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‘Therapeutic lies’ in the context of dementia care
Caregivers regularly face the challenge of whether to ‘tell the truth’ to a person with dementia. Navigating the grey area between truth-telling and therapeutic lying is a delicate balance. This article explores both sides of the issue and concludes with some recommendations.

By Annette Meeuwse, R.N., B.A., M.A.

Anosognosia
Lack of awareness of memory deficits could be predictive of dementia.

Exercise - A key to preventing Alzheimer’s
Increasing research suggests that physical activity not only improves executive function and cerebral blood flow, but may also reduce amyloid and tau levels. These new research efforts were presented at the 2017 Alzheimer’s Conference in London, England. Increasing research suggests that physical activity improves executive function, and may also reduce amyloid and tau levels.
Can A Lie Ever Be Considered Therapeutic?

Therapeutic lying, in the context of dementia care, is a heated and relevant ethical issue. Healthcare providers, as well as family members and caregivers, regularly face the challenging choice of whether to ‘tell the truth’ to a person living with dementia or ‘to lie’ to that person.

On the one hand, telling the truth can distress the person and lead to escalating behaviours as that person repeatedly processes the same difficult truths with their compromised cognition.

On the other hand, society values dignity and informed consent; thus, we feel we owe the truth - even when that may repeatedly distress a person who is living with dementia.

Navigating the grey area between truth-telling and therapeutic lying is a delicate balance. The following article explores both sides of the issue and concludes with some recommendations.

By Annette Meeuwse, R.N., B.A., M.A.

Therapeutic lies in the context of dementia care

Preamble
Barb sits down to breakfast one sunny morning. She wonders aloud when her husband will be joining her, and looking forward to his company at the table. To Barb’s absolute horror and dismay, a woman, who Barbara does not recognize, puts an arm around her shoulders, and with a warm smile, says: “Honey, your husband has died, remember? It’s ok; you’re safe here with us.”

The sheer shock of the news overwhelms and bewilders Barb. The last she knew, her husband had gone out for his morning walk. She panics, tears in her eyes, her appetite completely gone, wondering what on earth has happened to her husband. Dead? How? When?

She sobs aloud, forcefully pushing the stranger’s hand off her shoulder. Why had nobody told her sooner? What about the children? She must find the children! They should not hear this from anyone else! She quickly pushes the chair back from the table to go find the children, nearly losing her balance in her haste.

In response, the strange woman catches Barb by the elbow. Barb’s sorrow now gives way to anger. Who is this woman? Does she not understand the urgency of the situation, holding her back when she has to find her children? Giving her this dreadful news with a smile on her face?

Barb flings her arm free, briskly pushing away this woman. When she doesn’t leave her side, Barb hits out with a fist in frustration. She must find the children!

How to respond
Heart-wrenching scenes like this daily repeat themselves in various versions - and sometimes multiple times with the same person in the same day.

The feelings of restlessness, confusion and distress of those living with dementia in these situations are very real. The unmet needs associated with their verbalizations often lead to escalating behaviours, sometimes resulting in aggression against themselves or toward others around them.

And each time this situation arises, caregivers face the dilemma of how to respond. Professional staff and direct care staff express their concern in various ways such as, “I feel bad telling the person her son will come to visit, when we know he won’t. . . but otherwise she won’t eat.”

Circumventing distress
Physicians and managers express concern about “lying to avoid responsive behaviours, but we all know there is no other way to manage the person.”

Family members, too, ask in bewilderment, “How can I lie to my mother?”

The caregiver’s response to the resident/patient’s questioning and confusion is critical. The response can literally determine the direction and outcome of the situation. The caregiver must decide immediately whether to present the facts, such as a reminder that the person’s husband has passed away (or that the person no longer has a driver’s license, or that the house has been sold).

If the caregiver knows or intuits that this information will further distress the person, the caregiver might choose to respond by redirecting the person’s attention to another topic, by withholding information, or by telling a lie. This is where the concept of therapeutic lying enters the situation.

Therapeutic lying defined
Psychiatrist Michael Sperber (2015) defines therapeutic lying as “deliberately deceiving patients for reasons considered in their best interest.”

McElveen (2015) concurs with the preceding definition: “The term ‘therapeutic lie’ is a false statement or deception with the best interests of the patient.”

In order to more fully understand the issue, this article explores the constructs of
lying and truth-telling in healthcare; it also provides an overview of dementia’s affect on brain and behaviour, the possible pro’s and con’s of therapeutic lying to people living with dementia, and finally some recommendations related to therapeutic lying.

**Truth-telling and lying in healthcare**

Biomedical ethics is based on four main principles: 1. autonomy  2. justice  3. beneficence, and 4. non-malfeasance

Another way to express these principles is through asking the following questions:
- Is this situation allowing the person to freely make the best choices possible for himself/herself?
- Is this fair and just?
- Is this good for the person and in his/her best interest?
- Is all harm being avoided?

**Evolution of truth-telling**

While we would like to believe that, as professionals, we always tell the truth, truth-telling in healthcare has evolved over the years. This is evidenced in a landmark study in 1961 that showed 90% of a sample of 219 U.S. physicians reported that they would not disclose a diagnosis of cancer to a patient (Oken, 1961).

Of 264 physicians surveyed almost 20 years later, 97% stated they would disclose a diagnosis of cancer (Novack, et al., 1979).

Articles 21 and 22 of the Code of Ethics of the Canadian Medical Association state: “Provide your patients with the information they need to make informed decisions about their medical care, and answer their questions to the best of your ability. Make every reasonable effort to communicate with your patients in such a way that information exchanged is understood.”

Truth telling in healthcare has even made its way into Canadian Legislation through such cases as Hopp v. Lepp, 1980. This was the first case heard by the Supreme Court of Canada on a patient’s right to informed consent from a physician.

The court ruled in favour of the patient’s right to know. It set a precedent that other cases followed, such as Reibl v. Hughes (1980).

**Truth in a healthcare context**

Research, policy and legislation demonstrate the value our society places on truth telling in healthcare. As health professionals our motivation to tell the truth, to slightly bend the truth, or to withhold the truth with the people in our care can vary greatly. Our decision to tell or not tell can be influenced greatly by many factors including, but not limited to, our own and the resident’s cultural backgrounds, education, life experience or even basic stressors, such as amount of sleep experienced the night before.

In summary, truth telling in a healthcare context is highly valued - as well as expected. This is demonstrated by its evolution within healthcare and in the context of social expectations as well as professional codes of ethics, research and legislation.

**Dementia's effect on the brain and behaviour**

Truth telling intersects with dementia in a number of areas. Dementia is defined as a syndrome, a cluster of symptoms which result from diseased and dead brain cells. The effects of dementia are as diverse as the parts of the brain it affects.

Memory, decision-making, absorbing new information, personality changes, difficulty with self expression, as well as dealing with emotions, loss of ability to plan, including loss of abstract thinking, are all potential symptoms of dementia (Guidelines for Integrated Dementia Care (excerpt), 2009). Currently, the course of dementia is relentless and irreversible, with patients living an average of 8 years with the syndrome.

Studies show that the process of slow decline is often underway for a number of years before diagnosis, implying that people and their caregivers have been coping and compensating with the losses long before they officially enter the healthcare system (Understanding Dementia, 2016).

With the vast array of possible symptoms, as well as gloomy outcomes, one can only imagine how bewildered the person with dementia feels.

**Truth telling and dementia**

One area where truth telling intersects with dementia is from the persons with dementia themselves. They may sometimes intentionally use confabulation as a way of compensating for their losses - of which they are aware (Confabulation, 2017). They may also unintentionally confabulate, as in the case of Barb mentioned in the opening paragraph of this article. Their reality is what they understand it to be at that moment - however distant it appears from the reality the caregivers, family members and friends are seeing.

The second area where truth telling intersects with dementia - and the focus of this article - is in the response of caregivers to their loved one’s confusion and confabulation. Society’s emphasis on truth telling has already been established.

In the situation where a person with dementia has been suffering decline over time, even prior to entering the healthcare system, the family has been absorbing the impact and feeling the vast spectrum of emotional reactions along with the patients themselves.

Additionally, this stressed system often needs to simultaneously make difficult choices about care, finances and accommodations as the person declines.

The family system and caregivers often clearly know what ‘works’ for the person and, alternatively, what potentially triggers the anxious, upset or agitated responses. In a highly stressed system, already teetering on the delicate balance of tipping into overload, caregivers could understandably choose responses that calm, appease, and help maintain the status quo.

**Advantages of therapeutic lying**

This leads to the consideration of potential positive aspects related to therapeutic lying. It is safe to say that everybody, including the resident/patient and the caregiver(s), hope for positive outcomes. These can include everything from alleviating the patient/resident’s acute distress, through to the person being able to continue to live at home or in their current environment, rath-
er than a setting with higher security needed for more aggressive behaviours. There are a number of reasons why therapeutic lying could be advantageous.

**Motives for therapeutic lying**

One potential motivation for caregivers to engage in therapeutic lying could be to avoid escalating aggression. Aggressive behaviours are a serious concern both at home and in a care facility.

A report issued by the Ontario Long Term Care Association in 2016 states that 46% of residents exhibit some level of aggressive behaviour related to their cognitive impairment or mental health condition (“This is Long Term Care 2016”).

To reframe this, a personal support worker with ten residents assigned to her care could potentially face four to five residents who kick, bite, scratch, hit out, or verbally harass. To reframe it yet again, a caregiver who has an altered view of reality. Many approaches to dementia care focus on entering into a person’s reality.

For example, as early as the 1960s, social worker, Naomi Feil, developed ‘Validation’ as a state-of-the-art therapy for older people diagnosed as having Alzheimer’s type dementia or related disorders. Her early approach, as well as the many that have followed, focus on providing an empathetic and understanding response to a person’s confusion (Feil, et al., 2012).

The feelings of the patient/resident take priority over the facts. For example, if this patient/resident is feeling bored and lonely, their thoughts could focus on getting the car keys to get away from the boredom, or to approach their mother for comfort.

Using the approaches described, a caregiver listens to the heart of the issue, which is the person’s boredom and loneliness, rather than responding with the fact that he/she no longer owns a car or that their mother has died.

Another motivation to use therapeutic lying could be to maintain the current quality of life for that person. Moody (as cited in Pinner, 2000) wrote: “What sense really does it make to speak.”

Finally, the views of people with dementia have been canvassed by Day, et al. in regard to the use of lies in dementia care (Day, et al., 2011). This study had a small sample size of people with early stage Alzheimer’s, but it highlighted that the majority of participants considered lies to be acceptable if they were perceived to be in the person’s best interests.

Of the possible, positive benefits of therapeutic lying, psychiatrist Anthony McElvene (2015) makes a strong case with the following statement:

“Can a lie ever be considered therapeutic? I believe it can. I find that a difficult sentence to write because it does not fit with the deontological view that I would have espoused less than a year ago.

Yet, if the function of truth in a situation is to bring nothing but pain and distress to a confused, demented fellow human being, then its utilization in that instance is at best, futile, at worst, cruel.

“When we have exhausted all other possible therapeutic options - including truth-telling - and only when it is likely to enhance the person’s well-being, should a ‘best interests lie’ be trialed and then the benefit reassessed.”

**Disadvantages of therapeutic lying**

As with all care issues, there is more than one side to consider. Practice and literature suggest that there are potential disadvantages to therapeutic lying as well.

The first disadvantage can be the erosion of personhood and dignity (Kitwood, 1997). The person living with dementia has already lost, and will lose so many facets of function and control in their lives.

Someone deciding not to tell the truth to a person with dementia deprives that person of the dignity of even the option to make their own choices and have their individual reactions. This censoring of information prior to communicating with the person with dementia could be a return to the paternalistic medical view of personhood.

Another disadvantage could be that lying would lead to neglect and ill treatment of the individual with dementia (Kitwood, 1998). This line of thinking states that if lying is acceptable, perhaps other sorts of ill treatment could also become gradually acceptable in a certain workplace culture.

An article by McDonnell (2014) outlines how institutional abuse occurs when the “routines and the rituals of a service result in the lifestyles and needs of individuals being sacrificed in favour of the needs of the institution.” If the focus of the institution is to keep the environment smooth at all costs and caregiving as easy as possible, then there could be a motivation to lie to residents simply to keep them calm and avoid conflict and stress - which obviously does not have the best interest of the resident at heart.

**“Deception guilt”**

Yet, another disadvantage of therapeutic lying could be ‘Deception Guilt’. This concept is exactly what it sounds like - the guilt the caregiver feels about lying to a loved one or patient/resident.

A number of sources, including Abel (1990), Blum, (1994) and Corbin (1990), all speak to the concept of ‘deception guilt.’ The ‘guilt’ experienced can be heard firsthand from care staff as well as physicians and family members who express their bewilderment at lying. As previously established, society places high value on honesty, so being dishonest does not feel right to a professional, family member or friend.

‘Breach of trust’ is another potential disadvantage of therapeutic lying. People with dementia have very clear moments, hours, and days; others have decline in one or more certain areas of function, but still...
have an intact sense of emotional intelligence or perception. In any case, devastation, bewilderment, frustration, and a range of other potentially negative emotions could result from a person knowing they are being lied to.

Finally, it may be illegal in some situations to lie to a resident, whether therapeutic or not.

In discussing the right of residents/patients to gain access to their own medical records, the Supreme Court of Canada acknowledged that information can have value to patients for its own sake and that “non-disclosure can itself affect the patient’s well-being.” (Judgments - Supreme Court of Canada: McInerney v. MacDonald, 1992).

**Recommendations**

Whether one chooses to side with the advantages or disadvantages of therapeutic lying, there are a few recommendations that could bridge the grey area between ‘therapeutic’ and ‘lying’.

**- Terminology**

**Therapeutic lying** is a contradiction in terms; in fact, an ethical challenge is contained in the term itself.

Perhaps the first step toward a deeper understanding is to come up with a new phrase to describe the responses caregivers sometimes choose in response to the confabulations of people living with dementia: Is the person with dementia lying? Was Barbara, at the beginning of the article, ‘lying’? Does there need to be intent to lie and an awareness of lying in order to be considered a lie?

When a caregiver enters the reality of the resident/patient’s confabulation, and empathetically addressing the feelings behind the ‘lying’ words, is the caregiver really ‘lying’? Perhaps we need to entirely delete the word ‘lying’ from our lexicon.

Could we refer to ‘entering their reality with creativity and empathy’ simply as a therapeutic approach?

Could we refer to entering their reality with creativity and empathy and individualized decision-making, rather than putting responses into an either/or category? The dialogue definitely needs to continue and the wording we choose affects the dialogue.

**- Relational approaches**

Many dementia care approaches, or models, focus on feelings over fact. A recent visit to Dementia Village (Hogeweyk, 2017), showed an entire small Dutch town intentionally constructed as a dementia-friendly space with only two entrances/exits. This large scale environmental design shows an overt focus for the feelings of people living with dementia, honouring their need for both autonomy and safety.

Other models of relational care, in regards to dementia, focus solely on approach, such as Teepa Snow, a dementia-care education specialist, whose focus centres on relationship (Snow, 2017).

Another example is David Sheard’s ‘Butterfly Approach’ to dementia care, referring to identifying with the feelings of the person rather than the facts being expressed (Sheard, 2017).

In the example at the beginning of this article, the caregiver reminds Barbara of the fact that her husband has died. However, rather than having a comforting and reassuring effect, her well-intended factual comments set off a cascade of escalating distress for both Barbara and the caregiver and those around them in the dining room. These effects included physical and verbal aggression as well as disturbance to the other residents having their breakfast.

What would have happened if, instead, the caregiver had tuned to Barbara’s feelings of anxiety and loneliness? How could the outcome have been different if the caregiver had responded: “Tell me about your husband”? (Snow, 2017). A lie did not have to be told - but a truth could have been avoided.

**- Training**

This type of approach comes intuitively for some, however, not for everybody. Caregivers can have a knowledge gap about the disease process and what to expect. Training for staff and caregivers in managing challenging behaviours, as well as supportive communication techniques, is key to developing awareness. It is also important to be aware that, in the healthcare community, staff and caregivers regularly struggle with this ethical dilemma. Family members and professionals alike have expressed concern that the person with dementia is lying. There is equal concern that they themselves feel guilty lying to the person with dementia.

**- Person-centered responses**

How aware is the person with dementia? Is the chosen response comforting? Or, is it patronizing and demeaning?

As mentioned earlier, those living with dementia who were surveyed about their opinion of therapeutic lying essentially responded, ‘it depends;’ and it does depend! As with all aspects of care, responses need to be person-centred and individualized.

**Conclusion**

**Therapeutic lying** is defined as “deliberately deceiving residents/patients for reasons considered in their best interests” (Sperber, 2015).

The concept of therapeutic lying is complex. It can be considered a matter of semantics, a matter of approach, an ethical dilemma, and sometimes all three at once.

Our society places significant value on truth-telling. This is evidenced by social practices as well as laws. However, literature and practice also indicate that there are times when the truth may not be the most therapeutic approach in responding to the verbalizations and sometimes the confabulations of persons living with dementia.

McElveen (2015) concurs with the following definition: “The term ‘therapeutic lie’ is a false statement or deception with the best interests of the patient at heart.”

**The heart of the matter!**

The heart of the matter is that people living with dementia have lives that are constantly affected, day and night, by one or more negative effects of dementia. All would agree that the caring response is one that is empathetic, kind and supportive.

Hopefully, this article provides a thought-provoking expose of all sides of the issues,
as well as practical recommendations in dealing with the topic of Therapeutic Lying.

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Table 1 - Guidelines on Therapeutic Lies
1) Lies should only be told if they are in the best interests of the resident (to ease distress).
2) Specific areas, such as covert medication and aggressive behaviour, require individualized policies that are documented in the care plan.
3) A clear definition of what constitutes a lie should be agreed within each setting.
4) Mental capacity assessments should be performed on each resident/patient prior to use of therapeutic lies.
5) Communication with family should be required and family consent gained if a lie is to be told to the patient.
6) Once a lie has been agreed upon, it must be used consistently across people and settings.
7) Lies told should be documented to ensure they are told in resident/patients’ best interests.
8) An individualized approach should be adopted towards each case, that is, the relative costs and benefits established relating to the lie.
9) Staff should feel supported by management and the resident/patient’s family. They should not feel at risk of being accused of misconduct by telling lies if they have been agreed using these guidelines.
10) Circumstances in which lies should not be told need to be outlined and documented.
11) The act of telling lies should not lead to staff disrespecting the patient. The lies should be seen as a strategy to enhance the resident/patient’s well being, rather than an infringement of their basic rights.
12) Staff should receive training and supervision on the potential problems of lying, and taught alternative strategies to use when lies are not appropriate. 

Ian James, et al., 2006
Anosognosia

Lack of awareness of memory deficits is predictive of a dementia

While memory loss is an early symptom of Alzheimer’s, its presence doesn’t mean a person will develop dementia. A new study at the Centre for Addiction and Mental Health (CAMH) in Toronto has found a clinically useful way to predict who ‘won’t’ develop Alzheimer’s disease, based on the person’s awareness of memory problems.

Anosognosia

People who were unaware of their memory loss, a condition called anosognosia, were more likely to progress to Alzheimer’s disease, according to the study, published in the Journal of Clinical Psychiatry.

Those who were aware of memory problems were unlikely to develop dementia.

“If patients complain of memory problems, but their partner or caregiver isn’t concerned, it’s likely that the memory loss is due to other factors, possibly depression or anxiety,” says lead author Dr. Philip Gerretsen, clinician scientist in CAMH’s Geriatric Division and Campbell Family Mental Health Research Institute in Toronto. “They can be reassured that they are unlikely to develop dementia, and other causes of memory loss should be addressed.”

In other cases, the partner or caregiver is more likely to be distressed while patients don’t feel they have any memory problems.

In Alzheimer’s disease, lack of awareness is linked to more burden on caregivers. Both unawareness of illness (anosognosia) and memory loss MCI (mild cognitive impairment) can be objectively assessed using questionnaires.

Glucose and Alzheimer’s

The study, believed to be the largest of its kind on ‘illness awareness’, accumulated data on 1,062 people aged 55 to 90 from the Alzheimer’s Disease Neuroimaging Initiative (ADNI). The study included 191 people with Alzheimer’s disease, 499 with mild cognitive impairment and 372 as part of the healthy comparison group.

The researchers also wanted to identify which parts of the brain were affected in ‘impaired illness awareness’. They examined the brain’s uptake of glucose. Brain cells need glucose to function, but glucose uptake is impaired in Alzheimer’s disease.

Using Positron Emission Tomography, i.e., PET brain scans, they showed that those with impaired illness awareness also had reduced glucose uptake in specific brain regions, even when accounting for other factors linked to reduced glucose uptake, such as age and degree of memory loss.

Next research stage

At the next stage of the research, Dr. Gerretsen will be tracking older adults with mild cognitive impairment who are receiving an intervention to prevent Alzheimer’s dementia. This ongoing study, the ‘PACt-MD study’, combines brain training exercises and brain stimulation, using a mild electrical current to stimulate brain cells and improve learning and memory. While the main study is focused on dementia prevention, Dr. Gerretsen will be looking at whether the intervention improves illness awareness in conjunction with preventing progression to dementia.

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Understanding and caring for those with anosognosia

A neuropsychiatric explanation

More than simple denial, anosognosia is a lack of awareness of impairment; that is, most people do not even know they have an illness. It also affects over 80% of those with Alzheimer’s disease.

A ‘Place for Mom’ recently had the opportunity to speak with the Treatment Advocacy Center to learn more about anosognosia and Alzheimer’s. The Treatment Advocacy Center was started in 1998 in Arlington, Virginia, by Dr. Fuller Torrey.

Anosognosia described

When U.S. President Woodrow Wilson had a stroke in 1919, his physical health was only ‘slightly’ impacted, but his mental health suffered. Film director Errol Morris, in a New York Times opinion column, wrote that, “Wilson’s close associates noticed a change in his personality. He became increasingly suspicious, even paranoid, without having the dimmest awareness of the fact that he was becoming a different person.” Edwin Weinstein, a neuropsychiatrist who reviewed Wilson’s case in the 1970s, deemed it a classic case of anosognosia - a lack of awareness that one is impaired.

Alarmingly common

It is a very difficult for caregivers to make progress with a person’s illness when he or she is showing signs of anosognosia. Yet, the condition is alarmingly common. After stroke, some studies show that up to 77% will suffer anosognosia.

Anosognosia occurs frequently in those with mental illness, according to the Treatment Advocacy Center in Arlington, VA, and can also affect people who have suffered traumatic brain injury, as well as people with Alzheimer’s and other types of dementia.

Misperception of an illness

Anosognosia is still difficult to define, but researchers know it results from physi-
cal, anatomical changes, or damage to the part of the brain that affects perception of one’s own illness.

Studies suggest that deterioration in the frontal lobes may be involved, which “play an important role in problem-solving, planning and understanding the context and meaning of experiences and social interactions,” states the New York Times’ New Old Age blog.

How anosognosia occurs
To put it another way, “. . . our right brain is wired to detect anomalies and new information and incorporate these into our sense of reality,” says the neuroscientist Dr. V.S. Ramachandran, also in the New York Times. When something happens to damage that part of the (right) brain - a stroke or dementia, for instance - then “the left brain seeks to maintain continuity of belief, using denial, rationalization, confabulation and other tricks to keep one’s mental model of the world intact.”

Anosognosia and Alzheimer’s
Anosognosia has long been recognized in individuals with Alzheimer’s, brain tumors, Huntington’s disease and stroke, says the Treatment Advocacy Center.

According to the University of Florida’s health resource, AlzOnline, the prevalence of anosognosia in those with cognitive impairment or dementia can be very high.

“Some researchers have estimated that as many as 60% of people with MCI, and 81% of people with Alzheimer’s disease, have some form of anosognosia.”

This situation is difficult for caregivers, who are trying to help someone who essentially does not and cannot acknowledge they are ill. The anosognosic person with dementia may have evident problems with routine tasks, but they may insist they do not need help, or may even refuse medical evaluation or treatment - treatments which are often key to helping them realize they are impaired in the first place.

To make the situation even more challenging, anosognosia may be complete or selective. They may be entirely unaware of their impairment, for instance, or they may even react with anger and defensiveness if confronted about their illness. This makes it difficult to diagnose anosognosia, and tough to differentiate it from simple denial.

What to do if a resident has anosognosia
Whether the resident (or loved one) is in denial of their anosognosia, the most effective caregiver strategy is one of mitigation of the effects, rather than trying over and over to make the person understand. “Trying to make someone with this problem understand that they have changed and need to accept new limits is often an exercise in frustration,” says the New York Times. And, the controversial Treatment Advocacy Center in the U.S. concurs: “Nobody wants to take meds if they aren’t sick, and people with anosognosia are no exception.”

However, if not treated, the patient could put themselves and others in danger. AlzOnline, has the following suggestions for caring for someone with anosognosia:

Sedentariness and mortality across levels of frailty
Sedentary time, such as time spent sitting, increases the risk of death for middle-aged and older people who are frail and inactive - although it doesn’t appear to increase the risk for non-frail people who are inactive, according to a recent study published in the Canadian Medical Association Journal.

Benefits of physical activity
Many studies have looked at the benefits of physical activity on health, although little data exists on sedentary behaviour and risk of death linked to levels of frailty.

Frailty refers to a person’s overall health state as determined by the number of health problems he/she has. To understand if level of frailty and prolonged sitting are linked to a higher risk of death, researchers looked at data on 3141 adults aged 50 and over in the US National Health and Nutrition Examination Survey from 2003/04 and 2005/06.

Participants used activity trackers during waking hours and were assessed using a 46-item frailty index; they were then followed until 2011 - or date of death.

“We found that, in people who scored low on the frailty index, sitting time was not linked to risk of death,” states Dr. Olga Theou of the Department of Medicine, Dalhousie University, Halifax. “Prolonged sitting, however, was associated with a higher risk of death only in vulnerable or frail people who did not meet the weekly recommendation for 2.5 hours of moderate physical activity.”

The harms of inactivity
“Physicians should stress the harms of inactivity to encourage movement,” states Dr. Theou. “Even something as simple as getting up and walking around with a walker or cane can benefit frail people.”

See: <http://www.cmaj.ca/content/189/33/E1056.full?sid=49781f07-8aab-45ad-911e-d08febe61e6c>.
Can lack of sleep cause Alzheimer’s?

If someone suffers from sleep deprivation, the consequences can be far more serious than simple crankiness and fatigue - with studies showing that lack of sleep may cause Alzheimer’s disease

According to the U.S. Centers for Disease Control and Prevention, inadequate sleep is a public health epidemic. In fact, millions of adults have ‘sleep or wakefulness disorder’ which can affect daily functioning, such as driving, and overall mental health and long-term well-being.

In fact, sleep is just as important as exercise and diet when it comes to living a healthy life. Lack of sleep has been linked to a myriad of health problems, including:
- Cognitive dysfunction
- Depression
- Stress
- Type II diabetes
- Weight gain

Nocturnal cleansing system

More disconcerting is a new study indicating that lack of sleep may lead to Alzheimer’s disease. Led by Maiken Nedergaard, M.D., who co-directs the University of Rochester Medical Center for Translational Neuromedicine, the researchers discovered that a waste-draining system, the “glymphatic system” is ten times more active during sleep than while awake.

This nocturnal cleansing system removes proteins called amyloid-beta, which accumulate in the plaques that contribute to Alzheimer’s and dementia. Nedergaard and her team coined the term “glymphatic system” when they used new imaging technology known as ‘two-photon microscopy’ to discover a scrubbing process taking place around brain cells, known as glial cells.

The mechanism of this cleanup process is fascinating. Nedergaard and colleagues found that cerebrospinal fluid flows through the spaces between neurons, flushing proteins and other neural waste into the circulatory system and away.

“So, what if you’re not getting those eight hours of sleep? Are you destined to get Alzheimer’s disease,” asks Nedergaard. “Not necessarily - but don’t buy any sleep remedies just yet. You might just have leftover sleep patterns from your ancestors.”

The eight hour sleep myth

Before the advent of the light bulb, we went to sleep when it got dark. Interestingly, heading to bed around 7:00 or 8:00 p.m. meant that we had about 12 whole hours of restful time, so it was natural for us to wake up in the middle of the night.

The eight uninterrupted hours of sleep is a modern notion that, unfortunately, causes many sleepers a great deal of concern. But we need to ‘rethink’ the way we get sleep.

Quality, not quantity!

What is ultimately most important about sleep is the quality of it - not the quantity. Our modern lives simply don’t let us get the rest we need. Laptops and smart-phones naggingly chirp and glow at us all day long, ad nauseam. If we could truly ‘unplug’, we might find the peace of mind needed to get our rest; but most of us don’t have that luxury. So what can we do to get the sleep we so desperately need?

“It may simply mean accepting that ‘waking up during the night is part of normal human physiology,” say sleep psychologists.

Segmented sleep patterns

Furthermore, in 2001, historian/author, Roger Ekirch of Virginia Tech University, in a paper drawing on 16 years of research, revealed “a wealth of historical evidence that humans used to sleep in two distinct chunks” of time.

Ekirch went on to publish a book, “At Day’s Close: Night in Times Past,” in which he revealed more than 500 references to a segmented sleeping pattern by humans in the past. The references describe a first sleep, which began about two hours after dusk, which were followed by waking periods of one to two hours, then a second block of sleep.

Quality, not quantity

“This knowledge is comforting for the many of us today who, all too often, feel sleep-deprived.” But there are other ways to get rest, says Ekirch. If someone cannot sleep 8 continuous hours, then spread it out:
- sleep in four hour chunks, or,
- take naps

“Quality, not quantity, of sleep is the most restorative; so, do whatever it is that is most natural to get the quality shut-eye needed,” says Roger Ekirch.

* The glymphatic system - The brain’s waste disposal system

The glymphatic system (or glymphatic clearance pathway) is a functional ‘waste clearance pathway’ for the central nervous system (CNS).

This pathway consists of a para-arterial influx route for cerebrospinal fluid (CSF) to enter the brain parenchyma, coupled to a clearance mechanism for the removal of interstitial fluid (ISF) and extracellular solutes from the interstitial compartments of the brain and spinal cord. Exchange of solutes between the CSF and the ISF is driven by arterial pulsation and regulated during sleep by the expansion and contraction of extracellular brain space. Clearance of soluble proteins, waste products, and excess extracellular fluid is accomplished through convective bulk flow of the ISF, facilitated by astrocytic aquaporin 4 (AQP4) water channels.

* Parenchyma - refers to the functional tissue in the brain that is composed of the two types of brain cells, neurons and glial cells.
Understanding the link between poor sleep and Alzheimer’s!

A good night’s sleep refreshes body and mind, but a poor night’s sleep can do just the opposite. A study from Washington University School of Medicine in St. Louis, the Radboud University Medical Centre in The Netherlands, and Stanford University in California, has shown that disrupting just one night of sleep in healthy, middle-aged adults causes an increase in amyloid beta, a brain protein associated with Alzheimer’s. “We further showed that poor sleep is associated with higher levels of two Alzheimer’s-associated proteins,” said David Holtzman, M.D., head of the Department of Neurology at Washington University in St. Louis and the study’s senior author. “We think that perhaps chronic poor sleep during middle age may increase the risk of Alzheimer’s later in life.”

Poor sleep and dementia
These findings, published in July, 2017 in the journal *Brain*, may help explain why poor sleep has been associated with the development of dementias such as Alzheimer’s.

Millions of people globally are living with Alzheimer’s disease, which is characterized by gradual memory loss and cognitive decline. The brains of people with Alzheimer’s are dotted with plaques of amyloid beta protein and tangles of tau protein, which together cause brain tissue to atrophy and die. Currently there are no therapies that have been proven to prevent, slow or reverse the course of the disease.

Consequences of poor sleep
Previous studies by David Holtzman, Dr. Yo-El Ju, an assistant professor of neurology from The Netherlands, and others, have shown that poor sleep increases the risk of cognitive problems. People with sleep apnea, for example, a condition in which people repeatedly stop breathing at night, are at risk for developing mild cognitive impairment an average of 10 years earlier than people without the sleep disorder. Mild cognitive impairment is an early warning sign for Alzheimer’s.

But it wasn’t clear how poor sleep damages the brain. To find out, the researchers (Holtzman, Dr. Ju, graduate student Sharon Ooms, and Jurgen Claassen, M.D., Ph.D., of Radboud University, The Netherlands; Emmanuel Mignot, M.D., Ph.D., of Stanford University, and colleagues), studied 17 healthy adults ages 35-65 with no sleep problems or cognitive impairments. Each participant wore an activity monitor on the wrist for up to two weeks that measured the time they spent sleeping each night.

The ‘sleep room’
After five or more successive nights of wearing the monitor, each participant came to the School of Medicine at Washington University in St. Louis to spend a night in a specially designed sleep room. The room was dark, soundproof, climate-controlled and just big enough for one - a perfect place for sleeping - even as the participants wore headphones over the ears and electrodes on the scalp to monitor brain waves.

Half the participants were randomly assigned to have their sleep disrupted during the night they spent in the sleep room. Every time their brain signals settled into the slow-wave pattern characteristic of deep, dreamless sleep, the researchers sent a series of beeps through the headphones, gradually getting louder, until the participants’ slow-wave patterns dissipated and they entered shallower sleep.

The next morning, the participants who had been ‘beeped’ out of slow-wave sleep reported feeling tired and unrefreshed, even though they had slept just as long as usual and rarely recalled being awakened during the night. Each underwent a spinal tap so the researchers could measure the levels of amyloid beta and tau in the fluid surrounding the brain and spinal cord.

A month or more later, the process was repeated, except that those who had their sleep disrupted the first time were allowed to sleep through the night undisturbed, and those who had slept uninterrupted the first time were disturbed by beeps when they began to enter slow-wave sleep.

The researchers compared each participant’s amyloid beta and tau levels after the disrupted night to the levels after the uninterrupted night, and found a 10% increase in amyloid beta levels after a single night of interrupted sleep; there was no corresponding increase in tau levels; however, participants whose monitors showed they had slept poorly at home the week before, the spinal tap did show a spike in tau levels.

“We were not surprised to find that tau levels didn’t budge after just one night of disrupted sleep while amyloid levels did, because amyloid levels normally change more quickly than tau levels,” Dr. Ju said. “But we could see, when the participants had several bad nights in a row at home, that their tau levels had risen.”

Slow-wave sleep
Slow-wave sleep is the deep sleep that people need in order to wake up feeling rested. Sleep apnea disrupts slow-wave sleep, so people with the disorder often wake up feeling unrefreshed, even after a full eight hours of shut-eye.

Slow-wave sleep is also the time when
neurons rest and the brain clears away the molecular by-products of mental activity that accumulate during the day, when the brain is busily thinking and working.

Dr. Ju thinks it is unlikely that a single night or even a week of poor sleep, miserable though it may be, has much effect on overall risk of developing Alzheimer’s disease. Amyloid beta and tau levels probably go back down the next time the person has a good night’s sleep, she pointed out.

**Chronic sleep problems**

“The main concern is with people who have chronic sleep problems,” Dr. Ju said. “I think that may lead to chronically elevated amyloid levels, which animal studies have shown lead to increased risk of amyloid plaques and Alzheimer’s.”

**New insights into why sleep is good for our memory**

Researchers at the University of York in the UK shed new light on sleep’s vital role in helping us make the most of our memory.

Sleep, the researchers show, helps us to use our memory in the most flexible and adaptable manner possible - by strengthening new and old versions of the same memory to similar extents.

**‘Updated’ memory retrieval**

The researchers also demonstrate that when a memory is retrieved, i.e., when we remember something, it is updated with new information present at the time of remembering. The brain appears not to ‘overwrite’ the old version of the memory; instead, the brain generates and stores multiple (new and old) versions of the same ‘memorable’ experience.

The results of the research, carried out at York’s Sleep, Language and Memory (SLAM) Laboratory, were presented in the journal *Cortex* in mid-November, 2017.

**Sleep and memory efficiency**

Lead researcher, Dr. Scott Cairney of the University of York’s Department of Psychology said: “Previous studies have shown sleep’s importance for memory. Our research takes this a step further by demonstrating that sleep strengthens both old and new versions of an experience, helping us to use our memories adaptively.

“In this way, sleep is allowing us to use our memory in the most efficient way possible, enabling us to update our knowledge of the world and to adapt our memories for future experiences.”

**Memory strengthening**

In the study, two groups of subjects learned the location of words on a computer screen. In the test phase, participants were presented with each of the words in the centre of the screen and had to indicate where they thought they belonged.

One group then slept for 90 minutes, while a second group remained awake before each group repeated the test. In both groups, the location recalled at the second test was closer to that recalled at the first test than to the originally-learned location, indicating that memory updating had taken place and new memory traces formed.

However, when comparing the sleep and wake groups directly, the locations recalled by the sleep group were closer in distance to both the updated location (i.e., previously retrieved) and the original location, suggesting that sleep had strengthened both the new and old version of the memory.

Corresponding author, Professor Gareth Gaskell, also of York’s Department of Psychology, explained further: “Our study reveals that sleep has a protective effect on memory and facilitates the adaptive updating of memories... For the sleep group, we found that sleep strengthened both their memory of the original location as well as the new location. In this way, we were able to demonstrate that sleep benefits all the multiple representations of the same experience in our brain.”

**Incorrect information and memory**

The researchers point out that, although this process helps us by allowing our memories to adapt to changes in the world around us, it can also hinder us by incorporating incorrect information into our memory stores. Over time, our memory will draw on both accurate and inaccurate versions of the same experience, causing distortions in how we remember previous events. The study builds on a research model created by Ken Paller, Professor of Psychology at Northwestern University in Evanston, Illinois, and a co-author on the University of York study.

**Reference**

• Scott Cairney, Shane Lindsay, Ken Paller and Gareth Gaskell, Sleep preserves original and distorted memory traces, *Cortex*; 99; p.39-44; 2017. DOI: https://www.ncbi.nlm.nih.gov/pubmed/29145007.
**Highlight from the Alzheimer’s Association International Conference, July, 2017**

*London, England:* Increasing research suggests that physical activity not only improves executive function and cerebral blood flow but may also reduce amyloid and tau levels in the brain. This research was presented at the Alzheimer’s Association International Conference 2017.

## Exercise - a possible key to the prevention of Alzheimer’s Disease

Much of the focus of the 2017 AAIC meeting was on lifestyle interventions, i.e., healthy eating, reduced stress, adequate sleep, and increased physical activity to help ameliorate dementia and its symptoms. Some experts believe that, of all the lifestyle factors, exercise leads the way when it comes to preserving cognition.

In recent years, the identification of biomarkers for Alzheimer’s has made it possible to compare levels of amyloid-beta (Aβ) and tau, both hallmarks of AD in those who are and are not, physically active.

### Exercise and amyloid load

One study presented at the AAIC in London was led by Dr. Belinda Brown, Ph.D., of the School of Psychology and Exercise Science at Murdoch University in Perth, Australia. She evaluated the relationship between exercise levels and brain amyloid load in carriers of genetic mutations that cause autosomal-dominant AD, i.e., the genetic predisposition that leads to AD.

The analysis included data from the *Dominantly Inherited Alzheimer Network* for 139 pre-symptomatic mutation carriers. These patients are destined to develop AD and know approximately when they will start having symptoms. (See box below)

From self-reports of exercise, the researchers categorized patients into those reporting fewer than 150 minutes per week of (low exercise) and those reporting 150 minutes or more per week (high exercise).

Researchers also had information on brain amyloid load, as quantified by positron-emission tomography (PET). They stratified patients in order to investigate those with high brain amyloid plaque levels.

Compared to the high-exercise group, the low-exercise group was older (38.6 years vs. 33.7 years) and had more depressive symptoms as measured by the *Geriatric Depression Scale* (2.2 vs 1.4).

When the entire cohort of mutation carriers was examined, there were no differences in amyloid load between patients in the low-exercise group and those in the high-exercise group. However, for the 16 patients with beta-amyloid plaque in the low-exercise group, the mean level of brain plaque was higher than in the 55 patients with amyloid plaque who were in the high-exercise group.

### Higher exercise levels delaying AD pathology

The researchers were able to show that amyloid-beta (Aβ), in those in the high-exercise group, accumulated at a slower rate relative to what would be expected.

“In mutation carriers of Alzheimer’s disease, researchers can estimate how many years in the future an individual will be from developing Alzheimer’s symptoms, based on their age and the average age of onset of others with the same mutation,” Dr. Belinda Brown explained. “The results suggest that higher levels of exercise may delay the accumulation of Alzheimer’s pathology and subsequent symptom onset in Alzheimer’s disease mutation carriers. She also noted that these results require further validation using longitudinal analyses.

Nevertheless, the findings were particularly impressive for Dr. Christina Hugenschmidt, Ph.D., assistant professor of gerontology and geriatric medicine at Wake Forest School of Medicine in Winston-Salem, North Carolina.

The study finding that patients in the high-exercise group, who were estimated to be at the same stage of dementia development, had less Aβ, which was found ex-

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**Dominantly Inherited Alzheimer’s Disease (DIAD)**

*DIAD* - a rare form of Alzheimer’s disease that causes memory loss and dementia in people typically in their 30s to 50s. The disease affects less than 1% of the total population.

DIAD, also known as *autosomal dominant Alzheimer’s disease*, is a form of dementia caused by rare, inherited gene mutations. A person born with one of these gene mutations not only develops Alzheimer’s Disease typically before the age of 60, but has a 50-50 chance of passing the mutation on to each of his or her children. The disease is identifiable through genetic testing.

DIAD is not the more common form of Alzheimer’s that appears later in life.

Individuals should consider learning more about DIAD if:

- he/she is at least 18 years of age, AND, have a parent or other relative with dominantly inherited Alzheimer’s disease (i.e., someone with a genetic mutation on one of three genes: PSEN1, PSEN2, APP); and,
- the family has two generations of Alzheimer’s disease and symptoms that starts younger than age 60.

**Source:**
Washington University School of Medicine, St. Louis, MO.

**See:** [https://dian.wustl.edu](https://dian.wustl.edu).
In advanced Parkinson’s Disease, regular exercise can slow decline

For patients with Parkinson’s Disease (PD), regular exercise is associated with significant positive effects on health-related quality of life (HRQL), especially in advanced PD, says a study in the Journal of Parkinson’s Disease (March 29, 2017).

Dr. Miriam Rafferty, Ph.D., from Northwestern University in Chicago, and colleagues, identified a cohort of 3408 National Parkinson Foundation Quality Improvement participants with three visits, and compared health-related quality of life and mobility between self-reported regular exercisers and PD patients who did not exercise.

The changes in health-related quality of life and mobility associated with 30-minute increases in exercise were quantified across PD severity.

Researchers found that, compared with non-exercisers, consistent exercisers and patients who started to exercise regularly after their baseline visit, had smaller declines in HRQL and mobility after 2 years. Non-exercisers, per-year, had worsening points on the Parkinson’s Disease Questionnaire and on the Timed Up and Go.

Slower declines in health-related quality of life (HRQL) and mobility were seen by increasing exercise by 30 minutes per week. In advanced versus mild PD, the benefit of exercise was greater on HRQL.

“Consistently exercising and starting regular exercise after baseline was associated with small but significant positive effects on HRQL and mobility changes over 2 years,” the authors wrote.

Over 6,600 new PD cases per year in Canada

Parkinson’s disease affects 1 in every 500 people in Canada. Over 100,000 Canadians are living with Parkinson’s today and approximately 6,600 new cases of PD are diagnosed each year in Canada (based on annual incidence of 20 new cases per 100,000 people).

Investigators led by Dr. Laura Baker, Ph.D., also of Wake Forest University in Winston-Salem, investigated cerebral blood flow and cognitive outcomes from tests that measured executive function and working memory. These investigators also measured change in blood-sugar concentration and cerebral spinal fluid glucose levels during an oral glucose tolerance test, as well as change in insulin sensitivity. They found that for patients in the exercise group, executive function and cerebral blood flow were improved. These initial findings were presented a year earlier at the AAIC (2016) meeting held in Toronto.

Exercise and better neuron communication

Dr. Hugenschmidt also carried out a network analyses on a subset of these patients. Her analysis, presented at the 2017 AAIC meeting, found that for the patients who experienced an improvement in executive function, there was also improvement in connectivity between brain regions in the frontal cortex, an area believed to control executive function. “These new results,” said Dr. Hugenschmidt, “provide a neurobiological reason for the improved cognition. . . It seems that it’s because the neurons are communicating with each other in a more tightly connected way.”

Lower Aβ levels

In other research presented at the meeting, Dr. Brown’s group added to their previous work showing a relationship between physical activity and lower brain Aβ levels. With the recent advent of tau PET tracers, they were able to show that in a group of 88 cognitively healthy older adults, mean cortical tau burden was higher in those reporting low to moderate physical activity than in those reporting a high level of physical activity (P = .02).

It is believed that brain amyloid deposition precedes the pathological tau accumulation and that density of neurofibrillary tangles (composed of tau) is more closely associated with cognitive impairment and neurodegeneration, said Dr Brown. “So the investigation of both of these biomarkers in relation to physical activity is vital.” Exercise and other lifestyle interventions will be tested for their impact on cognitive function in a new 2-year, $20 million clinical study trial Dr. Brown said. The study will include 2500 older adults with no cognitive symptoms who are at increased risk for later cognitive decline.

In addition to physical activity, the study will assess nutritional counselling and modification, cognitive and social stimulation, and improved self-management of medical conditions. The study will begin recruiting in 2018.

Exercise and/or medications?

Evidence already exists that physical activity and other positive lifestyle changes bring about improvements in mental health that are similar to, and, in some cases, exceed the effects currently achieved with medications, said Dr. Keith Fargo, Ph.D, director of scientific programs and outreach, Alzheimer’s Association. “Exercise is relatively inexpensive and is almost uni-
versally accessible, he said. “Anyone can exercise, even those who have disabilities . . . Exercise does not have the side effects associated with some medications,” he noteded. “No one has ever had a side effect from eating a salad or running an extra 30 minutes.”

Of all lifestyle interventions aimed at preventing dementia, there seems to be more evidence for physical activity, possibly because it more directly affects cerebral blood flow. “When you give someone, say, 6 months of exercise, 6 months of moving their legs, you see more profound effects in their cognition than if they did 6 months of cognitive training, and that’s completely fascinating,” explained Dr. Hugenschmidt.

**Strength training and aerobics**

Nor does the activity have to be strenuous, Dr. Hugenschmidt pointed out. “Accumulating evidence suggests that yoga, tai chi, and mindful movement practices may also be helpful. . . and some studies have found positive effects from combining strength training with aerobics. . . I definitely think it’s within the realm of possibility that you need more than one kind of exercise and that combining them could be helpful,” said Dr. Hugenschmidt.

**Best therapeutic there is!**

Another expert conducting research in the field of dementia prevention, Steven Braithwaite, Ph.D., chief scientific officer, Alkahest, Inc. *, agreed that exercise “is the best therapeutic there is for Alzheimer’s disease. Of all the possible lifestyle changes, I think exercise has been proven to have the biggest impact on dementia risk,” Braithwaite added.

**High-intensity exercise boosts memory suggests McMaster University research**

The health advantages of high-intensity exercise are widely known but new research from McMaster University points to another major benefit: better memory.

**Implications**

The findings could have implications for an aging population which is grappling with the growing problem of catastrophic diseases such as dementia and Alzheimer’s.

Scientists have found that six weeks of intense exercise - short bouts of interval training over the course of 20 minutes - showed significant improvements in what is known as high-interference memory, which, for example, allows us to distinguish our car from another of the same make and model.

The study was published in the Journal of Cognitive Neuroscience.

The findings are important because memory performance of the study participants, who were all healthy young adults, increased over a relatively short period of time, say researchers.

**Increased neurotrophic factor**

They also found that participants who experienced greater fitness gains also experienced greater increases in BDNF (brain-derived neurotrophic factor)*, a protein in the CNS that supports the growth, function and survival of brain cells.

“Improvements in this type of memory from exercise might help to explain the previously established link between aerobic exercise and better academic performance,” says Jennifer Heisz, an assistant professor in the Department of Kinesiology at McMaster and lead author of the study.

“At the other end of our lifespan, as we reach our senior years, we might expect to see even greater benefits in individuals with memory impairment brought on by conditions such as dementia,” she says.

For the study, 95 participants completed six weeks of exercise training, combined exercise and cognitive training or no training (the control group which did neither and remained sedentary). Both the exercise and combined training groups improved performance on a high-interference memory task, while the control group did not.

Researchers measured changes in aerobic fitness, memory and neurotrophic factor, before and after the study protocol.

**Exercise and cognitive training**

The results reveal a potential mechanism for how exercise and cognitive training may be changing the brain to support cognition, suggesting that the two work together through complementary pathways of the brain to improve high-interference memory.

Researchers have begun to examine older adults to determine if they will experience the same positive results with the combination of exercise and cognitive training.

“One hypothesis is that we will see greater benefits for older adults given that this type of memory declines with age,” says Heisz. “However, the availability of neurotrophic factors also declines with age and this may mean that we do not get the synergistic effects.”

**Reference**

* Jennifer Heisz, Ilana Clark, Katiya Bonin, Emily Paolucci, Bernadeta Michalski, Suzanna Becker, Margaret Fahnestock, The Effects of Physical Exercise and Cognitive Training on Memory and Neurotrophic Factors, Journal of Cognitive Neuroscience; Vol. 29(11); p.1895-1907; November, 2017. DOI: <10.1162/jocn_a_01164>.

* Brain Derived Neurotrophic Factor (BDNF) acts on certain neurons of the central nervous system and peripheral nervous system, helping to support the survival of existing neurons, and encouraging the growth and differentiation of new neurons and synapses. In the brain, BDNF is active in the hippocampus, cortex, and basal forebrain - areas vital to learning, memory, and higher thinking. It is also expressed in the retina, motor neurons, the kidneys, saliva, and the prostate. BDNF is also important for long-term memory.
Can a blend of dietary supplements slow the progress of catastrophic neurological diseases?

A dietary supplement containing a blend of thirty vitamins and minerals - all natural ingredients widely available in health food stores - has shown remarkable anti-aging properties that can prevent and even reverse massive brain cell loss, according to new research from McMaster University in Hamilton, Ontario.

It’s a mixture scientists believe could someday slow the progress of catastrophic neurological diseases such as Alzheimer’s, ALS and Parkinson’s.

“The findings are dramatic,” says Jennifer Lemon, research associate in the Department of Biology and a lead author of the study. “Our hope is that this supplement could offset some very serious illnesses and ultimately improve quality of life.”

‘The formula’

The formula, which contains common ingredients such as vitamins B, C and D, folic acid, green tea extract, cod liver oil and other nutraceuticals,* was first designed by scientists in McMaster’s Department of Biology in 2000.

A series of studies published over the last ten-and-a-half years have shown its benefits in mice, in both normal mice and those specifically bred for such research because they age rapidly, experiencing dramatic declines in cognitive and motor function in a matter of months.

The mice used in this study had widespread loss of more than half of their brain cells, severely impacting multiple regions of the brain by one year of age, the human equivalent of severe Alzheimer’s disease.

The mice were fed the supplement on small pieces of bagel each day over the course of several months. Over time, researchers found that it completely eliminated the severe brain cell loss and abolished cognitive decline.

“The research suggests that there is tremendous potential with this supplement to help people who are suffering from some catastrophic neurological diseases,” says Lemon, who conducted the work with co-author Vadim Aksenov, a post-doctoral fellow in the Department of Biology at McMaster. “We know this because mice experience the same basic cell mechanisms that contribute to neuro-degeneration that humans do.”

Enhanced vision & smell, better motor activity

In addition to looking at the major markers of aging, they also discovered that the mice on the supplements experienced enhancement in vision and, most remarkably, in the sense of smell (loss of which is often associated with neurological disease), and improved balance and motor activity.

The next step in the research is to test the supplement on humans, likely within the next few years, and target those who are dealing with neurodegenerative diseases.

Reference

* Nutraceutical is a broad umbrella term that is used to describe any product derived from food sources with extra health benefits in addition to the basic nutritional value found in foods. The term “nutraceutical” combines two words - “nutrient” (a nourishing food component) and “pharmaceutical” (a medical drug).

Peanuts, tree nuts associated with reduced cardiovascular risk

Consumption of peanuts and tree nuts is associated with lower risk for cardiovascular disease, states a long-term study presented in a recent issue of the Journal of the American College of Cardiology (JACC).

20-year follow-up

Researchers studied over 200,000 health professionals, who completed multiple food-frequency questionnaires during more than 20 years’ follow-up. In that time, there were 14,100 cases of major cardiovascular disease (CVD), defined as myocardial infarction, stroke, or fatal CVD.

After multivariable adjustment, CVD risk was 14% lower among participants who ate one serving of nuts at least twice weekly, relative to those who almost never or never consumed nuts.

CVD risk reductions

In particular, significant risk reductions were found for peanuts, tree nuts, and walnuts (which are nutritionally distinct from other tree nuts). Risk decreased in a dose-dependent manner as intake increased.

Of note, peanut butter was not associated with CVD risk.

A journal editorial concluded that “raw nuts - if possible unpeeled and otherwise unprocessed - may be considered as natural health capsules that can be easily incorporated into any heart-protective diet to further cardiovascular well-being and promote healthy aging.”

In a ‘nut shell’!

Tree nuts include, but are not limited to, almonds, Brazil nuts, cashews, chestnuts, hazelnuts, macadamia nuts, pecans, pistachios, pine nuts, shea nuts and walnuts.

Tree nut allergies are distinct from peanut allergy, as peanuts are legumes, whereas a tree nut is a hard-shelled nut.
Depression linked to a higher long-term risk of early death

Despite increased awareness of mental illness, depression remains strongly linked to a higher risk of early death - a risk that has increased for women in recent years - according to results from the 60-year-long Stirling County Study that was published recently in the Canadian Medical Association Journal (CMAJ).

“There is less stigma now associated with depression. . . and the availability of better treatments;” but, “. . . despite these positives, depression’s link to mortality still persists” said Dr. Stephen Gilman of the U.S. National Institutes of Health. “At first, the association was limited to men, but in later years it was seen for women as well.”

Canadian Maritimes study

The Stirling County Study, begun in 1952 in the Canadian Maritimes, and is well-known internationally as one of the first community-based studies on mental illness. A researcher from the original study, Dr. Jane Murphy with Massachusetts General Hospital and Harvard Medical School in Boston, is a co-author.

An international team of researchers looked at 60 years of mental health data on 3,410 adults during 3 periods (1952-67; 1968-90; and 1991-2011).

The research centred in a region in Atlantic Canada and linked the data to deaths in the Canadian Mortality Database. The team found that the link between depression and an increased risk of death was observed in all decades of the study among men, whereas it emerged among women only in the 1990s during the third research period - 1991 to 2011.

Remission of depression

The risk of death associated with depression appeared strongest in the years following a depressive episode, leading the authors to speculate that risk could be reversed by achieving remission of depression.

At enrollment, the mean age of participants in the study was about 49 years. “The lifespan for young adults with depression at age 25 was markedly shorter over the 60-year period, ranging from 10 to 12 fewer years of life in the first group, 4 to 7 years in the second group and 7 to 18 fewer years of life in the 1992 group,” notes Dr. Ian Colman, Canada Research Chair, University of Ottawa. “Most disturbing is the 50% increase in the risk of death for women with depression between 1992 and 2011.”

Though depression has also been linked with poor diet, lack of exercise, smoking and alcohol consumption, the researchers did not explain in this study the increased risk of death that is linked to depression.

Increased risk explained

Societal change may help explain the emergent risk of death for women with depression. “During the last 20 years of the study in which women’s risk of death increased significantly, roles have changed dramatically both at home and in the workplace, and many women shoulder multiple responsibilities and expectations,” explains Dr. Colman.

The authors suggest that patients should be monitored for mood disturbances, especially recurrent episodes of depression, so that treatment and support could be offered.

Study limitations included a long interval between participant interviews which prevented determining the exact timing of depression and the participants’ experiences of recurrent episodes between interviews.

International involvement

The study was conducted by researchers from the U.S. National Institute of Child Health and Human Development in Bethesda, Maryland; the Johns Hopkins Bloomberg School of Public Health in Baltimore; the Massachusetts General Hospital and Harvard Medical School; the Amherst College, Amherst, Mass.; Dalhousie University Faculty of Medicine, Halifax; and the University of Ottawa.

Reference


Brisk walks crucial to cardiovascular health

It is not the amount of time spent sitting that matters, but the extent of physical activity that is essential in reducing the risk of elderly women developing cardiovascular disease (CVD), according to a study from Örebro University in Sweden, originally a college of Uppsala University.

Reality of a sedentary life

“We studied women over 65, the least active group of the population (who) run a high risk of developing CVD,” says Fawzi Kadi, Professor at Örebro University.

“The study shows how important it is to encourage more physical activity. We are not talking a slow, everyday pace, but at least one brisk walk or other physical activity requiring some exertion,” says Andreas Nilsson, a researcher at Örebro.

Over 120 women took part in the study. They had a medical exam and, over the course of one week, their physical activity was measured using an accelerometer.

“Our study points to the reality that the negative health effects of a sedentary lifestyle decreases with the extent of physical activity,” says Fawzi Kadi. “This means that if one person is jogging while another is doing less strenuous activities, the first person runs a lower risk of CVD - regardless of the extent of his/her sedentariness.

“Given our findings, increased amounts of physical activity with an emphasis on increased time in moderate-to-vigorous activity should be recommended in order to promote a favorable metabolic health profile in older women,” Kadi concludes.

Source

• Andreas Nilsson, Fawzi Kadi, et al., Physical activity, not sedentary time per se, influences metabolic risk in elderly community-dwelling women, PLOS ONE; vol. 12(4); April, 2017.
See: DOI: 10.1371/journal.pone.0175496.

Canadian Nursing Home
Inter-facility communication key to preventing drug-resistant pathogens

Poor communication between other care facilities can pave the way for outbreaks of infection, according to research on the spread of a drug-resistant bacterium.

The Oregon State University, and the Oregon Health & Science University - College of Pharmacy, teamed with the Oregon Health Authority and other collaborators on a two-year study of Acinetobacter baumannii, an opportunistic, multi-drug-resistant pathogen associated primarily with infections among those who have compromised immune systems and are in health care facilities - including nursing homes.

Researching multiple sites in the U.S. Pacific Northwest, researchers identified 21 cases, including 16 isolates, of A. baumannii that contained a rare gene responsible for resistance to carbapenem antibiotics.*

Interfacility communication

The patients’ transfer history among the studied facilities, and the isolates’ genetic profiles illustrated how the organism spreads from place to place aided by a lack of interfacility communication that patients who were infected or colonized by A. baumannii were being transferred.

Jon Furuno, co-author on the study and an associate professor in the College of Pharmacy, noted that the findings support a recent law requiring written notification, from the discharging facility to the receiving facility, anytime a patient carrying a multi-drug-resistant organism, is transferred.

Extensively drug-resistant A. baumannii can contain many antibiotic resistant genes that can be transmitted to other organisms, Furuno added. “It just makes sense that you would want to alert a receiving facility if patients have a specific drug-resistant organism... The discharging facility needs to include that information with the discharge summary or somewhere on the chart, and the receiving facility needs to know where to look for it.”

‘Contact precautions’

The lead author of the two-year study, Genevieve Buser, an infectious disease specialist who worked as an Epidemic Intelligence Service officer for the Centers for Disease Control and Prevention, was based at the Oregon Health Authority when the study was done. She emphasizes that communication can ensure appropriate contact precautions are taken.

“A chain of transmission can be avoided if receiving facility staff know about a patient’s multi-drug-resistant organism status,” Buser said. “This outbreak might not have been identified if not for a new, limited, voluntary surveillance system in Oregon and an astute infection preventionist.”

* Acinetobacter baumannii is a usually short, round, rod-shaped gram-negative bacterium. It can be an opportunistic pathogen in humans, affecting people with compromised immune systems, and is becoming increasingly important as a health-facility-derived (nosocomial) infection where it is almost exclusively isolated.

* Carbapenems are a class of beta-lactum, broad spectrum antibiotics which inhibit the synthesis of the cell wall of the pathogen and are known to be most effective against gram negative infections. Used in combination with other agents, Carbapenems are a mainstay of therapy in patients/ residents with facility-acquired infections.

Reference


See also:

• Infection Control & Hospital Epidemiology, 2017. DOI: 10.1017/ice.2017.189.

New eye technology detects symptoms of Alzheimer’s before symptoms expressed

Scientists may have overcome a major roadblock in the development of therapies for Alzheimer’s by creating a new technology to observe - in the back of the eye - progression of the disease before the onset of symptoms. Clinical trials were initiated last July (2016) to test the technology in humans according to a paper published in Investigative Ophthalmology & Visual Science.

The paper builds upon previous work that detects changes in the retina of mice predisposed to develop Alzheimer’s.

Early detection critical

Early detection of Alzheimer’s is critical for two reasons: “First, effective treatments need to be administered well before patients show actual neurological signs,” said author Robert Vince, Ph.D., of the Center for Drug Design at the University of Minnesota. “Second, since there are no available early detection techniques, drugs currently cannot be tested to determine if they are effective against early Alzheimer’s disease. . . An early diagnostic tool like ours could help the development of drugs as well.”

Looking through the eye to see the brain is a key advantage of the new technology. “The retina of the eye is not just ‘connected’ to the brain - it is part of the central nervous system,” explained author Swati More, Ph.D., also of the Center for Drug Design.

While the brain - and retina - undergo similar changes due to Alzheimer’s disease, “unlike the brain, the retina is easily accessible, thereby making changes in the retina easier to observe.”

“We saw changes in the retinas of Alzheimer’s mice before the typical age at which neurological signs are observed,” said Dr. More. “The results are close to our best-case scenario for outcomes of this project.”

Reference


See: <http://iovs.arvojournals.org/article.aspx?articleid=2530713>
Uncovering the link between diet, inflammation and dementia

Researchers believe they have uncovered a key piece of the puzzle in the connection between diet and dementia. They linked a specific dietary pattern to blood markers of inflammation.

In addition, they showed that in elderly adults who followed such a dietary pattern, the volume of brain gray matter was less - and they had worse visuo-spatial cognitive function.

Dietary factors and lower Alzheimer’s risk

“We found that people who consume less Omega 3, less calcium, vitamin E, vitamin D, and vitamin B2 and B5 have more inflammatory biomarkers,” explained study investigator, Ms. Yian Gu, Ph.D., Columbia University Medical Center, and Taub Institute for Research on Alzheimer’s Disease and the Aging Brain in New York City.

An inflammatory dietary pattern, said Dr. Gu, “is bad for both brain and cognition.”

The study was presented at the Alzheimer’s Association International Conference (AAIC) held in London in mid-July, 2017.

Evidence cited by Dr. Gu suggested that dietary factors, such as fish, nuts, omega-3 polyunsaturated fatty acids, and folate, as well as Mediterranean-type diets, are associated with lower risk for Alzheimer’s disease and better brain health in the elderly; additional evidence, she said, shows that many foods and nutrients modulate or control inflammatory processes.

Inflammation and AD risk

Other studies have linked chronic inflammation to an increased risk for Alzheimer’s disease. Dr. Gu’s group previously showed an association between increased C-reactive protein (CRP)* and Interleukin-6 (IL6) levels* as well as worse cognition and smaller brain volumes.

But none of this research addressed whether diet affects brain and cognitive health by modulating inflammation. “No study has formally tested whether the relationship of diet with cognition, or with the brain, is actually because of inflammation,” Dr. Gu stated.

The new cross-sectional study included 330 elderly New York City adults from the Washington Heights-Inwood Community Aging Project imaging study. With these participants, researchers carried out structural MRI scans and measured levels of the inflammatory biomarkers CRP (C-reactive protein) and IL6 (Interleukin-6).

“Inflammation-related Nutrient Pattern” (INP)

Study participants completed a 61-item food frequency questionnaire that asked about nutrient intake during the past year. From this information, the researchers used a statistical model to create the Inflammation-related Nutrient Pattern (INP).

“The INP is basically a linear combination of 24 nutrients, each with a different weight on the INP,” explained Dr. Gu. “For example, omega-3 is negatively ‘loaded’ - which is similar to ‘correlated’ - on this pattern. Lower consumption of omega-3 will contribute to a higher INP score.”

Study participants also underwent neuropsychological testing that assessed memory, language, executive speed, and visuospatial function. From these test scores, the researchers calculated a composite mean cognition score for each participant.

The study showed that the INP was positively correlated with C-reactive protein (CRP) level and interleukin-6 (IL6).

The analysis uncovered a significant association between INP and visuospatial function (P = .015) and total gray matter volume (P = .002) after adjusting for age, sex, race/ethnicity, APOE4 status, calorie intake, body mass index, and vascular comorbidity.

The researchers determined that having a smaller volume of brain grey matter might help explain why those who consume more inflammatory nutrients have worse visuospatial cognition.

Brain grey matter contains most of the brain’s neuronal cell bodies, and includes regions of the brain involved in muscle control, and sensory perception such as seeing and hearing, emotions, memory, speech, decision making, and self-control.

This study is important because “now we have a linkage to measurable biological differences,” said Keith Fargo, Ph.D., director of scientific programs at the U.S. Alzheimer’s Association. “If your cognition is poor, we know something has to be going on. But it’s not clear what’s going on. Now, however, we can measure the level of these proteins in the body,” i.e., the INP.

Possible targets

These new findings suggest that interventions that decrease inflammatory markers may be helpful. “It gives us some ideas of what pathways might be involved,” said Dr. Fargo. “Once that is known, it may be possible to intervene, not just through a healthier diet, but perhaps also with medications. At least, there may be some targets to work with,” he said.

Source:


* CRP (C-reactive protein) - is a substance produced by the liver in response to inflammation. A high level of C-reactive protein in the blood is a marker for inflammation, which can be caused by a wide variety of conditions, from infection to cancer.

* Cytokines - a broad and loose category of small proteins that are important in cell signalling. The release of cytokines has an effect on the behaviour of cells around them. Cytokines include chemokines, interferons, interleukins, lymphokines, and are especially important in the immune system.

* IL6 (Interleukin-6) - an endogenous chemical substance active in inflammation. Besides being an immune protein, it is also a pyrogen; that is, it originates from within an organism, tissue or cell.
Researchers may have discovered a way to use patient’s sense of smell to treat Alzheimer’s disease before symptoms develop

Researchers at Columbia University Medical Center and the New York State Psychiatric Institute may have discovered a way to use a patient’s sense of smell to treat Alzheimer’s disease before it ever develops.

**Impaired sense of smell an early sign of AD**

Having an impaired sense of smell is recognized as one of the early signs of cognitive decline, before the clinical onset of Alzheimer’s disease. The researchers at Columbia and NYSPI have found a way to use that effect to determine if patients with mild cognitive impairment may respond to cholinesterase inhibitor drugs to treat Alzheimer’s disease. The findings were published online, in early November, in the *Journal of Alzheimer’s Disease*.

Cholinesterase inhibitors, such as donepezil, enhance cholinergic function by increasing the transmission in the brain of the neurotransmitter, acetylcholine, the primary transmitter of nerve impulses in the brain. Cholinergic function is impaired in individuals with Alzheimer’s disease.

Cholinesterase inhibitors, which block an enzyme that breaks down acetylcholine, have shown some effectiveness in improving the cognitive symptoms of Alzheimer’s disease. However, they have not been proven effective as a treatment for individuals with mild cognitive impairment (MCI), a condition that markedly increases the risk of Alzheimer’s disease.

**Cholinesterase inhibitors can make a difference**

“We know that cholinesterase inhibitors can make a difference for Alzheimer’s patients, so we wanted to find out if we could identify patients at risk for Alzheimer’s who might also benefit from this treatment,” said D.P. Devanand, M.B.B.S., M.D., professor of psychiatry, scientist in residence, director of the Memory Disorders Clinic and the Late Life Depression Clinic at New York State Psychiatric Institute.

“Since odor identification tests have been shown to predict progression to Alzheimer’s, we hypothesized that these tests would also allow us to discover which patients with MCI would be more likely to improve with donepezil treatment.”

**Odour identification testing**

In this year-long study, 37 participants with MCI underwent odour identification testing with the University of Pennsylvania Smell Identification Test (UPSIT). The test was administered before and after using an atropine nasal spray that blocks cholinergic transmission.

The patients were then treated with donepezil for 52 weeks, and were periodically re-evaluated with the UPSIT (smell test) and with memory and cognitive function tests. Those who had a greater decline in UPSIT scores, indicating greater cholinergic deficits in the brain, after using the anticholinergic nasal spray test saw greater cognitive improvement with donepezil.

In addition, short-term improvement in odor identification, from baseline to eight weeks, tended to predict longer-term cognitive improvement with donepezil treatment over one year.

**Inexpensive strategies**

“These results, particularly if replicated in larger populations, suggest that these simple inexpensive strategies have the potential to improve the selection of patients with mild cognitive impairment who are likely to benefit from treatment with cholinesterase inhibitors like donepezil,” said Dr. Devanand.

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**Dry mouth could be side effect of certain medications**

For older adults, dry mouth (xerostomia) can be a common side effect of medications. Dry mouth means you don’t have enough saliva, or spit to keep the mouth wet. The condition can lead to problems chewing, eating, swallowing, and even talking. Dry mouth also puts one at higher risk for oral infections and tooth decay.

**Medications and dry mouth**

There’s much that is misunderstood about the connection between medications and dry mouth in elders. Recently, researchers examined 52 relevant studies to learn more and published their research in the *Journal of the American Geriatrics Society*.

The researchers reported that there are a number of medications linked to dry mouth. These include medications used to treat urinary incontinence, depression, insomnia, and anxiety, as well as diuretics used to treat high blood pressure. In fact, medications used to treat urinary incontinence were nearly six times more likely to cause dry mouth than a placebo.

**Monitor medications**

The researchers suggested that healthcare providers should regularly monitor and review all medications to identify potential side effects and to adjust doses or change medications when necessary.

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**Reference**

- D.P. Devanand, et al., Change in Odor Identification Impairment is Associated with Improvement with Cholinesterase Inhibitor Treatment in Mild Cognitive Impairment, *Journal of Alzheimer’s Disease*, 2017; vol. 60(4); p.1525-31; DOI: 10.3233/JAD-170497
Loss of multiple senses in older adults predictive of overall health and ability to function

Researchers have primarily focused on what happens after people lose one or two of their senses. However, it is known that losing more than two senses occurs frequently for older adults.

Until now, no studies have examined how losing multiple senses affects older adults. Recently, researchers designed a study to focus on the consequences of what happens when multiple senses are lost.

When the five senses (hearing, vision, smell, touch, and taste) begin to dim or are lost with age, one faces challenges dealing with everyday life; losing one’s senses can also cause serious health problems.

Researchers have mainly focused on what happens after the loss one or two senses. However, we know that losing more than two senses occurs frequently.

Until now, no studies have examined how losing multiple senses affects older adults; to learn more, a research team from the University of Chicago designed a study to focus on just that. The study was published in the Journal of the American Geriatrics Society.

The researchers conducted interviews among 3,005 older adults between the ages of 57 and 85. They checked participants’ abilities to hear, see, smell, touch, and taste. They also assessed the participants’ mobility, health behaviours, chronic diseases, cognitive function (the ability to think and make decisions), and BMI (body-mass index, a measure for obesity that compares height to one’s weight).

Reassessed 5 years later

Five years later, the researchers reassessed those participants who were still living in order to measure:
• mobility (measured with a timed 10-foot long walk);
• degree of difficulty performing eight key daily activities, including bathing, feeding and shopping for themselves;
• doing light housekeeping;
• managing their own finances;
• physical activity (measured with a tracking device used for research purposes);
• mental health status; and
• overall health.

Poor mobility performances

The researchers reported that the more sensory losses older adults experienced, the worse they performed on the mobility test. Participants with greater sensory problems were more likely to have trouble performing two or more daily activities.

Women, older participants, smokers, and people with more chronic illnesses had higher levels of disability than other participants.

After five years, the participants who had more sensory disabilities at the beginning of the study walked more slowly than participants who had fewer sensory problems.

Participants who were obese with BP and more chronic illnesses walked much slower than other participants.

Women, minorities, and people with less education also walked much slower than other participants.

People with more sensory losses at the beginning of the study also had:
• difficulty performing ADL’s;
• difficulty staying physically active;
• difficulty staying sharp mentally;
• overall worse health;
• unhealthy weight loss;
• increased risk for dying.

Close monitoring

The researchers concluded that older adults, including LTC residents with multiple sensory losses, should be closely monitored because they are at higher risk for poor health.

They also suggested that monitoring at-risk older adults sooner could help prevent problems such as cognitive impairment.

Reference


New treatment for osteoporosis provides better protection against fractures

A new treatment for osteoporosis provides major improvements in bone density and more effective protection against fractures than the current standard treatment.

These are the findings of a study, from the University of Gothenburg in Sweden, and the first that compares the effect of two osteoporosis medicines on fractures.

Significantly better protection

“With the new treatment, we could offer significantly better protection against fractures and could thereby help many patients with severe osteoporosis,” says study co-author Mattias Lorentzon, Professor of Geriatrics at the University of Gothenburg’s Institute of Medicine, and senior physician at Sahlgrenska University Hospital in Gothenburg, Sweden.

Many patients with severe osteoporosis and a high risk of fractures often cannot regain their original bone strength. They continue to have fractures even with treatment, according to current standards, with alendronate in tablet form every week.

Alendronate increases bone density by slowing the breakdown of bone and thereby decreasing fracture risk by 20-50%.

Many people with osteoporosis, especially elderly women, nonetheless continue to suffer broken bones, sometimes just by falling from a standing position. The fractures lead to disability and suffering, and with hip and vertebral fractures, often premature death.

The current study included 4,093 women, of an average age of 74 years, with osteoporosis and previous fractures. They were...
randomly allocated to 12 months’ treatment with either alendronate or the new medication, romosozumab, an antibody that blocks the substance, sclerostin, which slows the new formation of bone. Treatment with romosozumab thereby leads to rapid new bone formation. After the first 12 months, all patients received alendronate for 12 months.

48% lower with romosozumab

The risk of vertebral fracture in the course of the study proved to be 48 per cent lower for those who received romosozumab compared with the group that received alendronate the whole time. The proportions suffering fractures in the two groups were 6.2% and 11.9%, respectively.

The risk of a clinical fracture, such as an arm or leg fracture, was 27 per cent lower in the group that received romosozumab. Here, the proportions suffering fractures in the different groups were 9.7% and 13.0 %, respectively.

The proportion of side effects and serious side effects was generally just as common in both of the treatment groups. However, it was observed that serious cardiovascular events, such as heart attack or stroke, occurred in 2.5% of the patients that received romosozumab compared with 1.9% in the group that received alendronate during the first 12 months of the study.

Safety aspects considered

According to Dr. Mattias Lorentzon, the safety aspects of the new medication need to be studied further. However, an earlier study of nearly twice the size showed that romosozumab does not provide a greater risk of cardiovascular events compared with a placebo.

“With romosozumab in the treatment arsenal, we could prevent many fractures among the high-risk patients,” Dr. Lorentzon concludes.

Reference


Stopping one’s daily aspirin therapy shown to enhance second heart attack and stroke risk

For heart attack survivors and people at high risk for one, a low-dose aspirin is part of the daily routine to prevent a heart attack or stroke. But for those who don’t stick to that routine, the rate of heart attacks, strokes or deaths from one of those causes goes up almost 40 percent, a new study shows.

Stopping long-term, low-dose aspirin therapy may increase one’s risk of suffering a cardiovascular event, according to research from Sweden and described in Circulation.

Inhibits clotting

Aspirin, taken in low doses, is used to help reduce the risk of recurrent heart attack or stroke; it inhibits clotting, thereby lowering the risk of cardiovascular events.

Between 10% and 20% of heart attack survivors stop daily aspirin use within the first three years following their event. In broader settings, discontinuation rates of up to 30% and poor aspirin compliance in up to 50% of patients have been reported.

Effects of terminating therapy

To study the health effects of stopping aspirin therapy, Swedish researchers examined the records of over 600,000 adults who took low-dose aspirin for heart attack and stroke prevention. Participants were older than 40, cancer-free and had an adherence rate of greater than 80% in the first year of treatment. Some 54% were using aspirin to prevent recurrent events (or secondary prevention), while the rest were using it to prevent first events (primary prevention).

During roughly 3 years’ follow-up, patients who discontinued aspirin had a 37% increased risk for the composite of myocardial infarction, stroke, or CV death relative to those who adhered to treatment. In the secondary-prevention group, this translated to one additional CV event per year in every 36 patients who stopped aspirin.

In the primary-prevention group, this meant one additional CV event per year in every 146 patients who stopped treatment.

Increased clotting levels

“Low-dose aspirin therapy is a simple and inexpensive treatment,” said Johan Sundstrom, M.D., lead author and professor of epidemiology at Uppsala University in Sweden. “As long as there’s no bleeding or any major surgery scheduled, our research shows the significant public health benefits that can be gained when patients stay on aspirin therapy.”

“Rebound effect”

Studies have suggested patient’s experience a “rebound effect” after stopping aspirin treatment, which is possibly due to increased clotting levels from the loss of aspirin’s blood-thinning effects.

Because of the large number of patients on aspirin and the high number who stop their aspirin therapy, the importance of a rebound effect may be significant, Sundstrom said.

“We hope our research may help healthcare providers make informed decisions on whether or not to stop aspirin use,” Sundstrom concluded.

Reference

Ultrasound promising in treating Parkinson’s tremor

An initial test to determine if a scalpel-free form of brain surgery can reduce tremor caused by Parkinson’s disease has produced encouraging results. Further research is warranted, the researchers conclude in a paper published in the Journal of the American Medical Association.

The small pilot study was led by Jeff Elias, M.D., of the University of Virginia School of Medicine (UVA), and also was conducted at the Swedish Neuroscience Institute in Seattle.

Twenty-seven participants with tremor-dominant Parkinson’s disease were enrolled in the study. The research team randomly assigned 20 to be treated with focused ultrasound waves on their brains, while the others received a fake procedure, to account for any potential placebo effect. (All were offered the opportunity to have the actual procedure). All the participants had tremor that had resisted medical treatment, and all continued taking their existing Parkinson’s medication.

62% improvement

Participants who received the ultrasound procedure had a 62% median improvement in their hand tremor three months later.

Additional testing is needed to better establish the effectiveness of focused ultrasound for Parkinson’s tremor, the researchers concluded.

The median age of trial participants was 67.8 years, and 26 were male. The most significant side effects reported were mild numbness on one side of the body, which improved, and numbness of the face and finger, which were persistent.

Two subjects also experienced partial weakness that recovered or improved during the study. The procedure has since been modified to mitigate this risk of weakness, the researchers say.

Potential role of ultrasound

Researchers are also evaluating focused ultrasound’s potential for treating many other conditions, including breast cancer, brain tumors, epilepsy and pain.

Researchers believe that a larger, multicenter study is needed to better define the potential role of focused ultrasound in managing Parkinson’s disease.

“Our findings suggest that the patients likely to benefit from this approach are those for whom tremor reduction is enough to improve their quality of life,” said UVA researcher Binit Shah, MD.

Reference


Focused ultrasound - What it is and how it works

Focused ultrasound works by focusing sound waves inside the body to generate a tiny hot spot, much like a magnifying glass focuses light. By controlling this process, researchers can interrupt faulty brain circuits or destroy unwanted tissue. Unlike traditional brain surgery, there is no need to drill or cut into the skull.

Obesity increases dementia risk says new study

People who have a high body mass index (BMI) are more likely to develop dementia than those with a normal weight, according to a new study led by researchers from University College London (UCL). The study analysed data from 1.3 million adults living in the U.S. and Europe.

Lower weight at onset

Researchers also found that people near onset of dementia, who then go on to develop dementia, tend to have lower body weight than their dementia-free counterparts.

The BMI-dementia link observed in the studies is actually attributable to two processes, explained author, Mika Kivimäki of UCL. “One is an adverse effect of excess body fat on dementia risk. The other is weight loss due to pre-clinical dementia. For this reason, people who develop dementia may have a higher-than-average BMI some 20 years before dementia onset, but those close to overt dementia have a lower BMI than those who remain healthy.”

“The new study confirms both the adverse effect of obesity as well as weight loss caused by metabolic changes during the pre-dementia stage,” explained Kivimäki.

Conflicting results

Previous research on how a person’s weight influences their risk of dementia has produced conflicting results. Some findings have suggested that being over-weight poses a higher dementia risk, while other studies link lower weight to an increased incidence.

In the UCL study, researchers from across Europe pooled data from 39 longitudinal studies from the U.S., the United Kingdom, France, Sweden, and Finland. A total of 1,349,857 dementia-free adults partook of these studies.

Higher BMI predicted dementia

Dementia was ascertained using electronic health records obtained from hospitalisation, prescribed medication and death registries. A total of 6,894 participants developed dementia during up to 38 years of follow-up.

Two decades before symptomatic dementia, higher BMI predicted dementia occurrence: each 5-unit increase in BMI was associated with a 16-33% higher risk of this condition. (5 BMI units is 14.5 kg for a person 5’7” (170 cm.) tall, approximately the difference in weight between the overweight and normal weight categories, or between the obese and overweight categories).

In contrast, the mean level of BMI during the pre-clinical stage close to dementia onset was lower compared to that in participants who remained healthy.

This study suggests that maintaining a healthy weight could prevent, or at least delay, dementia.

Reference

Group exercises far better than individual workouts

Researchers found working out in a group lowers stress by 26% and significantly improves quality of life, while those who exercise individually put in more effort but experienced no significant changes in stress level and a limited improvement to quality of life, according to a study in The Journal of the American Osteopathic Association.

Superior to exercising alone

“The communal benefits of coming together with friends and colleagues, and doing something difficult, while encouraging one another, pays dividends beyond exercising alone,” said Dr. Dayna Yorks, lead researcher on the study. “The findings support the concept of a mental, physical and emotional approach to health that is necessary for student doctors and physicians.”

Dr. Yorks and her fellow researchers at the University of New England College of Osteopathic Medicine in Biddeford, Maine, recruited 69 medical students - a group known for high levels of stress and self-reported low quality of life - and allowed them to self-select into a twelve-week exercise program, either within a group setting or as individuals.

Every four weeks, participants completed a survey asking them to rate their levels of perceived stress and quality of life in three categories: mental, physical and emotional.

Those participating in group exercise spent 30 minutes weekly in a core strengthening and functional fitness program.

At the end of the 12 weeks, their mean monthly survey scores showed significant improvements in all three quality of life measures: mental (12.6%), physical (25%) and emotional (26%). They also reported a 26% reduction in perceived stress levels.

By comparison, individual fitness participants were allowed to maintain any preferred exercise regimen, which could include activities like running and weight lifting, but had to work out alone.

No significant changes

On average the solitary exercisers worked out twice as long, and saw no significant changes in any measure, except in mental quality of life (an increase of 11%).

Reference

• Dayna Yorks, Christopher Frothingham and Mark Schuenke, Effects of Group Fitness Classes on Stress and Quality of Life of Medical Students, The Journal of the American Osteopathic Association; 2017; vol. 117(11); 2017. DOI: 10.7556/jaoa.2017.140.

Study suggests sleep deprivation is an effective ‘anti-depressant’ for half of depressed patients

Total or partial sleep deprivation shown to produce clinical improvement in depression symptoms within 24 hours

Sleep deprivation, typically administered in a controlled setting, rapidly reduces symptoms of depression in roughly half of the depressed patients, according to an initial meta-analysis by researchers from the Perelman School of Medicine at the University of Pennsylvania.

Partial sleep deprivation (sleep for three to four hours followed by forced wakefulness for 20-21 hours) was equally as effective as total sleep deprivation (deprived of sleep for 36 hours), and medication did not appear to significantly influence these results. The study was published in September, 2017.

of the antidepressant effects of sleep deprivation, we still do not have an effective grasp on precisely how effective the treatment is and how to achieve the best clinical results,” said study senior author Philip Gehrman, Ph.D., an associate professor of Psychiatry at the Penn Sleep Center. “Our analysis precisely reports how effective sleep deprivation is and in which populations it should be administered.”

Timing of deprivation

Reviewing more than 2,000 studies, the team pulled data from a final group of 66 studies executed over a 36 year period to determine how response may be affected by the type and timing of sleep deprivation performed (total vs. early or late partial sleep deprivation), the clinical sample (having depressive or manic episodes, or a combination of both), medication status, and age and gender of the sample. They also explored how response to sleep deprivation may differ across studies according to how “response” is defined in each study.

“These studies show that sleep deprivation is effective for many populations,” said lead author Elaine Boland, Ph.D., a clinical associate and research psychologist. “Regardless of how the response was quantified, how the sleep deprivation was delivered, or the type of depression the subject was experiencing, we found a nearly equivalent response rate.”

Reduced depression severity

The authors note that further research is needed to identify precisely how sleep deprivation causes rapid and significant reductions in depression severity. Also, future studies are needed to include a more comprehensive assessment of potential predictors of treatment outcome to identify those patients most likely to benefit from sleep deprivation.

Reference

Physical frailty is common among the elderly and is strongly associated with cognitive impairment, dementia and adverse health outcomes such as disability, hospitalisation, and mortality.

A four-year study conducted by researchers from the National University of Singapore (NUS) showed that a combination of nutritional, physical and cognitive interventions can reverse physical frailty.

**Risk of cognitive impairment**

Associate Professor Ng Tze Pin, from the Department of Psychological Medicine at NUS, and the leader of the research team, said that their earlier research findings from the Singapore Longitudinal Aging Studies (SLAS) showed that physically frail elderly persons, compared to their robust counterparts, are eight times more likely to have cognitive impairment - and if they are not cognitively impaired, are more than five times at risk of becoming cognitively impaired on a follow-up three years later.

“In addition, physically frail elderly are up to 10 times more likely to become functionally disabled for ADLs, hospitalised and die than their more robust counterparts.

“With such compelling rationale, if it is possible to reduce, or even reverse, physical frailty in the elderly, we could greatly improve their quality of life,” Ng explained.

Professor Ng and team conducted a 4-year trial between 2010 and 2013, involving 250 community-living older persons in Singapore who showed signs of frailty.

“Our study shows that it is feasible to identify pre-frail and frail older persons in the community and care settings and provide them with lifestyle interventions to reverse frailty. We found that better nutrition, physical training and mental exercises can reverse frailty, enhance muscle strength and gait speed, reduce depressive symptoms and improve cognitive functioning. These interventions can go a long way to reducing the high prevalence of physical disability, hospitalisation and mortality in an aging society,” Prof. Ng explained.

Participants for the trial were recruited between 2009 to August, 2012 from various senior activity centres in Singapore. They were randomly allocated to receive lifestyle interventions in one of five groups for a period of six months.

Three groups of participants were provided with either physical training, nutritional enhancement, or cognitive training; a fourth group received a combination of all three interventions. The 5th group, was the control group, which did not receive any intervention. Assessment of all the participants’ frailty were made before the start of the interventions.

During the six-month trial, the participants’ progress was measured after three and six months. A follow-up assessment was also conducted 12 months after the initiation of interventions.

**Intervention results**

Researchers at the National University of Singapore found that the initial three types of interventions, as well as a combination of all three approaches, were able to reduce frailty and depressive symptoms, and improve cognitive functioning.

Professor Ng noted: “The important message from our studies is that frailty is not an inevitable part of aging. . . There is much that older people can do for themselves to avoid becoming frail and disabled, so it is vital that they pay attention to good quality diet and nutrition, engage in physical exercise, and participate in socially and cognitively stimulating activities.”

**Expanded frailty program**

The research team is currently working to develop and implement a ‘pilot frailty screening and multi-domain lifestyle intervention in a community-based programme.’

It is hoped that such a programme can help improve the physical, psychological and cognitive well-being of senior citizens.

### Source

- National University of Singapore (NUS),
  “Good nutrition, physical training and mental exercises can reverse physical frailty in the elderly,” June, 2017.

**Skipping breakfast linked to hardening of the arteries**

According to recent research from the American College of Cardiology, skipping breakfast is associated with an increased risk of atherosclerosis, or hardening and narrowing of arteries due to a build-up of plaque. Eating a healthy breakfast has been shown to promote greater heart health, including healthier weight and cholesterol.

While previous studies have linked skipping breakfast to the risk of coronary heart disease, this is the first study to evaluate the association between breakfast and the presence of sub-clinical atherosclerosis.

Prakash Deedwania, M.D., professor of medicine at the University of California, San Francisco, said that this study also provides clinically important information by demonstrating the evidence of sub-clinical atherosclerosis in people who skip breakfast.

“Between 20% and 30% of adults skip breakfast and these trends mirror the increasing prevalence of obesity and associated cardio-metabolic abnormalities,” Deedwania said.

“Poor dietary choices are generally made relatively early in life and, if unchanged, can lead to cardiovascular disease later on. Adverse effects of skipping breakfast can be seen early in childhood in the form of childhood obesity and although breakfast skippers are generally attempting to lose weight, they often end up eating more and unhealthy foods later in the day. . . Skipping breakfast can cause hormonal imbalances and alter circadian rhythms.

That breakfast is the most important meal of the day has been proven right in light of this evidence,” Deedwania concluded.
Blocki ng the ApoE4 genetic variant may be a therapeutic target for preventing Alzheimer’s

Nearly a quarter century ago, a genetic variant known as ApoE4 was identified as a major risk factor for Alzheimer’s disease - one that increases a person’s chances of developing the neurodegenerative disease by up to 12 times.

However, it was never clear why the ApoE4 genetic variant was so hazardous. When the ApoE4 protein is present, clumps of the protein, amyloid beta, accumulate in the brain. But such clumps alone do not kill brain cells or lead to characteristic symptoms of Alzheimer’s such as memory loss and confusion.

A different protein involved
A study, released in September, 2017, and led by researchers at Washington University School of Medicine in St. Louis, shows that the presence of ApoE4 exacerbates the brain damage caused by toxic tangles of a different Alzheimer’s-associated protein, namely, tau. In the absence of ApoE4, tau tangles did very little harm to brain cells.

The findings suggest that targeting ApoE4 could help prevent or treat the brain damage present in Alzheimer’s disease, for which there are currently no effective therapies. “Once tau accumulates, the brain degenerates,” said senior author David Holtzman, M.D., and head of the Department of Neurology at the Washington University School of Medicine.

“What we found was that when ApoE4 is there, it amplifies the toxic function of tau, which means that if we can reduce ApoE4 levels we may be able to stop the disease.”

Tauopathies
Alzheimer’s, which affects one in 10 people over age 65, is the most common example of a group of diseases called tauopathies. This group of diseases also includes chronic traumatic encephalopathy, which plagues professional boxers and football players, as well as several other neurodegenerative diseases.

To find out what effect the ApoE variants have on tauopathies, Holtzman and graduate student Yang Shi, and their colleagues, turned to genetically modified mice that carry a mutant form of human tau that are prone to forming toxic tangles.

Variants of human ApoE
They utilized mice that lacked their own version of the mouse ApoE gene or replaced it with one of the three variants of the human ApoE gene: ApoE2, ApoE3 or ApoE4.

Compared with the majority of people who have the more common ApoE3 variant, people with ApoE4 are at elevated risk of developing Alzheimer’s, and those with ApoE2 are protected from the disease.

By the time the mice were 9 months old, the ones carrying human ApoE variants had widespread brain damage. The hippocampus and entorhinal cortex, important for memory, were shrunken, and the fluid-filled space of the brain had enlarged where the dead cells had been.

ApoE4 mice exhibited the most severe neurodegeneration, and ApoE2 the least. The mice that lacked an ApoE variant entirely showed virtually no brain damage.

Further, the immune cells in the brains of mice with ApoE4 turned on a set of genes related to activation and inflammation much more strongly than those from ApoE3 mice. Immune cells from mice lacking ApoE were barely activated.

ApoE4 = higher inflammation
“ApoE4 seems to be causing more damage than the other variants because it is instigating a much higher inflammatory response, and it is likely the inflammation that is causing injury,” Holtzman said.

“But all forms of ApoE - even ApoE2 - are harmful to some extent when tau is aggregating and accumulating. The best thing seems to be, in this setting, is to have no ApoE at all in the brain,” Holtzman stated.

To find out whether ApoE in people similarly exacerbates neuronal damage triggered by tau, the researchers collaborated with Bill Seeley, M.D., from the University of California, San Francisco. Seeley identified autopsy samples from 79 people who had died from tauopathies other than Alzheimer’s disease in the past 10 years.

The researchers examined each brain for signs of injury and noted the deceased’s ApoE variants. They found that, at the time of death, people with ApoE4 had more damage than those that lacked ApoE4. ApoE transports cholesterol around the body via the bloodstream.

A few, rare individuals lack a functional ApoE gene. Such people have very high cholesterol levels and, if untreated, die young of cardiovascular disease. The lack of ApoE in their brains, however, creates no obvious problems. “There are people walking around who have no ApoE and they’re fine cognitively,” Holtzman said.

“It doesn’t appear to be required for normal brain function.”

Decreasing brain ApoE
These findings suggest that decreasing ApoE specifically in the brain could slow or block neuro-degeneration, even in people who already have accumulated tau tangles.

Most investigational therapies for Alzheimer’s have focused on amyloid beta or tau, and none has been successful yet in changing the trajectory of the disease.

Targeting ApoE has not yet been tried, according to Holtzman. “Assuming that our findings are replicated by others, I think that reducing ApoE in the brain in people who are in the earliest stages of disease could prevent further neurodegeneration,” Holtzman said.

Reference
Canola oil linked to worsened memory and learning ability in Alzheimer’s disease - research report

Canola oil is one of the most widely consumed vegetable oils in the world, yet surprisingly, little is known about its effects on health. Recently, a new study by researchers at the Lewis Katz School of Medicine at Temple University in Philadelphia associates the consumption of canola oil with worsened memory, worsened learning ability, and weight gain in mice which model Alzheimer’s disease.

‘More harmful than healthful’

The study is the first to suggest that canola oil is more harmful than healthful for the brain.

“Canola oil is appealing because it is less expensive than other vegetable oils, and is advertised as being healthy,” explained Domenico Praticò, M.D., Professor in the Departments of Pharmacology and Microbiology and Director of the Alzheimer’s Center at the Lewis Katz School of Medicine at Temple University, as well as senior investigator on the study. “Very few studies, however, have examined that claim, especially in terms of the brain.”

Curious about how canola oil affects brain function, Dr. Praticò and Elisabetta Lauretti, a graduate student in Dr. Praticó’s laboratory at Temple and co-author on the study, focused their work on memory impairment and the formation of amyloid plaques and neurofibrillary tangles in an Alzheimer’s disease mouse model.

Alzheimer’s replicated

Amyloid plaques, and phosphorylated tau, which is responsible for the formation of tau neurofibrillary tangles, contribute to neuronal dysfunction and degeneration and memory loss in Alzheimer’s disease. The animal model (i.e., the mice) was designed to replicate Alzheimer’s in humans, progressing from an asymptomatic phase in early life to full-blown disease.

Praticò and Lauretti had previously used the same mouse model in an investigation of olive oil, the results of which were published earlier. In that study, they found that Alzheimer mice fed a diet enriched with extra-virgin olive oil had reduced levels of amyloid plaques and phosphorylated tau and experienced memory improvement.

For their latest work, they wanted to determine whether canola oil is similarly beneficial for the brain.

The researchers started by dividing the Alzheimer’s mice into two groups at six months of age, before they developed signs of Alzheimer’s. One group was fed a normal diet, while the other was fed a diet supplemented with the equivalent of about two tablespoons of canola oil daily.

The researchers then assessed the animals at 12 months. One of the first differences observed was in body weight - animals on the canola-oil-enriched diet weighed significantly more than mice on the regular diet. Maze tests to assess working memory, short-term memory, and learning ability uncovered additional differences. Most significantly, mice that had consumed canola oil over a period of six months suffered impairments in working memory.

Decreased amyloid beta 1-40

Examination of brain tissue from the two groups of mice revealed that canola oil-treated animals had greatly reduced levels of amyloid beta 1-40, the more soluble form of the amyloid beta proteins, and generally considered to serve a beneficial role in the brain and act as a buffer for the more harmful insoluble form, amyloid beta 1-42.

As a result of decreased amyloid beta 1-40, animals on the canola oil diet further showed increased formation of amyloid plaques in the brain, with neurons engulfed in amyloid beta 1-42. The damage was accompanied by a significant decrease in the number of contacts between neurons, indicative of extensive synapse injury. Synapses, the areas where neurons come into contact with one another, play a central role in memory formation and retrieval.

“Amyloid beta 1-40 neutralizes the actions of amyloid 1-42, which means that a decrease in 1-40, like the one observed in our study, leaves 1-42 unchecked,” Dr. Praticò explained. “In our model, this change in ratio resulted in considerable neuronal damage, decreased neural contacts, and memory impairment.”

Not beneficial to brain health

The findings suggest that long-term consumption of canola oil is not beneficial to brain health. “Even though canola oil is a vegetable oil, we need to be careful before we say that it is healthy,” Dr. Praticò said.

“Based on the evidence from this study, canola oil should not be thought of as being equivalent to oils with proven health benefits.”

The next step is to carry out a study, of shorter duration, to determine the minimum extent of exposure necessary to produce observable changes in the ratio of amyloid beta 1-42 to 1-40 in the brain and alter synapse connections. A longer study may be warranted in order to determine whether canola oil also eventually impacts tau phosphorylation, since no effects on tau were observed over the six-month exposure period.

Onset of other neuro diseases

“We also want to know whether the negative effects of canola oil are specific to Alzheimer’s disease,” Dr. Praticò added. “There is a chance that the consumption of canola oil could also affect the onset and course of other neurodegenerative diseases or other forms of dementia.”

Source


Page 28 Canadian Nursing Home
Using iPads to comfort patients affected by dementia

The reassuring presence of a trusted loved one/family member makes every patient feel better. For patients affected by dementia, a familiar face may be a lifeline out of fear and confusion.

But family can’t always be present!

**Videos of family members**

So, staff at Vancouver General Hospital (VGH) were determined to find out if iPads might ‘fill the void’, and agreed on using video recordings of family members and monitor if this had a positive impact on the patients affected by a dementia.

Lillian Hung, a Vancouver Coastal Health Research Institute researcher and clinical nurse specialist in Gerontology & Tertiary Mental Health at VGH, came up with the idea after reading about the use of iPad recordings in residential settings.

“We are always looking for innovative ways to do the best we can for this patient group: and we’re always looking for non-pharmacological solutions wherever possible. This technology may be a safe and low-cost way of helping our clients.”

**Kicking, screaming at staff**

The patients at VGH often come from a residential program that can no longer care for them safely. These patients might have mental health issues - coupled with dementia; simple care interventions, such as being dressed or taking medication, can be a stressful experience for them. The stress and fear experienced can trigger responses, such as kicking and screaming at staff. Lillian Hung points out that such behaviours are physically and emotionally draining for both patients and staff.

Each time staff need to intervene to care for a patient - helping them dress, take medication or eat meals - for example, they will first play a recording of a trusted loved one reassuring the patient and urging them to be calm. The hope is that the sound and sight of a familiar loved one will reassure patients and make them feel secure.

When patients feel calm and reassured they are more inclined to allow staff to safely give them the care they need.

Lillian Hung says she is looking forward to giving families more involvement. “This approach places the family in the drivers seat. It relieves some of the burden people feel when they can’t be there. “Many of our patients have family far away, so this technology has the potential to really help patients feel less alone. It could be a win-win for everybody.”

All participation is voluntary and both staff and patients will be filmed to produce an educational program for training purposes. The research team will gather feedback from families and staff and publish their findings in the near future.

Project manager, Corrina Helmer, says recruitment for the study is underway and the staff is keen to try the new approach. “We’re hoping our iPad project will improve safety for our patients and our staff.”
Vancouver General Hospital’s iPad project impresses delegates at the 2017 Alzheimer Europe Conference

The iPad project from Vancouver General Hospital (VGH) impressed leading researchers from around the world at the 2017 Alzheimer Europe Conference held in Berlin, Germany in early October.

The VGH research team shared their use of iPads and family videos that serve to calm agitated patients with dementia. Corrina Helmer, project manager, and Alice Ip, program co-ordinator, presented the iPad project: “Using an iPad with Patients Affected by Dementia” at the international conference.

Berlin posters

The team also presented two posters by Lillian Hung and Alice Ip, clinical nurse specialists, Tertiary Mental Health, VGH. The posters were titled: ‘The Gentle Persuasive Approach Poster,’ and ‘Involving Patients in Research.’ “The experience has been inspiring and energizing,” Alice Ip commented.

Corrina Helmer and Alice Ip shared with their colleagues what they learned in Berlin about the latest Dementia research studies.

The VGH iPad research team is grateful for the support of the Vancouver Coastal Health Authority and the Vancouver Coastal Health Research Institute in conducting the ongoing project.

The viability of iPads

Designed for patients with dementia in the Older Adult Tertiary Mental Health Unit at VGH, the iPad research study explored the viability of iPads showing pre-recorded family videos.

The objective of the iPad program was to transform care into less stressful experiences for patients affected by dementia, as well as the staff who cared for them.

VGH team wins first prize at ‘Safety Den’

The B. C. Care Providers’ Association’s 40th Annual Conference in Whistler, B.C. earlier this year did something unique: it created the “Safety Den,” an event for ‘change agents’ in healthcare to pitch their ideas on “promoting staff & patient safety.”

Earlier this year, the “Safety Den” had put out a call for submissions. The submissions received were very creative and showed ‘commitment and passion’ that employees and organizations have for creating safe workplaces in health care venues.

A committee reviewed the submissions, and narrowed them down to four finalists who presented their innovations at the Conference’s “Safety Den”. VCH was one of the four finalists and won first place!

Team members

The team, from the Older Adult Tertiary Mental Health Program at VGH, included:

- Lillian Hung, clinical nurse specialist;
- Andy Au-Yeung, occupational therapist;
- Corrina Helmer, unit clerk;
- Levi Elija, care worker;
- Alice Ip, patient care coordinator; and
- Dr. M. Wilkins-Ho.

The winning project

The team pitched an innovative idea of using technology, an iPad, to improve safety and quality-of-care for patients with dementia. It involves showing a pre-recorded personalized family video to the client/patient prior to rendering care.

Early results showed the use of the iPad could lead to between 77% and 93% reduction in responsive behaviours in patients with dementia, compared to baseline.

Lillian Hung says, “Our iPad Project was one of the four finalists who presented at the ‘Safety Den.’” The selection committee was impressed by our idea because:

1. It promotes patient safety (a pre-recorded family video helps patients feel safe);
2. It prevents staff injury; and
3. It improves quality of care by inviting family to become “partners in care”

The $1,000 prize will be used “to scale up the iPad project”.

The $1,000 prize will be used “to scale up the iPad project.”

Corrina Helmer, project manager, (left) and Alice Ip, patient care coordinator; rubbed shoulders with leading researchers in Berlin during the 2017 Alzheimer Conference.
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