Healthy Aging

Understanding the Process and What We Can Do About It

To age is to be ill, or is it? Despite population–wide issues with poor health, there are examples of older adults who have maintained health and wellness — in some cases even outliving their children. What are those factors that are contributing to this and why do they seem to matter?

Topics: Healthy Aging — Usual Aging — Pathological Aging — Coronary Heart Disease — Congestive Heart Failure — Chronic Obstructive Pulmonary Disease — Osteoporosis — Bone Remodeling — Bone Modeling — Reserve Theory — Sarcopenia

Healthy Aging — The process of developing and maintaining the functional ability [health] that enables wellbeing in older age. [World Health Organization]

Over the past several decades a disproportionate amount of research has focused on older adult populations. This, in part, was due to federal emphasis on aging which allocated funds to research in this area out of concern for the aging 'baby boomer' generation and their parents, the 'silent' generation; but was also a reflection on the necessities of research focused on diseases, dysfunctions, disorders, and health. That is, there is considerably greater occurrence of poor health and wellbeing within older adults than in younger populations. The general proximity to projected lifespan limits also enabled better alignment between research on disease/disorder morbidity and mortality as research was able to track individuals with identified morbidities until mortality occurred. A critical premise that evolved out of this work was that while there are some changes that naturally occur as a part of the aging process, poor health is not necessarily an expected outcome.

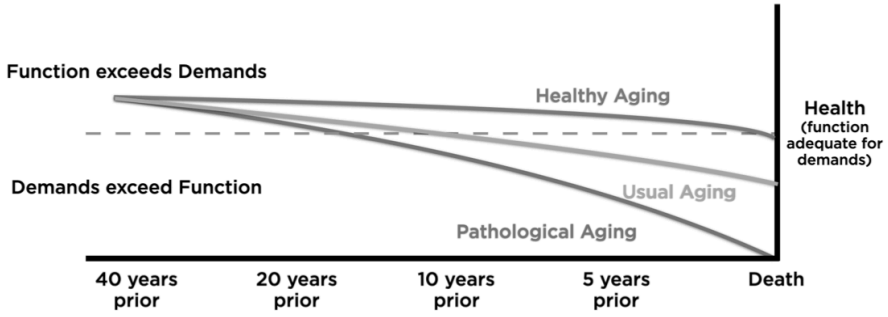
Healthy aging (also referred to in some work as successful aging) reflects the retainment of health throughout the lifespan to enable optimal wellbeing. This view aligns with modern perspectives on health as the point of homeostasis between the demands placed upon an individual and their ability to effectively respond to those demands. Although normal age–related changes may reduce an individuals functional abilities, the demands that they are exposed to are also typically diminished proportionately. Healthy aging would then be reflected by the maintenance of health (function adequate

for demands) and wellbeing (positive subjective evaluation) up until a physiological system failure results in death.

Usual aging reflects the trajectory of healthy aging, with the added influence of detriments in functional abilities (health) occurring due to system–related dysfunctions. The nomenclature (formal term) as 'usual' is meant to reference this trajectory as the one most adults will follow as they age, in some ways aligning with historic views of health as a state of optimal rather than adequate function. Thus, as a result of host and environment factors, individuals will exhibit some diminished capacity beyond that expected with healthy aging but will be free of disease. Usual aging reflects a state of diminished health that might negatively influence wellbeing that occurs prior to the physiological system failure results in death.

Pathological aging reflects the trajectory of healthy aging, with the added influence of detriments in functional abilities (health) occurring due to system–related dysfunctions as well as disease states. Combined, these dysfunctions and disease states result in much more progressive deterioration in functional ability which are also likely to negatively impact upon wellbeing. Pathological aging would then be reflected by greatly diminished health and wellbeing that occurs well before the physiological system failure results in death.

Figure: Trajectories of Aging.



The nuance of the healthy aging framework is that diminished health/wellbeing are distinct from the point at which death occurs, with pathological aging reflecting onset of diminished health/wellbeing well before death and healthy aging reflecting the maintenance of health/wellbeing up to the point of death. Research on aging consistently observes that there is substantial variation in the extent to which health is retained as a person reaches projected lifespan limits. This healthy aging framework attempts to explain this variation as a cumulative result of system–level trajectories — acknowledging the influence of genetic predispositions. Specifically, an individual with only minimal dysfunction in a small subset of areas would likely follow a more optimal trajectory of 'usual aging' that is nearer 'healthy aging'; whereas an individual with dysfunction across multiple systems would

likely follow a suboptimal trajectory of 'usual aging' that is nearer 'pathological aging.'

The greater number of systems that are impacted and the more severely they are dysfunctional/diseased the worse the aging trajectory. The benefit of this framework is that separates health/wellbeing from mortality (as it is possible to continue to live in a diminished health state), and further suggests that processes along the life course can alter the aging trajectory through optimization and compensation processes. So altering lifestyle behaviors can result in systemic changes to reduce disease risk, enhance function, and allow for more optimal aging trajectories.

Major Disease States

One criticism of the encompassing nature of the Healthy People initiative — which represents evidence and recommendations across a broad number of disciplines — is that it can make it difficult to hone in on the most relevant factors to promote better health and wellbeing. However, there are a number of target health conditions which are particularly relevant within the field of Kinesiology.

Coronary heart disease — An arterial disease characterized by the accumulation of fatty deposits on the walls of the arteries that partially or totally block blood flow in the heart.

Congestive heart failure — A disease characterized by an inability of the heart to effectively pump blood resulting in the accumulation of blood within the arms, legs, and lungs.

Heart disease represents the singular leading cause of death within the US for the past century (100 years), representing the underlying cause of death in nearly 30% of deaths. According to the Centers for Disease Control, one person dies every 33 seconds from heart disease, with a prevalence of nearly 20% in adults aged 65 and older. Heart disease, in particular, is by far the leading cause of death in men; with nearly half of men who die suddenly of coronary heart disease presenting with no prior symptomatology. Although the International Classification of Diseases recognizes a large number of diagnostic codes associated with heart disease, there are a few variants that are worth clarifying.

Coronary heart disease (also referred to as ischemic heart disease and represents a subtype of coronary artery disease) occurs when **atheroma** — abnormal accumulation of fatty deposits — occur within

the arteries in a process known as atherosclerosis that partially or totally blocks blood flow in the heart. Symptoms of coronary heart disease typically include pain or pressure in the chest/shoulder/neck, fatigue, shortness of breath, irregular or rapid heart beats, and poor peripheral perfusion (press down on your finger tip, hold for 30 seconds, when you let go the color should rapidly come back). For most individuals however, the first sign of a problem occurs when the individual has a **Myocardial Infarction** (heart attack). Tertiary Prevention for coronary heart disease typically employs angioplasty (the insertion of balloons and stents to combat the arterial narrowing) and blood thinning medication for less severe blockages. More severe accumulation may require coronary artery bypass grafting (CABG, referred to as cabbage) to redirect blood around the blockage.

Congestive heart failure (also referred to as heart failure) occurs when the heart is unable to properly circulate blood. Because blood is being returned to the heart faster than it can be pumped out, the system becomes congested; resulting in edema (fluid accumulation) in the arms, legs, and lungs (referred to as pulmonary edema). Symptoms of congestive heart failure typically include shortness of breath, fatigue, persistent coughing, fluid in the lungs or abdomen, and pitting edema within swollen areas. Pitting edema describes when pushing against a swollen area of the arms or legs results in an indentation. Tertiary Prevention for congestive heart failure typically employs vasodilator medications and diuretics to correct fluid retention.

> **Grade 1 Pitting** — Pitting edema indentations less than 2mm in depth which typically are observed to return to normal (rebound) immediately.
>
> **Grade 2 Pitting** — Pitting edema indentations 3 to 4mm in depth which take 15 seconds or less to rebound.
>
> **Grade 3 Pitting** — Pitting edema indentations 5 to 6mm in depth which take up to 1 minute to rebound.
>
> **Grade 4 Pitting** — Pitting edema indentations around 8mm in depth that can take 2 to 3 minutes to rebound.

Coronary heart disease and congestive heart failure both exhibit very high levels of comorbidity with conditions such as high cholesterol, hypertension (high blood pressure), and diabetes. Similarly, the risk factors associated with these diseases have substantial overlap (male, 65 years old or older, poor diet, family history of heart disease, history of smoking, lack of physical activity). Accordingly, secondary prevention relies upon screening measures such as the American Heart Association's Life's Essential 8, which assesses four health behaviors (nutrition, physical activity, tobacco, and sleep) and four health factors

(obesity, blood sugar, blood pressure, cholesterol) to identify individuals in need of follow up.

In terms of primary prevention efforts, physical activity (really the lack of physical activity at any intensity above very light) in particular appears to be the second strongest predictor of risk, accounting for nearly 12% of the risk of developing heart disease — falling in between hypertension (at 18%) and diabetes (at 10%). Accumulating just 10 minutes per day (70 minutes per week) of physical activity (at any intensity above very light) is associated with a reduced risk of developing heart disease by nearly half (54%). Although the mechanistic pathway by which physical activity serves to prevent cardiovascular disease is complex and multifaceted, the overarching perspective is that the heart is fundamentally a muscle. Therefore, it would be expected to atrophy (degenerate, decline in effectiveness) with lack of use. Thus, engaging in even small amounts of physical activity support the health of the heart muscle enabling it to sustain adequate function.

However, three particular pathways encompassing both direct and indirect effects have articulated. **Vasodilation and Angiogenesis** pathways highlight the effects of physical activity on inducing vascular adaptations which result in increases in the diameter of blood vessels, stimulates capillary growth, and facilitates vascular remodeling. These factors protect against vascular stress and facilitate cardiopulmonary oxygen delivery. **Mitochondrial Biogenesis** pathways highlight the role of physical activity for increasing the cellular renewal/regeneration of mitochondria within fat cells, skeletal muscle cells, and cardiac muscle cells increasing their ability to create energy and sustain function. **Anti–inflammatory** pathways highlight the effect of physical activity for creating favorable conditions for cellular repair processes to work, ultimately reducing the need for inflammatory responses that can cause additional damage to cardiac tissue.

Chronic obstructive pulmonary disease — A chronic lung disease characterized by airflow restriction and reduced gas exchange.

Chronic obstructive pulmonary disease (COPD) comprises the majority of deaths due to chronic lower respiratory diseases and represents the 3rd leading cause of death worldwide. COPD is a chronic inflammatory lung disease that traditionally encompasses a group of diseases but most specifically emphysema and chronic bronchitis. **Emphysema** is a condition in which the alveoli at the end of the smallest air passages of the lungs are destroyed as a result of exposure to cigarette smoke and other irritating gases and

particulate matter in the air. This ultimately reduces the ability to exchange gases (Respiration) between the alveoli and the blood stream. **Chronic bronchitis** is inflammation of the lining of the bronchial tubes, narrowing the airway passages to the lungs making it more difficult to get air to the alveoli (Ventilation) and triggering spontaneous cough reflexes. Symptoms of COPD typically do not present until after significant irreparable lung damage has occurred and worsen over time. Problematically, nearly 50% of individuals who die of COPD never received diagnosis or treatment. Symptoms include shortness of breath, wheezing, tightness in the chest, a chronic cough, and fatigue.

> **Stage 1** — Mild or no symptoms. Airflow is 80% of normal. Individual may be short of breath with moderate exertion.
>
> **Stage 2** — Frequent coughing, wheezing, shortness of breath. Airflow is 50 to 79% of normal.
>
> **Stage 3** — Frequent coughing, wheezing, shortness of breath that requires hospital or clinical treatment to manage. Airflow is 30 to 50% of normal.
>
> **Stage 4** — Constant coughing, wheezing, shortness of breath despite treatment. Exacerbation of symptoms (flair ups) may be life threatening. Airflow is less than 30% of normal.

Evidence surrounding the tertiary prevention of COPD indicates that current efforts which focus on pulmonary medications and respiratory therapy are suboptimal for restoring function, resulting in most individuals requiring continuous oxygen supplementation. Comorbidities of COPD include heart disease, diabetes, high cholesterol, hypertension (high blood pressure). Asthma is frequently cited as a comorbid condition in nearly 50% of individuals with COPD; however, other definitions of COPD now consider chronic asthma conditions within the broad umbrella of COPD. Secondary prevention efforts oriented towards COPD primarily focus on screening for exposure to tobacco, occupational exposure to chemical fumes, and history of excessive respiratory infections.

The relationship between physical activity and COPD appears to be bidirectional. Although aerobic fitness is associated with greater efficiency of metabolic systems (reducing the demand for oxygen) and superior gas exchange/oxygen extraction, engaging in physical activity can exacerbate (make worse) symptoms of COPD. As a result, although there is evidence that low levels of physical activity engagement are associated with greater severity of COPD; it is important to consider that the low levels of activity may be a symptom of respiratory difficulty rather than an antecedent behavior that resulted in COPD. Similarly, although individuals with COPD who engage in greater

levels of physical activity have reduced risk of mortality; this may be a correlated but non–causal finding as reduced issues associated with COPD may allow for greater activity as well.

The general perspective is that as COPD has a particularly strong exogenous (developing from external causes) component, that the role of physical activity in primary prevention efforts centers around the development of greater aerobic fitness which would enable the individual to enhance their respiratory and metabolic functions. Having enhanced systemic function would make it less likely that those exogenous factors would degrade function to a clinical level. Interestingly, short bursts of light intensity physical activity (such as walking from the parking lot into the doctors office) have been observed to result in temporary reduction in symptoms of COPD. While problematic for masking symptoms for diagnosis, such findings do suggest there may be opportunities for utilizing physical activity in tertiary prevention efforts.

Osteoporosis — A bone disease characterized by loss of bone mineral density that results in increased risk of fracture and prolonged healing time.

Osteoporosis and the related state **Osteopenia** refer to an inflammatory bone disease associated with abnormally low bone mineral density that places the individual at high risk of bone fracture. Typically diagnosed through the use of dual–energy x–ray absorptiometry (DEXA) bone scans, the World Health Organization defines osteoporosis as having bone mineral density less than a t–score of –2.5 while osteopenia is defined as a bone mineral density between –1 to –2.5. In this way the critical difference between osteoporosis and osteopenia is the quantitative reduction in bone mineral density below what would be expected for an individual of that age and biological sex. In the US, 54% of postmenopausal women are osteopenic, and an additional 30% are already considered osteoporotic; with females having a four–times higher overall prevalence compared to males.

The physiological mechanism responsible for osteoporosis result in a dysregulation of bone remodeling. The process of **bone remodeling** maintains the health of bone by renewing old bone tissue; bone resorbing **osteoclasts** remove old bone tissue and bone forming **osteoblasts** then immediately replace it with new healthy bone tissue. Bone mass is maintained in healthy individuals as this process carefully ensures that the amount of bone resorbed by osteoclasts equals the amount of bone formed by osteoblasts. In osteopenic and osteoporotic individuals, dysregulation of this process leads to increased activity

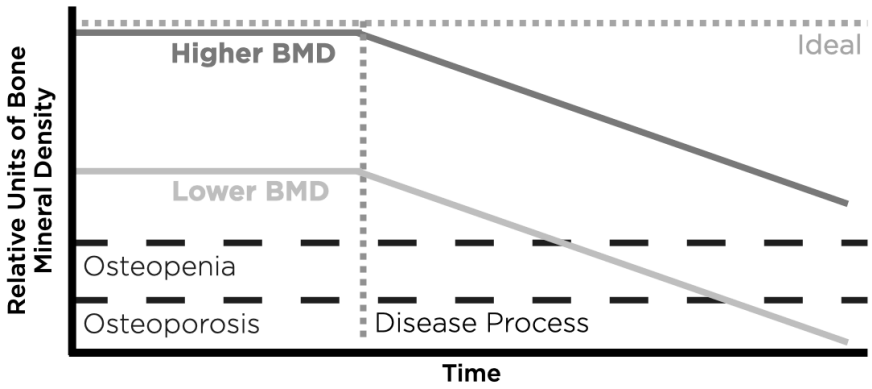
of bone resorbing osteoclasts and decreased activity of bone forming osteoblasts resulting in reduced bone mineral density. Beyond greater fragility of bones, osteopenia and osteoporosis are also associated with prolonged time necessary for healing as the bone forming osteoblasts exhibit decreased activity.

Osteoporosis usually does not present with any symptoms beyond the individual being prone to bone fractures. Tertiary prevention of osteoporosis through pharmaceutical approaches largely focus on slowing the continued decline of bone mineral density; while rehabilitation approaches focus on 'fall–proofing' the individual and their environment to reduce the risk of future injury. Efforts to enhance secondary prevention approaches have promoted regular DEXA bone scans for all individuals over the age of 40, with most medical insurance covering the test once every two years. Comorbidities of osteoporosis include arthritis, chronic lower back pain, and heart disease. The potential role of physical activity in primary prevention of osteopenia and osteoporosis conceptually aligns with explanations for both 'fixed' and 'modifiable' risk factors. Specifically, risk factors associated with osteoporosis (being female, having a small body frame, having a parent or sibling with a history of osteoporosis or history of fractures, low early life calcium intake, tobacco use, history of disordered eating) are all situations in which the individual would be predisposed to having lower bone mineral density to begin with. An individual with lower bone mineral density would be at higher risk of osteoporosis as they have less bone mineral density that they can lose before the bone mineral density reaches clinical levels of degradation.

Physical activity as a primary prevention approach focuses on the process by which bones alter to optimize their strength. The process of **bone modeling** reshapes existing healthy bone structures to optimize the strength and flexibility of the bone to the demands placed on it. Conceptualized by the **Theory of Trajectory Architecture**, the optimal strength and flexibility of a bone is achieved by only placing bone tissue along the paths of transmission of forces. So as the bone is mechanically loaded, it responds through the process of bone modeling whereby bone resorbing **osteoclasts** remove bone tissue that is not aligned with the forces being placed on the bone and bone forming **osteoblasts** build bone along the path of forces. As physical activity is the primary means by which bones are mechanically loaded (other than gravitational effects), engaging in physical activity activates the process of bone modeling to result in increases in bone mineral density. The **Reserve Theory** suggests that the negative impacts of a disease process can be reduced by altering the pre–disease levels (e.g., the reserve). If the reserve is sufficiently large (such as in the case of an individual with very high levels of bone mineral density) then the function of a given system is less likely to diminish to clinically

significant levels (i.e., meet diagnostic criteria for osteoporosis) when the disease process occurs. However, that same disease process is likely to result in clinically significant declines in systemic function in an individual with very little reserve (such as in the case of an individual with a small body size who has low levels of bone mineral density).

Figure: Reserve Theory of Bone Density.

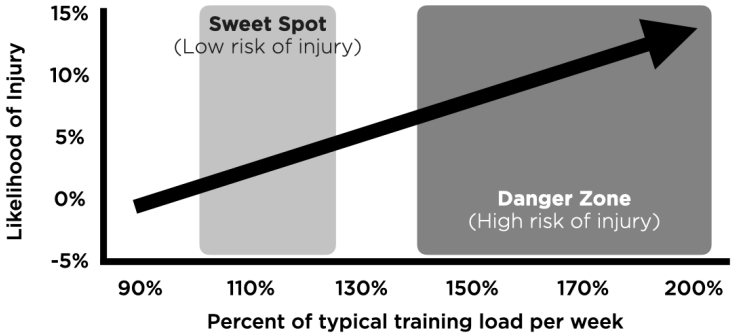


There are some important caveats (stipulations, clarifications) that are necessary to be aware of however. First, an individual's peak bone mineral density usually presents between the ages of 20 and 25, with gradual declines in density associated with aging. Mechanical loading of bones appears to be the most effective in increasing bone mineral density when the loading occurs prior to the onset of puberty, with lesser effectiveness between 11 and 19 years of age, and greatly diminished effectiveness after this point. So from a prevention standpoint, mechanical loading of bones to increase bone mineral density is best implemented as a Primordial approach, although the potential benefits may still occur to a lesser extent as a Primary approach.

Second, research into the cellular signaling cascade that allows mechanical loading of bones to result in increases in bone mineral density gives us some perspective as to how long it might take to experience such benefits and why some individuals may experience reductions in bone mineral density instead. Research in both human and non–human models suggests that it may take as long as 4 to 6 months for bone modeling processes to begin, so brief, irregular, and non–habitual mechanical loading of bones through physical activity are not likely to observe any potential benefits. Conversely, excessive mechanical loading beyond 130% of typical weekly workload levels are associated with elevated risk of injury. The risk of injury increases from 5% at 130% of typical weekly workload to 15% at 200% of typical weekly workloads.

The cellular signaling cascade also appears to become 'blind' to further stimulation within a few hundred exposures, taking 3 to 4 hours to restore mechanosensitivity. So a runner (with a cadence of 160 steps per minute) likely causes the cellular signaling cascade to go 'blind' within a few minutes. When that runner doubles their weekly running distance they are likely to cause additional stress/damage to their bones without incurring any additional benefits for enhanced bone modeling. The greater workload without additional benefit is likely to result in reductions in bone mineral density. This provides some insight into the occurrence of overuse injuries and stress fractures within highly active individuals as well as those individuals who are just starting increasing their physical activity levels.

Figure: Risk of Injury.

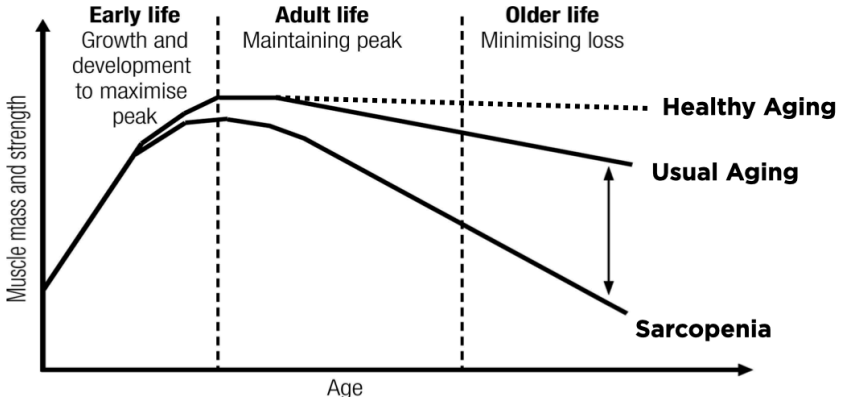


Sarcopenia — A muscle disease characterized by loss of muscle mass and strength.

While sarcopenia is not specifically included by name as an objective within the Healthy People initiative, it is a disease that is critical for maintaining health and wellbeing and is an underlying factor in many prominent disease states. Classically, sarcopenia has been characterized as a normal process associated with aging whereby the individual experiences progressive degeneration of muscle mass and loss of strength. Most estimates suggest that between the ages of 30 and 50 there is a 3 to 10% reduction in muscle fiber mass across both fast and slow twitch muscle fibers. Between the ages of 60 and 80, muscle fiber mass reduces by approximately 15% each decade; with a 30% reduction each decade observed after age 80. Similar to perspectives on bone mineral density however, **modern conceptualizations of sarcopenia suggest that this condition/disease reflects a unique dysregulation (separate from general age–related declines in muscle mass) that results in**

abnormally severe muscle degeneration.

Figure: Trajectory of Sarcopenia.



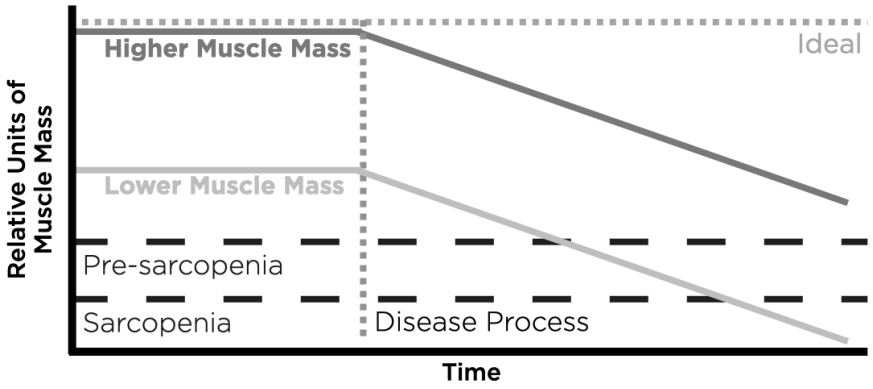
Although specific diagnostic criteria are still being debated and developed, this disease state perspective was officially codified by the International Classification of Diseases in 2016. As the diagnostic criteria are not fully formed, secondary prevention efforts have not been validated; but a number of international working groups have proposed screening metrics using walking speed, hand grip strength, the need for aids to assist with standing/walking, as well as the need for sweaters/blankets to maintain thermoregulation that appear to provide good predictive power. Such metrics have been used to suggest that as many as 10 to 20% of individuals over the age of 50 would meet criteria for clinically significant sarcopenia; 90% of whom are not actively seeking treatment or intervening to prevent the continued degeneration of muscle. In long-term rehabilitation units, the prevalence of sarcopenia is over 50%. This disease state places individuals at increased risk of falls, fractures, and greater susceptibility to disability. Sarcopenia exhibits frequent comorbidities with heart disease, respiratory diseases such as COPD, diabetes, and osteoporosis.

Tertiary prevention of sarcopenia has predominately focused upon physical and occupational therapy to maintain muscle function integrated alongside nutritional interventions to address protein insufficiencies. Pharmaceutical approaches have also yielded some success through early intervention with low–dose steroids to slow muscle wasting. Further a number of clinical trials are ongoing targeting hypothesized mechanisms which may lead to the disease state of sarcopenia. The potential role of physical activity in the primary prevention of sarcopenia is arguably the most clear cut as the disease is characterized by muscle degeneration. As a lack of physical activity results in muscle atrophy (degeneration), engaging in resistance exercises promotes increases in muscular strength, endurance, and increases muscle mass. These increases specifically relate to better functional outcomes as individuals have additional

support for bones, lower fall risk, and enhanced thermoregulation.

Again **Reserve Theory** is employed as an underlying basis for primary prevention and to explain associated risk factors (being female, having a small body frame, having a parent or sibling with a history of frequent falls or muscle issues, history of disordered eating, protein insufficiency). If the reserve is sufficiently large (such as in the case of an individual with very high muscle mass) then the function of a given system is less likely to diminish to clinically significant levels when the disease process occurs. However, that same disease process is likely to result in clinically significant declines in systemic function in an individual with very little reserve (such as in the case of an individual with low muscle mass).

Figure: Reserve Theory of Muscle Mass.



Additional Resources:

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Research into Aging

Federal emphasis on aging allocated substantial funds to enhance understanding of aging-related processes.

Population Need

Massive growth of the population over the age of 65 and projected further growth meant that it was a critical topic of importance.

Alignment of Focus

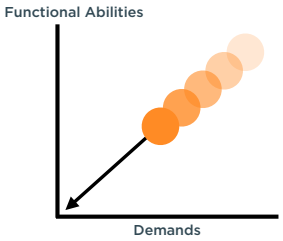
Young people have lower prevalence of disease/ disorder making it harder to study.

Focusing on elderly populations means that we can follow a disease from morbidity until mortality.

Perspective of

Healthy Aging

Retainment of health throughout the lifespan.

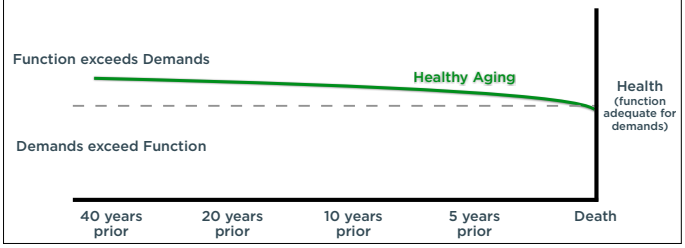


- Although normal age-related changes may reduce an individuals functional abilities, the demands that they are exposed to are also typically diminished proportionately.
  - Retirement.
  - Reduced activities.

Perspective of

Healthy Aging

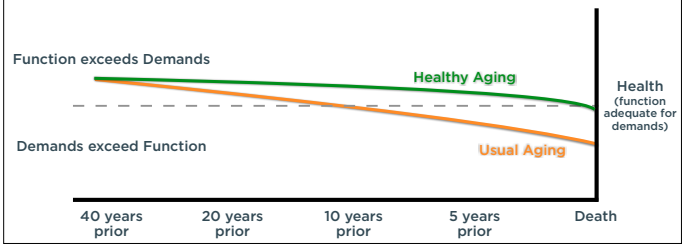
Healthy Aging: Maintenance of health and wellbeing up until the point of death.



Perspective of

Healthy Aging

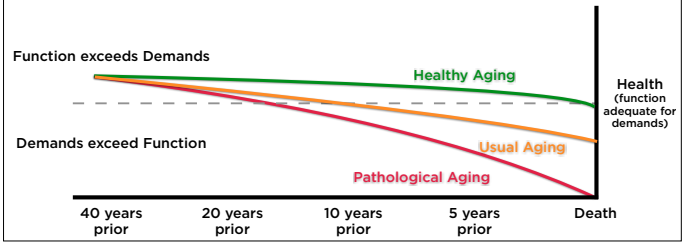
Usual Aging: Aging trajectory + System-related dysfunctions



Perspective of

Healthy Aging

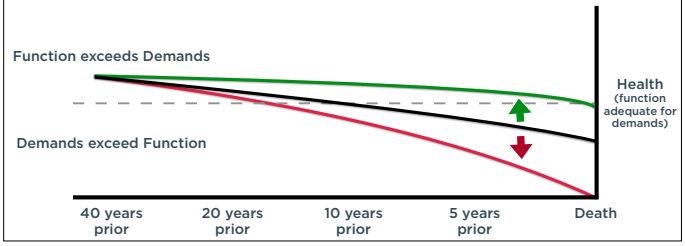
Pathological Aging: Aging trajectory + System-related dysfunctions + Disease



Perspective of

Healthy Aging

Altering lifestyle behaviors can result in systemic changes to reduce disease risk, enhance function, and allow for more optimal aging trajectories.



Perspective of
Healthy Aging

Healthy Aging Framework

- Diminished health and wellbeing are distinct from the point at which death occurs.
  - Healthy Aging: Health/wellbeing is maintained up to the point of death.
  - Pathological Aging: Health/wellbeing is compromised well before death occurs.
- Acknowledges influence of genetics on potential life span and disease/dysfunction resistance.
- Suggests that variability in health during aging reflects a pseudo-cumulative process.
  - The more systems that are compromised, the less health is possible.

Heart
Disease

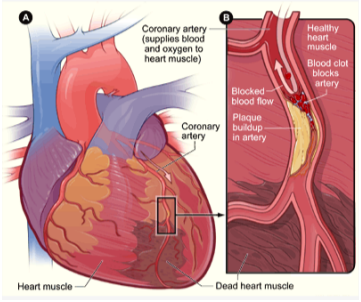
Leading Cause of Death for the past 100 years.

Every 33 seconds someone in the US dies from heart disease.

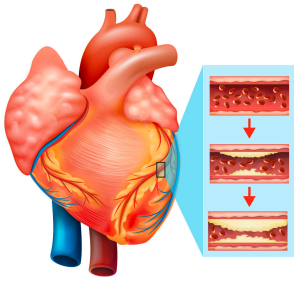
- Heart Disease represents a broad classification encompassing several different types of diseases:
  - Abnormal heart rhythms
  - Abnormal development of the heart
  - Blood pressure related diseases
  - Valve related diseases
  - Blood flow related diseases
  - Failure of heart muscles

Coronary
Heart Disease

An arterial disease characterized by the accumulation of fatty deposits on the walls of the arteries that partially or totally block blood flow in the heart.



Coronary
Heart Disease

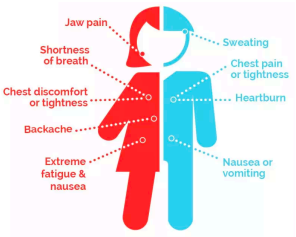


- **Atheroma** - abnormal accumulation of fatty deposits
- Accumulation of atheroma on the arterial walls (Athero-sclerosis) begins to block blood flow to the heart.
- As blockage progresses, insufficient blood flow is able to get to the heart muscle which causes that muscle to die.

Coronary
Heart Disease

Symptoms

- Pain or pressure in the chest, shoulder, neck, jaw
- Fatigue
- Shortness of breath
- Irregular or rapid heart beats
- Poor peripheral perfusion



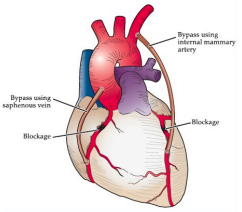
Coronary
Heart Disease

- Nearly 50% of men who die from Coronary Heart Disease never reported any symptomatology.
  - This could be a reflection on social environment.
  - This could reflect asymptomatic disease states.
  - This may also reflect attributing symptoms to other issues.
- Most individuals never know they have Coronary Heart Disease until they have a Myocardial Infarction.

Coronary
Heart Disease

- 12% of individuals who suffer a Myocardial Infarction die within 1 year.
- 66% of individuals who suffer a Myocardial Infarction die within 5 years.
- After the first Myocardial Infarction, 53% of individuals die within 1 year following the 2nd Myocardial Infarction.
- 30% of Myocardial Infarctions are considered 'Silent'
  - No symptom presentation.
  - Individual is unaware.
  - Irreparable cellular death of heart muscle still occurs.
  - More common among women.

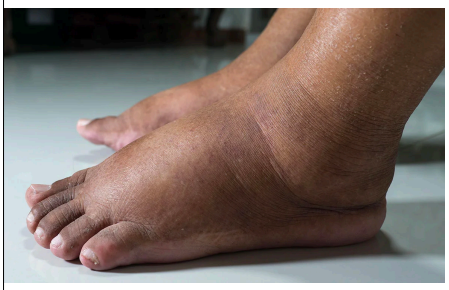
Coronary
Heart Disease



Tertiary Prevention

- For less severe blockages, blood thinning medication and angioplasty are typically used.
- More serious blockages require grafting blood vessels onto the heart to bypass the blockage.
  - Coronary artery bypass grafting (cabbage)
  - More commonly known as single/double/triple bypass surgery.

Congestive
Heart Failure



A disease characterized by an inability of the heart to effectively pump blood resulting in the accumulation of blood within the arms, legs, and lungs.

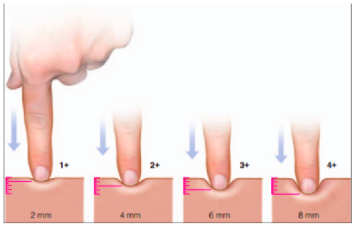
Congestive
Heart Failure

- When the heart is no longer able to properly circulate blood, it begins to accumulate.
  - The left side of the heart takes blood circulated from the lungs and sends it to the rest of the body.
    - Left sided heart failure results in blood pooling into the lungs causing **Pulmonary Edema**.
  - The right side of the heart take blood circulated from the body and sends it to lungs.
    - Right sided heart failure results in blood pooling in the arms and legs causing **Peripheral Edema**.

Congestive
Heart Failure

Symptoms

- Shortness of breath
- Fatigue
- Persistent coughing
- Fluid in the lungs or abdomen
- Pitting edema within swollen areas



Congestive
Heart Failure

Tertiary Prevention

- The more severe the heart failure, the fewer the options available for treatment.
- Most tertiary prevention uses vasodilation medications to reduce blood pressure and ease the strain on the heart.
- Diuretics to attempt to reduce edema.

Coronary Heart Disease & Congestive Heart Failure

Risk Factors

Comorbid Conditions

- High cholesterol
- Hypertension (high blood pressure)
- Diabetes

Relative Risks

- Male
- 65 years old or older
- Poor diet
- Family history of heart disease
- History of smoking
- Lack of physical activity

Coronary Heart Disease & Congestive Heart Failure

Secondary Prevention

American Heart Association's Life's Essential 8

Health Behaviors

- Nutrition
  - Diet consistent with AHA recommendations
- Physical Activity
  - 150 minutes of Moderate Intensity PA
- Tobacco
  - No usage of any kind
- Sleep
  - 7 to 9 hours per night

Health Factors

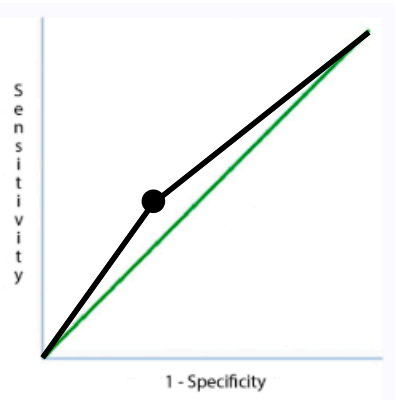
- Obesity
  - BMI under 25 (normal)
- Blood Sugar
  - Fasting below 100 mg/dL, A1C less than 5.7
- Blood Pressure
  - Below 120/80 mmHg
- Cholesterol
  - Blood lipids consistent with AHA criteria

Coronary Heart Disease & Congestive Heart Failure

Secondary Prevention

American Heart Association's Life's Essential 8

- Online tools for self-screening + tools integrated into medical records
- Provides an Overall Score with lower numbers relating to poorer heart health.
- Every 5 point increase in overall score is associated with 17% lower risk of dying from cardiovascular disease.
- As a screening tool, the AHA LE8 exhibits a 48.8% sensitivity and 64.8% specificity.

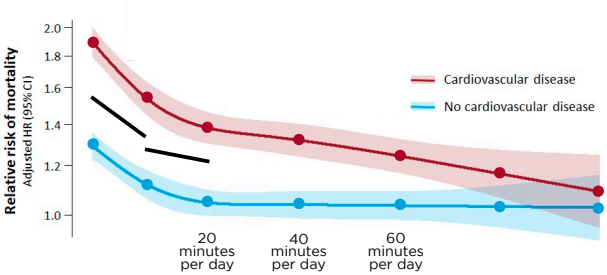


Although American Heart Association's Life's Essential 8 provides one of the best screening tools for early detection of heart disease, it is objectively a poor tool.

Coronary Heart Disease & Congestive Heart Failure

Primary Prevention

- Physical activity is the second strongest predictor of risk, accounting for nearly 12% of the risk of developing heart disease.
  - Falling in between hypertension (at 18%) and diabetes (at 10%).
  - Physical activity is also related to the AHA Health Factors.
- 10 minutes per day of physical activity at any intensity above very light effectively reduces the risk of developing heart disease by half.



Of PA at an intensity above Very Light

10 minutes of physical activity is great,
20 provides marginal additional risk reduction.

How does Physical Activity Prevent
Cardiovascular Disease

The heart is fundamentally a muscle. Trained muscles work better.

- Unlike medications, physical activity simultaneously activates multiple pathways to enhance cardiovascular function.
- These pathways work through both direct and indirect mechanisms.
  - Direct mechanisms specifically act upon cardiovascular systems.
  - Indirect mechanisms have intermediary steps (sometimes multiple) before acting upon cardiovascular systems.

How does Physical Activity Prevent
Cardiovascular Disease

The heart is fundamentally a muscle. Trained muscles work better.

Vasodilation and Angiogenesis

- Physical activity induces vascular adaptations:
  - Increases in the diameter of blood vessels
  - Stimulates capillary growth
  - Facilitates vascular remodeling

These factors protect against vascular stress and facilitate cardiopulmonary oxygen delivery.

How does Physical Activity Prevent
Cardiovascular Disease

The heart is fundamentally a muscle. Trained muscles work better.

Mitochondrial Biogenesis

- Physical activity increases the cellular renewal/regeneration of mitochondria within
  - Fat cells
  - Skeletal muscle cells
  - Cardiac muscle cells

This increases their ability to create energy and sustain function.

How does Physical Activity Prevent
Cardiovascular Disease

The heart is fundamentally a muscle. Trained muscles work better.

Anti-Inflammatory

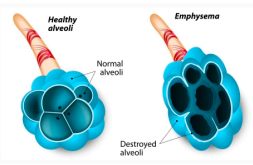
- Physical activity creates favorable conditions for cellular repair processes to work.

This decreases the need for inflammatory responses which can induce additional damage to cardiac tissue.

Chronic Obstructive
Pulmonary Disease

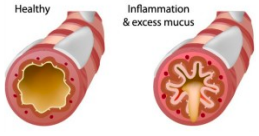
Emphysema

Condition in which the alveoli at the end of the smallest air passages of the lungs are destroyed as a result of exposure to cigarette smoke and other irritating gases and particulate matter in the air.



Chronic Bronchitis

Inflammation of the lining of the bronchial tubes, narrowing the airway passages to the lungs making it more difficult to get air to the alveoli and triggering spontaneous cough reflexes.



Ventilation

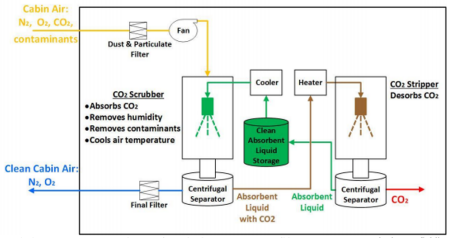
Movement of Air

When you open the door to your car, whatever is in the air comes inside.

Respiration

Movement of Gases

Carbon Dioxide Scrubber Only allows specific gases to pass through.



Chronic Obstructive

Pulmonary Disease

Emphysema

Condition in which the alveoli at the end of the smallest air passages of the lungs are destroyed as a result of exposure to cigarette smoke and other irritating gases and particulate matter in the air.

Reduces the ability to exchange oxygen and carbon dioxide between the alveoli and the blood stream.

Impairs Respiration.

Chronic Bronchitis

Inflammation of the lining of the bronchial tubes, narrowing the airway passages to the lungs making it more difficult to get air to the alveoli and triggering spontaneous cough reflexes.

Reduces the flow of air that can get to the alveoli.

Impairs Ventilation.

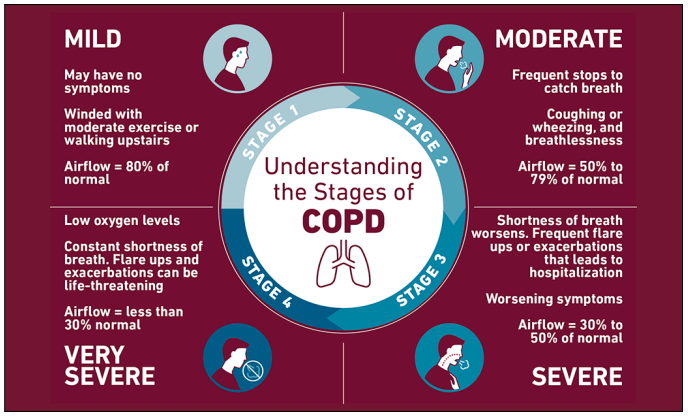
Chronic Obstructive

Pulmonary Disease

Symptoms

Shortness of breath
Wheezing
Tightness in the chest
Chronic cough
Fatigue

- Symptoms of COPD typically do not present until after significant irreparable lung damage has occurred
- Symptom severity worsens over time.
- Nearly 50% of individuals who die of COPD never received diagnosis or treatment.

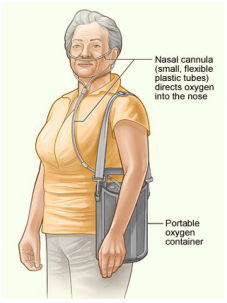


Chronic Obstructive

Pulmonary Disease

Tertiary Prevention

- Primary approach involves pulmonary medications and respiratory therapy.
  - Evidence suggests that while these are effective in slowing down disease progression they are suboptimal for restoring function.
  - Most individuals will require continuous oxygen supplementation or the availability of supplemental oxygen.



Chronic Obstructive

Pulmonary Disease

Comorbid Conditions

- Heart disease
- Diabetes
- High cholesterol
- Hypertension (high blood pressure).
- Asthma*

Risk Factors

- Tobacco use/exposure
- Exposure to chemical fumes
- Excessive respiratory infections

Why might low levels of Physical Activity be associated with

Greater Severity of COPD?

Risk Factor

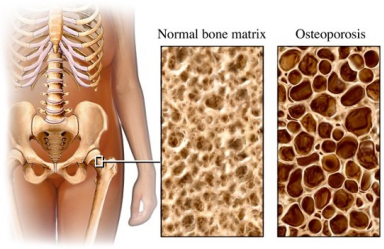
- Aerobic fitness is associated with greater efficiency of metabolic systems.
- Cardiorespiratory exercise can enhance aerobic fitness.
- Lack of cardiorespiratory exercise would then relate to poorer efficiency of metabolic systems.
- This would result in poorer gas exchange/ oxygen extraction.

Symptom

- An individual with more severe COPD would have diminished gas exchange/ oxygen extraction.
- This would limit their ability to engage in cardiorespiratory exercise.
- Lack of cardiorespiratory exercise would lead to lower aerobic fitness.

Osteoporosis

A bone disease characterized by loss of bone mineral density that results in increased risk of fracture and prolonged healing time.



Osteoporosis

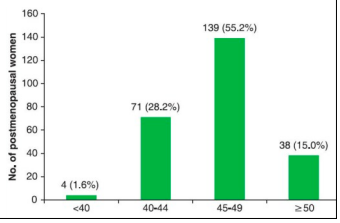
- Bone (osteo) that is porous (porosis)
- Inflammatory bone disease that places the individual at high risk for fractures.
- Characterized by low bone mass and deterioration of bone tissue.

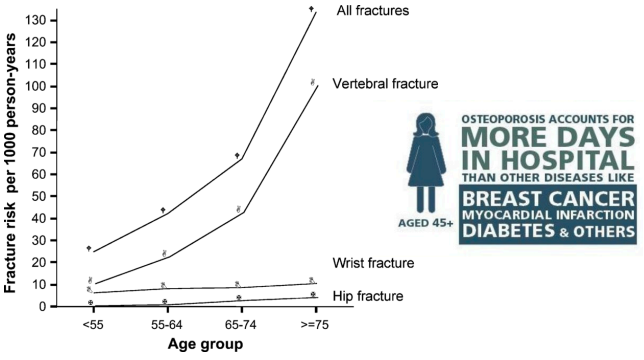
- **Osteopenia**: bone mineral density is lower than normal but not low enough to be considered osteoporosis.
  - "pre-osteoporosis"

Osteoporosis

- Women have 4 times the overall prevalence of osteoporosis/osteopenia than men.
- Within postmenopausal women, 84% exhibit disordered bone density:
  - 54% meet criteria for osteopenia.
  - 30% meet criteria for osteoporosis.

50% of women are projected to suffer an Osteoporosis related fracture in their lifetime.





Osteoporosis

Morbidity and Mortality

within 1 year following osteoporosis related fracture

24%
Died

30%
Confined to Nursing home

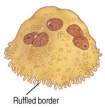
80%
Unable to carry out at least one ADL

Osteoporosis is associated with

Prolonged Time Necessary for Healing

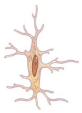
Bone Remodeling: The process that maintains the health of the bone by renewing old bone tissue.

- Bone cells work on the same area of a bone in a coupled fashion.



Osteoclasts

Remove old bone tissue.



Osteocyte

Osteoblasts

Build new bone tissue.

Osteoporosis is associated with

Prolonged Time Necessary for Healing

Bone Remodeling: The process that maintains the health of the bone by renewing old bone tissue.

- Bone cells work on the same area of a bone in a coupled fashion.
- Bone mass is maintained in healthy individuals as this process carefully ensures that the amount of bone resorbed by osteoclasts equals the amount of bone formed by osteoblasts.

Osteoporosis is associated with

Prolonged Time Necessary for Healing

Bone Remodeling: The process that maintains the health of the bone by renewing old bone tissue.

- In osteopenic and osteoporotic individuals,
  - **Osteoclasts** become **overactive,** removing excess bone.
  - **Osteoblasts** become **less active,** reducing how much bone is restored.
    - The decreased activity of osteoblasts contributes to prolonged healing time.

Osteoporosis

Tertiary Prevention

- Pharmaceutical approaches largely focus on slowing the continued decline of bone mineral density.
- Rehabilitation approaches focus on 'fall-proofing' the individual and their environment to reduce the risk of future injury.

Symptoms

Prone to bone fractures

Osteoporosis is associated with

Prolonged Time Necessary for Healing

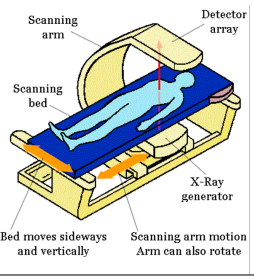
Bone Remodeling: The process that maintains the health of the bone by renewing old bone tissue.

- In osteopenic and osteoporotic individuals,
  - **Osteoclasts** become **overactive,** removing excess bone.
  - **Osteoblasts** become **less active,** reducing how much bone is restored.
    - The decreased activity of osteoblasts contributes to prolonged healing time.

Osteoporosis

Secondary Prevention

- Regular dual-energy x-ray absorptiometry (DEXA) bone scans for all individuals over the age of 40
  - Most medical insurance will cover the test once every two years.
  - Although dose of x-ray is 1/10th the dose of chest x-ray, concern over potential risks limit the frequency of screening.
- Diagnostic ability in addition to screening.



Osteoporosis

Comorbid Conditions

- Arthritis
- Chronic lower back pain
- Heart disease

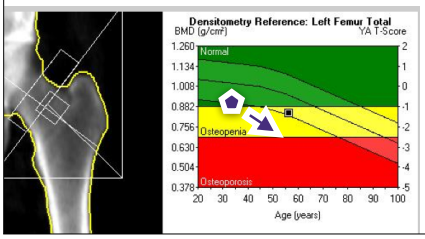
Risk Factors

Situations in which the individual would be predisposed to having lower bone mineral density.

- Being female
- Having a small body frame
- Having a parent or sibling with a history of osteoporosis or history of fractures
- Low early life calcium intake
- Tobacco use
- History of disordered eating

Why would predisposition to having lower bone mineral density

Be a Risk Factor for Osteoporosis

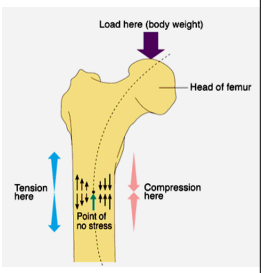


Starting off with lower bone mineral density means that you have less to lose before it reaches clinical levels.

Osteoporosis

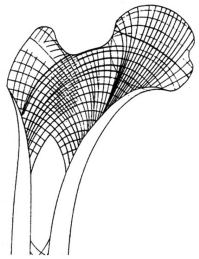
Primary Prevention

- Primary prevention of osteoporosis focuses on the process that enables bones to change to optimize their strength.
  - Solid bone would be very brittle and exceptionally heavy.
  - Bones must be strong and flexible.



Trajectory Architecture

The optimal strength and flexibility of a bone is achieved by only placing bone tissue along the paths of transmission of forces.



Optimal Model

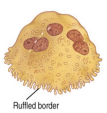
Actual Bone

Osteoporosis

Primary Prevention

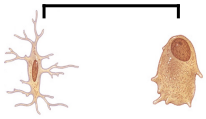
Bone Modeling: The process that reshapes existing bone structures to optimize the strength and flexibility of the bone to the demands placed on it.

- Bone cells work on different areas of a bone in an independent fashion.



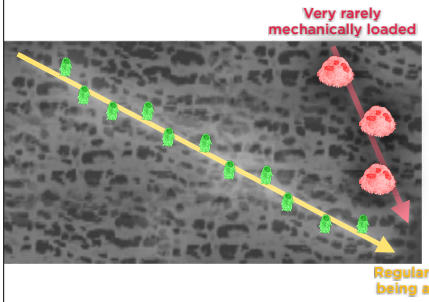
Osteoclasts

Remove bone not regularly loaded.



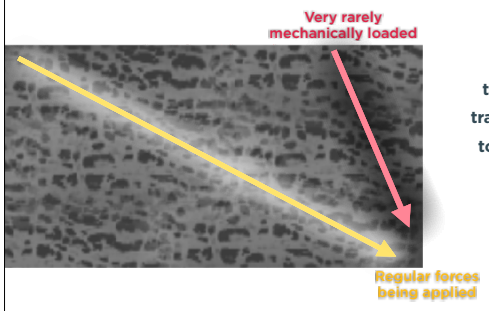
Osteocyte **Osteoblasts**

Build new bone tissue along paths where forces are placed.



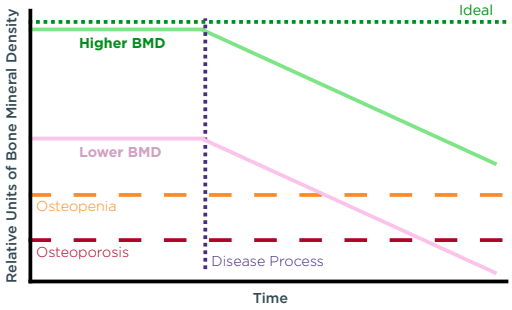
Osteoclasts remove bone tissue not aligned with the forces placed on the bone.

Osteoblasts build additional bone along the path of forces.



Bone modeling therefore allows for trajectory architecture to occur based upon how the bone is regularly loaded.

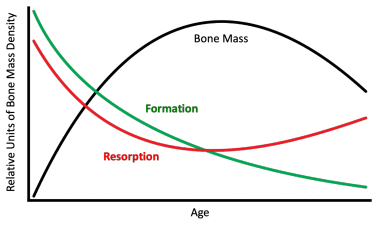
Reserve Theory



Osteoporosis

Primary Prevention

- There is a critical window when bone mineral density is most responsive to bone modeling.
- Peak density occurs between 20 and 25 years of age.
- Effects of mechanical loading of bones:
  - Prior to Puberty (optimal)
  - 11 to 19 (lesser effectiveness)
  - 20+ (greatly diminished effectiveness)



Osteoporosis

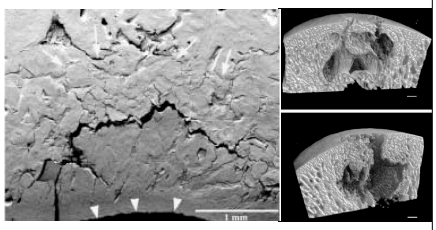
Primary Prevention

- Research in both human and non-human models suggests that it may take as long as **4 to 6 months** for bone modeling processes to begin.
  - Requires regular habitual mechanical loading of bones.
  - Brief, irregular, and non-habitual mechanical loading of bones is unlikely to observe any potential benefits.

Osteoporosis

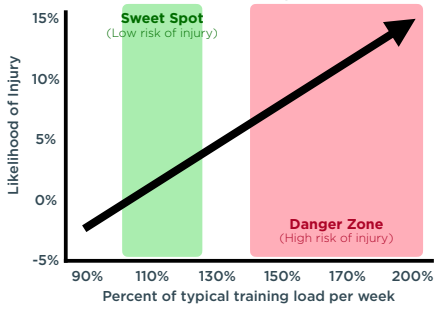
Primary Prevention

- Excessive mechanical loading results in small microscopic areas of damage to the bone.
- This is usually repaired by Bone Remodeling.
- If not remodeled this can contribute to lower bone density.



Osteoporosis

Primary Prevention



Dramatic increases in workload above typical training increases the risk of those microscopic areas of damage resulting in injury.

Increases should be kept below 130% of typical volume.

Osteoporosis

Primary Prevention

- Evidence from ulnar loading studies have found that after a few hundred exposures, the cellular signaling cascade that initiates Bone Modeling becomes 'blind'.
  - Additional loading beyond this point only causes damage.
- It can take 3 to 4 hours after the cessation of loading for this 'blindness' to wear off.

If regular chronic physical activity results in greater bone density

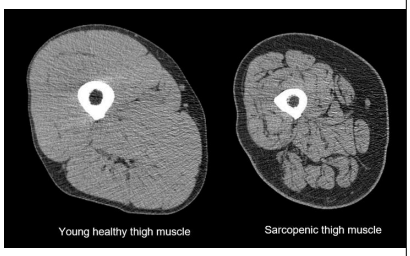
How do we explain stress fractures

A runner (with a cadence of 160 steps per minute) likely causes the cellular signaling cascade to go 'blind' within a few minutes.

When that runner doubles their weekly running distance they are likely to cause additional stress/damage to their bones without incurring any additional benefits for enhanced bone modeling.

Sarcopenia

A muscle disease characterized by loss of muscle mass and strength.



Sarcopenia

Historic Perspective

- Sarcopenia has been characterized as a normal process associated with aging whereby the individual experiences progressive degeneration of muscle mass and loss of strength.

Modern Perspective

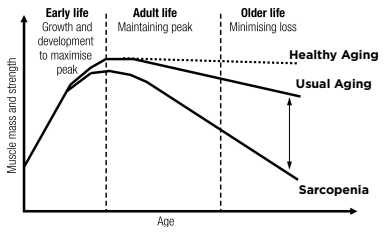
- Sarcopenia reflects a unique dysregulation that results in abnormally severe muscle degeneration.
  - Separate from general age–related declines in muscle mass.

Sarcopenia

Between the ages of 30 and 50 there is a 3 to 10% reduction in muscle fiber mass across both fast and slow twitch muscle fibers.

Between the ages of 60 and 80, muscle fiber mass reduces by approximately 15% each decade;

After age 80, muscle fiber mass reduced by approximately 30%.



Sarcopenia

- Although formally codified as a disease by the International Classification of Diseases in 2016, no formal diagnostic criteria exist.
  - Loss of muscle strength.
  - Loss of muscle size
  - Impaired ability to maintain function.

Without formal diagnostic criteria, screening effectiveness and prevalence is difficult to assess.

Sarcopenia

Proposed screening metrics:

Walking Speed

Different from typical walking tests to assess aerobic fitness, these tests would use a short distance such as from the waiting room to the clinical room.

Need for mobility aid

Either need for assistance moving from sitting to standing, or requiring use of walker/cane.

Handgrip Strength

Handgrip strength is relatively well correlated with muscular strength of major muscle groups in older adults.

Need for sweater/blanket

Individuals with very low levels of muscle mass tend to exhibit difficulty maintaining thermoregulation.

Sarcopenia

- Maintaining muscular strength is critical for maintaining health and wellbeing and is an underlying factor in many prominent disease states.
- This disease state places individuals at increased risk of falls, fractures, and greater susceptibility to disability.
- An individual who lacks the muscular strength for completing activities of daily living cannot typically live alone and may be confined to a nursing home.

10 to 20% of individuals over the age of 50 would meet criteria for clinically significant sarcopenia.

In long-term rehabilitation units, the prevalence of sarcopenia is over 50%.

Sarcopenia

Comorbid Conditions

Heart disease

Respiratory diseases

Diabetes

Osteoporosis

Risk Factors

Female

Small body frame

Family history of frequent falls or muscle disorder

Eating disorder

Protein insufficiency

Sarcopenia

Tertiary Prevention

- Physical and occupational therapy to maintain muscle function are predominately used.
  - Frequently incorporate nutritional interventions to address protein insufficiencies.
- Pharmaceutical approaches using low-dose steroids have been effective with early intervention to slow muscle wasting.
- A number of clinical trials are ongoing targeting hypothesized mechanisms which may lead to the disease state of sarcopenia.

Tertiary prevention efforts have only been effective at slowing disease progression, not reversal.

Reserve Theory