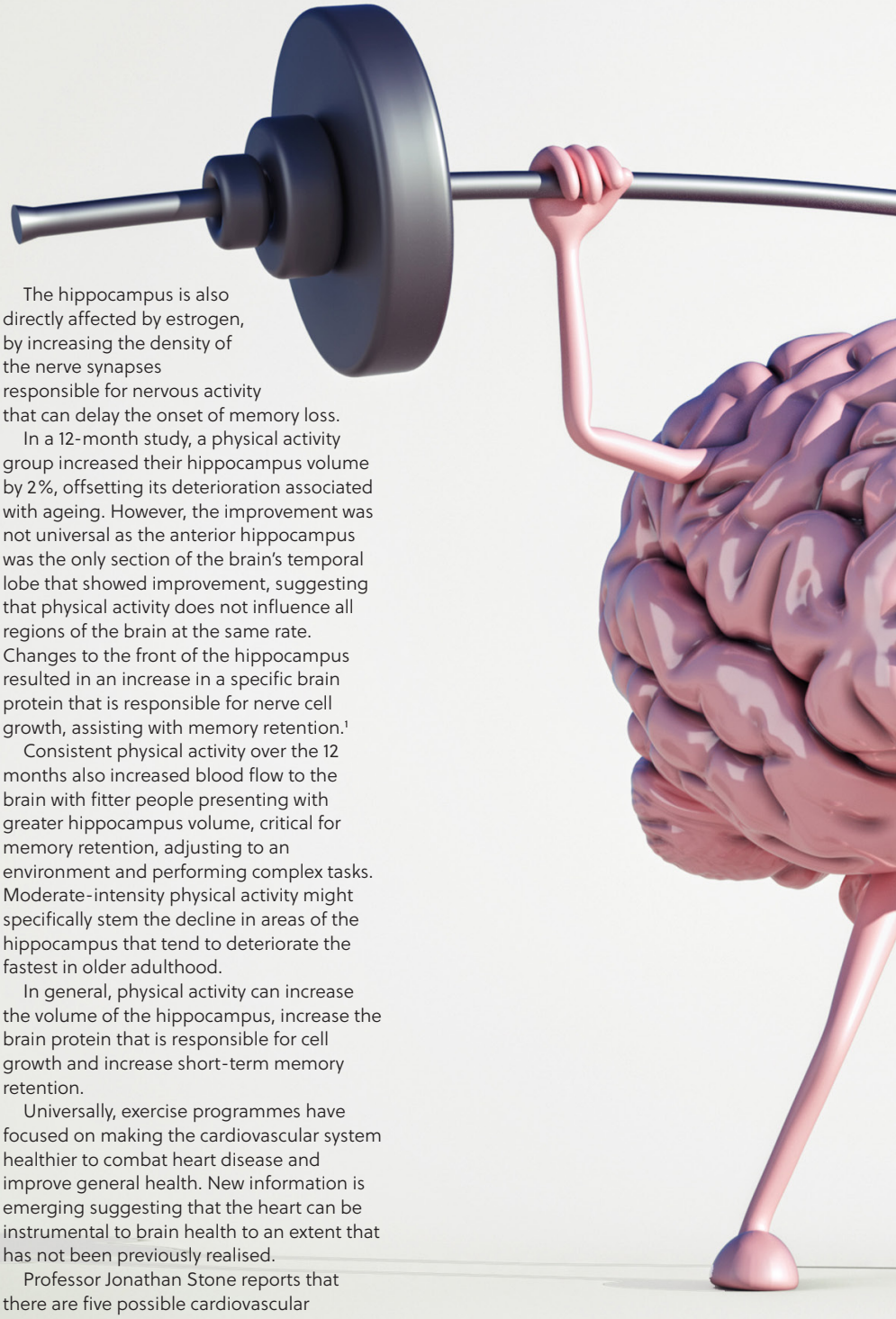
LET YOUR BRAIN DO THE WALKING

Dr Paul Batman is here with another enlightening **SIX-PART SERIES**, this time on physical activity and mental health. In part one, he looks at the impact exercise has on the hippocampus and overall brain function.



As a teacher, I always prided myself on knowing the names of every student in all my classes. I learned very early in my teaching career that knowing a student's name was a very powerful motivator. I was told that the most important possession a person owns is their name and to be recognised by name is a sign of respect and interest.

What is it about people that makes us remember them? Is it their appearance, their clothing, their facial expressions, the way they talk ...?

The part of the brain responsible for memory is the hippocampus, located on the innermost fold of the temporal lobe just under the temple. Names of people, machines, animals, etc. are stored in this temporal lobe of the brain.

The hippocampus is important for making new memories in the present and not in the past. Alzheimer's disease affects the hippocampus, initially causing those in the early stages of the illness to forget things that happen in the moment, while still remembering things from the past. In old age, the hippocampus starts to shrink, increasing the risk of dementia or memory loss. Even in older people who don't suffer from dementia, it will still shrink approximately 1-2% per year, eventually causing some memory loss[1].

The hippocampus is also an important site in the development of mental illness such as depression, schizophrenia and bi-polar as it appears to start shrinking.

The hippocampus is also directly affected by estrogen, by increasing the density of the nerve synapses responsible for nervous activity that can delay the onset of memory loss.

In a 12-month study, a physical activity group increased their hippocampus volume by 2%, offsetting its deterioration associated with ageing. However, the improvement was not universal as the anterior hippocampus was the only section of the brain's temporal lobe that showed improvement, suggesting that physical activity does not influence all regions of the brain at the same rate. Changes to the front of the hippocampus resulted in an increase in a specific brain protein that is responsible for nerve cell growth, assisting with memory retention.[1]

Consistent physical activity over the 12 months also increased blood flow to the brain with fitter people presenting with greater hippocampus volume, critical for memory retention, adjusting to an environment and performing complex tasks. Moderate-intensity physical activity might specifically stem the decline in areas of the hippocampus that tend to deteriorate the fastest in older adulthood.

In general, physical activity can increase the volume of the hippocampus, increase the brain protein that is responsible for cell growth and increase short-term memory retention.

Universally, exercise programmes have focused on making the cardiovascular system healthier to combat heart disease and improve general health. New information is emerging suggesting that the heart can be instrumental to brain health to an extent that has not been previously realised.

Professor Jonathan Stone reports that there are five possible cardiovascular

contributors to age-related memory loss and dementia that have not been previously identified. These are the heart, brain, aorta, small vessels supplying the brain and, lastly, time[2].

The heart is a very trainable organ that responds extensively to physical activity. It beats 60 to 70 times per minute, with an approximate volume of 50-60 millilitres of blood per beat. In 60 seconds, it pumps about five to six litres of blood carrying oxygen around the body, while also transporting carbon dioxide to the lungs for removal.

In a rested state, the heart pumps 35 million beats per year, equivalent to two to three billion beats in a lifetime. During intense exercise, the heart can pump 150-200 beats per minute with a volume of blood up to 200 millilitres per beat, equivalent to a cardiac output of 30-40 litres of blood per minute.

For the brain to function optimally, the heart must pump oxygen and nutrients to the smallest blood vessels that surround and feed it, and it is now regarded as potentially significant in improving brain health. Conversely, as heart function deteriorates, so does brain health.

Age-related memory loss and dementia has previously been regarded as an old person's disease. However, there is now a growing awareness that it might have its roots in childhood and a sedentary lifestyle, provoking an interest in inactivity and brain health.

> People leading sedentary lives have a brain that will form a sedentary structure, due to its interaction with its inactive environment

In a landmark study examining the effect that sedentary behaviour has on brain function, Dr Catherine Kotz injected a neurochemical called Orexin into half of her rodent subjects, while the other half received just a water injection[3]. In other animal studies, it was found that without Orexin animals were always falling asleep. The results of this study showed that the rats injected with Orexin moved considerably more than those injected with the water, confirming Dr Kotz's theory that there are specific neurochemicals that can control activity patterns.

To investigate this further, another researcher – Dr Colleen Novak – studied the brain networks and chemicals responsible for sitting in a chair or moving. Dr Novak mated multi-generational obese rats with obese rats, and lean rats with lean rats, and discovered that the obese rats did not have the ability to initiate movement[4].

Irrespective of the types and amounts of neurochemicals, these animals did not have the wiring in the brain to respond to the injected chemicals and so remained sedentary. Alternatively, when the brain of the lean rats was injected, their movement became almost uncontrollable. The lean rats were bred to move, while the obese rats were bred to sit.

She also discovered that the muscle of the moving rats was different to the muscles of the sedentary rats, as was the signalling to the brain. The sedentary sitters did not respond the same way. Their muscles were trained to be sedentary and, as such, the signal to move was stopped.

Obese rats tended to be less sensitive and unresponsive to moving signals from either the muscles or the brain. Their muscles were trained to sit while the other lean rats were trained to move, suggesting they were more sensitive to moving neurochemicals and had a stronger feedback loop from the muscles to the brain. It could be that the movers' brains were hardwired to move.

While we are the product of our DNA, the environment that we exist in still influences our physiological function. It is common knowledge that a brain must be kept active to maintain its optimal function. This neuroplasticity allows the brain to change with its environment. As with other organs of the body, if the brain is not stimulated it will eventually turn off and become as dormant as the lifestyle it leads.

People leading sedentary lives have a brain that will form a sedentary structure, due to its interaction with its inactive environment leading to a sedentary lifestyle. Just as a muscle adapts to movement, so will the brain.

Chronically inactive people are faced with a double-edged sword. They have a brain that is not responsive to the neurochemicals for movement and have muscles that are trained to sit that do not send feedback signals to the brain to get them moving.

As the need to move is further eliminated, our brains will form to create a more sedentary lifestyle that can contribute to increased memory loss and, potentially, mental illness. *fp*

DR PAUL BATMAN
has been involved in health and fitness for more than 40 years as a university lecturer, vocational educator, author, researcher, international conference presenter and workshop facilitator. Over the last 18 years, Paul has built, owned, operated and sold two leading health and fitness vocational training institutes, and has received a Lifetime Achievement award for his services to the Australian fitness industry. Paul originally contributed to our *Network* articles back in the 1990s.
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PTSD AND PHYSICAL ACTIVITY

In **PART TWO** of **Dr Paul Batman's** series on physical activity and mental health, he shares his experience of post-traumatic stress disorder and the research suggesting exercise can help.

In June 2012, my life changed forever. I had just been informed that my only son had been killed in a one-car rollover accident, leaving behind three small children. At the news of my son's passing, my body went into a state that I had never experienced before. The shock of the accident left me numb, bewildered, confused and trance like.

Over the next 12 months, I went through shock, denial, anger, bargaining, depression and waited for acceptance and processing to occur, but couldn't shake off the helplessness, anxiety, loneliness, sadness, social withdrawal, avoidance, unhappiness and general malaise. I seemed to be stuck in the grief cycle. My medical practitioner diagnosed me with PTSD.

I did not think it could be PTSD, as this was a condition generally reported in combat veterans, police officers, paramedics and firefighters who have been subjected to high rates of trauma on an ongoing basis. The estimated prevalence of PTSD among combat veterans alone has been reported as between 7% and 23%.[1] As I found out more about PTSD, I realised that no one was immune, including those who had experienced the unexpected death of a child.

People with PTSD typically present with intrusive thoughts, avoiding people, places, events, negative thoughts and feelings, ongoing fear, anger and guilt, irritability, anxiety, angry outbursts, depressive symptoms and difficulty sleeping. PTSD symptoms can last for months to many years as there is no definitive recovery period, and it is regarded as one of the most disabling mental health conditions[1].

People who suffer from PTSD experience a two to three times' increased mortality rate than the general population and present with a significant increase in cardiovascular disease and metabolic syndrome. They tend to adopt unhealthy lifestyle behaviours, including sedentary behaviour, smoking, alcohol abuse and unhealthy eating habits, and have low levels of fitness.

PTSD people often present with autonomic dysregulation, where either the sympathetic nervous system responsible for speeding up cardiovascular functions and the parasympathetic nervous system responsible for slowing down or conserving cardiovascular functions are disrupted, causing an imbalance in the regulation of heart rate and blood pressure. They also demonstrate elevated heart rates and blood pressure for longer periods, particularly in response to flashbacks, trauma cues and environmental stimuli[2].

Basal blood pressure responses tend to be higher in PTSD, with elevated systolic and diastolic pressures during stressful situations. Due to the reduced parasympathetic nervous system control of the heart, the baroreceptors responsible for regulating this system are vulnerable to controlling blood pressure.

Other PTSD cardiovascular and metabolic complications include increased endothelial dysfunction, limiting the movement of blood through the arteries, potentially leading to an increased risk of atherosclerosis, elevated cholesterol levels, low-density lipoproteins and triglycerides and decreased high-density lipoproteins, potentially leading to an increased risk of type 2 diabetes, as well as low levels of cardiovascular fitness and increased whole-body inflammation[2].

PTSD sufferers tend to be high users of healthcare services and generally incur greater healthcare costs compared to other psychiatric disorders and are four times more likely to develop type 2 diabetes, with higher rates of obesity and being overweight.

Current treatment for PTSD includes pharmacotherapies, as well as cognitive behavioural therapy, exposure therapy, eye movement desensitisation and reprocessing[5]. Although evidence-based psychological and pharmacological interventions have proven successful, some PTSD people view them as barriers due to the cost, access to mental

> People who suffer from PTSD experience a two to three times' increased mortality rate than the general population

health professionals, motivation to remain in treatment and the stigma. Sadly, fewer than 30% of those with PTSD seek any mental health assistance[3].

The role of physical activity

One intervention that is rapidly gaining a reputation as a viable treatment intervention is physical activity, defined as "any bodily movement produced by muscle contractions resulting energy expenditure"[3].

A 12-week study following the American College of Sports Medicine guidelines, reflective of clinical practice, reported significant improvements in both physical and mental health outcomes, suggesting that exercise has the power to reduce the symptoms of PTSD. Classified as a vulnerable group, exercise interventions must also be accompanied by individualised prescriptions and motivational techniques tailored to their specific needs[1].

The focus for any physical activity intervention for improved health should be based on the importance of intermittent muscle contractions. The most potent mechanism at the root cause of many chronic and psychological problems is muscular inactivity and is specifically linked to the need for frequent muscle contractions throughout the whole day. Muscle contractions at any intensity (2-9 METs) control oxidative stress, release anti-inflammatory molecules, activate insulin receptors, control blood fats, activate mitochondrial respiration, increase capillarisation, control blood sugar, activate the immune system and constantly change the shape of cell membranes. Physical activity can take any form, whether as part of a formal fitness programme or by substituting daily sedentary behaviour with low- to moderate-intensity alternatives, sport, recreational, household or occupational activities.

Some of the possible mechanisms explaining the role of physical activity in reducing PTSD symptoms include[3]:

- Exposure to increased heart rates can desensitise the impact of stress events, as well as improving stroke volume and cardiac output to improve overall aerobic fitness.
- PTSD is associated with changes in the brain's grey matter, especially the reduction in hippocampus volume, anterior cingulate and lateral prefrontal cortex. Physical activity can increase grey matter by increasing the volume of the pre-frontal cortex, cingulate and the hippocampus.
- The hypothalamic/pituitary axis (HPA) is responsible for regulating hormonal reactions to stress and is adversely affected in PTSD by reducing basal cortisol levels. Regular moderate-intensity aerobic exercise normalises the HPA by stimulating the release of cortisol[3].
- PTSD is characterised by an increase in pro-inflammatory molecules contributing to a low-grade chronic inflammatory response. Regular exercise also promotes the release of anti-inflammatory molecules, creating a balance between both inflammatory molecules, improving the immune system response.
- Physical activity creates a distraction from everyday PTSD triggers by forcing the person to concentrate on the activity and the environment.

Personally, my own journey has improved significantly through daily regular physical activity across all lifestyle domains: by reducing sitting time to fewer than six hours per day; by standing; by strolling for five minutes every 60 minutes; by going to the gym three times per week for more moderate to vigorous aerobic workouts; and by incorporating two days of resistance training. I use heart rate to calculate METs or oxygen cost for every activity I perform to reach a workload goal of 2,000-5,000 MET minutes per week at any intensity between 2-9 METs.

I also set performance goals based on outdoor physical activities, often culminating in long-distance trekking trips. Although it's early days, limited studies on the role of outdoor activities and PTSD have revealed significant improvements in PTSD symptoms, including depression, anxiety and stress.

Outdoor activities typically provide an attention reversal for the PTSD, with the focus on natural environments that require less directed attention compared to busy urban settings, reducing the hyper vigilance to stimuli. Outdoor activities often require the learning of new recreational skills, rather than focusing on formal psychological interventions, and can cause a distraction from everyday concerns and a sense of purpose and achievement[4].

While the evidence of the effects of physical activity and muscle contractions on PTSD are in the early stages, there is strong emerging evidence that it can provide significant clinical benefits. *fp*

DR PAUL BATMAN
has been involved in health and fitness for more than 40 years as a university lecturer, vocational educator, author, researcher, international conference presenter and workshop facilitator. Over the last 18 years, Paul has built, owned, operated and sold two leading health and fitness vocational training institutes, and has received a Lifetime Achievement award for his services to the Australian fitness industry. Paul originally contributed to our *Network* articles back in the 1990s.
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CROSS-BODY TALK:
THE BRAIN'S EFFECT ON THE BODY DURING EXERCISE

In **PART THREE** of his series on physical activity and mental health, **Dr Paul Batman** discusses how your best anti-depressant might be the cross talk between the brain and muscle contractions.

Health professionals working with patients suffering from mental health issues have long recognised the importance of exercise as a non-pharmacological intervention. Many more health practitioners are prescribing a combination of medication and exercise for conditions such as major depressive disorders. There have been reports that the effects of anti-depressant medications can be reduced if the body and brain develop a tolerance to it, as well as possibly producing side-effects. If this does occur, the dosage would need to be increased to produce the same effect or an additional intervention prescribed.

In an eight-week exercise intervention study, subjects were divided into either a medication plus exercise group or a medication without exercise group. The exercise group performed 150 minutes of moderate to vigorous exercise per week, while the non-exercise group performed just group behavioural therapy. The exercise plus medication group demonstrated a 75% reduction in depression compared to 25% in those who did no exercise, supporting the notion that physical activity and medication in combination could be a more effective long-term alternative[1].

Conclusions have been drawn that exercise releases specific hormones that create a 'feel-good' effect that lessens the symptoms of depression and anxiety. Popular literature reports that exercise can aid in the release of endorphins and opioid-like hormones that produce an almost euphoric effect, also responsible for the 'runner's high' often experienced by addicted runners.

During acute exercise, the heart rate increases causing the hypothalamus to release a corticotrophin-releasing hormone that travels to the anterior pituitary gland and releases the adrenocorticotrophic hormone into general circulation. This stimulates the release of cortisol that acts to inhibit or reduce the stress response. The cortisol level can remain high for approximately two hours post exercise and is generally proportionate to the amount of exercise performed after it returns to pre-exercise levels[1].

While this can partly explain the significant acute effects of exercise, it does not account for the euphoric after-effects reported from exercise. The Runners High Theory or Endorphin Hypothesis has been the most popular explanation for these positive long-term effects[2].

Endorphins are naturally occurring opiates in the body that are released in the brain, improving mood and increasing performance.

> "Extended aerobic exercise at >60% VO_2max can decrease depressive symptoms"

The release of these mood-altering opiates is increased with longer and more intense exercise, possibly lessening the perception of pain sometimes produced by exercise. It has been reported that the greatest release of endorphins occurs at intensities of 75% VO_2max and 80% heart rate max, suggesting the intense aerobic exercise produces the most significant results. Extended aerobic exercise at >60% VO_2max can also decrease depressive symptoms.

Another theory that attempts to explain the positive effects of exercise is the Neurogenesis Theory. Rodent studies report that exercise and medication can stimulate the growth of neurons in the hippocampus and the sub ventricular zones, reducing depressive symptoms. The increased volume of the hippocampus was noted in rodents that engaged in long-term aerobic exercise. Apparently, the increased endorphin levels from exercise also contribute to the growth of new neurons.

The Endorphin Hypothesis, while a distinct possibility, has been challenged in recent years. Beta endorphins are large molecules that could have difficulty in passing through the blood brain barrier (BBB). The BBB is a semi-permeable border separating the blood circulation from the brain and the extracellular fluid. The BBB screens every substance to ensure that no harmful destructive microbes can enter the brain. If the larger endorphins are unable to penetrate the BBB, they might not play the pivotal role in reducing depressive symptoms and

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improving mood that was once thought, while still being released during exercise to reduce pain sensations.

New information has focused on the role that endocannabinoids might play in improving mood. Endocannabinoids are natural chemicals produced in the body that can cause a euphoric and analgesic effect. These molecules are small enough to pass through the BBB and bind to the brain receptors and are released on demand. Exercise at intensities of 50-80% of maximal aerobic capacity induced sharp increases in endocannabinoid levels in the frontal cortex and hippocampus, increasing dopamine levels and resulting in more pleasurable feelings, potentially reducing depressive symptoms and stabilising moods[2].

A Cross Talk model reported to improve brain function and reduce depressive symptoms is the important link between muscle contractions and organs, resulting in improvements in cognition, lipid and glucose metabolism, endothelial function and mental health conditions.

The contraction of skeletal muscle results in the release of myokines or cytokines (small proteins or peptides) produced by muscle tissues that regulate metabolism and also produce a paracrine and endocrine function on other tissues and organs including the liver, adipose tissue and the brain.

The muscles are regarded as a key endocrine organ that forms a muscle-endocrine loop affecting memory, cognition, mood and decision making. Exercise activates signalling pathways resulting in improvements in synaptic transmission and development of new neurons.

Brain derived neurotrophic factor (BDNF) is the most studied myokine released during muscle contractions that plays a dominant role in regulating the effect of exercise on the hippocampus, impacting on memory and learning, and the maintenance, functioning and survival of nerve cells and synapses, as well as improving sleep quality. Sleep disturbances have been reported in 90% of depressed patients[3].

> "Exercise produces an anti-inflammatory response for up two hours post exercise, contributing to lowered levels of depressive symptoms"

Low levels of BDNF have been reported in those with depressive symptoms, resulting in altered synapses and poor synaptic transmission. Exercise increases hippocampus volume and circulating BDNF concentrations, particularly in older adults, resulting in reduced depressive symptoms[1].

The increase in BDNF is partly regulated by the release of IGF-1, a myokine released from the liver that can cross the blood brain barrier and is stimulated by exercise. Exercise also improves the IGF-1 signalling pathway to the brain, contributing to improved neural functioning. Cathepsin and irisin are two other myokines that are released during exercise and reportedly have a positive effect on the hippocampus volume and BDNF release and stimulating nerve cell production. Irisin also plays a role in muscle and liver cell metabolism.

As the muscle contracts, it induces rapid changes in stroke volume and cardiac output to improve capillarisation to the working muscle and reduce the blood flow to inactive tissues. Irrespective of the muscles requiring the additional oxygen, the blood flow to the brain increases. The increased blood flow stimulates the development of new nerve cells in the hippocampus, as well as increasing the availability of BDNK, nutrients, oxygen and other myokines to the brain, as well as improving cerebral circulation.

Low-grade chronic inflammation has been identified as a potential contributor to depressive symptoms led by pro-inflammatory molecules IL-6 and TNF-a. Exercise produces an anti-inflammatory response for up two hours post exercise with the release of key myokines IL-1 and IL-10 from the innate immune system, reducing inflammation and contributing to lowered levels of anxiety and depressive symptoms.

As depressive patients are generally less active, an increase in muscle contractions can assist in reducing other associated conditions by releasing other myokines throughout the body that lead to mitochondrial biogenesis, decreased chronic inflammation, lower oxidative stress, greater serotonin production, improved insulin sensitivity, reduced pain sensitivity, enhanced neurogenesis and increased BDNF production. fp

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THE DOWNSTREAM CONSEQUENCES OF DEPRESSION AND ANXIETY

PART FOUR of **Dr Paul Batman's** series on physical activity and mental health explores how physical activity can improve the downstream cardiovascular risk factors of people with depression and anxiety.

In a recent study, it was reported that if current global physical inactivity levels do not change, there will be an estimated 499.2 million new cases of preventable non-communicable chronic diseases (NCD) and mental health cases between 2020 and 2030, resulting in a total global cost of $520 billion. Of these new cases, 47% – or 234.6 million – would be from hypertension and 43% – or 217.7 million – will be due to depression and anxiety, which steadily increased as a consequence of the COVID-19 pandemic[1].

Despite a relatively low incidence of dementia and cancers reported, they do incur higher healthcare costs due to the requirements for diagnosis, treatment and long-term healthcare management. To overcome some of these costs, physical activity has long been recognised as a major modifiable factor for NCD, mental health conditions, all-cause mortality, cardiovascular disease and metabolic conditions. However, despite the *WHO Global Action Plan on Physical Activity*, which includes intervention strategies and national guidelines, there has been little change in global physical activity levels and, as a consequence, there are increased levels of NCD and mental health conditions.

When physical activity has been recommended as a viable intervention for improvements in mental health, the emphasis has been on upstream effects including increases in concentration of brain-derived neurotrophic factor (BDNF), reduced inflammation, increased blood flow to the brain, increase in endocannabinoids, neuroplasticity and increased dopamine levels. In a first ever study on dose response between physical activity and a decrease in depression, the greatest results occurred between "no activity to at least some activity". Accumulating 2.5 hours of moderate-intensity physical activity reduced depressive symptoms by 25%, while 1.25 hours per week of moderate physical activity reduced symptoms by 18%[2]. These results indicate that beneficial upstream effects of physical activity can occur at levels below current public health recommendations.

While these changes are critical to mental health improvements, it is also reported that those suffering from depression and anxiety are among the most sedentary or inactive in our community. Conversely, people leading a sedentary lifestyle or who are long-term inactive are more likely to experience depression and anxiety, especially in adolescents and older adults[3]. This is reportedly due to the reduced communication between people due to isolation and the reduced time spent in daily physical activity. Interestingly, there was an increased prevalence in depression in those watching TV compared to those involved in active sedentary activities such as computer use. Results show that moderate to severe depression is associated with high levels of TV viewing and computer use, generally in excess of six hours per day, culminating in a prolonged duration of sitting time.

In the older population, there is a higher

> Sedentary and inactive people are three times more likely to have higher levels of depression than active groups

mortality rate in those who suffer from depression. Combining depressive symptoms and inactivity, each independently increases the risk of cardiovascular disease and they are strongly correlated with each other[4]. Physical inactivity approximately accounts for 25% of the increased risk of cardiovascular mortality due to depression in older adults. Sedentary and inactive people are three times more likely to have higher levels of depression than active groups[4]. Giving rise to the suggestion is that a downstream effect of depression and anxiety is an increased risk of heart disease mortality and hypertension, indicating that physical activity is not only important for cognitive changes but also to reduce the effects of prolonged inactivity and its effects on cardiovascular disease[5].

The two areas of emerging physical activity research are the apparent differences between physical inactivity and sedentary lifestyle. It is possible to be sedentary and still be active, while it is possible to reduce sedentary time but still be inactive. This is due to physical inactivity being defined as not meeting the national guidelines of 150 minutes per week, while being sedentary refers to any waking behaviour with an energy expenditure <1.5 METs as represented by sitting or reclining[5].

People suffering from depression have difficulty in maintaining an exercise programme due to lack of motivation, poor self-efficacy, low dopamine levels and self-isolation, and they generally perform poorly in formal exercise programmes. An alternative might be to initially focus on reducing sedentary time and replacing it with intermittent low-intensity physical activities throughout the day in whatever activities they choose to do, with the emphasis on sitting less and moving more contributing to an improvement in cardiovascular health. By replacing sedentary time by 30 minutes per day, an initial 2-4% improvement in major cardiovascular risk factors can occur, with the greatest effects seen in the most inactive population.

Once a movement pattern of replacing 30 minutes of sedentary time daily with light-intensity habitual activity has been established, the focus could then shift to include more moderate to vigorous activities towards more significant cardiovascular benefits. Groups that exercise 60-75 minutes per day at moderate intensity and are still sedentary for more than eight hours per day report significantly lower cardiovascular risk factors[5].

Prolonged sitting time and increased TV viewing time can increase cardiovascular risk factors by reducing blood flow and shear force in the arteries resulting in blood pooling in the large veins of the lower leg. The reduced shear force decreases the availability of nitric oxide, increasing the constriction of the arterial walls, further reducing blood flow to the tissues. The increased vasoconstriction of these vessels in the large muscle of the legs increases peripheral resistance of the blood in the arteries, increasing blood pressure and hypertension.

Skeletal muscle is designed to contract to increase energy expenditure through the uptake of glucose from the blood to the muscle through insulin-dependent glucose-mediated pathways. Prolonged sedentary time decreases the activation of the GLUT 4 transporter on the plasma membrane, increasing glucose concentration in the blood and reducing the effectiveness of insulin, as well as causing a down regulation of genes responsible for carbohydrate metabolism. The increase in blood pressure and hyperglycaemia caused by sedentary time can also reduce blood flow to the brain.

The increased levels of circulating glucose have been known to increase the low-grade inflammation in all tissues, contributing to cardiovascular disease. Concomitantly, increased inflammation can contribute to reduced levels of nitric acid and increased vasoconstriction of blood vessels, resulting in an increase in blood pressure[5].

One interesting phenomenon reported is the possibility of "sitting induced exercise resistance", where prolonged uninterrupted sitting (> four days) can dilute the significant glucose uptake, insulin secretion and lowered triglyceride levels of a single bout of moderate to vigorous exercise. This would suggest that an exercise programme of two or three times per week might not fully mitigate the effects of uninterrupted prolonged sedentary time. Similar effects have been noted on blood pressure responses after three to four days of prolonged sedentary time. This suggests the frequency of exercise bouts is an important variable when attenuating the effects of prolonged sedentary time.

Although regular structured exercise programmes of moderate to vigorous intensity will produce the most significant cardiovascular benefits, historically they are also the most difficult to maintain long term in depression and anxiety groups. Perhaps reducing daily sedentary time would be a more sustainable and feasible behaviour change to initially improve their downstream cardiovascular risk factors.

DR PAUL BATMAN
has been involved in health and fitness for more than 40 years as a university lecturer, vocational educator, author, researcher, international conference presenter and workshop facilitator. Over the last 18 years, Paul has built, owned, operated and sold two leading health and fitness vocational training institutes, and has received a Lifetime Achievement award for his services to the Australian fitness industry. Paul originally contributed to our *Network* articles back in the 1990s.
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DEPRESSION, ANXIETY AND CANCER

PART FIVE of **Dr Paul Batman's** series on physical activity and mental health looks at the efficacy of a physical activity programme on reducing cancer-specific mortality.

A cancer diagnosis is a life-changing event and a major source of psychological and emotional stress impacting on mental health and wellbeing, with 90% of cancers diagnosed in those >50 years. It has been reported that 25% of cancer patients suffer from depression (three times the general population) within the first five years, with only 5% seeking any effective treatment by a mental health professional. The cancer diagnosis not only affects the patient but also has negative consequences for family and friends[1].

After diagnosis, a cancer patient's initial emotional response is brief, extending from days to weeks, with feelings of sadness, denial and disbelief. For some, this progresses to fear of death, negative changes in lifestyle, financial costs, appearance, the effect on family, and anxiety related to the prescribed treatment and the eventual outcome. This is further compounded in people who have poor coping strategies, previous mental illness and poor communication abilities. In a study of 256 females undertaking chemotherapy for early breast cancer, 26% presented with severe depression and 41% with increased levels of anxiety[2].

In a study of 149 women with nonmetastatic breast cancer, 40% reported mild depression symptoms at the conclusion of their chemotherapy treatment, with family and caregivers also reporting increased feelings of depression. Within five years post-chemotherapy treatment, 23% had been diagnosed with a new mental health diagnosis, 62% were prescribed medication and 21% engaged in mental health care.

The prevalence of depression among cancer patients was highest in those with lung cancer (13%), gynaecological cancer (11%), breast cancer (9%), colorectal cancer (7%) and genitourinary cancer (6%), with 73% not receiving mental health treatment[2].

In a study of 20,582 patients being treated with colorectal cancer, breast and prostate cancers, those diagnosed with major

> **Compelling evidence suggests that both depression and cancer are responsible for increased levels of pro-inflammatory cytokines**

depression had significantly lower survival rates. The impact of mood and wellbeing has been reported as a major determinant of survival, with 70% of oncologists and 85% of patients believing that mental health plays a significant role in cancer recovery[3].

Even with extensive reporting of depression being a comorbidity in cancer patients, it is still under recognised in healthcare and mental health interventions. In a meta-analysis of over 10,000 cancer patients across 14 countries, 38.7% of cancer patients developed a comorbid mental health disorder within five years of their cancer diagnosis.

A common link reported for both depression and cancer is the role that the innate immune system and inflammation play in the progress of both diseases. Ironically, chronic depression is associated with an increased cancer risk and shorter survival rates.

Compelling evidence suggests that both depression and cancer are responsible for increased levels of pro-inflammatory cytokines. As cancer cells grow, the increase in inflammatory cells promotes tumour growth, reduces immune system function and creates a chronic inflammatory environment.

While it is initially important for inflammation to be part of the immune response, when maintained at chronically low levels for long periods it can cause an immune-suppression response, making it difficult for the immune system to fulfil its function. The increased levels of pro-inflammatory cytokines, particularly C reactive protein (CRP), can reduce levels of brain-derived neurotrophic factor (BDNF), which is a key factor for brain health contributing to depression. Another inflammatory marker (IL-6) is found in medically depressed patients as well as depressed patients with various cancers.

In both animal and human studies, high levels of stress have been associated with a decrease in cell-mediated immunity, highlighted by a decrease in the activity of natural killer cells (NK). NK activity is reported to be blunted in depression and cancer patients. Women with breast and ovarian cancer who have experienced heightened levels of stress have presented with impaired NK activity, as well as reduced levels of T helper cells and T lymphocyte cells all playing a key role in immune system functioning[5].

The hypothalamic-pituitary-adrenal axis is crucial in combating the effects of chronic stress. During stress, the sympathetic nervous system is stimulated, the parasympathetic nervous system is inhibited and the HPA axis is activated. However, in both depression and cancer patients, due to an increase in pro-inflammatory cytokines there is dysregulation of the HPA axis, resulting in thyroid dysfunction, increases in blood pressure, poor immune system activation, poor glucose control, adrenal fatigue and sustained inflammation[4].

The treatment of depression among cancer patients is given little attention. This is mainly due to the lack of time and expertise of the oncologist to recognise mental health complications and the sometimes reluctance of the patient to disclose their mental health condition. There are also considerable additional costs to engage a mental health professional at the same time as being treated by a cancer specialist. Ironically, depression in cancer patients is associated with low compliance to chemotherapy and an increased risk of death.

Alongside pharmacological and psychosocial treatment, another intervention that can have a profound effect on both cancer and depression as comorbidities is exercise as medicine. Exercise can have an effect on all tissues of the body by forcing the muscles to release myokines that cause anti-inflammatory responses, reducing low-grade systemic chronic inflammation and suppressing tumour growth[6].

Exercise also mobilises the innate immune system, reduces pain and fatigue and limits the production of pro-inflammatory molecules in the hippocampus. Recent studies have also reported that even a single bout of exercise can elevate myokine levels to an extent that suppresses cancer cell output. Physical activity can enable the cancer patient to tolerate greater dosages of cancer treatment, reduce the side effects of treatment and improve its effectiveness.

Exercise also releases monoamine transmitters, including serotonin, dopamine and epinephrine, which have a positive effect on neurotrophic factors in the brain and improve mood and coping strategies.

In a recent landmark study, nine patients with advanced stage prostate cancer who participated in 34 minutes of high-intensity indoor cycling per day over six months presented with elevated levels of anti-cancer myokines that suppressed prostate cancer cell growth by 17%. These myokines also allow the muscle to communicate with the brain, triggering a wave of responses that improve memory, learning and mood[6].

Cancer patients with depression reportedly have lower levels of muscle mass, increasing their mortality risk when compared to those with average muscle mass. This will contribute to a further decrease in the release of myokines and anti-inflammatory responses. They also have a higher fat mass, contributing further to the pro-inflammatory environment.

The general physical activity recommendation for cancer patients with depression includes breaking up prolonged sedentary periods throughout the day; participating in low to moderate aerobic exercise on as many days of the week as possible; participating in two to three days of vigorous physical activity; and participating in two days per week of resistance training. For some cancer patients, multiple bouts of physical activity of any duration throughout the day can be very effective for myokine release.

An effective physical activity programme has the potential to reduce cancer-specific mortality by 28-44%, lower the risk of cancer recurrence by 21-35%, and provide a 25-48% reduced risk of all-cause mortality, while at the same time reducing the chances of anxiety and depression[7]. fp

DR PAUL BATMAN has been involved in health and fitness for more than 40 years as a university lecturer, vocational educator, author, researcher, international conference presenter and workshop facilitator. Over the last 18 years, Paul has built, owned, operated and sold two leading health and fitness vocational training institutes, and has received a Lifetime Achievement award for his services to the Australian fitness industry. Paul originally contributed to our *Network* articles back in the 1990s.
www.neatfitcoaching.com

DEPRESSION, ANXIETY AND AGEING

In the **FINAL PART** of his series on physical activity and mental health, **Dr Paul Batman** looks at how exercise can relieve many of the symptoms associated with depression and ageing.

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During the COVID-19 epidemic, a controversial intervention mandated to control the spread of the disease was social distancing and self-isolation. A 50% increase in rates of depression was reported, particularly in those aged 50 years and over, during this period.

This was further exacerbated in the elderly by the decrease or suspension of essential services, the high risk of contracting COVID-19 and the inability to see family and friends significantly impacting on their mental health[1].

The increased focus on the elderly highlighted the increased incidence of anxiety and depression reported with normal ageing. With the prediction that the >60 years group will increase from one billion people in 2019 to 1.2 billion by 2030 and to 2.1 billion by 2050, the incidence of mental health problems is expected to increase to dramatic levels. By 2035, it is estimated that 84% of healthcare expenditure will go to people >65 years, representing 19% of the population.

In 2015 it was reported that 300 million people globally suffered from major depressive disorder and that 7.5-12.6% in this group were aged 65 years and above. The World Health Organization estimates that the prevalence of depression in the elderly is between 10% and 20%, far higher than the global average across all ages. The results of a meta-analysis revealed depression in the elderly to be closer to 31.7%, with higher values of 40.78% reported in developing countries compared to 17.05% in developed countries, possibly due to poverty, poor public health services, civil unrest and gender inequality[2].

Even though this would suggest that depression is a common condition in the elderly, it is left undiagnosed in more than 50% of cases, with depressive symptoms often attributed to other comorbidities such as cardiovascular disease, diabetes and major neurocognitive disorders. In many cases,

depression co-exists with these other comorbidities, often leading to increased disability, poor medical outcomes, cognitive decline and increased mortality risk.

The common predictors of depression in the elderly can be found by examining the sociodemographic, behavioural, health and life events of individuals. The sociodemographic predictors include sex (females are more at risk than males), age (depression increases with age >60), education (depression is identified in those with lower education levels), marital status (higher incidence among those divorced or widowed), stressful events (loss of family members or friends) and social support (low social connection with others). Behavioural predictors include poor nutritional status and low physical exercise levels. The health predictors include chronic diseases and poor cognitive ability. These predictors all contribute to higher levels of depression and anxiety, further decreasing their quality of life and affecting their cognitive functioning[3].

These predictors indicate that depression alone is not solely caused by psychopathology but rather in tandem with physiological and molecular changes that are related to normal biological ageing.

Anxiety and depression are now linked to an increase in cardiovascular risk factors such as atherosclerosis, metabolic syndrome and coronary artery disease. The root causes of these chronic conditions include mitochondrial dysfunction, telomere attrition, vascular disruption, cellular senescence, deregulated nutrient sensing and a low-grade chronic inflammation persistent throughout the body[4].

While mitochondrial dysfunction is a consequence of ageing, depressed people present with intense levels of oxidative stress and free radical production leading to increased mitochondrial, cellular and DNA damage to potentially all cells and tissues in the body.

Telomeres are found at the ends of chromosomes and are responsible for protecting the genetic data, making it possible for cells to divide and hold the secrets to ageing and chronic diseases. Individuals with depression present with shorter telomeres. Without telomeres, chromosome ends can fuse together and corrupt the cell's genetic blueprint, possibly causing cell malfunctions or even cell death. As broken DNA is dangerous, a cell will sense and repair the damaged chromosomes. With shorter telomeres, the end of chromosomes resembles broken DNA. The cell tries to repair something that wasn't broken, forcing the cell from dividing and, eventually, dying.

Cerebrovascular blood flow disruptions are common in normal ageing. They affect brain blood flow and cause leakage of the brain blood barrier, triggering widespread inflammation contributing to depressive symptoms.

> "Substituting inactivity with light physical activity produces positive changes to the inflammatory profile"

Ageing causes an increase in insulin resistance and decreased release of insulin-like growth factor-1. The changes in these anabolic and catabolic processes contribute to metabolic disorders, sarcopenia and low muscle strength. There is a strong correlation between the severity of depressive symptoms and metabolic disorders.

Intercellular communication is defined as the transfer of information from one to another through signalling mechanisms. Changes in these communication pathways are common in both normal ageing and depression. Senescent cells are ageing, poorly functioning cells that self-sacrifice and are cleared to make way for newer functional cells. If they are not cleared, they accumulate and take up space and activate a signalling reaction that releases pro-inflammatory cytokines. Depressed people report higher concentrations of senescent cells and increased signalling reactions, causing a chronic low-grade inflammatory response[5].

The elevated levels of pro-inflammatory cytokines and lower anti-inflammatory cytokines found in depressed people can affect memory, mood and cognitive health. These levels are compounded by the increased low-grade inflammation in ageing termed 'inflammaging' that contributes to an increase in cardiovascular risk factors and alters the cellular environment in the brain.

Over 50% of all deaths globally are related to inflammation-caused diseases, including heart disease, metabolic disease, cancers, Alzheimer's disease, autoimmune diseases, depression and neurological diseases.

The increased inflammatory response in ageing is caused by the chronic stimulation of innate immune system-damaging sensory receptors that become hypersensitive to damaging signals and increase levels of oxidative stress, resulting in insulin insensitivity, atherosclerosis and tumour growth and auto-immune reactions in the brain leading to depression, autism, poor memory, Alzheimer's disease and multiple sclerosis in older populations[6].

The key pro-inflammatory cytokines include interleukin-6 (IL-6), tumour necrosis factor (TNF) and C reactive protein (CRP) that act as messengers between the immune system, blood and vessels and the endocrine system. For example, the accumulation of abdominal fat with ageing causes an increase in pro-inflammatory immune cells that release inflammatory substances leading to low-grade inflammation throughout the body. These pro-inflammatory markers cause a cross-talk between the immune system, adipose tissue, muscle cells and the brain's hypothalamus.

Substituting inactivity with light physical activity produces positive changes to the inflammatory profile by overriding the chronically active immune system through the release of anti-inflammatory myokines that slow the system down. This relaxation response allows the immune system to react to normal danger signals rather than be in a constant state of alert, which not only requires additional energy but also down regulates the pro-inflammation response.

More significant changes can occur in moderate-to-vigorous activity for middle-aged adults, causing lower levels of pro-inflammatory cytokines particularly when the volume is kept constant. The three possible explanations supporting physical activity as a key player in reducing and controlling systemic low-level inflammation include a reduction in visceral fat, an increased production and release of anti-inflammatory cytokines from the contracting skeletal muscle, and a reduction in the hyperactivity of the immune system's white blood cells.

Physical activity can induce both positive cognitive effects and physiological effects of improving memory, mood, coping mechanisms, executive function and attention, relieving many of the symptoms associated with depression and ageing.

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