to function."
 - "The disease is inevitable for all aging clients."
 - "Genetic testing may only predict Early-Onset AD"
- A definitive diagnosis of Alzheimer's Disease is made by which of the following?
 - Complete Blood Count
 - Post-mortem tissue examination
 - Complete Metabolic Panel
 - Magnetic Resonance Imaging
- Alzheimer's Disease is subdivided into which two types?
 - Early and late onset
 - Primary and secondary
 - Genetic and non-genetic
 - External and internal
- Policies regarding nutrigenomics protects consumers by which of the following?
 - Predicting how genes interact with food
 - Providing intense scrutiny of food labels
 - Mandating food manufacturers to submit research claims
 - Supporting consumer privacy
- The quality of life for client's diagnosed with Alzheimer's Disease are associated with what type of prevention intervention?
 - Primary
 - Secondary

- c. Tertiary
- d. Quaternary
8. How would A clinician plan for primary prevention interventions?
- a. Create educational brochures
- b. Provide family support groups
- c. Provide genetic counseling
- d. Assess family history
9. Which of the following is correct about the cause of Alzheimer's Disease?
- a. Nutrigenomics
- b. Complex multifactorial genetics
- c. High Density Lipoproteins
- d. Low Density Lipoproteins
10. The Affordable Healthcare Act supports chronic illnesses like Alzheimer's Disease by...
- a. Increasing funds for skilled nursing facilities
- b. Providing researchers with funds for clinical trials
- c. Increasing funds for preventative services
- d. Decreasing the cost of specialized healthcare visits

Answers

- | | |
|------|-------|
| 1. C | 6. B |
| 2. A | 7. B |
| 3. D | 8. A |
| 4. B | 9. B |
| 5. A | 10. C |

References

- Aarnio, P., Kulmala, T., & Olsson, P. (2018). Husband's role in handling pregnancy complications in Mangochi District, Malawi: A call for increased focus on community level male involvement. *Sexual & Reproductive Healthcare, 16*, 61–66. <https://doi-org.lopes.idm.oclc.org/10.1016/j.srhc.2018.02.005>
- Albarrati, A. M., Alghamdi, M. S. M., Nazer, R. I., Alkorashy, M. M., Alshowier, N., & Gale, N. (2018). Effectiveness of Low to Moderate Physical Exercise Training on the Level of Low-Density Lipoproteins: A Systematic Review. *BioMed Research International, 1*–16. <https://doi-org.lopes.idm.oclc.org/10.1155/2018/5982980>
- Anamika Misra, Sankha Shubhra Chakrabarti, & Indrajeet Singh Gambhir. (2018). New genetic players in late-onset Alzheimer's disease: Findings of genome-wide association studies. *Indian Journal of Medical Research, Vol 148, Iss 2, Pp 135-144 (2018)*, (2), 135. https://doi-org.lopes.idm.oclc.org/10.4103/ijmr.IJMRpass:1473_17
- American College of Physicians, (2012), Patient-Centered Medical Home, Retrieved November 17, 2018 from https://web.archive.org/web/20120114063028/http://www.acponline.org/running_practice/pcmh/understanding/index.html
- American Heart Association, (2018), American Heart Association Recommendations for Physical Activity in Adults and Kids, Retrieved on 30 November, 2018 from <http://www.heart.org/en/healthy-living/fitness/fitness-basics/aha-recs-for-physical-activity-in-adults>
- Archontaki, M., E. K. Symvoulakis, J. K. Hajjioannou, et al. 2009. Increased frequency of rhinitis medicamentosa due to media advertising for nasal topical decongestants. *B-ENT 5*(3): 159–162.
- Ballard C, Gauthier S, Corbett A, Brayne C, Aarsland D, Jones E. (2014) Alzheimer's disease.; 377:1019–1031
- Ballke, C., & Meisterernst, A. (2012). Nutrigenomics -- A New Trend from a Legal Perspective. *European Food & Feed Law Review, 7*(1), 14–21. Retrieved from <https://lopes.idm.oclc.org/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=a9h&AN=72444568&site=ehost-live&scope=site>
- Beronio, K., Glied, S., & Frank, R. (2014). How the Affordable Care Act and Mental Health Parity and Addiction Equity Act greatly expand coverage of behavioral health care. *The journal of behavioral health services & research, 41*(4), 410-428.
- Buonuomo, P. S., Bartuli, A., Rabacchi, C., Bertolini, S., & Calandra, S. (2015). A 3-day-old neonate with severe hypertriglyceridemia from novel mutations of the GPIIb/IIIa gene. *Journal of Clinical Lipidology, 9*(2), 265–270. <https://doi-org.lopes.idm.oclc.org/10.1016/j.jacl.2014.10.003>
- Canevelli, M., Bruno, G., Vanacore, N., De Lena, C., & Cesari, M. (2016). Are we really tackling the “evidence-based medicine issue” in Alzheimer's disease? *European Journal of Internal Medicine, 29*. <https://doi-org.lopes.idm.oclc.org/10.1016/j.ejim.2016.07.009>

- Castillo-Carranza DL, Guerrero-Muñoz MJ, Kaye R., 2014 Immunotherapy for the treatment of Alzheimer's disease: amyloid- β or tau, which is the right target? *ImmunoTargets and Therapy*; 3:19–28
- Cognitive Assessment. (2018). Retrieved December 28, 2018, from <https://www.alz.org/professionals/healthcare-professionals/cognitive-assessment>
- Costa, P. T., Whitfield, J. R., & Stewart, D. (1989). *Alzheimer's disease: Abstracts of the psychological and behavioral literature*. Washington, DC: American Psychological Association.
- Elmegeed, G. A., Ahmed, H. H., Hashash, M. A., Abd-Elhalim, M. M., & El-kady, D. S. (2015). Synthesis of novel steroidal curcumin derivatives as anti-Alzheimer's disease candidates: Evidences-based on in vivo study. *Steroids*, 101, 78–89. <https://doi-org.lop.es.idm.oclc.org/10.1016/j.steroids.2015.06.003>
- EUR-Lex Access to European Union Law, Document 31995L0046, 24 October, 1995, retrieved on 1 December, 2018 from <https://eur-lex.europa.eu/legal-content/EN/TXT/?uri=CELEX%3A31995L0046>
- Fowler NR, Perkins AJ, Gao S, Sachs GA, Uebelhor AK, & Boustani MA. (2018). Patient characteristics associated with screening positive for Alzheimer's disease and related dementia. *Clinical Interventions in Aging, Vol Volume 13, Pp 1779-1785 (2018)*, 1779. Retrieved from <https://lop.es.idm.oclc.org/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=edsdoj&AN=edsdoj.16bd70e9f8b34e42b89b9c9ca90d19ee&site=eds-live&scope=site>
- Giau VV, Pyun JM, Bagyinszky E, An SSA, & Kim SY. (2018). A pathogenic PSEN2 p.His169Asn mutation associated with early-onset Alzheimer's disease. *Clinical Interventions in Aging, Vol Volume 13, Pp 1321-1329 (2018)*, 1321. Retrieved from <https://lop.es.idm.oclc.org/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=edsdoj&AN=edsdoj.5a1fa85e0da44dd939b077609e10873&site=eds-live&scope=site>
- Gu, Y., Nieves, J. W., Stern, Y., Luchsinger, J. A., & Scarmeas, N. (2010). Food combination and Alzheimer disease risk: a protective diet. *Archives of neurology*, 67(6), 699-706.
- Hui, L., Chen, X., & Geiger, J. D. (2012). Endolysosome involvement in LDL cholesterol-induced Alzheimer's disease-like pathology in primary cultured neurons. *Life Sciences*, 91, 1159–1168. <https://doi-org.lop.es.idm.oclc.org/10.1016/j.lfs.2012.04.039>
- Knapp, M., Iemmi, V., & Romeo, R. (2013). Dementia care costs and outcomes: a systematic review. *International journal of geriatric psychiatry*, 28(6), 551-561.
- Konigorski, S., Khorasani, S., & Lippert, C. (2018). Integrating omics and MRI data with kernel-based tests and CNNs to identify rare genetic markers for Alzheimer's disease. Retrieved from <https://lop.es.idm.oclc.org/login?url=https://search.ebscohost.com/login.aspx?direct=true&db=edsarx&AN=edsarx.1812.00448&site=eds-live&scope=site>
- Kunkle, B. W., Jaworski, J., Barral, S., Vardarajan, B., Beecham, G. W., Martin, E. R., ... Pericak-Vance, M. A. (2016). Genome-wide linkage analyses of non-Hispanic white families identify novel loci for familial late-onset Alzheimer's disease. *Alzheimer's & Dementia: The Journal of the Alzheimer's Association*, 12(1), 2–10. <https://doi-org.lop.es.idm.oclc.org/10.1016/j.jalz.2015.05.020>
- Lalli, M. A., Cox, H. C., Arcila, M. L., Cadavid, L., Moreno, S., Garcia, G., ... Lopera, F. (2014). Featured Article: Origin of the PSEN1 E280A mutation causing early-onset Alzheimer's disease. *Alzheimer's & Dementia: The Journal of the Alzheimer's Association*, 10(Supplement), S277–S283.e10. <https://doi-org.lop.es.idm.oclc.org/10.1016/j.jalz.2013.09.005>
- Lalić, K., Rajković, N., Popović, L., Lukač, S. S., Stošić, L., Rasulić, I., & Lalić, N. M. (2018). The effects of 3-year statin therapy and the achievement of LDL cholesterol target values in familial hypercholesterolemia patients: An experience from Serbia. *Atherosclerosis*, 277, 298–303. <https://doi-org.lop.es.idm.oclc.org/10.1016/j.atherosclerosis.2018.08.014>
- Lanoiselee, H.-M., Nicolas, G., Wallon, D., Rovelet-Lecrux, A., Lacour, M., Rousseau, S., ... Champion, D. (2017). APP, PSEN1, and PSEN2 mutations in early-onset Alzheimer disease: A genetic screening study of familial and sporadic cases. *PLoS Medicine*, (3). <https://doi-org.lop.es.idm.oclc.org/10.1371/journal.pmed.1002270>
- Ling, J., Huang, Y., Zhang, L., Wei, D., & Cheng, W. (2018). Research article: Association of KIBRA polymorphism with risk of Alzheimer's disease: Evidence based on 20 case-control studies. *Neuroscience Letters*, 662, 77–83. <https://doi-org.lop.es.idm.oclc.org/10.1016/j.neulet.2017.08.057>
- Mohammad Vahid Jorat, Reza Tabrizi, Naghme Mirhosseini, Kamran B. Lankarani, Maryam Akbari, Seyed Taghi Heydari, ... Zatollah Asemi. (2018). The effects of coenzyme Q10 supplementation on lipid profiles among patients with coronary artery disease: a systematic review and meta-analysis of randomized controlled trials. *Lipids in Health and Disease, Vol 17, Iss 1, Pp 1-9 (2018)*, (1), 1. <https://doi->

- [org.lobes.idm.oclc.org/10.1186/s12944-018-0876-4](https://doi.org/10.1186/s12944-018-0876-4)
- Mohr, D. C., Burns, M. N., Schueller, S. M., Clarke, G., & Klinkman, M. (2013). Behavioral intervention technologies: evidence review and recommendations for future research in mental health. *General hospital psychiatry*, 35(4), 332-338.
- Morland, K., Roux, A. V. D., & Wing, S. (2006). Supermarkets, other food stores, and obesity: the atherosclerosis risk in communities study. *American journal of preventive medicine*, 30(4), 333-339.
- Negi, S. I., Brautbar, A., Virani, S. S., Anand, A., Polisecki, E., Asztalos, B. F., ... Jones, P. H. (2013). A novel mutation in the ABCA1 gene causing an atypical phenotype of Tangier disease. *Journal of Clinical Lipidology*, 7(1), 82. Retrieved from <https://lobes.idm.oclc.org/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=edo&AN=85155634&site=eds-live&scope=site>
- Norton, S., Matthews, F. E., Barnes, D. E., Yaffe, K., & Brayne, C. (2014). Articles: Potential for primary prevention of Alzheimer's disease: an analysis of population-based data. *Lancet Neurology*, 13, 788-794. [https://doi.org/10.1016/S1474-4422\(14\)70136-X](https://doi.org/10.1016/S1474-4422(14)70136-X)
- Office of the Commissioner. (2018, March 28). What We Do. Retrieved November 14, 2018, from <https://www.fda.gov/aboutfda/whatwedo/default.htm>
- Radhika Nair, Virginia S. Haynes, Mir Siadaty, Nick C. Patel, Adam S. Fleisher, Derek Van Amerongen, ... Daniel E. Ball. (2018). Retrospective assessment of patient characteristics and healthcare costs prior to a diagnosis of Alzheimer's disease in an administrative claims database. *BMC Geriatrics*, Vol 18, Iss 1, Pp 1-12 (2018), (1), 1. <https://doi.org/10.1186/s12877-018-0920-2>
- Rastogi, N., Tyagi, N., Singh, O., Hemanth Kumar, B. S., Singh, U. P., Ghosh, K., & Roy, R. (2017). Mn(II) based T1 and T2 potential MRI contrast agent appended with tryptamine: Recognition moiety for A β -plaques. *Journal of Inorganic Biochemistry*, 177, 76-81. <https://doi.org/10.1016/j.jinorgbio.2017.08.007>
- Reduced penetrance of the PSEN1 H163Y autosomal dominant Alzheimer mutation: a 22-year follow-up study. (2018). *Alzheimer's Research & Therapy*. <https://doi.org/10.1186/s13195-018-0374-y>
- Robert Nussbaum, Roderick R. McInnes, Huntington F. Willard, & Ada Hamosh (2007) Genetics in Medicine, Retrieved from <https://viewer.gcu.edu/PGBPMV>
- Roberts, J. S., & Uhlmann, W. R. (2013). Genetic susceptibility testing for neurodegenerative diseases: Ethical and practice issues. *Progress in Neurobiology*, 110, 89-101. <https://doi.org/10.1016/j.pneurobio.2013.02.005>
- Saunders, C. C. (2017). Balancing Innovation and Regulation: Why the Fda Should Adopt a More Dynamic Risk-Based System for Wearables. *Jurimetrics: The Journal of Law, Science & Technology*, 58(1), 83-103. Retrieved from <https://lobes.idm.oclc.org/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=a9h&AN=129653732&site=eds-live&scope=site>
- Saurabh Ghosh, & David W. Fardo. (2018). Association analyses of repeated measures on triglyceride and high-density lipoprotein levels: insights from GAW20. *BMC Genetics*, Vol 19, Iss S1, Pp 127-131 (2018), (S1), 127. <https://doi.org/10.1186/s12863-018-0651-6>
- Schoonenboom, N. S. M., Kalisvaart, K., & Akarriou, M. (2017). Use of Pet Scan in Early Diagnosis of Alzheimer's Disease in a Secondary Care Memory Clinic. *Alzheimer's & Dementia: The Journal of the Alzheimer's Association*, (7). <https://doi.org/10.1016/j.jalz.2017.06.265>
- Smalley, L. P., Kenney, M. K., Denboba, D., & Strickland, B. (2014). Family perceptions of shared decision-making with health care providers: Results of the National Survey of Children with Special Health Care Needs, 2009-2010. *Maternal and Child Health Journal*, 18(6), 1316-1327. <https://doi.org/10.1007/s10995-013-1365-z>
- Takahashi, H., Mercken, M., Honda, T., Saito, Y., Murayama, M., Song, S., & Takashima, A. (1999). Impaired proteolytic processing of Presenilin-1 in chromosome 14-linked familial Alzheimer's disease patient lymphocytes. *Neuroscience Letters*, 260, 121-124. [https://doi.org/10.1016/S0304-3940\(98\)00966-5](https://doi.org/10.1016/S0304-3940(98)00966-5)
- US Food & Drug. (2018, September). Code of Federal Regulations Title 21. Retrieved November 14, 2018 from <https://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfrcfr/cfrsearch.cfm>
- Waldmann, E., Vogt, A., Crispin, A., Altenhofer, J., Rijs, I., & Parhofer, K. G. (2017). Effect of mipomersen on LDL-cholesterol in patients with severe LDL-hypercholesterolaemia and atherosclerosis treated by lipoprotein apheresis (The MICA-Study). *Atherosclerosis*, 259, 20-25. <https://doi.org/10.1016/j.atherosclerosis.2017.02.019>

Vickers, K. S., Ridgeway, J. L., Hathaway, J. C., Egginton, J. S., Kaderlik, A. B., & Katzelnick, D. J. (2013). Integration of mental health resources in a primary care setting leads to increased provider satisfaction and patient access. *General hospital psychiatry*, 35(5), 461-467.

Welch, E. W., Fusi, F., Louafi, S., & Siciliano, M. (2017). Genetic resource policies in international collaborative research for food and agriculture: A study of USAID-funded innovation labs. *Global Food Security*. <https://doi-org.lopes.idm.oclc.org/10.1016/j.gfs.2017.04.004>

Wiseman, F. K., Al-Janabi, T., Hardy, J., Karmiloff-Smith, A., Nizetic, D., Tybulewicz, V. L., ... & Strydom, A. (2015). A genetic cause of Alzheimer disease: mechanistic insights from Down syndrome. *Nature Reviews Neuroscience*, 16(9), 564.

Zivotofsky, A. Z., & Zivotofsky, N. T. S. (2014). Capitalism Works for Health Care Too. *American Journal of Bioethics*, 14(3), 56–58. <https://doi-org.lopes.idm.oclc.org/10.1080/15265161.2013.879956>