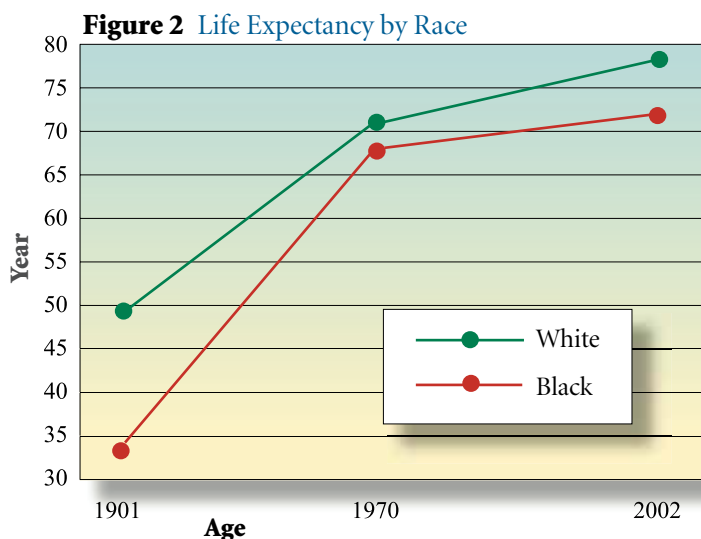
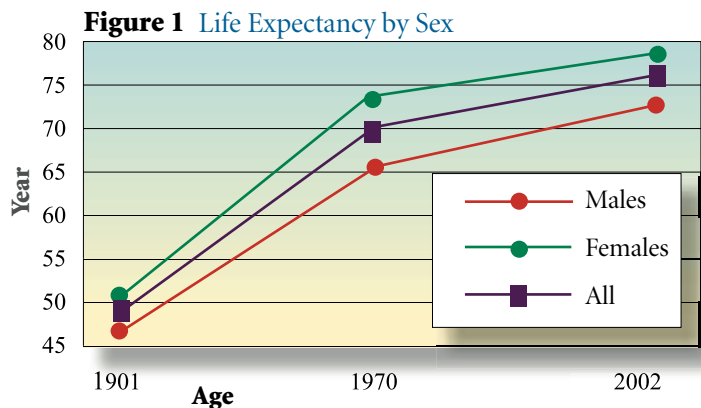


## Aging and Brain Injury

We are all getting older – and I don't mean to state the obvious. Rather, I am speaking about Life Expectancy, a gauge of overall health, which has increased linearly in the U.S. over the last century. According to the National Center for Health Statistics, the average lifespan was 49.3 years in 1901, 70.8 years in 1970 and 77.3 years in 2002. Moreover, rates differ by sex (Figure 1) and race (Figure 2; Arias, 2004). By the year 2030, it is expected that one in four Americans (70 million people) will be over the age of 65 (Lange and Paul, 2004).

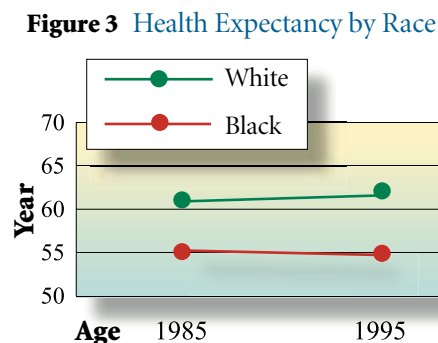
As we live longer, the consequences of aging and corresponding health problems have become a topic of great interest. As life expectancy steadily rises, we must also watch another population health index—Health Expectancy. Figure 3 illustrates rates of health expectancy by race (Wagner et al., 2001), and highlights

*continued*



**Life expectancy** is the average number of years of life remaining to a person at a particular age. It is based on a given set of age-specific death rates, generally the mortality conditions existing in the period mentioned. Life expectancy may be determined by race, sex, or other characteristics using age-specific death rates for the population with that characteristic (Center for Health Statistics).

**Health Expectancy** is defined as the number of years an average person is expected to live in good health without activity limitations due to impairments or health problems typically of a chronic nature – or disability-free life expectancy (Wagner et al., 2001).



## Aging and Brain Injury *continued*

that, while we are living longer, we may not be living healthier. Looking at life expectancy and health expectancy together, on average, we may live 76.7 years, but nearly 17.7 of those years are not necessarily spent in good health.

How the process of aging affects brain health is becoming more and more relevant and raises the following questions: Does aging in general impact brain functioning? Does age relate differentially to the incidence and prevalence of acquired brain injury (ABI)? For those affected by brain injury, the question may be: Does having a brain injury impact or accelerate the aging process? The purpose of this article is to take a rudimentary examination of these questions and determine what we do and don't know about aging and brain injury.

### In general, does aging impact brain functioning?

As life expectancy has increased and technological advances have improved our ability to study the brain, researchers have begun to learn more about how the brain changes over time. The advances in technology over the last few decades include: Computed tomography scans (CT), positive emission tomography scans (PET) and magnetic resonance (MR) imaging scans, to name a few. What these technological advances provide is the ability to see and quantify changes to the brain. This allows scientists and medical professionals to: (1) Determine the extent of damage to brain structures for an individual given some type of neurological event, (2) Examine brain structures over time to see if changes occur with age, and (3) Examine brain structure in healthy versus non-healthy individuals to determine disease etiology

### What is considered 'normal' in terms of aging and brain health?

First, we know that select areas of cognitive function are affected by aging. Some researchers have theorized that this decline may start as early as age 30 (Victoroff, 2002). Pickholtz and Malamut (2002) summarized studies, which showed areas of cognition affected by the normal aging process. Some of these studies found reductions in:

1. Short term memory - retention of information shortly after it is presented
2. Long-term episodic memory - acquisition of memory for specific personal events where declines in performance may be related to

problems with memory acquisition rather than memory retrieval, and

3. Processing speed, which includes changes in both motoric response, and mental processing speed.

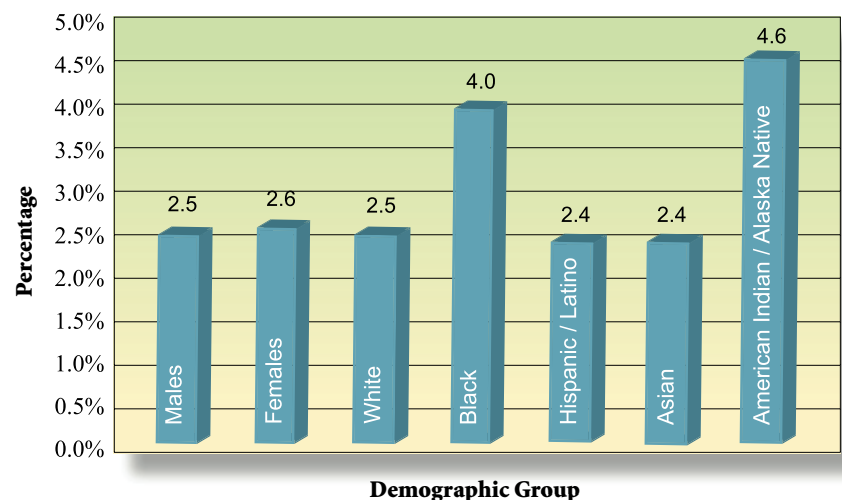
Second, we know that physical changes to the brain relate to the normal process of aging. For example, changes in brain structure as people age have been found to differ by sex and by area of the brain. Pickholtz and Malamut (2002) noted that changes in ventricle volume occur earlier in men (5th decade) than women (6th decade), and females show less cortical loss than males. In an article in USC Health, Guttman (2001) noted the following physical changes to the brain as we age:

1. Decreases in brain weight & volume by 5-10% between the ages of 20 and 90
2. Changes to the grooves in the brain
3. Increases in neurofibrillary tangles
4. Formation of senile plaques.

Moreover, decreases in white and gray matter and increases in ventricle or cerebrospinal fluid (CSF) volumes associated with aging have been documented. Bigler et al. (2002), using quantitative MR imaging techniques, measured volumes of brain matter and CSF in areas within the temporal lobe. The subjects were healthy individuals spanning 51/2 decades across a series of age groups (16-25, 26-35, 36-45, 46-55 and 56-72 years). When comparing the first age group (16-25) to the fifth (56-72), they found that overall temporal lobe volumes were reduced by 13%, reaching statistical significance. This translates to a modest

*continued*

**Figure 4** Stroke Prevalence 2002



## Aging and Brain Injury *continued*

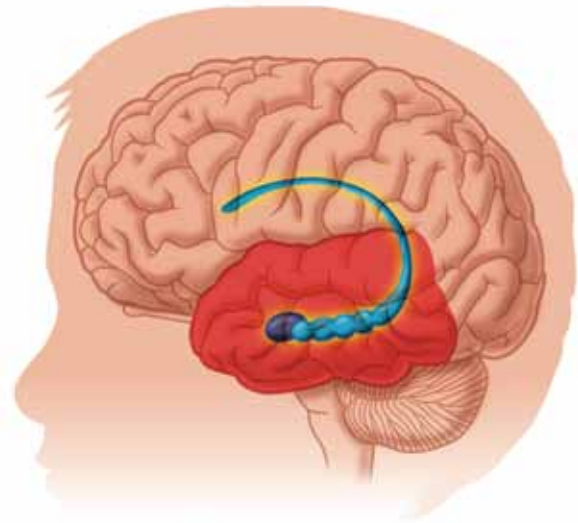
reduction in white matter volume of .26% per year. In specific ventricular areas, particularly subarachnoid sulcal CSF volumes, they found a doubling of the CSF, and thus an increase in ventricular size within the temporal lobe.

We know that cognition and some areas of brain structure can be negatively affected by age. But can we make the leap that changes in brain structure cause cognitive dysfunction? It seems that more and more evidence points to an unequivocal yes. Bigler et al. (2002) examined a sample of individuals without any type of neurological condition and a sample of individuals with TBI. They found atrophy in the hippocampus, a structure significant to memory encoding and retrieval, and increases in CSF in the temporal lobe. In essence, they found that changes in CSF volumes related to decreases in white matter and that these physical changes correlated to reductions in memory functions as measured in standards testing.

Additional evidence to support that affirmative answer relates to brain changes noted early—specifically in regards to neurofibrillary tangles and senile plaques. In essence, neurofibrillary tangles and senile plaques are neurodegenerative changes (Alzheimer's Association). They are brought about by proteins that, when they errantly fold, are responsible for their formation. For example, when beta amyloid folds, senile plaques, can form which harm neurons. Another protein by the name of tau can also mis-fold, producing tangles. When tangles combine with senile plaques the end result is neuronal death. And neuronal death means changes to our brains and changes in our functioning, like loss of memory, or movement disorders. It was not too long ago that plaques and tangles were considered the cause of Alzheimer's disease (AD). But recent research has demonstrated that inflammatory proteins, rather than plaques and tangles per se, may be implicated in AD (Guttman, 2001).

While researchers are making progress in understanding the factors that cause neurodegenerative diseases like AD, that is little comfort when one considers that today there are more than four million people diagnosed with AD (Victoroff, 2002). As scientists make greater strides in understanding neuro-degenerative processes and look to find cures for diseases like Parkinson's and Alzheimer's, it is important to note that what we view as "disease", and thus potentially curable afflictions, may well be far more complicated than that.

In his book titled "Saving Your Brain," Dr. Jeff Victoroff makes a compelling argument against the notion of neuro-degeneration as disease, but rather a normal consequence of aging. Because



**Hppocampus Diagram**

we continue to push the envelope on longevity, his thesis is that neuro-degeneration is going to happen if we live long enough. So, at this point as the reader you are probably thinking, "great, we're all doomed!" But, the second notion forwarded by Victoroff is that there are protective steps we can take now to delay the onset. While Victoroff covers numerous aspects of brain health, I will mention a few to offer perspective on the subject.

When looking at the two most common types of dementia, neurodegenerative (e.g., AD or Parkinson's), and vascular dementia (e.g., stroke), Victoroff pointed to many factors, which can either be causally related to the dementia or neuro-protective, thus reducing the risk of dementia. Some of these factors are decided by our genetic makeup but many can be considered lifestyle factors, providing us some ability to impact and hence reduce the risks. For example, Victoroff (2002) cited a study looking at risk factors in AD, which found the following: Psychosocial inactivity, physical inactivity, head injury, loss of teeth and low education. Likewise, there are many factors that contribute to stroke such as high levels of low-density lipoproteins (or LDL-Cholesterol), and hypertension, which has its own set of risk factors like obesity and diabetes. When one looks at these risk factors, it is apparent that many of them are of our own doing. So the bad news is that we are all at risk and the good news is that there are steps we can take to reduce that risk.

The answer to the question: Does aging in general impact brain functioning? The answer is yes. However, that answer comes with

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## Aging and Brain Injury *continued*

the understanding that the impact is variable, and to some degree, under our volitional control.

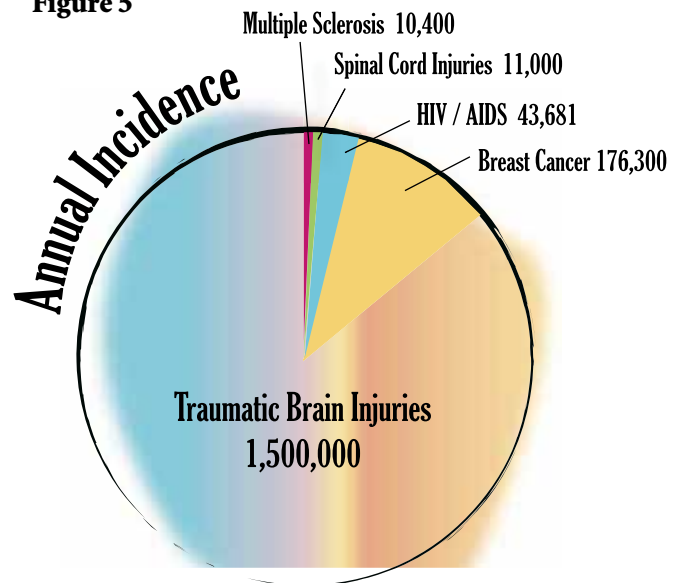
### Does age relate differentially to the incidence and prevalence of acquired brain injury (ABI)?

In the United States, every 21 seconds someone incurs a traumatic brain injury (TBI), and 5.3 million people are disabled due to traumatic brain injury (Centers for Disease Control – TBI Fact Sheet). Every 45 seconds someone suffers a stroke, and 4.7 million people are disabled secondary to stroke (American Heart Association). Thus, 10 million people are disabled secondary to these two types of acquired brain injury. In some categories of morbidity, persons over 65 are disproportionately at risk for, and acquire, a brain injury. For persons 65 years or older, falls are the leading cause of TBI related hospitalizations (CDC, Morbidity and Mortality Weekly Report, April 4, 2003), followed by motor vehicle crashes. In terms of deaths related to TBI, falls are the leading cause of death for women over 75 years of age and for men over 85 years of age (AACBIS Training Manual, Edition 3). Persons over 75 years of age constitute the second highest group in terms of risk for TBI (CDC – TBI Fact Sheet), and age over 75 is one of the highest risk factors for stroke (American Heart Association). For adults over 55, the lifetime risk of stroke is greater than one in six. Like TBI, there are differential rates for men and women, with women having higher risk than males. Figure 4 provides these rates as well as prevalence by race. Lange and Paul (2004) noted that in the 1990's stroke prevalence increased by almost 20%, and that life expectancy is reduced by as many as 12 years post-stroke. Thus, to answer the question, it is fair to state that age is a substantial factor in both the risk of and incidence of acquired brain injury.

### Does having a brain injury impact or accelerate the aging process?

This brings us to our final question - perhaps the hardest yet to answer. It is difficult to answer because the research findings on this topic have been inconclusive. Over the course of time, there have been numerous theories and anecdotal evidence to suggest that brain injury accelerates aging. But for all the anecdotal evidence, there have also been many research studies that have shed light on whether TBI is a factor in accelerating aging. For the purposes of this article, I will discuss two main areas, and in doing so will stoke the fires of the age-old debate of nature versus nurture. By nature, I of course reference one's genetic makeup. By

**Figure 5**



#### Comparison of Annual Incidence

Data compiled and arranged by the Brain Injury Association of America based on the data from the Centers for Disease Control and prevention, American Cancer Society and the National Multiple Sclerosis Society.

nurture, I speak of environmental factors that shape who we are and influence behavior. First, let's talk about Nurture. While there are many ways that a brain can be harmed – through trauma, toxicity, and lack of oxygen or vital nutrients – the end result is relatively the same; neuronal deterioration or neuronal death. When those injuries result in neuro-degeneration in brain structures the end result is a change in function and/or behavior. Take for instance a coup-contra coup injury where focal damage occurs when the brain rapidly accelerates and hits the very bony structures of the skull, and where the rapid acceleration and deceleration results in the ripping or shearing of the neurons structures. This phenomenon is termed diffuse axonal injury, and as the name implies, injury occurs throughout the brain. The frontal lobes are particularly vulnerable to a coup-contra coup injury as described. It is the part of our brain that provides such functions as impulse control, working memory, the ability to plan or organize, attention and motor function to name a few. Damage can potentially result in problems such as motor

*continued*

## Aging and Brain Injury *continued*

dysfunction, issues with judgment and problem solving, lack of awareness, behavioral discontrol or change our ability to learn. The consequences of this type of injury can have a profound effect on how we behave and the judgments we make: What we eat, whether we exercise, whether we adhere to physician recommendations, whether we engage in risky behavior or a whole host of other lifestyle factors. Thus, the presence of neurological injury can influence all of these factors, which in turn increases the risk of secondary morbidity. Now let's turn to "Nature."

Brain injury has often been assumed to be a risk factor for other, age related neurodegenerative diseases – namely that of Alzheimer's. Much research has been done in this area, and Victoroff (2002) summarized studies that examined the role of previous head injury as a risk factor for dementia and Alzheimer's. Overall, when examining patients with Alzheimer's versus non-Alzheimer's elderly controls, it was found that head trauma with a loss of consciousness was more common by almost 80% for the Alzheimer's groups. However, as Victoroff notes, it is important to understand that a history of head trauma does not equate to a diagnosis of Alzheimer's or dementia down the road. There are many factors that moderate the relationship of brain injury and dementia/ Alzheimer's. Some of these include:

1. Age at injury – the effects of injury at a younger age may result in less of a chance of neuro-degeneration than injuries at greater ages
2. Severity of injury where the higher the severity the more likely the chances of neuro-degeneration over time
3. Area of injury within the brain where damage to certain areas have more harmful effects than others with respect to future neuro-degeneration
4. Type of injury – diffuse versus focal
5. Genetic makeup

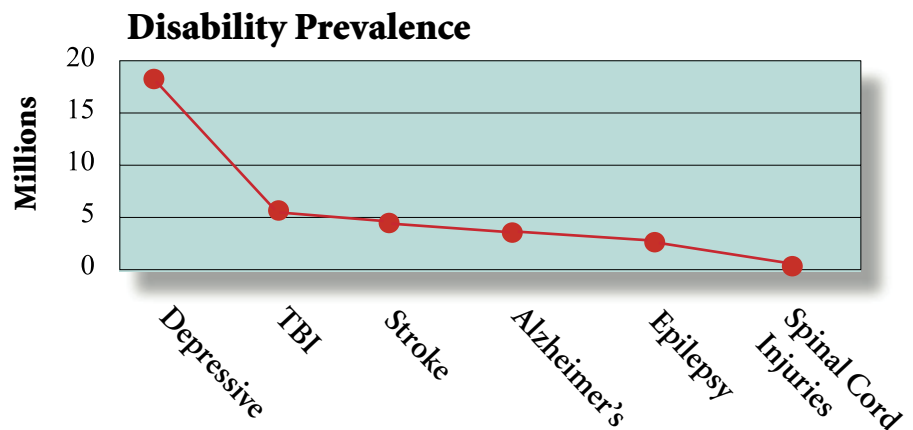
It is this last area that may have the most influence on the relationship of brain injury and dementia/ Alzheimer's. To begin, there are two distinctly different forms of Alzheimer's: One that is associated with familial history of the disease (5% of cases), and one where there is no familial history (95% of cases). But, even for the much more common form, that without familial history, our genes may play a substantive role in our

risk of neuro-degeneration of the Alzheimer's type. Specifically, I am referring to a gene by the name of apolipoprotein E, or APOE ε4. This gene has been identified as a substantial risk factor in Alzheimer's. The level of risk for Alzheimer's depends upon how many copies of the APOE ε4 allele one carries (Victoroff, 2002). The more copies of the allele, the greater the risk. Mayeux (1995; cited in Zansler) found that a history of brain injury alone did not increase the risk of Alzheimer's, but a combination of the APOE ε4 allele and brain trauma resulted in significantly greater risk (ten fold) of the non-familial type. Thus, a previous history of brain injury does increase the risk of Alzheimer's, but only if one was already pre-disposed.

At this point, it is fair to say that both environmental—secondary factors related to brain injury—and genetic factors can affect age related processes for individuals with brain injury. However, there are other factors to be considered and include physical changes that can accompany brain injury which in turn can affect a term already discussed; that of life expectancy.

Research has found two factors that reduce life expectancy rates: A lack of mobility and the need for a gastrostomy feeding tube (MacMillan and Greenwood, 2003). In a study on long-term survival after traumatic brain injury, Brown et al. (2004), found that in cases of moderate to severe brain injury, for those surviving the first six months post-injury, rates of mortality did not differ significantly from that of the general population. For cases of mild brain injury, they found a small but statistically significant reduction in life expectancy. However, they did report a finding that was somewhat surprising. When comparing the rates of mortality between moderate to severe injuries versus mild injuries

*continued*



## Aging and Brain Injury *continued*

six months post-injury, they did not find a lack of reduction in long-term survival rates as was expected for the mild group. The authors ventured that a better focus on an aging population of people with mild brain injuries was warranted.

One can thus conclude that brain injury can result in secondary health issues, which can affect the health and welfare of those persons with TBI. We must then focus on what can we do to change the course of these secondary health issues. One answer may be to determine which health issues pose the greatest risks and design care to mitigate these risks. Another is to ensure for those individuals affected by injury, that care planning take into account the lifelong effects of a brain injury, regardless of the severity. When we realize that 80,000-90,000 Americans are disabled following traumatic brain injury each year (Centers for Disease Control, Traumatic Brain Injury – Fact Sheet, May 2) one can conclude that brain injury certainly impacts health expectancy figures and life expectancy figures. Does having a brain injury impact or accelerate the aging process? The answer to this final question is a conditional yes. Not all injuries lend themselves to accelerated aging or dementia, and like non-head injured counterparts, there are steps one can take to diminish this acceleration: Stay active, nourish your mind and eat a healthy balanced diet. Advice we could all use! ❖

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