AGEING AND SKELETAL CHANGES

In **Paul Batman's NEW SIX-PART SERIES**, he looks at the effects of ageing on the human body. Here, in part one, he examines the skeletal changes that occur as we age.

Important information on space travel has been collected over the years to understand the abnormal physiology that occurs with inactivity and ageing.

The data collected and interpreted has been extensive, with parallels drawn between space flight, inactivity and ageing. Changes to the mechano-skeletal system, cardiovascular system and the vestibula-neuromuscular system occur in a similar fashion across all three only 10 times faster in zero gravity than during ageing. The major difference is that the long-term effects are reversed in the following weeks after the space flight. What they have in common is the reduced effect of gravity – called gravity deprivation – and the problems associated with sedentary lifestyles and ageing resulting from gravity withdrawal.

From birth, the mechanical forces of gravity shape our physical development. By the time we start walking, we are constantly attempting to overcome its forces. This battle with gravity continues throughout our lives but, as we age, we begin to withdraw gravity from our lives and, consequently, our bodies age at a faster rate. Our new sedentary society has accelerated these adverse changes to the point where we are living longer but with greater disabilities and diseases due to this mismatch.

The concept of tensegrity theory states that molecules, cells, tissues, organs and systems need to be exposed to constant intermittent tension and mechanical forces in order to develop[1]. When this stops, there is a breakdown in their structure leading to problems with cell and tissue growth and development.

Our bodies have evolved over many millions of years to work against gravity. It differentiates up from down and causes fluid shifts throughout our cells that regulate homeostasis in all our systems.

Immobility, falls and fractures are major health risk factors for older adults and are all related to muscle and bone weakness. The most common fractures are at the hip, vertebrae and the wrist, with fragility fractures costing the NHS £4.4 billion per year and USD $57 billion, which is projected to grow to USD $95 billion by 2040[2].

On average, humans lose 10-20% of their total bone mass by the age of 65 years, with women experiencing a three-to-five-year acceleration after menopause[3].

The two major types of bone structure are cortical and cancellous bone. Cortical bone accounts for approximately 80% of the total skeletal structure, while cancellous makes up the remaining 20%. Cortical bone forms the hard outer layer of the long bones of the body, while cancellous bone is found in the spine, pelvis and the ends of the long bones and is made of a latticework structure.

The decrease in bone mass that accompanies ageing is site specific and dependent upon the content of cortical and cancellous bone. It is at these sites that the bone remodelling process, which accompanies bone health, declines with age, increasing the skeleton's proportion of 'old bone' or fatigued, damaged bone.

The bones lose density and become more porous and more liable to fracture. Up to 50% of the total bone loss in the vertebrae in women occurs before menopause, while cortical bone is primarily lost after menopause. At age 50 years, both men and women experience a significant bone loss at the head of the femur, making their hips particularly vulnerable.

> "On average, humans lose 10-20% of their total bone mass by the age of 65 years"

Ageing has a serious effect on the structure of cancellous bone, resulting in significant malformations. The horizontal framework of the cancellous bone becomes very brittle, leaving the strength of the bone to be maintained by the vertical columns, particularly in the vertebral bodies of the spine. The decrease in strength at this site is much greater than the overall bone loss throughout the body.

Cancellous bone is usually found at the end of long bones forming weight-bearing joints. Damage to this section of bone can cause a crumbling effect within the joint, limiting movement.

Cortical bone tends to be maintained particularly in older men as they add new bone to the existing outer ring, increasing the width of the bone. At approximately 75 years, even cortical bone is severely affected in both old men and women.

As we age, our height reduces, mainly due to degenerative changes in the discs in the spine, along with increased curvature of the spine. The intervertebral discs dry out and shrink with age or disuse, reducing their capacity to act as shock absorbers for the vertebral column. Over time, there is an increase in body fat (about 10%) accompanied by a decreased muscle mass, which is more apparent in men because of the reduction in testosterone that occurs in the mid 50s.

Osteopenia is the loss of bone mass without a loss of bone quality and can progress to osteoporosis. Women over 65

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years present with a continual loss of cortical bone in the femur, radius and the heel bone up to the age of 90 years[3].

Inactivity and sedentary lifestyles can cause a further decrease in bone mineral density, while weight-bearing activities produce favourable changes. Bodyweight and the tension exerted on muscles, tendons and ligaments play an important role in maintaining bone integrity.

Physical activity plays a significant role in maintaining or slowing down the normal loss of bone density with ageing and inactivity. However, if physical activity is stopped, bone mineral density can revert to pre-training levels. Physical activity must be maintained for life for maximum benefit[4].

People who have been active throughout their life tend to maintain bone mineral density more than those who begin moving later in life. In the first one to three years after menopause, women experience a rapid loss of bone mineral density. Following the decrease in oestrogen secretion, there is an increased inability of the bone to absorb calcium, increasing calcium in the bloodstream. Calcium supplementation during this period is usually ineffective in maintaining bone mineral density. Once the new steady state bone mass is reached, modest calcium supplementation is more effective. Both men and women over the age of 65 years should consume at least 1,500mg of calcium per day.

Tendons and ligaments that support the skeleton are made up of 70-80% collagen fibres. As the body ages, the concentration of collagen in both ligaments and tendons is reduced. Ageing, inactivity and sedentary lifestyles have a detrimental effect on the function of ligaments and tendons. The rate and the magnitude of strength loss within these structures is rapid, while their retraining is very slow[5].

Physical activity maintains the level of strength required for the ligaments and tendons to operate near their functional level. When daily loading is reduced, the collagen fibrils lose their typical structure, suggesting that ageing changes are due more to disuse than any other factors. Old collagen fibrils are not being replaced at the same rate as new collagen fibrils are being produced, causing changes in the overall structure.

After exposure to physical activity, there is an increase in the stiffness of the collagen fibres, resulting in an increase in strength. The musculotendinous junction also increases in strength, allowing more force to be generated through the tendon attachment. The improvement in strength only occurs in the ligaments and tendons of the muscles and joints that are specifically recruited.

In older adults, strength training, aerobic exercise, flexibility, balance and stability exercises have all been recommended to maintain bone mineral density by reducing sarcopenia and increasing muscle mass, and producing mechanical stressors on the skeleton that increases osteoblast activity[6]. fp

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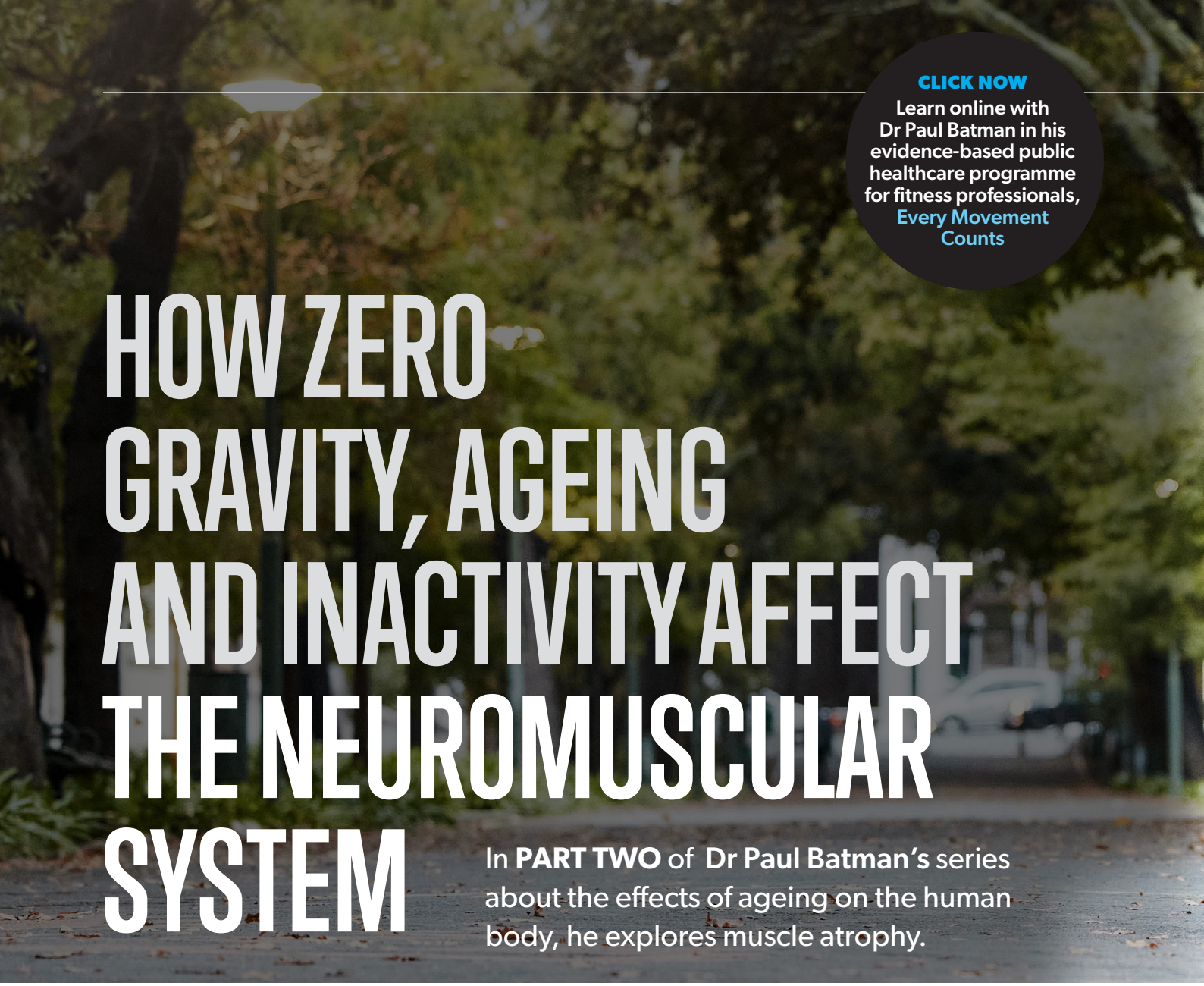

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HOW ZERO GRAVITY, AGEING AND INACTIVITY AFFECT THE NEUROMUSCULAR SYSTEM

In **PART TWO** of **Dr Paul Batman's** series about the effects of ageing on the human body, he explores muscle atrophy.

An inevitable consequence of growing old and inactivity is the possibility of suffering from physical frailty, caused by a severe reduction in strength, mobility, endurance and balance.

Over 65% of older adults in the US die from problems caused by falls, mainly due to muscle atrophy, diminution of the nerve reflex arcs and a loss of balance. While the inner ear mechanism is primarily responsible for balance, so too are proprioceptors, muscle, joint, tendon and skin receptors. All these sensory receptors supply information to the brain about the loading of limbs as they change position and undergo deformations, different force vectors and weight distributions.

In space, as there is no difference between 'up or down', the brain receives far less – and confusing – information from the proprioceptors that inform the body where it is in relation to gravity. Astronauts can only determine their position by observing their limbs and cannot rely on their inner ear balancing mechanism. The problems with the inner ear balancing are also seen in older adults as they begin to experience body balance problems[1].

In older adults, many of these balance problems are associated with gravity withdrawal as they voluntarily choose to become more inactive, particularly with an increase in prolonged sitting, leading to a decrease in muscle mass. The progressive loss of muscle tissue over time is called sarcopenia, with the loss ranging from 4-27% depending on the country and gender.

In micro gravity the body has mass but no weight, causing a reduction in blood flow to the legs. Without gravity, the nervous innervation to the legs is minimal, making it difficult to bear load or contract, leading to muscle atrophy particularly in the postural muscles, with little atrophy observed in the arm muscles as they are in continual use.

After the age of 30 years, there is an 8% decline in the strength per decade in the arms, back and legs. While these changes are unavoidable, they can be controlled as even elite athletes experience a decline in performance but at a much later stage[2].

The reduction in performance mirrors the reduction in muscle mass, which is approximately 7% per decade up to 50 years and then 11% per decade up to 70 years. Muscle mass is decreased by approximately 25-30% by the age of 65 years. Muscle force declines at a 10% faster rate than strength. At age 45 years, there is a critical period of muscle mass reduction and a lowering of resting metabolic rate.

Older adults with poor muscle strength have a 2.6-fold risk of severe immobility, a 4.3-fold increase of slow gait speed and a 2.1-fold greater risk for mortality.

The decline in muscle mass is a function of the difference between protein synthesis and protein degradation, ultimately leading to muscle atrophy. In short space flights, astronauts lose approximately 1% of muscle mass per month and generally about 1.8-2% of bone. This is equivalent to the muscle and body mass lost through ageing in 12 months.

The rate of muscle atrophy can be very specific to the muscles and their role in habitual movement. Many muscles that contract continuously to maintain normal functioning appear to hold the cross-sectional size more effectively than muscles that are deprived of gravity or used very little. The common variable in both astronauts and inactive subjects is the reduced influence of gravity on their bodies that leads to a reduction in muscle strength and mass. Ageing is accelerated with inactivity and a sedentary lifestyle[1].

> "The decline in muscle mass is a function of the difference between protein synthesis and protein degradation"

With ageing and inactivity, muscle atrophy can result from changes within motor units. Motor end plate activity is reduced by the decline in the activity of the synaptic neurotransmitter acetylcholine and the enzyme acetylcholinesterase. Acetylcholinesterase is responsible for cleaning up the synaptic gutter after acetylcholine has been released, initiating the nerve impulse on the dendrites of the adjacent neuron.

During the normal turnover of synaptic junctions, new neurons are linked to their previous muscle fibre type. With ageing and inactivity there is a denervation of type 2 fibres, meaning that neurons previously innervating type 2 fibres are now supplying type 1 fibres. The result is an atrophy and loss of force generated by type 2 fibres. The number of motor units also decreases, reducing the fibre's action potential by over 20%, as well as voluntary force by 30%.

Muscle atrophy can also be caused by a decrease in the number of muscle fibres. Muscle fibre number is said to begin declining at approximately 30 years, continuing to approximately 80 years.

The cross-sectional area of type 1 fibres appears to remain the same over the lifespan, whereas type 2 fibres can decrease by up to 28%. The maximum number of muscle fibres is reached at age 25 years and then slowly decreases by approximately 40% until age 80 years.

While the number of muscle fibres changes significantly from a relatively early age, fibre diameter remains stable until approximately 70 years of age. It is at this age that the fibre diameter of type 2 fibres atrophies more than type 1 fibres, mainly due to the habitual use of these muscles. Those muscles that are constantly in use have fibres that remain constant in diameter, while those that are subject to disuse or inactivity show the greatest atrophy[2].

Sarcopenia has also been associated with a reduction in the number of satellite cells. The function of these cells is the assist in muscle regeneration as well as maintaining cell homeostasis. The loss of satellite cells is particularly evident in the type 2 fibres and is significantly blunted in older males[3].

Type 2 fibres produce a four-fold increase in power output above and beyond that of type 1 fibres. When there is a decrease in the number and size of type 2 fibres, there will be a greater reduction in power output. This can have significant implications for performing daily functional movements, as the speed of walking, climbing stairs and standing from a seated position is heavily dependent on the power output from type 2 fibres. Type 2 and their power output play a significant role in reducing the risk of falling[4].

In space, 15-20% of type 1 fibres are converted to type 2 fibres caused by the reduced need for postural stability and weight-bearing ability.

The exercise programmes currently prescribed during space travel, including treadmills, elastic bands, bicycle ergometers and different types of resistance equipment, have only been partially successful in combating sarcopenia changes.

NASA scientist Dr Joan Vernikos states, "In spite of strenuous exercise for many hours every day, loss of bone, muscle and cardiovascular fashion happens 10 times faster in space than on earth. Exercise without gravity is not as effective." Further to this statement, exercise is regarded as only 50% effective in zero gravity compared to on earth[5].

During her tenure at NASA, Dr Vernikos prescribed many different exercise protocols, including exercising vigorously over varying durations, finally instituting an intermittent postural change protocol that required astronauts to rise from a reclining position to sitting and then to standing once every 15-20 minutes over eight hours. Astronauts completed this transitional movement 24-32 times per day. It was found that this transitional movement stimulated vascular reflexes that were disabled during spaceflight and prolonged sitting. Astronauts responded to this protocol better than any other exercise regime.

Daily intermittent standing and walking at different speeds and over different terrains has widespread application for breaking up prolonged sitting and reducing the time spent in sedentary activities, and should be implemented in addition to physical activity programmes across all age groups.

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AGEING'S EFFECT ON THE CARDIOVASCULAR AND RESPIRATORY SYSTEMS

Dr Paul Batman looks at the changes in cardiovascular and respiratory physiology that happen with age in **PART THREE** of his series on the effects of ageing.

Ageing causes changes in the cardiovascular system resulting in alterations in cardiovascular physiology. These changes must be differentiated from pathology effects, such as coronary artery disease, that occur with increasing frequency during ageing. The changes with age are universal but do not necessarily occur at the same rate, accounting for the difference between chronological age and physiological age.

Cardiac output is the amount of blood pumped from the heart per minute and is calculated by heart rate multiplied by stroke volume. Advancing age and inactivity causes a decrease in cardiac output, mainly due to a decrease in stroke volume. Stroke volume is strongly related to blood volume. In detraining or inactivity, blood volume decreases causing a concomitant decrease in stroke volume.

The size of the heart decreases with ageing, reducing its pumping ability and stroke volume with a 14% decrease in left ventricular mass observed during space flight.

Space flight, inactivity and head-down bed rest mimic ageing, with cardiac function initially reduced by a fluid shift from the lower body to the upper body, increasing the plasma volume in the upper body. The receptors in the carotid arteries, aorta and the myocardium are stimulated causing an increase in kidney activity, leading to a reduction in body mass and overall plasma volume. The lower plasma volume levels lead to a decreased heart size, reduced cardiac filling, reduced stroke volume, cardiac output and VO_2max. The sensitivity of the baroreceptors is significantly decreased within 10-12 days of inactivity, ageing, head-down bed rest or space flight.

When an astronaut returns to earth or standing after prolonged bed rest or inactivity, the change in the cardiovascular system leads to orthostatic hypotension (low blood pressure). Ageing also results in a desensitising of the baroreceptors that regulate blood pressure. The cardiac output changes that occur due to prolonged bed rest (three weeks) are equivalent to those seen after 30-40 years of ageing.

In the case of a healthy active older adult, cardiac output may not decrease as significantly due to an unchanged stroke volume or left ventricular volume. Some evidence suggests that left ventricular volume can increase in active older males but not in active females, reportedly due to a lack of oestrogen after menopause in the older females.

While the size of the left ventricle might remain the same, there are changes in cardiac function that can affect health, including an increase in after load, an increase in heart wall thickness and overall mass and a reduction in diastolic filling time. An ageing pigment called lipofuscin is deposited throughout the heart, causing muscle cells to degenerate.

Ageing also causes a decrease in resting and maximum heart rate and an increase in the prevalence of arrhythmias. Some of the heart's pathways begin to develop fibrous tissue and fat deposits, causing the sinoatrial node (SA) or pacemaker to lose some of its cells, resulting in a slower heart rate.

The mechanism for the reduced heart rate has yet to be determined but could be caused by the reduced conduction velocity of impulses generated from the sinoatrial node as it passes through the heart's myocardium.

> "The closing capacity of the lungs increases with age, resulting in airway obstruction during normal breathing at rest"

Mean arterial blood pressure increases with age, especially after menopause in females. This is amplified in inactive older adults, while healthy active older adults only show a slight increase in systolic blood pressure. The main artery of the heart (called the aorta) becomes thicker and less flexible, contributing to higher blood pressure.

In older adults, there is an increase in vascular resistance leading to a decrease in blood flow to specific body regions, including an increase in blood flow to the heart's myocardium. The capillary walls become thicker, slowing the rate of exchange of nutrients and wastes. Contributing to this resistance is the increased stiffness and reduced compliance and elasticity of the arterial walls. Sympathetic nerve discharge increases to the skeletal muscle as well as elevated epinephrine and norepinephrine levels, all contributing to the increase in vascular resistance.

In ageing, space flight and head-down bed rest, the arterial walls are no longer stimulated by the shear force of the blood pushing against the endothelial lining of the arteries, resulting in rigid structural wall remodelling.

Maximum oxygen consumption declines with advancing age and inactivity. The rate of decline with age is 50% less in active healthy people. Longitudinal studies suggest that VO_2max can be maintained over middle age for 10-20 years.

There is little doubt that older adults can improve their VO_2max to a significant degree, often double that of untrained peers. Studies have reported VO_2max of approximately 49ml.kg.min-1 in older female endurance athletes, which is 20-25% greater than in untrained females. While these increases are significant in both males and females, the magnitude of the increase is greatest in males, varying dramatically from person to person with ranges of between 2% and 49% reported.

Based on the Fick principle (heart rate X stroke volume X arterio-venous oxygen difference) to determine VO_2max, the age-related decrease in maximum heart rate reduces the maximum cardiac output required to reach VO_2max. The improvement in VO_2max in older males is due to a greater maximal cardiac output, caused by a greater stroke volume. The stroke volume changes are due to an increased filling time of the left ventricle and an improved left ventricle contractile performance.

Approximately 50% of VO_2max improvements in older males is due to an increase in arterial venous oxygen difference (A-vod). In older females, 100% of VO_2max improvements is due to this same increase. The improvement in A-vod is due to an increase in capillary density and oxidative enzymes in the skeletal muscle. In addition, there is also an increase in the dilation ability of the arteries and an increase in blood flow to the muscle.

The decrease in stroke volume in ageing is caused by the reduction in blood volume that normally occurs with age, resulting in approximately a 50%-100% reduction in VO_2max.

The remaining contributor is the relatively small reduction in the arterial venous oxygen difference (A-vod). It is possible that the maintenance of A-vod is due to the high levels of oxidative ability of the skeletal muscle.

The decline in VO_2max in ageing is substantially affected by changes in body composition. VO_2max is related to muscle mass, more specifically due to a denervation of type 2 fibres. A decrease in the ability of these fibres will reduce the intensity of the exercise, contributing to a decrease in caloric expenditure and an increase in fat mass. Alternatively, any reduction in bodyweight will result in an increase in VO_2max when expressed relative to bodyweight (ml.kg.min-1).

During physical activity, blood pressure remains unchanged or increases with age. Systolic blood pressure is higher during physical activity in older clients because of their elevated resting levels and increased vascular resistance. While this increase

occurs in both sexes at maximal levels of exercise, it is greater in females.

Myoglobin is a protein found within the muscle tissue that stores and releases oxygen from within the muscle and helps transport oxygen from the capillary to the mitochondria or the powerhouse of the cell. Myoglobin increases with physical activity and decreases with inactivity and ageing, with the slow degradation of myoglobin contributing to the body's poor performance in any continuous physical activity.

Haemoglobin is found in the red blood cells and is responsible for transporting oxygen. In ageing, the speed of the red blood cells moving around the body is slowed dramatically, causing a slower response to blood loss or infection.

White blood cells used in immunity generally remain at the same level, although neutrophils reduce in number, inhibiting their ability to fight off bacterial infections.

Pulmonary system changes

Pulmonary ventilation, alveolar ventilation and gas exchange are regarded as some of the most important functions of the respiratory system. Pulmonary function is affected by the characteristics of the chest wall, strength and endurance of the respiratory muscles, diffusion capabilities, perfusion and neurochemical regulation.

Changes in pulmonary function are affected by the inability of the lung tissue to recoil and a stiffening of the chest wall. As the lung tissue is comprised of elastin and collagen fibres, the elastic recoil of the lung during exhalation is adversely affected by a reduced cross-linking of these fibres, increasing the total work completed by the respiratory muscles, especially at moderate workloads.

The lungs begin to age as early as 35 years. Initially, the diaphragm becomes weaker, causing a shortness of breath. As the lung ages, there is a decline in surface area from approximately 70 square metres at age 20 years to 60 square metres at age 60 years, reducing the capillarisation of the lungs and gas exchange capabilities. This is compounded by the reduced compliance of the arteries, affecting blood flow. The small alveoli, where diffusion occurs, begin to collapse and lose their shape.

The increased chest wall stiffness is a function of costal cartilage calcification, narrowing of the intervertebral discs, changes to the joint where the ribs and vertebrae articulate and a change in the shape of the chest wall. Part of the increased stiffness is due to an atrophy of the type 2 fibres that comprise the diaphragm muscle.

The most significant change in lung function that affects physical activity is the increased limitation in expiration. This limitation causes a greater airway closure at higher levels of lung volume, resulting in a decline in the amount of air that can be expired in one second (FEV 1.0). The inability of the lungs to exhale the carbon dioxide will cause an increased concentration in the blood and reduced exercise tolerance.

The closing capacity of the lungs (the lung volume where the small airways begin to close) increases with age, resulting in airway obstruction during normal breathing at rest.

There is a decrease in the sensitivity of nerves in the airways, causing the lungs to become more prone to tissue damage. When particles enter the lungs, the airways trigger a coughing response to clear the particles. During the ageing process, the desensitised nerves stop the coughing response, causing damage to the lung tissue.

These many changes to the respiratory system generally cause a shortness of breath and an increased susceptibility to infection.

Even though the long-term effects of ageing on the cardiovascular system and respiratory system cannot be eliminated entirely and are inevitable, they can be controlled and slowed by lifestyle and exercise choices. *fp*

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TELOMERES COULD BE THE ANSWER TO LIVING LONGER

PART FOUR of **Dr Paul Batman's** series on the effects of ageing explores how the telomere has become a significant marker of ageing.

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There is a growing body of evidence that identifies lifestyle factors as a contributor to long-term health through changes to the cells' telomeres[1].

In the nucleus of each cell, genes are stacked into DNA molecules, tightly packed into thread-like structures called chromosomes surrounded by specific proteins that support its structure, with every cell having 23 pairs of chromosomes.

At the end of the chromosomes are sections of DNA called telomeres. Telomeres protect genetic data, make it possible for the cells to divide and hold the secrets to ageing and diseases.

Telomeres are similar to plastic tips on shoelaces. They keep chromosome ends from fraying and sticking to each other, which could destroy or scramble genetic information. More recently, the telomere has become a significant marker of ageing. With each division of the cell, the telomere shortens and, over time, they can become habitually shortened, causing many age-related diseases as well as the more common wrinkles and grey hair.

The shortening of the telomere reflects the number of times the cell has divided. When they get too short and the cell can no longer divide, they become inactive, 'old' or die. This shortening process is associated with ageing, cancer and an increased risk of death[1]. Telomeres are like a time bomb ready to go off at any time!

Cells can normally divide only about 50 to 70 times, with telomeres getting progressively shorter until the cell becomes old or dies. Longer telomeres mean the cell can divide more often before entering senescence or dying, thereby increasing longevity.

Without telomeres, the genes essential for life would get shorter each time a cell divides. Telomeres allow cells to divide without losing genes. Cell division is necessary for growing new skin, blood, bone and other cells.

Telomerase is the enzyme that ignites many of these reactions and remains active in sperm and eggs, which are passed from one generation to the next. If reproductive cells did not have telomerase to maintain the length of their telomeres, any organism with these cells would soon become extinct[2].

As a cell becomes cancerous, it divides more often and its telomeres become very short. If the telomere gets too short, the cell may die. These cells escape death by making more telomerase enzyme, which prevents the telomeres from getting even shorter.

Successful ageing requires an ability to balance damage with repair of cells. When the damage to the cell overcomes the repairing of the cell, subsequent long-term degeneration will occur, resulting in Alzheimer's disease, cardiovascular disease, Type 2 diabetes and sarcopenia[2].

Many cancers present with shortened telomeres, including pancreatic, bone, prostate, bladder, lung, kidney, and head and neck[3].

Geneticist Richard Cawthon from the University of Utah found shorter telomeres were associated with shorter lives. In a sample of people over 60 years, those with shorter telomeres were three times more likely to die from heart disease and eight times more likely to die from infectious disease.

Without telomeres, chromosome ends can fuse together and corrupt the cell's genetic blueprint, possibly causing malfunctions or cell death. As broken DNA is dangerous, a cell will sense and repair the damaged chromosomes. Without telomeres, the ends of chromosomes would look like broken DNA. The cell would try to repair something that wasn't broken, forcing the cell from dividing and eventually dying.

Cawthon's study found that people with longer telomeres lived longer than those with shorter telomeres, suggesting that increasing the length of telomeres could increase the lifespan by approximately five years.

A major result of ageing is 'oxidative stress'. Oxidative stress is the damage to DNA, proteins and lipids (fats) caused by oxidants, which are highly reactive substances containing oxygen[3].

> "Active individuals have longer telomeres in their immune cells compared to sedentary individuals"

The free radicals that are produced in combination with the consumption of alcohol and smoking can result in inflammation and infection, all of which has an impact on shortening the telomere, while a diet rich in antioxidants such as vitamin E, vitamin C and beta carotene have been reported to increase the length of telomeres and lower the risk of breast cancer[4].

Smoking is significantly associated with telomere shortening, with one packet a day for 40 years reducing the lifespan by 7.4 years. Telomeres could be used as a biomarker for oxidative stress caused by smoking and can also predict the rate of ageing[5].

Obesity is also associated with increased oxidative stress and DNA damage, mainly caused by the increased release of adipocytokines from the white adipose tissue contributing to a decrease in the length of the telomeres. Obese tissue releases an array of hormones that send messages to key organs creating a state of low-grade inflammation that increases free radical production in the body.

Long-term exposure to air pollutants as indicated by the increased presence of toluene and benzene can also reduce telomere length. Researchers have reported that traffic police officers exposed to pollution have shorter telomeres than office workers of a similar age.

Increased stress levels cause the release of glucocorticoid hormones from the adrenal glands, causing a reduction in antioxidant levels and increasing oxidative stress levels, thus accelerating telomere shortening.

If people with a low fitness level and who are obese, older or suffering from a chronic disease undertake high-intensity exercise, there is the potential for the unregulated production of free radicals (ROS) that can affect the cell membrane.

Several studies have reported a positive association between physical activity and telomere length. Active individuals have longer telomeres in their immune cells compared to sedentary individuals, with differences in telomere length equating up to approximately 10 years' difference in biological age. Some studies have reported that moderately active individuals have longer telomeres compared to both sedentary and extremely active individuals.

A group of 50-70-year-old subjects in both the lowest <990kcal/wk-1 and highest energy expenditure >3,541kcal/wk-1 groups reported shorter telomeres than subjects in the middle group who expended between 991-2,340kcal/wk-1, even when controlling for age, gender and bodyweight.

Older people identified with shorter telomeres have a three to eight times' increased risk of dying from heart disease and infections. This is exacerbated by older obese people smoking, being unfit, being highly stressed and living in polluted environments, putting them at a further risk of telomere shortening[5].

Moderate-intensity physical activity (3-6 METs) is more likely to not cause skeletal muscle damage, does not result in an excess ROS and inflammation, and does not shorten telomeres. Consequently, moderate-intensity activity would appear to at least maintain or more likely increase telomere length with age. Exercise increases the level of telomerase and antioxidant levels and suppresses several proteins responsible for cell dying.

The available research data supports that physical activity in combination with stress reduction, dietary modifications, limiting alcohol intake, maintaining a healthy weight and a good night's sleep can slow cellular ageing and improve health by reducing telomere shortening. *fp*

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INFLAMMAGEING: AGEING AND INFLAMMATION

In **PART FIVE** of **Dr Paul Batman's** series on the effects of ageing, he explores the concept of inflammageing, which refers to inflammation in the aged.

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While the National Physical Activity Guidelines recommend at least 150 minutes of moderate or 75 minutes of vigorous physical activity per week, there are many older adults, obese or overweight, unhealthy and unfit who are unable to complete this prescription. This can be due to the constant low level of chronic inflammation in the tissues of their body that predisposes them to cardiovascular disease, colorectal cancer, type 2 diabetes, chronic obstructive disease and different types of dementia that can persist for decades.

Inflammation in the aged is often referred to as inflammageing, a condition that is highlighted by high levels of blood inflammatory molecules leading to higher mortality rates and chronic diseases, even in the absence of known risk factors.[1]

Under normal conditions, inflammation is important to fight the spread of diseases or infection and is at the centre of the body's basic survival instincts. During any infection, the immune system is activated and mobilised by releasing white blood cells that attack the irritant. After the battle, the white blood cells clean up the affected area, promoting and assisting with healing, and finally return to normal levels.

The role of inflammation is to eliminate the initial cause of cell injury, remove damaged tissues and begin the repair and restoration process. Heart disease, cancer, stroke, Alzheimer's disease, diabetes and kidney disease are the consequences of this low-grade chronic inflammation that can be caused by high blood sugar levels, increased food intake, inactivity and oxidative stress and ageing.

In the aged, overweight, obese, inactive and unhealthy, inflammageing is always present and becomes progressively more difficult to control and neutralise with the cells always sensing they are being attacked. This causes the immune system to become overworked and unable to fight legitimate infections, potentially leading to chronic diseases. As the inflammation progresses, it is possible that the immune system will begin to react against itself, as seen in autoimmune diseases such as Lupus, Graves' disease and Crohn's disease.

Obesity in the aged is associated with a low-grade inflammation that is similar yet different from normal inflammation. To make a distinction between normal inflammation and obesity-related inflammation, the term 'metaflammation' was coined to describe a milder inflammation that has a direct relationship between obesity and chronic disease, caused in response to excessive nutrients and energy.[1]

The accumulation of abdominal fat that often accompanies ageing causes an increase in pro-inflammatory immune cells that release inflammatory substances leading to low-grade inflammation, causing a cross talk between the immune system, adipose tissue, muscle cells and the brain's hypothalamus.

> "It is not necessary to undertake high-intensity exercise for the anti-inflammatory response to occur"

Anti-inflammatory agents have been in existence for thousands of years, while the pro-inflammatory agents are relatively new, possibly from industrial changes in the late 19th century. Given that the modern body has only had a couple of hundred years to adapt to these new pro-inflammatory agents, the immune system has developed its holding pattern of metaflammation to combat these side effects, including reducing the effectiveness of insulin released from the pancreas to control and transport glucose, potentially leading to type 2 diabetes and heart disease.

Those most 'at risk' of this low-grade chronic inflammation are the aged who are unfit, obese and sedentary, who do not regularly engage in physical activity and whose immune system is in an overactive state, resulting in a high-energy demand.[2]

Low-grade systemic inflammation is associated with a decrease in insulin sensitivity, atherosclerosis and tumor growth, all of which have been termed 'diseases of physical inactivity', and is regarded as a strong predictor of all-cause mortality and cardiovascular disease in older populations.

Inflammageing is generally measured by an increase in inflammatory circulating markers such as cytokines, growth factors and reactive oxygen species. The key cytokines include interleukin-6 (IL-6), tumor necrosis factor (TNF) and C-reactive protein (CRP), all acting as messengers between the immune system, blood and vessels and the endocrine system. Active older adults often present with low levels of IL-A, TNF and CRP, as well as higher anti-inflammatory markers such as IL-10, while the more sedentary older adults have higher mortality rates and lower anti-inflammatory markers.

Physical activity overrides the chronically active immune system and reduces inflammation across the ageing populations through the increased release of anti-inflammatory myokines that inform the immune system to slow down and relax. 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.

In studies that cross-reference different age groups with activity levels and inflammation it was found that, regardless of age, the critical variable was still physical activity. Those at the higher physical activity levels had the lower markers of inflammation, irrespective of BMI.

Other factors that exacerbate the inflammatory process in the aged include smoking, increased number of fat cells, sedentary lifestyle, obesity, poor diet and high blood pressure.[3]

Ironically, in the early stages of a fitness programme, a pro-inflammatory response has been noted, but as fitness levels improve the response becomes anti-inflammatory, indicating that the best results occur with a strong adherence to a physical activity programme over time.

There are three possible explanations as to why physical activity is a key player in reducing and controlling systemic low-level inflammation in the aged. These include a reduction in visceral fat, an increased production and release of anti-inflammatory cytokines from the contracting skeletal muscle, as well as a reduction in the activity of the immune system's white blood cells.

As the skeletal muscles contract during physical activity, the muscle fibres produce and distribute anti-inflammatory molecules that act on all regions of the body and protect the arteries against atherosclerosis and stenosis (narrowing). It is also possible to receive an anti-inflammatory response from physical activity without any significant decrease in obesity or fat levels. Regular low to moderate muscle contractions promote anti-inflammatory signals to other distant regions of the body in addition to skeletal muscle.[4]

It is not necessary to undertake high-intensity exercise for the anti-inflammatory response to occur. In the aged, unhealthy, unfit, obese and sedentary populations, high-intensity exercise can add to their already inflamed state due an increase in oxidative stress through the release of rebel oxygen molecules that can damage the internal structure of the cell by increasing pro-inflammatory molecules.[5]

Resistance training also plays a significant role in reducing low-grade inflammation in the aged by increasing lean body mass and reducing fat mass, both of which are central to maintaining a strong anti-inflammatory profile.

The mental health of older adults has also been identified as a potential contributor to inflammageing, with a greater level of pro-inflammatory molecules observed in those with mood disorders, anxiety, loneliness and depression. *fp*

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AGEING AND MITOCHONDRIAL DYSFUNCTION

In the **FINAL PART** of **Dr Paul Batman's** series on ageing, he looks at the powerhouse of the cell – the mitochondria.

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Mitochondria are very small organelles found within all cells producing energy for the remake of ATP, a molecule necessary for muscle contractions. There are over 400 mitochondria in one cell, totalling over 10 million billion in the entire adult body, accounting for approximately 10% of bodyweight.

To get some idea of their size, approximately one billion mitochondria would fit into a grain of sand. There are organelles within the cell that have dedicated jobs to ensure that each of the 50 trillion cells in the body can function normally in all the chemical processes required to maintain life. Mitochondria constantly move around the cell, rarely remain stationary and are often referred to as the powerhouse of the cell.

The number of mitochondria within each cell is governed by the metabolic demand of that specific cell (e.g., the liver, kidneys, muscles and brain have hundreds of thousands of mitochondria, while sperm have less than 100).

Older adults have enlarged mitochondria that are less dense and more rounded in shape with reduced structural strength, with a greater proportion of non-functional mitochondria leading to increased physical inactivity[1].

A greater number of mitochondria exist in slow-twitch or Type 1 muscle fibres due to their need to utilise oxygen and contract for long periods of time. Type 2a muscle fibres have a significant number of mitochondria, while Type 2b fibres have very few.

The importance of mitochondria is now being investigated for its DNA and its ability to track changes in DNA mutations over time. Based on the mitochondria's ability to carry genes, it is possible to trace genetic ancestry down through the female relatives. These genes are passed down through the female egg cell and not in the sperm of the male, with approximately 100,000 mitochondria being passed down in the egg cell to the next generation.

Mitochondria contain approximately five to 10 copies of genes, with many thousands of copies of the same gene in each cell, with the nucleus also storing critical genes.

Mitochondria also play a pivotal role in maintaining health over the lifespan. Diseases of the mitochondria have a profound impact on ageing as they regulate substrate utilisation, energy production and muscle size and function. Changes in muscle mitochondrial functioning and development significantly contribute to the onset of chronic diseases and health issues in an ageing population.

To maintain the balance in all cells, mitochondria voluntarily commit suicide for the benefit of other tissues and organs, a process called 'apoptosis'. Without the programmed death of cells, degenerative diseases would occur with greater regularity.

Humans over the past >100,000 years have increased their life span from early 20s to over 100 years mainly due to how the environment has forced cells to make changes resulting in genetic modifications.

Ageing and disease are intrinsically linked. While generally reported that ageing increases the chances of chronic diseases, it is more a case of how genes are affected by their environment[1]. Some people age very quickly, while others remain functional into old age. There is no set point when these changes will occur, with degenerative diseases not always a function of ageing by itself.

A key function of the mitochondria is to burn fuel to create energy for the cell to function normally. As the burning of fuel occurs, the mitochondria leak free radicals (FR) or reactive oxygen species (ROS) caused by the release of electrons at rest and/or during higher intensity physical activity. Over time, the increased free radicals attack the cell's structures and cause irreversible damage to the genes within the mitochondria, causing a gene mutation and decreasing the performance of the whole cell.

Mitochondrial mutations can accumulate over time and spiral uncontrollably, with the free radical leakage causing accelerated ageing and death. In healthy mitochondria, the increase in free radical production is a warning sign to the cell to make changes to eliminate any unwanted free radicals.

High blood pressure, diabetes, high cholesterol and sarcopenia are the result of a gene mutation found in the mitochondria. In ageing, mutations that have developed in the mitochondria are due to being hereditary, sickness and/or an inactive lifestyle[2].

Mitochondrial disease is diagnosed by a malfunctioning of the mitochondria in the most metabolically active organs such as the heart, muscle and brain, while they are less serious in low metabolic tissues such as skin. These diseases can lay dormant for many years and begin to surface during ageing.

Mitochondrial disease is characterised by muscle weakness and increased fatigue at low levels of physical activity and reduced exercise tolerance. Due to the reduced exercise tolerance, those with mitochondrial disease become very inactive, increasing the risk of heart disease by 2.5 times.

In healthy, young mitochondria, the leakage and production of free radicals can be neutralised by the cell's antioxidant system and little damage occurs.

Approximately 85% of functioning mitochondria is considered normal and performs well during the early years of growth but, as ageing occurs, the mitochondria's performance declines, placing additional stress on the remaining normal functioning mitochondria. What once caused low stress in the young mitochondria can now cause devastating results in old mitochondria.

One way to combat the production and leakage of free radicals is to produce more mitochondria or increase the production of existing mitochondria, taking the pressure off the remaining mitochondria and spreading the workload.

Each cell has a tipping point where its functioning limits are reached, and any additional insult can push them over the top. It is possible that at this point other conditions such as smoking, changing environment and infections can contribute to the problem[3].

If the cell increases mitochondria, they cope better when required to produce more energy and neutralise the accompanying increase in free radicals. Alternatively, with less mitochondrion, the cell is placed under additional stress to produce more energy and then to control the additional increase in oxidative stress.

Physical activity improves neuromuscular function, decreases oxidative stress, causes an increase in antioxidant enzymes and increases the activity of the mitochondrial enzymes. This allows the cell to control the increase in free radicals without any detrimental effects, leading to accelerated ageing or degenerative diseases, and can assist in increasing longevity. If the flow of free radicals is reduced at rest, then the body is less vulnerable to degenerative diseases.

Physical inactivity also has a dramatic effect on the performance of mitochondria by decreasing blood flow, reducing the size of existing mitochondria and reducing the number of enzymes. This will lead to an increase in the leakage of free radicals and ultimately to damage to genes, cell membranes and the performance of other components within the cell.

Inactivity combined with ageing results in an accelerated decrease in mitochondrial functioning, contributing to insulin resistance and Type 2 diabetes. In general, inactivity accelerates muscle breakdown, mitochondrial dysfunction and oxidative stress, and reduces aerobic fitness.

As these changes also occur with ageing, inactive older adults create further additional problems as they begin to suffer more muscle injuries, bone disorders and general frailty, which causes them to become even more inactive!

Multiple bouts of muscle contractions are an excellent stimulus to increase the production and surface area of existing mitochondria, as well as increase the production of key aerobic enzymes in all cells at any age of the lifecycle[4].

One of the most effective modifiable interventions that can combat chronic disease in ageing is to maintain mitochondrial health by being physically active at any time throughout the ageing process. *fp*

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