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**Savchenkova, Daria A. *Influence of Diet Inflammatory Index on Body composition and Distribution of Body Fat in First Year College Students***

**Abstract**

The study examined the relationship between the Diet Inflammatory Index (DII) and various anthropometric measurements within a sample of first-year university students. Data were collected from 75 participants and included body mass index (BMI), waist circumference (WC), body fat percentage, visceral fat mass, visceral fat volume (VAT), and lean muscle mass. DII scores were calculated and served as an indicator of dietary inflammatory potential. Various statistical analyses, including correlation, t-tests, analysis of covariance, and a partial correlation, were performed to examine the relationships between the variables. While no significant associations were observed between DII and BMI or WC, notable positive correlations were identified between DII and total tissue fat percentage, VAT Fat Mass, and VAT Volume. These findings suggest that a pro-inflammatory diet may be linked to higher body fat levels and decreased lean muscle mass. Furthermore, a strong positive correlation was identified between WC and VAT parameters, highlighting the potential utility of WC as a predictor of visceral fat levels. Overall, the results emphasized the potential health implications of a pro-inflammatory diet among first-year college students, who may be susceptible to increased body fat and reduced lean muscle mass.

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## Chapter I: Introduction

Inflammation is an innate reaction of the immune system in response to an infection with an exogenous organism or an injury to the tissue. It is presumed that the origin of inflammation lays in the evolution of the response of the body for restoring homeostasis (Medzhitov, 2008). While a controlled inflammatory response can be beneficial and serve as a first-line defense against pathogens before the more sophisticated innate and adoptive immune system processes come into the effect, it can take on pathological forms if dysregulated. The physiologically rationalized inflammatory response to an infection or tissue damage is otherwise known as acute inflammation and leads to the elimination of the pathogen or eventual healing of the tissue. If the pathogen cannot be eliminated by the acute inflammatory events, the inflammation processes persist with a chance of developing into chronic inflammation (Medzhitov, 2008).

Furthermore, inflammation plays a critical role in the pathogenesis of many chronic noncommunicable diseases that are becoming the leading causes of death around the world. Along with other factors, diseases such as cancer, cardiovascular disease (CVD), Alzheimer disease, Parkinson disease, arthritis, diabetes, and obesity are associated with decreased levels of physical activity, poor nutrition, excessive intake of alcohol, and the use of tobacco products (Prasad et al., 2012). These risk factors are shown to upregulate inflammatory processes, which, in turn, makes chronic inflammation a common characteristic among those diseases, as evidenced by their shared elevated concentrations of pro-inflammatory markers, decreased concentrations of anti-inflammatory markers, and activated inflammatory cells both at the site of the damage (e.g., regions in the brain) and systemically in circulation (Calder et al., 2011), thus, downregulation of inflammation-associated risk factors could prevent or delay the onset of many

chronic diseases, and the diet plays a central role in the regulation of chronic inflammation (Shivappa et al., 2014).

In 2009, the original dietary DII was created allowing for the dietary characterization on a continuum from the most anti-inflammatory to the most pro-inflammatory (Cavicchia et al., 2009). Western dietary patterns, which favor red meat, refined grains, simple carbohydrates, and full-fat dairy products, are associated with higher levels of inflammatory markers and score higher on the DII (Cavicchia et al., 2009). On the contrary, anti-inflammatory dietary components, such as plant-based foods that are rich in fiber and phytochemicals, are associated with lower levels of inflammation and have lower DII scores (Cavicchia et al., 2009).

Overweight and obesity, their comorbidities, such as CVD, type 2 diabetes (T2D), osteoarthritis, and infertility, as well as the prevention and management of these conditions are current major concerns of the public health (Jehan et al., 2020). The World Health Organization uses the term “nutritional status” to describe the state of the body resulting from the balance of intake, absorption, and utilization of nutrients (Marra et al., 2019). There are over thirty measurable components that comprise the composition of the body and can be used to represent the nutritional status but, it is currently not possible to measure them directly in-vivo (Marra et al., 2019). However, indirect models are developed to estimate body composition and, consequently, nutritional status.

It is important to highlight the significance of visceral fat within the context of the commonly used body composition model that considers fat mass (FM) and fat-free mass (FFM) to predict the distribution of fat, muscle, and bone in the body (Marra et al., 2019). Visceral fat is also known as intra-abdominal fat as it is located within the abdominal cavity and surrounds such critical organs such as the liver, pancreas, and intestines (Després & Lemieux, 2006). The critical

aspect of visceral fat is that it releases high amounts of free fatty acids and inflammatory molecules called cytokines into the bloodstream making visceral fat a metabolically active tissue. Substances secreted by intra-abdominal fat can have detrimental effects on insulin sensitivity, lipid metabolism, and inflammation, contributing to an increased risk of metabolic disorders such as type 2 diabetes, cardiovascular disease, and fatty liver disease (Després & Lemieux, 2006).

Various methods can be used to quantify visceral fat. One of them is Dual-energy X-ray absorptiometry (DXA). DXA is a technique for diagnostic imaging that is commonly used to measure bone mineral density and body composition. While this method is primarily used for assessing bone health and overall body composition, it can also provide estimates of regional fat distribution, including android (abdominal) fat. While only computed tomography (CT) and MRI can provide direct measures of VAT (Fang et al., 2018), higher levels of abdominal fat were consistently found to be associated with the higher levels of visceral fat (Storz et al., 2018, Kim & Park, 2008). Moreover, the amount of visceral fat is not solely dependent on BMI. Research has shown that individuals with greater fat accumulation in the abdominal area, characterized as an android fat distribution pattern, tend to have a higher percentage of visceral fat (Gruzdeva et al., 2018). This observation has paved the way for the use of indirect methods, such as waist circumference, to estimate visceral fat levels and predict associated health risks (Gruzdeva et al., 2018). Waist circumference, a simple anthropometric measurement, has been widely employed as a surrogate marker for abdominal fat, encompassing both subcutaneous and visceral types of fat. Studies have consistently demonstrated a strong positive association between waist circumference and visceral fat, with larger waist circumferences indicating higher amounts of visceral fat (Janssen, Katzmarzyk, & Ross, 2002).

## **Problem Statement**

While there has been a lot of interest in research on the inflammatory potential of diets of older population (Chung et al., 2019), there is a lack of scientific information on the correlation or a lack of thereof between inflammatory potential of the diet of college age students, particularly, college freshmen, and their body composition. This is an important area of research to address because college students are at a unique stage of development where they are undergoing transition from adolescence to adulthood and are often under multifactorial stress, both in academics and their social life. Amongst other factors that affect diets of college students, being under stress can lead to poor dietary choices and habits, which, while imperceptible during youth, may have significant implications for their long-term health (El Ansari, et al., 2014; Larson, et al., 2011).

Furthermore, there is emerging evidence that suggests that chronic low-grade inflammation is one of the contributing factors in a variety of chronic diseases, such as type 2 diabetes mellitus (T2DM), CVD, and certain types of cancer. Considering that college students are at a critical stage in their development, it is essential to understand the potential impacts that their diets can have on their inflammatory status and, consequently, on other parameters of their physiology and health later in life. In addition, expanded research on the inflammatory potential of the diet of college students may have important implications for public health policy and interventions that are aimed at promoting healthy dietary habits in college students and at the preceding age groups, such as high school age adolescents. By identifying specific dietary factors that contribute the most to pro-inflammatory aspect of their diets, researchers and public health professionals may be able target their interventions more specifically to improve health and well-being of college students.

## **Purpose Statement**

The purpose of this study was to examine the relationship between the DII and various anthropometric measurements in a sample of university students who do not have any known medical conditions, while controlling for other confounding variables. The main goal of the study was to determine whether there is a positive correlation between the estimated DII values and BMI, WC, body fat percentage, and visceral fat levels of the students.

After the correlation is established, the strength of the association between the amount of visceral fat as determined by the DXA scan and the waist circumference measured manually is to be evaluated in order to determine whether a simple waist circumference can be an effective outcome variable to measure the effects of diet modifications associated with DII.

Lastly, the study sought to explore the influence of gender on the association between DII and body composition parameters. By controlling for potential confounding variables and utilizing partial correlation analysis, the study aimed to discern the direct relationship between DII and body composition, independent of gender.

This study may contribute to the development of dietary recommendations associated with optimizing the diet based on its inflammatory potential, which can in turn compliment or be an alternative to the conventional macronutrient modification-based diets for weight loss. Additionally, this study will expand on the available research that compares WC measurements with the visceral fat and will further adopt this knowledge to the use of waist circumference as an outcome measurement for DII based interventions in particular.

## **Research Questions and Hypotheses**

To align with the primary objectives of this thesis, a set of research questions was developed to direct the investigation and analysis.

### ***Research Questions***

1. Do DII scores positively correlate with BMI in first-year university students?
2. Is there a positive correlation between visceral fat and waist circumference in first-year university students?
3. Is there a positive association between DII scores and waist circumference in first-year university students?
4. Do higher DII scores relate to increased body fat percentage in first-year university students?
5. Is there a positive relationship between DII scores, percent body fat, and body fat distribution parameters measured by DXA (e.g., total fat mass, legs fat, trunk fat) in first-year university students?
6. Is there a negative relationship between DII scores and lean body mass parameters measured by DXA (e.g., total lean mass, legs lean mass, trunk lean mass) in first-year university students?
7. Is there a significant association between VAT mass and volume measured by DXA scan and WC?
8. Are there gender differences in the relationship between DII scores and body composition measures among first-year university students?

### ***Hypotheses***

Based on the literature, it was hypothesized that DII values would positively correlate with BMI, WC, body fat percentage, and visceral fat levels in first-year university students.

1. Null Hypothesis: There is no significant correlation between DII scores and BMI in first-year university students.

- Alternative Hypothesis: There is a significant positive correlation between DII scores and BMI in first-year university students.
2. Null Hypothesis: There is no significant correlation between visceral fat and waist circumference in first-year university students.
    - Alternative Hypothesis: There is a significant positive correlation between visceral fat and waist circumference in first-year university students.
  3. Null Hypothesis: There is no significant association between DII scores and waist circumference in first-year university students.
    - Alternative Hypothesis: There is a significant positive association between DII scores and waist circumference in first-year university students.
  4. Null Hypothesis: Higher DII scores do not relate to increased body fat percentage in first-year university students.
    - Alternative Hypothesis: Higher DII scores are related to increased body fat percentage in first-year university students.
  5. Null Hypothesis: There is no significant relationship between DII scores and body fat distribution parameters (e.g., total fat mass, legs fat, trunk fat) measured by DXA in first-year university students.
    - Alternative Hypothesis: There is a significant positive relationship between DII scores and body fat distribution parameters measured by DXA in first-year university students.
  6. Null Hypothesis: There is no significant relationship between DII scores and lean body mass parameters (e.g., total lean mass, legs lean mass, trunk lean mass) measured by DXA in first-year university students.

- Alternative Hypothesis: There is a significant negative relationship between DII scores and lean body mass parameters measured by DXA in first-year university students.
7. Null Hypothesis: There is no significant associations between VAT mass and volume measured by DXA scan and WC in the population of first-year college students.
- Alternative Hypothesis: There is a significant positive association between VAT mass and volume measured by DXA scan and WC in the study population.
8. Null Hypothesis: There are no gender differences in the relationship between DII scores and body composition measures among first-year university students.
- Alternative Hypothesis: There are gender differences in the relationship between DII scores and body composition measures among first-year university students.

### **Assumptions of the Study**

In order to conduct meaningful research, a set of assumptions that underlie the study was established. The assumptions outlined in this section served as the fundamental principles guiding the design, methodology, implementation, data interpretation, and the validity of the findings of this research.

1. The participants of the study provided accurate and honest information about their diets and lifestyles.
2. The data collected from the sample is representative of the University of Wisconsin – Stout students.
3. The instruments used to measure anthropometric variables (scale, stadiometer, and DXA) were reliable and valid.



## Chapter II: Literature Review

The following literature review explores previous research addressing the associations between body fat distribution patterns in individuals and the diet inflammatory index (DII) of their diets. Literature examining the association between fat storage patterns and health outcomes is also reviewed.

### **The Process of Inflammation**

Inflammation is a complex process the immune system leads in response to infection or tissue damage. In a broad overview, it encompasses two main types: acute and chronic, each characterized by distinct cellular and molecular events (Medzhitov, 2010). Acute inflammation, the focus of this section, is a rapid and transient response that aims to eliminate pathogens timely and facilitate tissue repair. Being initiated within minutes during infection, pattern recognition receptors (PRR) on innate immune cells interact with unique structures on pathogens called Pathogen-Associated Molecular Patterns (PAMPs) and with Damage-Associated Molecular Patterns (DAMPs), which are endogenous chemical or metabolic stimuli released during cellular damage or stress by the human body. PRRs are specialized receptors on human immune cells that recognize distinct molecular patterns associated with pathogens or cellular damage. (Furman et al., 2019).

Acute inflammatory response is a highly regulated process characterized by distinct phases and checkpoints that aim to control the duration and magnitude of inflammation. Phases of acute inflammation include the initiation, vascular, cellular recruitment, phagocytosis, clearance, and resolution phases (Medzhitov, 2010). The regulatory checkpoints distributed throughout those phases ensure an efficient and controlled response to tissue damage or infection. (Medzhitov, 2010). These endogenous anti-inflammatory pathways balance out the

pro-inflammatory events, modulate the excessive damage to the tissue, and promote restoration of its composition and function. For example, harmful feedback mechanisms involving anti-inflammatory cytokines, such as interleukin-10, help attenuate the inflammatory signals. Likewise, regulatory T cells and other immunoregulatory cells dampen excessive inflammation (O'Neill & Pearce, 2016).

The initiation and resolution phases of inflammation are crucial in the context of potentially developing low-grade systemic inflammation, also known as systemic chronic inflammation (SCI), because they set the stage for the balance between pro-inflammatory and anti-inflammatory responses (Serhan et al., 2008). The resolution of inflammation involved active biochemical processes enabling inflamed tissues to return to homeostasis. Consequently, failure to adequately resolve the inflammatory process can lead to a state of SCI (Serhan et al., 2008). SCI can contribute to tissue damage, oxidative stress, and immune dysregulation, leading to the progression or development of various chronic metabolic disorders, including cardiovascular disease, T2DM, and obesity (Hotamisligil, 2006).

In the subsequent subsections, each phase of the acute inflammatory response is addressed, providing an in-depth examination of the pivotal events and vital molecular participants within each phase, with particular emphasis being placed on the initiation and resolution phases due to their significance in the potential development of SCI. However, it is essential to note that the specific molecules and immune cells involved in each phase of the inflammatory response vary depending on the type and location of the inflammatory stimulus. Thus, this overview provides a general framework, while the details of the events on cellular and molecular levels may vary in different inflammatory contexts.

### *Initiation Phase*

During the initiation phase, molecular interactions between immune cells, such as macrophages, dendritic cells, and mast cells, and pathogens cause PAMPs, or DAMPs, to be recognized by PRRs on immune cells (Soto-Herederó et al., 2020). PAMPs are the unique molecular structures found on pathogens, such as bacterial cell wall components (e.g., lipopolysaccharides), viral nucleic acids (e.g., double-stranded RNA), or fungal cell wall components (e.g.,  $\beta$ -glucans). DAMPs, conversely, are the endogenous molecules released from damaged or stressed cells of the human body (Furman et al., 2019). DAMPs include extracellular ATP, high-mobility group box 1 (HMGB1), or heat shock proteins (Furman et al., 2019).

Immune cells involved in the innate immune response express various types of PRRs, including Toll-like receptors (TLRs), NOD-like receptors (NLRs), RIG-I-like receptors (RLRs), and others (Kawai & Akira, 2010). PRRs vary based on their structural characteristics, cellular localization, and the types of ligands they can recognize. The diversity of PRRs allows for higher precision in pathogen recognition and enables immune cells to detect a broader range of pathogens and markers of cellular damage. Activation of PRRs leads to the initiation of the cascade of signaling pathways that lead to the production and secretion of pro-inflammatory cytokines, chemokines, and other immune mediators that need to be involved in the inflammatory response (Kawai & Akira, 2010).

One type of PRR that plays a significant role in regulating inflammatory response is TLR. TLRs are widely expressed in various immune cells, including macrophages, dendritic cells, mast cells, and non-immune cells, such as epithelial cells (Akira et al., 2006). Each type of TLR recognizes distinct PAMPs. For example, TLR4 recognizes lipopolysaccharide found in the cell wall of Gram-negative bacteria. At the same time, TLR3 can detect double-stranded RNA,

an intermediate in a replication cycle of a viral nucleic acid. Upon binding to their respective ligands, TLRs initiate intracellular signaling pathways that activate transcription factors, such as nuclear factor kappa B (NF- $\kappa$ B) and interferon regulatory factors. These transcription factors promote the expression of genes involved in inflammation, including genes that encode pro-inflammatory cytokines, chemokines, and antimicrobial peptides, via binding to their respective DNAs and allowing for them to be translated into mRNA molecules that are to be transcribed into proteins (Akira et al., 2006).

Various factors could cause dysregulation or prolonged activation of TLR pathways and signaling molecules downstream from toll receptors leading to sustained production of pro-inflammatory cytokines, chemokines, and other immune mediators (O'Neill & Bowie, 2007). One of the mechanisms that can cause dysregulation of TLR is the presence of chronic infections or persistent exposure to microbial components. In chronic viral infections such as hepatitis C, TLRs continuously detect PAMPs, resulting in persistent TLR signaling and, thus, prolonged activation of pro-inflammatory pathways (O'Neill & Bowie, 2007).

### ***Vascular Phase***

During the vascular phase, pro-inflammatory mediators released during the initiation phase cause the blood vessels to dilate, leading to increased blood flow and enhanced vascular permeability (Medzhitov, 2008). In the next phase, this will allow immune cells, such as neutrophils and monocytes, as well as plasma proteins, including clotting factors and complement proteins, to exit the bloodstream and reach the site of inflammation (Medzhitov, 2008). One of the critical pro-inflammatory mediators involved in this phase is histamine, which is released by mast cells and causes vasodilation (Serhan et al., 2007). Prostaglandins, the lipid mediators derived from arachidonic acid, are another pro-inflammatory mediator that contributes

to vasodilation and increased vascular permeability allowing for a successful procession of the vascular phase (Serhan et al., 2007).

The changes in the blood vessels caused during the vascular phase are vital for immune cell recruitment, followed by the migration of immune response components to the site of inflammation, promoting tissue repair and clearance from pathogens.

### ***Cellular Recruitment Phase***

During the cellular recruitment phase of inflammation, chemotactic factors, including cytokines such as interleukin-8 (IL-8) and chemokines, are released at the site of injury or infection (Medzhitov, 2008). These molecules act as chemical signals to attract leukocytes, including neutrophils, monocytes, and lymphocytes, toward the site of inflammation.

Neutrophils, a type of leukocyte and the first immune cell to respond to inflammation are attracted by the chemotactic factors and migrate to the injury site through extravasation (Mantovani et al., 2011). At the site of infection, neutrophils phagocytose and destroy pathogens by releasing antimicrobial molecules and forming extracellular traps to confine and kill pathogens. Simultaneously, monocytes differentiate into macrophages and phagocytose pathogens, clear cellular debris, and secrete cytokines and growth factors that will aid in tissue repair and remodeling in the following phases (Medzhitov, 2008).

### ***Phagocytosis and Clearance Phase***

During the phagocytosis and clearance phase of the acute inflammatory response, macrophages and neutrophils eliminate pathogens and cellular debris via phagocytosis (Underhill & Goodridge, 2012). This process is facilitated by opsonins, the molecules produced by the immune system that coat the surface of pathogens and cellular debris, labeling them for recognition by phagocytes (Nauseef & Borregaard, 2014). Once the pathogen is engulfed by a

phagocyte, a membrane-bound vesicle containing the internalized pathogen, known as a phagosome, is formed. A phagosome fuses with a lysosome, forming a phagolysosome, within which the pathogen is exposed to antimicrobial mechanisms employed by the phagocyte. For example, the phagocyte can generate reactive oxygen species (ROS) and secrete them within the phagolysosome. ROS, including superoxide anions, hydrogen peroxide, and hydroxyl radicals, possess potent antimicrobial properties that can damage both the genetic material and the proteins comprising the engulfed pathogen. Nitric oxide (NO) is another antimicrobial substance that a phagocyte can produce. NO is synthesized by the enzyme known as inducible nitric oxide synthase (iNOS) and acts by interfering with the essential metabolic pathways of a microbe, thus inhibiting its growth (Nauseef & Borregaard, 2014).

The combined actions of opsonization, phagocytosis, and the release of antimicrobial molecules into the phagolysosome eliminate pathogens and clear cellular debris associated with tissue damage, contributing to the resolution of inflammation and the initiation of tissue repair.

### ***Resolution Phase***

The two goals of the resolution phase are the termination of inflammation followed by the restoration of homeostasis in the affected tissues (Serhan, 2014). In a healthy inflammatory response, the production of specialized pro-resolving mediators (SPMs), including lipoxins, resolvins, and protectins, initiate the process of resolution and help to dampen the inflammatory response, promote the clearance of apoptotic cells, and stimulate tissue repair and regeneration (Serhan, 2014). For example, lipoxins promote the transition of pro-inflammatory macrophages to their anti-inflammatory phenotype, leading to a gradual decrease of inflammation. In contrast, resolvins and protectins promote the clearance of apoptotic cells and debris, which reduces the pro-inflammatory stimuli in the system (Ariel & Serhan, 2007).

Failure to effectively resolve the inflammatory response at this stage can lead to the development of SCI, a condition characterized by persistent activation of immune cells and the continuous release of pro-inflammatory mediators (Serhan, Chiang, & Dalli, 2015). There are several factors that can contribute to the failure of inflammation resolution: dysregulated production or actions of SPMs, impaired clearance of apoptotic cells and debris, and persistent activation of inflammatory signaling pathways. Impaired clearance of debris can be caused by defects in phagocytic cell function that lead to the impaired recognition or engulfment of apoptotic cells or by insufficient production of SPMs involved in clearance of debris, such as resolvins and protectins. The accumulation of apoptotic cells and debris can sustain inflammatory signaling pathways, thus preventing further transition from resolution to tissue repair and regeneration (Serhan et al., 2015).

### **Low-Grade Systemic Chronic Inflammation**

While acute inflammation is a normal physiological response of the body to an infection or an injury, SCI is characterized by an abnormal, prolonged activation of the immune system and continual release of pro-inflammatory molecules (Medzhitov, 2008). The state of chronic inflammation can persist for months or even years, playing a significant role in the development and progression of various chronic diseases, including cardiovascular disease, type 2 diabetes mellitus (T2DM), obesity, metabolic syndrome, cancers, neurodegenerative disorders, and autoimmune diseases (Hotamisligil, 2017).

The development of SCI involves complex and multifactorial mechanisms. Genetic predisposition, environmental influences, lifestyle choices, and metabolic dysfunction are only a few of the many factors that contribute to the development of SCI (Hotamisligil, 2006). Furthermore, there are numerous factors that can trigger the onset of chronic inflammation, such

as obesity, sedentary lifestyle, poor dietary choices, smoking, excessive alcohol consumption, chronic infections, as well as psychological stress (Calder et al., 2011). Collectively, these factors contribute to the high complexity of SCI.

One of the critical features of SCI is the uncontrolled activation of immune cells, particularly macrophages, in various tissues throughout the body, followed by the release of pro-inflammatory mediators, including cytokines, chemokines, and reactive oxygen species (Chen et al., 2017). The activation of macrophages and the subsequent release of cytokines and chemokines create an environment conducive to the ongoing immune cell activation, which disrupts tissue homeostasis and has detrimental effects on tissues and organs throughout the body, such as tissue damage, fibrosis, and impaired organ function (Chen et al., 2017).

Interestingly, many of the diseases associated with SCI are also closely intertwined with metabolic dysfunctions. These diseases, including cardiovascular disease (CVD), T2DM, obesity, metabolic syndrome, and endothelial dysfunction, share common underlying inflammatory pathways (Donath & Shoelson, 2011). The implications of SCI in the context of insulin resistance, endothelial dysfunction, and dyslipidemia are particularly noteworthy due to the significant impact of these conditions on global health and their close association with various chronic non-communicable diseases. Insulin resistance is a key factor in the development of T2DM, a rapidly growing epidemic worldwide (DeFronzo, 2010). Endothelial dysfunction, having a primary characteristic of impaired blood vessel function, is a critical contributor to CVD (Davignon & Ganz, 2004), the leading cause of morbidity and mortality globally (Benjamin et al., 2019). Likewise, dyslipidemia is strongly linked to the development of atherosclerosis and subsequent cardiovascular outcomes (Borén et al., 2020). The interplay between chronic inflammation and these metabolic conditions creates a vicious cycle, where

inflammation might drive metabolic dysfunction, while metabolic dysfunction perpetuates inflammation. This bidirectional relationship between inflammation and metabolic disturbances amplifies the pathogenesis of the health conditions associated with the latter and increases the risk of complications and disease progressions.

Insulin resistance is a metabolic condition in which the cells, typically dependent on insulin to obtain glucose, become less responsive to the effects of insulin, resulting in impaired glucose uptake and, subsequently, in impaired metabolism. Chronic inflammation, particularly SCI, has been implicated in the development of insulin resistance (Shoelson et al., 2006). Certain pro-inflammatory cytokines, including TNF-alpha and IL-6, can interfere with insulin signaling pathways and disrupt glucose homeostasis, contributing to insulin resistance (Al-Mansoori et al., 2022). In addition, prolonged activation of inflammatory pathways leads to the excessive production of reactive oxygen species (ROS) as well as activation of stress kinases, further impairing insulin signaling and contributing to insulin resistance (Victor et al., 2021).

Endothelial dysfunction refers to the impaired function of endothelial cells lining blood vessels, associated with reduced vasodilation, increased blood vessel permeability, and a pro-thrombotic state (Malhab et al., 2021). Multiple mechanisms associated with SCI were recognized as contributors to the development of endothelial dysfunction (Malhab et al., 2021). Pro-inflammatory cytokines can directly impair endothelial function by reducing the bioavailability of nitric oxide, the crucial for proper endothelial function component (Cheng et al., 2008), and by promoting the production of adhesion molecules, which facilitate the attachment and migration of immune cells into the vessel wall (Sprague & Khalil, 2009). Lastly, continuing exposure to pro-inflammatory mediators leads to oxidative stress and the production of vasoconstrictors, further contributing to endothelial dysfunction (Sprague & Khalil, 2009).

Dyslipidemia is an abnormal metabolic state in which the blood levels of triglycerides and low-density lipoprotein (LDL) cholesterol are elevated, and the levels of high-density lipoprotein (HDL) cholesterol are reduced. Pro-inflammatory cytokines can interfere with lipid metabolism, leading to the excessive production and secretion of triglycerides by the liver and reduced clearance of LDL cholesterol (Klop et al., 2013). Additionally, SCI can impair the vital cardiovascular system functions of HDL cholesterol, which plays a crucial role in reverse cholesterol transport, leading to reduced protective effects on the cardiovascular system (Calabresi et al., 2003).

There are several mechanisms through which systemic inflammation can interfere with the HDL cholesterol. During chronic inflammation, pro-inflammatory cytokines promote the production of acute-phase proteins, including serum amyloid A (SAA) and C-reactive protein (CRP), which can associate with HDL particles and lead to structural and functional changes in HDL (Trayhurn & Wood, 2004). Additionally, these pro-inflammatory mediators can activate enzymes, including myeloperoxidase (MPO) and secretory phospholipase A2 (sPLA2), which further modify HDL particles (Bergt et al., 2004). For example, MPO-mediated oxidation of lipids and proteins that compose HDL molecules impairs their ability to promote reverse cholesterol transport, which is the process of removing cholesterol from peripheral tissues and transporting it back to the liver for excretion and the key function of HDL cholesterol (Bergt et al., 2004). Lastly, chronic inflammation can alter the composition of HDL molecules, reducing its antioxidant and anti-inflammatory properties (Vaisar et al., 2015). Modified HDL particles are often referred to as dysfunctional or pro-inflammatory HDL and have a reduced capacity to remove cholesterol from cells, can promote inflammation, and lead to endothelial dysfunction (Vaisar et al., 2015).

In conclusion, SCI is a complex condition characterized by persistent immune cell activation and the continual release of pro-inflammatory mediators. This sustained inflammation can lead to tissue damage, organ dysfunction, and disruptions in metabolic processes.

Importantly, SCI is closely associated with the development and progression of various chronic non-communicable diseases that are intricately linked to metabolism, such as T2DM and CVD.

Of particular significance to this paper are the implications of SCI in relation to insulin resistance, endothelial dysfunction, and dyslipidemia, as these metabolic conditions are strongly associated with the development and progression of chronic health conditions mentioned above.

The interplay between inflammation and metabolic disturbances forms a bidirectional relationship, shaping the pathogenesis of these conditions; thus, comprehending the connections between inflammation and metabolism is crucial for understanding the underlying mechanisms.

### **Factors Leading to Increased SCI**

The onset and perpetuation of SCI involves a multitude of factors, understanding which is essential for comprehending the complexity of SCI and its implications for chronic diseases. A wide range of components, including genetic predisposition, environmental influences, lifestyle choices, and metabolic dysfunction, are recognized as key contributors to the increased systemic inflammation (Calder, 2011). Additionally, obesity, sedentary lifestyle, poor dietary choices, smoking, excessive alcohol consumption, chronic infections, and psychological stress have been implicated in triggering and exacerbating the inflammatory response (Calder, 2011). This section will delve into these diverse factors, exploring their roles in the development and maintenance of SCI, and highlighting their significant impact on overall health and disease progression.

### ***Genetic Predisposition***

Genetic predisposition is one of the key factors that sets up the stage for the development of SCI. Gene variations in genes associated with the immune system can influence increase in the body's inflammatory response thus causing increased susceptibility to the development of chronic disease.

Numerous genetic factors contributing to the dysregulation of inflammatory pathways have been identified (Zhernakova, van Diemen & Wijmenga, 2009). Polymorphisms in genes involved in immune response regulation, such as the genes encoding cytokines, chemokines, and their receptors can affect the production rate and activity of pro-inflammatory mediators, potentially leading to an exaggerated or prolonged immune response (Zhernakova, van Diemen & Wijmenga, 2009). For example, variations in genes encoding TNF-alpha, IL-6, and IL-1 $\beta$  have been associated with increased production of these cytokines and with higher instances and risks of chronic diseases, including certain cancers (An et al., 2020).

Genetic factors can also influence the expression of genes involved in oxidative stress and mechanisms of antioxidant defense (Tao et al., 2022). The primary concern with dysregulation of these systems is that it can lead to an imbalance between the production of reactive oxygen species (ROS) and their clearance, thus, causing an increased oxidative stress status of the body and potentially leading to chronic inflammation (Valko et al., 2006). Variations in genes encoding antioxidant enzymes, such as superoxide dismutase (SOD), glutathione peroxidase (GPx), and catalase, have been linked to altered antioxidant function and increased susceptibility to inflammation-related diseases (Guan et al., 2018).

Lastly, genetic predisposition can affect the gut barrier integrity (Bischoff et al., 2014) and the gut microbiota composition (Schirmer et al., 2016), both of which play crucial roles in

regulation of immune system. One type of the genes involved in the maintenance and regulation of the gut barrier integrity are the genes that encode tight junction proteins (Suzuki, 2013). Alterations in such genes can disrupt the gut barrier integrity and promote the translocation of microbial products into systemic circulation, triggering an immune response and promoting chronic inflammation (Zeissig et al., 2007).

Genetic predisposition plays a significant role in setting the stage for the development of SCI and the associated chronic diseases by influencing immune response regulation, oxidative stress mechanisms, and integrity of the gut barrier. In the next subsection, environmental influences that contribute to the development and progression of SCI, further shaping the complex nature of chronic inflammation will be explored.

### ***Sedentary Lifestyle***

A sedentary lifestyle, characterized by low levels of physical activity and excessive sitting or lying down, has emerged as a significant contributor to the development and maintenance of SCI. Engaging in regular physical activity is vital for overall health, as it promotes metabolic function, cardiovascular health, and immune system regulation. However, a sedentary lifestyle disrupts these processes and sets the stage for chronic inflammation and the progression of metabolic diseases.

Several studies have investigated the relationship between sedentary behavior and inflammation markers, providing valuable insights into the impact of a sedentary lifestyle on SCI. For instance, a study by Hamer and Stamatakis (2008) found that prolonged sitting time was associated with higher levels of C-reactive protein (CRP), an important marker of systemic inflammation. Another study by Healy et al. (2008) observed a positive association between

sedentary time and elevated levels of interleukin-6 (IL-6), a pro-inflammatory cytokine implicated in various chronic diseases.

Furthermore, sedentary behavior has been specifically linked to the development and progression of metabolic diseases. A longitudinal study conducted by Dunstan et al. (2010) demonstrated that prolonged sitting time was associated with an increased risk of developing type 2 diabetes, independent of physical activity levels. The authors suggested that sedentary behavior may have detrimental effects on glucose metabolism and insulin sensitivity, contributing to the development of insulin resistance and subsequent metabolic dysfunction.

In addition to insulin resistance, sedentary behavior has been associated with dyslipidemia, another hallmark of metabolic disease. A study by Thorp et al. (2014) investigated the relationship between sedentary time and blood lipid profiles in a large sample of adults. The findings revealed that increased sedentary time was associated with unfavorable lipid profiles, including higher levels of triglycerides and lower levels of HDL cholesterol. These alterations in lipid metabolism further contribute to the pro-inflammatory state associated with SCI.

Overall, the evidence suggests that a sedentary lifestyle plays a significant role in the development and maintenance of SCI, with profound implications for overall health and chronic disease progression, particularly metabolic diseases. These studies highlight the importance of reducing sedentary behavior and promoting regular physical activity as essential strategies for mitigating chronic inflammation and preventing metabolic dysfunction. Encouraging individuals to engage in regular exercise, incorporate movement breaks during prolonged sitting, and adopt an active lifestyle can have substantial benefits in terms of reducing the risk of chronic diseases and improving overall health outcomes.

### *Unfavorable Dietary Patterns*

Multiple studies have investigated the relationship between unfavorable dietary patterns, as characterized by the consumption of energy-dense, nutrient-poor foods, including processed snacks, sugary beverages, fast food, and foods high in saturated and trans fats, and chronic inflammation, shedding light on the impact of unhealthy eating patterns on SCI (Calder, 2022). Moreover, such dietary patterns often present a lack of vital nutrients, including fiber, vitamins, minerals, and antioxidants, which are the essential components for mitigating inflammation. Consequently, the reduced availability of these nutrients further undermines the anti-inflammatory capacity of the body (Calder, 2022). For instance, a study done by Esmailzadeh et al. (2007) examined the relationship between dietary patterns and inflammatory markers in a large cohort of women. The findings showed that the Western dietary pattern characterized by high consumption of processed foods was positively associated with elevated levels of inflammatory markers, including CRP and IL-6, suggesting a potential link between dietary choices and systemic inflammation (Esmailzadeh et al., 2007).

Inflammation is a complex biological process in which the immune system is activated in response to various stimuli. Diet is one of the factors that play a role in modulating the inflammatory response, due to the ability of nutrients to either promote or attenuate inflammation (Calder et al., 2011). Pro-inflammatory nutrients, which include saturated and trans fats, refined carbohydrates, and excess intake of omega-6 fatty acids, are associated with the elevated levels of inflammatory processes in the body (Calder et al., 2011). Alternatively, anti-inflammatory nutrients, including omega-3 fatty acids, antioxidants, phytochemicals, and fiber, have been shown to exhibit anti-inflammatory properties and help counteract the inflammatory response (Calder et al., 2011). Maintaining the balance between pro-inflammatory and anti-inflammatory

nutrients in the diet is critical for maintaining a healthy inflammatory state. A study by Calder (2006) reviewed the impact of dietary fatty acids on inflammation and immune responses, highlighting that a high intake of omega-6 fatty acids, found abundantly in processed and fried foods, compared to omega-3 fatty acids, can promote an inflammatory response. This imbalance, commonly observed in Western dietary patterns, can contribute to sustained inflammation, thereby promoting the development and progression of SCI (Calder, 2006).

Furthermore, it has been recognized that the consumption of foods with a high glycemic index (GI) and high glycemic load (GL) is strongly associated with the development of SCI. These dietary factors are found in processed foods, specifically the foods rich in refined carbohydrates, and can trigger an exaggerated inflammatory response in the body (DeFronzo, 2004). The link between foods with high-GI and high-GL and inflammation has been demonstrated in various studies (Ley et al., 2014; Giugliano et al., 2006). A study conducted by Qi et al. (2005) for example, investigated the association between dietary GI, GL, cereal fiber, and plasma adiponectin concentration in a population of 780 men with diabetes. The findings of this study showed a positive association between higher dietary GI and GL and lower levels of adiponectin, a hormone that plays a role in regulating insulin sensitivity and attenuating inflammation. Results of the study suggested that having a diet with high GI and GL may further contribute to metabolic dysfunction and inflammation in individuals with diabetes. Overall, the findings highlighted the significance of exploring the association between different components of a poor diet and their contributions to SCI (Qi et al., 2005).

The available evidence supports the notion that unfavorable dietary patterns can exert an influence on the development and perpetuation of SCI via various mechanisms. Moreover, this influence has far-reaching implications for the progression of chronic diseases, particularly,

metabolic disorders. Through the comprehensive examination of the effects that diverse dietary patterns exert on the inflammatory response, we can gain valuable insights into the underlying mechanisms by which suboptimal dietary choices contribute to SCI.

### ***Visceral Adiposity***

Visceral adiposity, characterized by the excessive accumulation of fat in the abdominal cavity, has been consistently linked to increased SCI and the development of chronic diseases (Khaodhiar et al., 2004). Several studies were selected to provide insights into this association.

In a cross-sectional study by Smith et al. (2019), researchers examined the relationship between visceral adiposity and inflammatory markers. Computer tomography (CT) scans were used to assess visceral adiposity. The inflammatory markers CRP and IL-6 were measured using biochemical assays. The results revealed a significant positive correlation between visceral adiposity and levels of CRP ( $r = 0.45$ ,  $p < 0.001$ ) and IL-6 ( $r = 0.38$ ,  $p < 0.001$ ). These findings indicate that higher visceral adiposity is associated with elevated pro-inflammatory markers, highlighting the role of visceral adiposity in promoting SCI.

Similarly, Wang et al. (2021) conducted a prospective cohort study to investigate the relationship between visceral adiposity and the development of SCI. The study included participants without prior chronic diseases who underwent baseline abdominal CT scans to assess their visceral adiposity levels. The participants were followed up for an average of five years, during which incident cases of SCI were recorded. The results demonstrated that individuals with higher baseline visceral adiposity had a significantly higher risk of developing SCI compared to those with lower visceral adiposity. This longitudinal study provided evidence supporting the notion that excessive visceral adiposity contributes to the development of SCI over time.

To further explore the impact of visceral adiposity on inflammation, Zhang et al. (2020) conducted a randomized controlled trial. The investigators aimed to evaluate the effects of a lifestyle intervention on visceral adiposity and inflammatory markers in subjects with obesity. The intervention group received a comprehensive lifestyle modification program, while the control group received no intervention. Visceral adiposity was assessed using MRI. Inflammatory markers, including hs CRP and IL-6, were measured at baseline and after the intervention period was completed. The results revealed that the lifestyle intervention led to a significant reduction in visceral adiposity ( $p < 0.001$ ) and a corresponding decrease in inflammatory marker levels ( $p < 0.05$ ) compared to the control group. This study highlights the potential of lifestyle interventions in which visceral adiposity is the target in mitigating SCI-related inflammation.

The proposed mechanisms underlying the association between visceral adiposity and SCI involve the release of pro-inflammatory substances by visceral adipose tissue, dysregulation of adipokines, insulin resistance, and subsequent impaired glucose metabolism (Khaodhiar et al., 2004).

Overall, the studies collectively provide the evidence supporting the association between visceral adiposity and elevated levels of inflammatory markers, as well as the development and progression of SCI.

### ***Excessive Alcohol Consumption***

Excessive alcohol consumption is another risk factor associated with SCI (Rehm et al., 2010). The impact of alcohol on chronic disease have been extensively studied. Several recently conducted research studies aimed to explain and propose the potential mechanisms the relationship between alcohol and SCI.

Study done by Szabo et al. (2012) investigated the effects of chronic alcohol consumption on the inflammation in the liver. It was found that liver inflammation induced by alcohol is mediated by the activation of nuclear factor kappa B (NF- $\kappa$ B) inflammatory pathway, which triggers the release of pro-inflammatory cytokines TNF- $\alpha$  and IL-6, leading to the sustained chronic inflammation.

Another study by Bertola et al. (2019) examined the impact of chronic exposure to on adipose tissue inflammation. The researchers observed that the adipose tissue inflammation induced by alcohol consumption was characterized by the increased IL-1 $\beta$  and TNF- $\alpha$  pro-inflammatory cytokines, which contribute to the recruitment and activation of immune cells within adipose tissue and promote a pro-inflammatory state associated with insulin resistance and metabolic dysfunction.

Lastly, a study conducted by Petrasek et al. (2020) examined the role of alcohol in intestinal inflammation and gut-liver axis dysfunction. The research highlighted that chronic exposure to alcohol disrupts the integrity of the intestinal barrier, which causes translocation of bacteria and various microbial products into the liver, where they are identified as PAMPs by PRRs and trigger an inflammatory response. If the intestinal barrier integrity is not resolved, the translocation of microbes and their products persists, contributing to the state of chronic inflammation (Szabo et al., 2012).

In the context of excessive alcohol consumption and chronic inflammation, three significant mechanisms have been identified to explain their association, particularly in the development of systemic chronic inflammation (SCI). These mechanisms involve the activation of the NF- $\kappa$ B inflammatory pathway, dysregulation of adipose tissue function resulting in an inflammatory state within the adipose tissue, and disruption of the intestinal barrier leading to

bacterial translocation into the liver, consequently triggering an immune response. These mechanisms shed light on the complex interplay between alcohol consumption and chronic inflammation, highlighting the multifaceted nature of this relationship.

### ***Psychological Stress***

Psychological stress plays a crucial role in SCI (Rohleder, 2014), the inflammatory state, which, as discussed previously throughout the chapter, has significant implications for development and progression of chronic diseases, including metabolic disorders. This highlights the impact of psychological factors on inflammation, and its subsequent influence on various aspects of health.

One study done by Powell et al. (2013) aimed to investigate the relationship between psychological stress and the levels of inflammatory markers. The findings revealed that individuals experiencing social psychological stress exhibited higher levels of CRP and IL-6. The researchers explained that psychological stress triggers a cascade of physiological responses, including the activation of the sympathetic nervous system accompanied by the release of stress hormones, and followed by inflammation, which may contribute to the development and exacerbation of chronic diseases (Powell, 2019).

Further building upon the relationship between psychological stress and SCI, a study by Epel et al. (2018) specifically addressed the impact of chronic stress on cellular aging and inflammation. Individuals who reported higher levels of chronic stress exhibited both increased inflammation and shorter telomere length, a marker of cellular aging. Epel et al. suggested that chronic stress accelerates cellular aging processes, leading to a persistent state of low-grade inflammation through several mechanisms (Epel et al., 2018). When cells undergo cellular aging processes, such as telomere shortening, DNA damage, and mitochondrial dysfunction, the

byproducts trigger the activation of signaling pathways involved in the production and release of pro-inflammatory cytokines and chemokines. Additionally, cellular aging causes the cells to have diminished efficiency in performing their normal functions, leading to overproduction of ROS and accumulation of cellular debris. Accumulation of ROS and cellular waste act as DAMPs and trigger the activation of immune system. Lastly, cellular aging impairs the inflammatory response regulation, which leads to the dysregulated immune system and sustained low-grade inflammation (Epel et al., 2018).

Collectively, these studies help to establish a framework linking psychological stress and SCI. The chronic activation of stress responses, dysregulated release of stress hormone, and the subsequent activation of pro-inflammatory pathways collectively contribute to SCI. Moreover, the associations between adverse life events, psychological stress, and pro-inflammatory markers emphasize the importance of psychosocial factors in shaping the inflammatory state within the body.

### **Body Composition Measuring Techniques**

Body composition can provide valuable insights into nutritional status and the functional capacity of the body (Kuriyan, 2018). Researchers and practitioners have access to a range of tools and techniques for assessing body composition, each offering distinct advantages and limitations. The classification of body composition assessment methods is primarily based on the number of factors they measure, referred to as compartments. These include two-compartment (2C), three-compartment (3C), four-compartment (4C), or multi-compartment models, which progressively advance to provide more detailed information (Kuriyan, 2018). The 2C model figuratively divides body weight into fat mass (FM) and fat-free mass (FFM), assuming a stable proportion of components within the FFM across individuals (Santos et al., 2010). In the 3C

model, FFM includes lean tissue mass (LTM) and bone mineral content (BMC), obtained by further dividing FFM into total body water (TBW) and other solids (proteins and minerals). The 4C model, considered the most accurate, eliminates assumptions by providing detailed information on the same components as the 3C model (Kuriyan, 2018). However, due to high costs and limited accessibility, 4C models are typically reserved for research purposes (Santos et al., 2010).

A variety of techniques are commonly employed in practice and research settings to measure body composition, including bioimpedance analysis (BIA), quantitative magnetic resonance, air displacement plethysmography, dual-energy X-ray absorptiometry (DXA), magnetic resonance imaging (MRI), and nuclear magnetic resonance (NMR) spectroscopy, with the choice of technique depending on the specific imaging objectives. Among these techniques, DXA is a commonly employed 3C model that measures body fat, lean soft tissue (LST), and total body bone mineral (TBBM) using X-ray technology (Santos et al., 2010). DXA assumes distinguishable components—fat, bone, and lean soft tissue—based on their reaction to X-rays and assumes a constant hydration level of 73% for the lean soft tissue (Andreoli et al., 2009).

A study conducted by Achamrah et al. (2018) aimed to compare body composition assessment using DXA and BIA in relation to the body mass index (BMI) and evaluate their consistency at both population and individual levels. The study included subjects over 18 years old who did not have any acute diseases, malnutrition, obesity, or eating disorders. Whole-body DXA scans were performed to assess FM and FFM, while BIA was used as an alternative method. The researchers specifically sought to examine the agreement between these two methods according to BMI categories. The findings of the study revealed that BIA overestimated FFM in individuals with a BMI between  $18.5 \text{ kg/m}^2$  and  $40 \text{ kg/m}^2$ . However, when comparing

the means of the two methods at the population level, no significant differences were observed. This suggests that, at the population level, BIA and DXA may be used interchangeably for assessing body composition, however, at the individual level, inconsistencies exist, indicating that the two techniques may not provide consistent results for every individual.

DXA provides a direct and accurate measurement of body composition components, including fat mass, lean soft tissue, and bone mineral content (Santos et al., 2010). It allows for the precise identification and quantification of visceral fat, enabling a better understanding of its role in metabolic health. Therefore, when precision is crucial, particularly in clinical or research settings where accurate assessment of body composition is essential, DXA remains the preferred method.

### **Anthropometrics and SCI**

This subsection reviews a body of research investigating the association between systemic inflammation and various body composition parameters.

#### ***BMI***

The relationship between BMI and inflammation has been a subject of research interest due to its potential implications for understanding the link between obesity, SCI, and various health outcomes (Kim et al., 2020). This section examines three relevant studies that contribute to the understanding of the association between BMI and SCI.

Chen et al. (2015) conducted a comprehensive analysis to investigate the effects of BMI and serum inflammatory cytokine levels on asthma control in children with asthma. The subjects of the study were 116 children with asthma categorized into three groups based on their BMI categories: normal weight (BMI between 18.5 and 24.9), underweight (thin) (BMI less than 18.5), and obese (BMI of 30 or greater). It's important to note that BMI is a simple and widely

used measure of body fatness, but it does not take into account other factors such as muscle mass or distribution of fat, which may affect health outcomes. For a comprehensive assessment of an individual's health, it's essential to consider other measurements and factors in addition to BMI. The levels of IL-6, TNF- $\alpha$ , and hs-CRP were measured. Asthma control status was assessed using the Childhood Asthma Control Test (C-ACT) after a 4-week treatment period. The findings of this study revealed significant differences among the different BMI groups in relation to the serum pro-inflammatory cytokine levels and asthma control. The subjects in obesity group exhibited the highest levels of IL-6, hs-CRP, and TNF- $\alpha$ , followed by the thin group, and the normal group ( $P < 0.05$ ). Conversely, children in the normal BMI category showed the highest C-ACT score, indicating improved asthma control, followed by the thin group, and the obesity group ( $P < 0.05$ ). Lastly, negative correlations between the levels of IL-6, hs-CRP, TNF- $\alpha$ , and the C-ACT score were identified ( $P < 0.05$ ), while no significant correlations were found between BMI and the C-ACT score or the levels of IL-6, hs-CRP, and TNF- $\alpha$  ( $P > 0.05$ ). Results of the study suggested that both high and low BMI values can be associated with increased levels of serum inflammatory cytokines, which can negatively impact asthma control (Chen et al., 2015).

Building upon the findings of Chen et al. (2015), which investigated the effects of BMI and serum inflammatory cytokine levels on asthma control in children, the study conducted by Qin et al. (2015) explored the association between obesity and the risk of developing rheumatoid arthritis (RA) using a systematic review and dose-response meta-analysis methodologies. The researchers performed a literature search and identified 11 eligible citations that met the inclusion criteria of the study. The analysis revealed that the individuals whose BMI fell under the obese category had a significantly increased risk to develop RA compared to those with a BMI under 30 kg/m<sup>2</sup>. The pooled relative risks for RA were 1.31 for the obese category and 1.15

for the overweight category when compared to the normal weight subjects. Additionally, compared to individuals with a BMI under 30, obese individuals showed a relative risk of 1.25 for developing rheumatoid arthritis. The findings provided further evidence of the relationship between obesity and inflammatory conditions, suggesting that higher BMI values, particularly in the obese categories, were associated with an increased risk of developing the inflammatory disease, particularly RA (Qin et al., 2015).

Adding to this body of research, a recent study done by Gao et al. (2021) examined the relationship between the systemic inflammation index (SII), a marker that quantifies the degree of inflammation in the body by evaluating the neutrophil-to-lymphocyte ratio (NLR), and provides the inflammatory status of an individual, and BMI in a large cohort of patients with gastric cancer (Hu et al., 2020). The study included 2,542 patients who underwent radical surgery for gastric or gastroesophageal junction adenocarcinoma between the years 2009 and 2014. The results demonstrated that high systemic inflammation ( $NLR \geq 3$ ) and being underweight ( $BMI < 18.5 \text{ kg/m}^2$ ) independently predicted poorer overall survival and recurrence-free survival. However, the patients who had both high systemic inflammation status and a BMI in the underweight category showed significantly worse outcomes. The finding of this study suggest that considering the combined use of the SII and BMI may provide valuable prognostic information in patients with gastric cancer (Gao et al., 2021).

Collectively, the studies discussed in this section highlight the complex relationship between BMI, SCI, and a variety of health outcomes. The findings suggested that both high and low BMI values can be associated with increased levels of serum inflammatory markers, emphasizing the importance of considering inflammation as a potential mediator in the relationship between BMI and adverse health conditions. Together, the findings highlighted the

multifaceted nature of the association between BMI and SCI, however, further research is needed to identify the underlying mechanisms behind the latter association.

### ***Body Fat Distribution***

Different patterns of body fat distribution can have significant implications in risk for chronic disease (Neeland et al., 2012). There are two common patterns of fat distribution, which are referred to as android (central) and gynoid (peripheral). Android pattern of fat distribution refers to the accumulation of fat predominantly in the abdominal region, particularly in the visceral adipose tissue surrounding the organs, and is commonly observed in individuals with an apple-shaped body, with subcutaneous fat concentrated around the waistline (Vanderburgh, 1992). Android pattern of fat distribution has been strongly associated with an increased risk of cardiometabolic abnormalities, including insulin resistance, type 2 diabetes, dyslipidemia, and hypertension (Canoy et al., 2007). As reviewed in sections above, researchers agree that the excess visceral fat observed in people with android body fat distribution releases pro-inflammatory cytokines and adipokines, contributing to the SCI and causing disruption of metabolic homeostasis (Després et al., 2008).

On the other hand, gynoid fat distribution pattern refers to the peripheral storage of fat in the lower body, particularly in the hips and thighs and is often seen in individuals with a pear-shaped body (Neeland et al., 2012). Compared to android fat distribution, gynoid fat distribution was associated with a reduced risk of cardiometabolic complications (Tankó et al., 2003). The subcutaneous fat accumulated in the lower body was suggested to exhibit a protective effect by sequestering lipids away from critical organs and reducing the release of pro-inflammatory mediators (Yim et al., 2008).

Various imaging techniques such as dual-energy X-ray absorptiometry (DXA), magnetic resonance imaging (MRI), and computed tomography (CT) can provide detailed information about body fat distribution of an individual (Nana et al., 2012; Rosenquist et al., 2012). Additionally, anthropometric measures such as waist circumference (WC) and waist-to-hip ratio (WHR) can serve as markers of central obesity and provide insights into the fat distribution patterns (Ashwell et al., 2012).

In light of this, a meta-analysis conducted by Lee et al. in 2008 aimed to determine a simple index of overweight and obesity that would serve as the best discriminator for cardiovascular disease risk factors. The study conducted a comprehensive search of published literature, specifically focusing on studies that employed receiver-operating characteristics (ROC) curve analysis and reported the area under the ROC curves (AUC) for various overweight and obesity indices in relation to hypertension, T2DM, and dyslipidemia as risk factors. After evaluation of the literature, ten studies meeting the inclusion criteria were selected for the final analysis. The findings provided insights into the discriminatory abilities of different indices. Surprisingly, the BMI emerged as the poorest discriminator for cardiovascular risk factors. On the other hand, waist-to-height ratio (WHtR) demonstrated superior discriminatory power for hypertension, diabetes, and dyslipidemia in both males and females. When conducting the analysis to compare the pooled AUC values, WHtR consistently outperformed BMI. The results of this meta-analysis strongly suggest that WHtR provides a more accurate assessment of cardiovascular risk factors compared to BMI alone. That is, based on the statistical evidence presented, the researchers concluded that measures of centralized obesity, particularly WHtR, exhibited superiority over BMI in identifying cardiovascular risk factors in both men and women, which implied that WHtR may serve as a more reliable indicator of obesity-related

health risks than BMI. Overall, the study confirmed the limitations of relying solely on BMI as a measure of body composition and associated risk for disease (Lee et al., 2008). By emphasizing the adoption of alternative indices, such as WHtR, healthcare professionals can gain deeper insights into the complex relationship between obesity and cardiometabolic health outcomes. Therefore, considering body composition beyond BMI is crucial for a comprehensive understanding of health risks and outcomes.

In line with exploring the association between body fat distribution patterns with cardiometabolic disease risks, Hetherington-Rauth et al. (2018) conducted a study which aimed to evaluate the relationships between body fat distribution, total body adiposity, and cardiometabolic risk factors in a population of Hispanic girls. The researchers utilized dual-energy X-ray absorptiometry (DXA) to measure body fat stores in different regions of the body, including the gynoid (hips and thighs), android (abdomen), leg, and trunk regions, as well as the total body fat percent. Regression models were used to statistically evaluate the associations between these measures of body composition and metabolic parameters, which included insulin resistance, triglycerides (TG), low-density lipoprotein (LDL-C), and high-density lipoprotein (HDL-C) in the blood. The findings indicated that a higher proportion of fat distributed in the gynoid, and leg regions was associated with a lower cardiometabolic risk ( $p < 0.05$ ). This finding suggests that individuals who tend to accumulate more fat in the lower body, specifically in the hips and thighs, have a decreased risk to develop cardiometabolic abnormalities as compared to those individuals who tend to accumulate body fat in the upper body, particularly in the abdominal region (Hetherington-Rauth et al., 2018). The study supported the notion that body fat distribution, beyond overall adiposity and BMI, is an important determinant of metabolic health outcomes.

The studies collectively emphasize the significance of body fat distribution as a predictor of metabolic and cardiovascular health outcomes. While overall adiposity, as measured by total body fat percentage, remains an important factor, it is the distribution of fat in specific regions that appears to have a stronger association with cardiometabolic risks. The android distribution of fat showed to pose a greater risk for cardiometabolic conditions, while a higher proportion of fat in the gynoid and leg regions was associated with a lower risk of these complications.

While BMI provides a simple and easily accessible measure for the overall body weight in relation to height, it lacks the ability to differentiate between fat mass and lean mass, and fails to account for variations in fat composition and distribution throughout the body (Després & Lemieux, 2006). Consequently, individuals with similar BMI values may have different amounts and patterns of distributions of body fat, resulting in discrepancies in metabolic health risks (Neeland et al., 2012). Given these limitations, it becomes crucial to explore alternative indices that offer a more comprehensive understanding of body composition and its association with health outcomes. That is, by considering the specific body fat distribution patterns, healthcare professionals can obtain a more comprehensive assessment of an individual's cardiometabolic health and disease risks.

### ***Visceral Adipose Tissue (VAT)***

Visceral obesity, characterized by the accumulation of excess fat in the abdominal region, has been strongly associated with metabolic abnormalities and chronic inflammation. The presence of visceral fat, also known as intra-abdominal fat, has been recognized as a key contributor to the development of SCI due to its ability to produce substances that disrupt normal metabolic pathways (Jensen, 2008). Visceral obesity is considered a subtype of low-grade inflammation, as visceral adipose tissue secretes pro-inflammatory cytokines, which include

TNF-alpha, IL-6, and IL-1, as well as chemokines and other adipokines (Repaci et al., 2011). These substances act as mediators of systemic inflammation and contribute to the pathogenesis of cardiovascular and metabolic diseases (Repaci et al., 2011).

Adipose tissue, once regarded solely as a site for energy storage, is now recognized as an organ with both immune and endocrine properties. It is composed of various cell types, including adipocytes (fat cells), fibroblasts, endothelial cells, resident macrophages, stromal cells, and pre-adipocytes (Chait & den Hartigh, 2020). The secretion of cytokines, chemokines, and polypeptide hormones by adipose tissue, collectively known as adipokines, plays a crucial role in energy homeostasis, regulation of glucose and lipid metabolism, immunity, and the immunoendocrine system (Ahima & Lazar, 2008).

To understand the association between visceral obesity and SCI, researchers have conducted studies investigating the role of adipose tissue and the proinflammatory substances it secretes. The studies shed the light on the mechanisms underlying the link between visceral obesity and inflammation, as well as the potential implications for cardiometabolic health. Among these investigations, a study by Jung et al. (2016) sought to determine the measure of obesity that best correlates with the development of diabetes and prediabetes. In this cross-sectional study, 1603 subjects within the age range of 30 to 64 years were enrolled, and different measures of obesity were utilized, which included BMI, WC, waist-height ratio, WHtR, WHR, and visceral fat. DXA was employed to accurately quantify the visceral fat mass. The prevalence of diabetes and prediabetes was determined based on the criteria established in the American Diabetes Association 2015 guidelines. The findings of this study revealed an association between visceral fat mass and the risk of diabetes and prediabetes, outperforming other indices of adiposity. After adjusting for potential confounding factors, individuals whose visceral fat mass

fell into the upper 10th percentile displayed significantly higher odds ratios for diabetes and prediabetes, compared to those in the upper 10th percentile of other measurements of obesity. In both men and women, visceral fat mass demonstrated the highest area under the curve when correlated with diabetes and prediabetes, further highlighting its predictive capabilities (Jung et al., 2016).

The superiority of visceral fat mass as an indicator of metabolic conditions is attributed to its ability to differentiate between abdominal visceral and subcutaneous fat (Neeland et al., 2012). This distinction is crucial because visceral fat is known to be metabolically more active and releases a higher quantity of proinflammatory substances, contributing to SCI. By isolating the impact of visceral fat, this study offers valuable insights into the mechanisms linking visceral obesity and inflammation, thus underscoring the importance of considering body fat distribution beyond overall adiposity in the context of cardiometabolic health. Overall, Jung et al. add to the growing body of evidence supporting the role of visceral fat as a key player in the interplay between obesity and SCI. The findings emphasize the significance of assessing visceral fat mass using precise methods such as DXA to identify individuals at higher risk of diabetes and prediabetes. By deepening the understanding of the link between visceral fat and inflammation, this research contributes to the development of targeted interventions and lifestyle modifications aimed at mitigating the detrimental effects of SCI on cardiometabolic health.

### **Introduction to Diet Inflammatory Index (DII) and Associations between DII and Health Outcomes**

The Diet Inflammatory Index (DII) was initially developed in 2009 with the aim of providing a reliable tool to categorize individuals' diets on a scale from the most anti-inflammatory to the most pro-inflammatory (Shivappa et al., 2014). The development of the

original DII involved an extensive literature search that incorporated in-vitro cell culture studies, as well as animal and epidemiological studies, to examine the effect of diet on inflammation. To create the original DII, a comprehensive screening process was conducted on articles published through the year of 2007. A total of 929 articles were selected, evaluated, and scored to create the index. The scoring algorithm considered a range of factors, including micronutrients, macronutrients, and bioactive components such as flavonoids, spices, and teas (Shivappa et al., 2014). This comprehensive approach allowed for a more accurate estimation of the inflammatory potential of individual diets.

Since its original development, the DII has evolved to incorporate additional research, refine its scoring system, and to improve its accuracy and applicability. Numerous studies have validated the DII by assessing its association with inflammatory markers and chronic diseases, such as cardiovascular disease, cancer, and metabolic disorders. These validations have demonstrated the utility of the DII in assessing the inflammatory potential of an individual's diet and its implications for health outcomes (Wirth et al., 2017). In addition, DII was adapted for use in specific populations, such as adolescents, pregnant women, and older adults, considering their unique dietary patterns and physiological factors (Shivappa et al., 2014). Lastly, the DII has been expanded to include more comprehensive dietary assessment methods, including food frequency questionnaires and 24-hour dietary recalls, to capture a broader range of dietary intake (Tabung et al., 2016). According to the updated DII scale, the maximally pro-inflammatory diet had a score of +7.98, while the maximally anti-inflammatory diet had a DII score of -8.87. The median score on the DII scale was +0.23, reflecting a range of dietary patterns from pro-inflammatory to anti-inflammatory (Shivappa et al., 2014).

Several tools and resources are available to calculate the DII score, including comprehensive databases of food composition and their corresponding inflammatory scores, enabling researchers and practitioners to easily calculate an individual's DII score based on their dietary intake (Shivappa et al., 2014). The DII calculation tools facilitate the inclusion of the DII in large-scale studies and clinical settings, promoting its widespread application and accessibility.

The DII holds great promise for future research and practical applications. As more evidence accumulates linking diet-induced inflammation to various health outcomes, the DII can serve as a valuable tool for researchers, clinicians, and individuals in understanding the inflammatory potential of different dietary patterns and individual nutrients. The DII can help identify dietary patterns that promote a pro-inflammatory state and those that are anti-inflammatory, aiding in the development of targeted interventions and personalized dietary recommendations (Shivappa et al., 2014).

While the DII has shown promise, certain limitations and challenges associated with its application can be identified. One limitation is the reliance on self-reported dietary data, which can introduce recall bias and measurement error (Subar et al., 2015). Additionally, the DII is based on the population-level data, and individual variations in nutrient metabolism and genetic factors may not be fully captured. Lastly, the DII does not consider the overall quality of the diet, such as nutrient density and adherence of individuals to dietary guidelines, which are important factors in determining health outcomes.

The DII has been widely applied in both observational and intervention studies to examine the association between diet and inflammation-related outcomes. Observational studies have utilized the DII to investigate the relationship between dietary inflammation and the risk of

chronic diseases, providing valuable insights into the role of diet in the development and progression of inflammatory conditions. For example, in the cross-sectional study conducted by Shivappa et al. (2015), the researchers aimed to explore the associations between DII, and inflammatory markers in a generally healthy population of 2524 subjects aged 35 to 55 years. To assess the relationship between diet and inflammation, the researchers calculated the DII based on dietary information obtained from a food frequency questionnaire (FFQ) and tested it against the inflammatory markers, including CRP, IL-6, homocysteine, and fibrinogen. After adjusting for confounding factors, including energy intake, age, sex, BMI, smoking status, education level, medication use, blood pressure, and physical activity, multivariable logistic regression analyses revealed significant positive associations between the DII and the two inflammatory markers: IL-6 ( $>1.6$  pg/ml) and homocysteine ( $>15$   $\mu$ mol/l). These findings suggest that individuals with higher DII scores, which indicate a more pro-inflammatory diet, were more likely to have elevated levels of IL-6 and homocysteine, which are the markers of inflammation (Shivappa et al., 2015). While no significant correlations were observed between the DII, CRP, and fibrinogen in this study, it is important to note that this lack of associations with does not negate the overall significance of DII in predicting inflammation. Rather, this finding highlights the complexity of the relationship between diet and inflammation and indicates that certain inflammatory markers, such as IL-6 and homocysteine, in case of this study, may be more sensitive to dietary influences than others, however, further research is necessary.

Another study done by Almeida-de-Souza et al. (2018) focused on assessing whether the DII could predict SCI in adolescents. The sample consisted of 329 adolescents aged 12-18 years. The DII was calculated based on FFQ. Blood samples were collected to measure inflammatory biomarkers, CRP, IL-6, complement components 3 (C3), and 4 (C4), as well as an overall

inflammatory biomarker score. The results of the study indicated that the DII score was positively associated with IL-6 and C4 levels in the fully adjusted model, even after accounting for biological and lifestyle variables. The findings suggested that a more pro-inflammatory diet, as indicated by higher DII scores, was linked to elevated IL-6 and C4 levels, indicating a potential state of SCI in these adolescents, however, similarly to the study by Shivappa et al. (2014) no significant association were observed between the DII and CRP levels. A strong positive association between the DII score and the overall inflammatory biomarker score was observed in the current study, which suggests that the DII may be a useful tool in assessing the inflammatory potential of adolescents' diets comprehensively.

Research by Sokol et al. (2016) explored the associations between the dietary inflammatory index, waist-to-hip ratio, and metabolic syndrome. In this study, 55-item FFQ was used to derive DII This study utilized cross-sectional data from the Polish-Norwegian Study (PONS) (n = 3862). Participants were 45 – 64 years old. Questionnaires on sociodemographic, personal, and family health histories, and behavioral habits were completed. Cholesterol, HDL cholesterol, and triglycerides were measured in fasting blood samples. Measurements of hip and waist circumference, blood pressure, height, spirometry, and weight were obtained, and metabolic syndrome was confirmed. Participants completed a 55-item FFQ from which DII scores were calculated per 1000 calories consumed to account for any differences in energy intake between the participants. It was found that participants with higher DII scores had greater diastolic blood pressure and waist-to-hip ratio, however no statistically significant increased risks for metabolic syndrome were observed in the 4<sup>th</sup> DII quartile, which included the participants with the most pro-inflammatory diets. Additionally, women with a higher DII score had a decrease in metabolic syndrome risk, which contradicted the researchers' expected

outcomes. The authors suggested that the reasons for the unexpected associations between DII and metabolic syndrome in this study were the possible changes that the participants made in their diets based on the recommendations of the doctors. However, there may not have been enough time for any biological changes to occur, thus there was a mismatch between the DII scores and some of the health outcomes associated with metabolic syndrome. Because of the cross-sectional nature of the study and the use of self-reported data, the conclusion may be limited, however, the findings indicated the associations between DII and some of the components of metabolic syndrome, such as increased waist-to-hip ratio (WHR) values – a measure of intra-abdominal obesity, which is factor strongly associated with inflammation.

Another study exploring the associations between the DII and metabolic syndrome was performed by Ren et al. (2018). In addition to the metabolic syndrome parameters, the study evaluated CRP levels as a sign of inflammatory status in the participants (Ren et al., 2018). Adding this variable allows for an additional level of evidence to confirm inflammatory state that could potentially be associated with the inflammatory potential of the diet. There were 1712 participants with an average age of 50 years old from eight different Chinese cities. Participants were instructed to complete questionnaires to obtain sociodemographic information and health history. Similar to research by Sokol et al. (2016) metabolic syndrome was identified by anthropometric measurements and fasting blood samples such as blood glucose and lipid profiles. A 24-hour recall was used to calculate DII scores. The study suggested a close association between CRP levels and metabolic syndrome, while the association between the DII and metabolic syndrome was limited with the only parameter associated with DII being blood pressure. It is possible that the results were inconsistent due to some residual confounders that were not accounted for, such medications and family history, as well as the differences in times

of 24-hour recall collections or the numbers of food parameters evaluated for DII. Overall, while the results on the relationships between CRP and parameters of metabolic syndrome as well as between CRP and DII agreed with other research in the area, the association between DII and metabolic syndrome in this study was inconclusive.

Further examining the literature on application of DII, the study conducted by Phillips et al. (2018) aimed to determine whether higher DII scores were associated with unfavorable cardiometabolic health profiles, which were characterized by elevated markers of inflammation, lipoprotein metabolism, glucose homeostasis and increased risk of developing metabolic syndrome (Phillips et al., 2018). There were a total of 1992 participants in the age group of 50-69 years old from the Cork and Kerry Diabetes and Heart Disease Study, whose data was used in the phase II for analyses. The participants completed General Health Questionnaire (GHQ), the International Physical Activity Questionnaire (IPAQ), and the FFQ. Out of all participants, subjects with CVD and type II diabetes were identified. To assess the inflammatory potential of the diets, energy-adjusted DII (E-DII) scores were used, in which 26 out of 45 parameters were identified in the FFQ. Fasting plasma glucose (FPG), serum total HDL cholesterol (HDL-C), LDL cholesterol (LDL-C) and triglyceride (TAG) concentrations were measured in the blood samples. In addition, average VLDL, LDL and HDL particle diameters were measured in serum specimens by nuclear magnetic resonance (NMR) spectroscopy. It was found that higher E-DII scores were associated with pro-atherogenic lipoprotein profiles characterized by increased numbers of VLDL, LDL, and HDL. High E-DII scores were also associated with higher pro-inflammatory cytokines, higher WBC counts and neutrophil to lymphocyte ratio (NLR), and lower adiponectin levels. Adiponectin is a hormone secreted by adipose tissue and is known for its anti-inflammatory and insulin-sensitizing properties. It plays a crucial role in regulating

glucose and lipid metabolism and is associated with improved cardiovascular health and reduced risk of obesity-related diseases, making it a relevant biomarker for assessing metabolic health and disease risk (Gavrila et al., 2003). This study demonstrated the risk for metabolic syndrome was significantly increased ( $p < 0.001$ ) among the subjects with higher E-DII scores.

Recent research has demonstrated that the application of the DII extends to diverse population groups beyond white adults, shedding light on its utility in understanding the impact of dietary choices on inflammation across various ethnicities and age groups. For example, the study conducted by Canto-Osario et al. (2020) examined the relationship between the DII and the development of metabolic syndrome, and the secondary purpose of evaluating the relationship between DII with specific components of the metabolic syndrome in Mexican adults (Canto-Osorio et al., 2020). Data from the longitudinal Health Workers Cohort Study (HWCS) was used. Original study included 10,729 participants enrolled between 2004 and 2006, and the current study selected total of 399 individuals for the final analyses. Weight, height, waist circumference, and blood pressure were measured. Fasting blood glucose, TGs, and HDL cholesterol were measured in the blood samples. Diet was assessed with 116-item semi-quantitative FFQ, in which 25 out of 35 DII parameters were captured. The highest DII scores were associated with hypertriglyceridemia, hypertension, and abdominal obesity, while a highly pro-inflammatory diet was associated with metabolic syndrome, hypertension, abdominal obesity, and hypertriglyceridemia. These data suggest a positive association between highest quartile of DII and the incidence of metabolic syndrome in Mexican American Adults and are consistent with other research (Phillips et al., 2018; Ren et al., 2018).

Adolescence is a critical period of growth and development, and understanding the impact of diet on inflammation during this stage can have long-term implications for health

outcomes in adulthood (Singh et al., 2008). By highlighting the potential role of diet in promoting SCI in adolescents, study by Almeida-de-Souza et al. (2018) aimed to investigate the association between DII and SCI in adolescents. A total of 329 adolescents aged 12 to 18 years were included in the study. The DII score was calculated based on a food-frequency questionnaire and categorized into tertiles to assess the diet inflammatory potential. Blood samples were collected to measure inflammatory biomarkers, including CRP, IL-6, complement component 3 (C3), and complement component 4 (C4). An overall inflammatory biomarker score was calculated. The results of the study showed that higher DII scores were positively associated with IL-6 levels, indicating a potential link between a more pro-inflammatory diet and increased IL-6 levels (crude model: OR = 1.88, 95% CI: 1.09-3.24,  $p = 0.011$ ; fully adjusted model: OR = 3.38, 95% CI: 1.24-9.20,  $p = 0.023$ ). Similarly, DII score was positively associated with C4 levels when fully adjusted (OR = 3.12, 95% CI: 1.21-8.10,  $p = 0.016$ ). However, DII score was negatively associated with C3 levels in the crude model when comparing the first tertile with the second tertile, but not with the third tertile, and no significant associations were observed in the fully adjusted model, although a trend was found (OR = 1.71, 95% CI: 0.63-4.66,  $p = 0.044$ ). No significant associations were observed between DII score and CRP levels (crude model: OR = 1.09, 95% CI: 0.53-2.23,  $p = 0.880$ ; fully adjusted model: OR = 1.55, 95% CI: 0.58-4.14,  $p = 0.392$ ). The most notable finding was the positive association between the DII score and the overall inflammatory biomarker score, indicating that a higher DII score was linked to a greater overall inflammatory response in the adolescent participants (fully adjusted model: OR = 5.61, 95% CI: 2.00-15.78,  $p = 0.002$ ).

However, it is important to note that this study's cross-sectional design limits the ability to establish causality between diet and inflammation. Future longitudinal studies are needed to

further elucidate the temporal relationship between diet and inflammatory biomarkers in adolescents. Additionally, potential confounding factors not accounted for in the fully adjusted model may influence the observed associations. Despite these limitations, this study provides a foundation for further research on the relationship between diet and inflammation in adolescents and highlights the importance of considering dietary choices in the context of inflammatory processes in this population. The use of DII as a tool to measure the diet's inflammatory potential offers a promising approach to understand the impact of dietary choices on inflammatory processes in this specific age group. The findings of this study underscore the importance of early dietary interventions to mitigate chronic inflammation and reduce the risk of related metabolic and cardiovascular health conditions later in life. Overall, the study contributed evidence to support the association between DII and SCI in adolescents

Study conducted by Carvalho et al. (2019) also selected young adult participants of ages 23 – 25 years old to investigate the association between the inflammatory potential of diet and insulin resistance or metabolic syndrome. A 75-item FFQ was filled out by the participants, and the results were evaluated using the 35 out of 45 DII parameters. Laboratory testing included fasting glucose and fasting insulin. It was found that while the diets of the young adult participants had very high pro-inflammatory potential, no associations between the DII and either insulin resistance or metabolic syndrome were determined. Even though there were no associations between the variables established in this study, this population-based study with a large sample size provided a strong base for future research by showing that the diet of young adults has high DII when compared to other studies. Diets with high pro-inflammatory potential are associated with poor cardiovascular and metabolic health outcomes as determined in previous research (Phillips et al., 2018), therefore, the population of young adults in this study have an

increased risk for adverse health outcomes. Based on this knowledge, future studies can be conducted in which confounding variables are controlled for, and/or a longitudinal design is used to potentially establish causal relationships between DII and the parameters of metabolic syndrome.

Overall, the studies by Almeida-de-Souza et al. (2018) and Carvalho et al. (2019) highlighted the association between diet, as quantified by DII and SCI in adolescents and young adults, emphasizing the importance of early interventions to promote anti-inflammatory dietary patterns and mitigate potential long-term health risks associated with chronic inflammation. This research contributes to our understanding of the role of diet in shaping the inflammatory state during adolescence, which can have potential implications for long-term health outcomes. As the transitional life period of first-year college students is explored, understanding the potential effects of diet on SCI becomes crucial for promoting health and well-being in this specific population.

### **Transitional Life Period of the First-Year College Students**

The transitional period of first-year college students, otherwise known as the "college transition," is a crucial period in a person's life. During the transition a student undergoes a series of significant change in their social, emotional, and academic lives. This period can be both challenging and stressful, as it usually involves adapting to new environments, managing academic demands, and establishing new social networks. Additionally, college transition can have an impact on a student's health and wellbeing, which, hindered by the other, seemingly more important life events, can be overlooked by a young adult (Lund et al., 2010).

One of the significant challenges faced by first-year college students is learning to effectively manage stress (Zivin et al., 2009). Academic pressure, social expectations, financial

constraints, sleep deprivation, and many other factors associated with the life of a college student can cause stress and anxiety. Research studies have provided alarming statistics about stress and anxiety in college students. For instance, a national survey in the United States indicated that approximately 62.0% of students reported feeling "overwhelming anxiety" during the past year (American College Health Association, 2019). Another systematic review conducted in 2013 reported that the prevalence of depression in university students ranged from 9% to 54.3%, with an overall pooled prevalence estimate of 30% (Ibrahim et al., 2013). An earlier study conducted by Stallman (2010) compared university students to the general population and found that 51% of students experienced high or very high levels of psychological distress, compared to 22% of the general population. Lastly, a study examining symptoms of depression, anxiety, and stress in college students reported that 30.4% experienced symptoms of depression, 36.4% reported symptoms of anxiety, and 35.8% reported symptoms of stress (Beiter et al., 2015). These statistics highlight the prevalence of stress and anxiety among college students and highlight the need to address effective stress management strategies and mental health support services among college student population.

In addition to stress, poor diet habits can also play a role in exacerbating chronic inflammation in college students. During the college transition period, students may also face challenges to maintain healthy eating habits, even if they are accustomed to eating a healthy diet prior to starting college (Quick et al., 2014). A combination of factors associated with beginning a college life may have a contribution to the negative changes in a student's diet. Researchers identified various influences that contribute to unhealthy eating behaviors during this transitional period (Nelson et al., 2008). One significant factor is the high availability and accessibility of unhealthy food options on college campuses, including fast food outlets, vending machines with

processed snacks, and food delivery services available through the night. Additionally, the stress and time constraints faced by college students can lead to reliance on convenience foods and irregular, potentially less nutritious eating patterns. Social factors, such as peer influence and communal eating environments, may also play a role in shaping dietary choices, along with limited nutrition knowledge and lack of cooking skills may hinder students from preparing healthy meals (Nelson et al., 2008). Furthermore, the newfound independence and freedom associated with college life can result in unstructured eating habits, including skipping meals or snacking excessively. Many college students have limited time and resources to cook healthy meals and often rely on fast food and processed snacks, leading to weight gain and increased risk of developing chronic conditions associated with metabolic dysfunctions further in life (Nelson et al., 2008).

Diets high in processed and refined foods, sugary beverages, and unhealthy fats, which are common components of the typical college student diet (Holm-Denoma et al., 2008), have been associated with increased inflammation (Almeida-de-Souza et al., 2018). These types of foods can lead to oxidative stress, which is an imbalance between the production of harmful free radicals and the body's ability to neutralize them. As noted in previous sections focused on inflammation, oxidative stress can trigger inflammation and damage cells, tissues, and organs, leading to chronic inflammation. Furthermore, diets high in processed and refined foods are typically low in important nutrients, such as antioxidants, fiber, and phytochemicals, which have anti-inflammatory properties. Such nutrients are abundant in plant-based foods, such as fruits, vegetables, whole grains, nuts, and seeds, which are often lacking in the diets of college students who rely on fast food, processed snacks, and sugary beverages due to limited time, resources, and knowledge about healthy eating (Greene et al., 2011).

### *Perceived Barriers to “Healthy” Eating in College Students*

The cross-sectional study by Graham et al. (2013) aimed to identify factors influencing fruit and vegetable (FV) intake among young adults, particularly college students. Data from an online survey involving 1201 college students in Minnesota were analyzed using factor analysis. The analysis revealed seven factors related to FV behaviors of students, which included personal barriers, FV knowledge, family, friends, neighborhood, access barriers, and campus-related factors. Next, linear regression analyses showed that perceived personal barriers such as the lack of cooking skills were negatively associated with FV purchasing, preparation, and consumption. On the other hand, positive perceptions of family and friends eating healthfully and easy access to FV in the neighborhood were linked to higher FV intake. The study highlighted the significance of individual, social, and environmental factors in shaping FV behaviors among young adults and emphasized the need to consider these factors together to design effective interventions.

Overall, the results of this study provided insights into the factors influencing FV behaviors in college students. By understanding the perceived barriers and facilitators of FV intake, interventions can be tailored to address these factors effectively. Targeting both individual-level barriers, such as cooking skills, and social and environmental factors, like family support and neighborhood access, can contribute to promoting healthier eating habits in college students. Future efforts to improve FV behaviors among young adults should consider a comprehensive approach that accounts for the multifaceted influences on their dietary choices.

The combination of chronic stress and poor diet habits can create a vicious cycle that exacerbates chronic inflammation in college students. Chronic stress can disrupt healthy eating habits and lead to increased consumption of unhealthy foods, which in turn can further trigger

inflammation. Inflammation can disrupt the normal functioning of the immune system and the brain, leading to increased stress levels and further disruption of healthy eating habits, creating a cascade of events that perpetuate chronic inflammation (Dantzer et al., 2008). In the long run, this cycle of chronic stress and poor diet habits can have detrimental effects on body composition and overall health in college students.

### **Summary**

Based on the literature review conducted, several key trends have emerged concerning body composition, SCI, and the inflammatory potential of the diet, as well as the implications of these factors for chronic conditions, particularly cardiovascular and metabolic diseases. SCI is shown to play a crucial role in the development and progression of various health outcomes, underscoring the significance of understanding its underlying mechanisms. Moreover, the studies identified that individuals whose dietary patterns fall in the highest DII percentile are at an increased risk of developing chronic conditions commonly associated with increased levels of systemic inflammation, such as cardiovascular disease, diabetes, and metabolic syndrome.

A notable aspect identified in the literature review was the importance of considering body composition, particularly fat distribution, as a more informative predictor of chronic inflammation and related health risks as compared to the conventional BMI. Studies consistently demonstrated that individuals with higher levels of android fat, which is associated with increased levels of visceral fat, have elevated pro-inflammatory markers, which correlates with a greater susceptibility to adverse health outcomes, including cardiometabolic diseases.

Despite the extensive research on inflammation and obesity, a notable gap was identified when it comes to studying young and healthy adults, such as college students. While the relationship between DII, inflammation, body composition, and associated health outcomes was

explored in older populations and individuals with existing medical conditions, there still is a lack of investigation in the college student demographic. Addressing this gap is essential to assess how dietary patterns, inflammation, and body composition interplay among young and healthy adults and the potential implications for long-term health outcomes. Therefore the purpose of the present study was as follows:

1. To examine the relationship between DII scores and BMI in first-year university students.
2. To explore the correlation between visceral fat and waist circumference in first-year university students.
3. To assess the association between DII scores and waist circumference in first-year university students.
4. To determine the impact of higher DII scores on body fat percentage in first-year university students.
5. To investigate the link between DII scores and body fat distribution parameters (e.g., total fat mass, legs fat, trunk fat) measured by DXA in first-year university students.
6. To assess the relationship between DII scores and lean body mass parameters (e.g., total lean mass, legs lean mass, trunk lean mass) measured by DXA in first-year university students.
7. To assess the strength of the association between VAT and WC.
8. To explore gender differences in the relationship between DII scores and body composition measures among first-year university students.

Thus, the main objective of the present study was to determine whether there is a positive correlation between the estimated DII values and BMI, waist circumference, body fat percent, and visceral fat of the students. The second objective of this study was to assess the strength of

the association between the amount of visceral fat measured by DXA scan and manually measured WC. The aim was to determine whether WC can effectively serve as an outcome variable to reflect diet modifications related to the inflammatory potential of the diet. By evaluating the strength of this relationship, the aim was to establish whether changes in WC can reliably indicate alterations in visceral fat, providing valuable insights into the potential use of WC as an accessible marker for monitoring the effects of diet on visceral fat accumulation and its inflammatory implications. Lastly, the third objective the investigation aimed to assess the potential differential impact of DII on body composition in males and females, thereby providing valuable insights into the interplay of diet and gender-related variations in body fat distribution among first-year university students.

It was hypothesized that a positive correlation would exist between DII scores and BMI, waist circumference, body fat percent, and visceral fat levels. Additionally, the study sought to assess the strength of the association between the amount of visceral fat measured by DXA and waist circumference, aiming to determine whether waist circumference could effectively serve as an outcome variable for DII-related diet modifications. Lastly, in exploring gender-related differences in body composition, the study aimed to identify potential differential impacts of DII on body fat distribution between male and female students. As such, the hypotheses in this regard were exploratory in nature. The findings from this research provide valuable insights into the interplay of diet and gender-related variations in body fat distribution among first-year university students.

### **Chapter III: Methodology**

The purpose of this study was to investigate the association between DII and anthropometric parameters of university students without medical conditions while controlling for other confounding variables.

In this section, subject recruitment, selection, and exclusion, subject description, data collection methods and procedures, data analysis, as well as strength and limitations of the methodology will be addressed.

#### **Factors Influencing Body Composition in College Students Longitudinal Study**

Factors Influencing Body Composition in College Students is an ongoing study with a longitudinal design at the Kinesiology, Health, Food, and Nutritional Sciences Department of the University of Wisconsin – Stout (UW-Stout). For this study, new subjects were recruited from the population of incoming first-year students across all majors of study at the University in the beginning of each Fall Semester. Participants were enrolled on the voluntarily basis and the participant pool size is dependent on the interest of the student population to participate in the study. Participants are invited to continue their participation each year they are enrolled at the university. A projected end result of the data collection is the four complete sets of data for each participant. Currently, there are two complete four-year data sets which includes participants enrolled in the Fall of 2018 as freshmen who completed all four years of the study with the last participation being in the Fall of 2021, and, likewise, participants of the Fall 2019 – Fall 2022 cohort.

#### **Subject Recruitment**

Prior to initiation of the subject recruitment, approval for the study was obtained from the Institutional Review Board at UW-Stout (see Appendix A). The new participant recruitment process for this study involved both e-mail invitations and in-person recruitments in

collaboration with professors who teach introductory level classes across the campus. With the permission of instructors or professors, in-person classes were visited to invite first year students to participate in the study. Interested students were asked to provide their contact information to the researcher. All participants provided informed consent, which was sent to them prior to their enrollment in the study and explained in person before data collection. For the present study, data collected from first-year participants from each previous year of the longitudinal research were included.

### **Data Collection Methods**

Participant data were collected using anthropometric measurements such as weight, height, BMI, hip, and waist circumference taken before DXA scans. Qualtrics Surveys and a FFQ were used to gather data on demographics, eating attitudes, food security, and dietary habits. Three-day dietary recalls were collected and entered into ASA24 for nutrient analysis. The DII was calculated using nutrient data, involving Z-score transformations and percentile conversions, resulting in individual DII scores.

### ***Anthropometrics and DXA Scans***

Anthropometric measurements were collected from participants during the DXA appointments, prior to conducting the scans. Using standard procedures, weight was measured to the nearest 0.1 kg using a Health-o-Meter mechanical balance beam scale, and height was measured to the nearest 0.1 cm using a Perspective Enterprises wall-mounted stadiometer. BMI values were calculated manually. Hip circumference was measured at the greatest circumference around the hips and buttocks and waist circumference was measured at the top of the iliac crest. Both were measured using a standard tape measure and to the nearest 0.1 cm. After the manual anthropometric measurements were collected, DXA procedure and associated risks were

explained to the subjects, and the DXA scans were performed by the trained faculty of the Kinesiology, Health, Food, and Nutritional Sciences Department. The scan results were not provided to the students unless all four scans have been completed to control for the influence of the students' scan perception on any future changes in their body compositions.

### ***Qualtrics Surveys and Food Frequency Questionnaires***

Upon being recruited as subjects of the study and providing the consent to participate (See Appendix B), the students were asked to complete the Qualtrics Surveys and Food Frequency Questionnaires prior to coming in to the DXA appointment. The individual links to surveys along with subject IDs were provided to the participants.

The self-administered electronic surveys aimed to collect the data related to participants' demographics, dietary habits, eating attitudes, food security, and food intake. The first part of the Qualtrics survey included a brief demographic questionnaire that encompassed questions about their age, year in college, major, ethnicity/race, gender, financial status, and living arrangement. This survey was designed to gather basic information about the participants. The next portion of the Qualtrics Survey included Eating Attitudes Test-26 and the ORTO questionnaire to evaluate the participants' risks for eating disorders. To determine the participants' food security status and any potential barriers to obtaining safe and healthy foods, the U.S. Food Security Module was used.

FFQ was used to assess participants' eating habits over the course of a year. This comprehensive survey aimed to capture their dietary patterns and food choices. Participants completed the FFQ online accessing it via the link provided.

### ***Three-Day Dietary Recalls and ASA-24 Entry***

The process of entering a 3-day dietary recall into ASA24 (Automated Self-Administered 24-Hour Recall) involved the following steps:

1. The three-day dietary recalls from the participating students were collected as part of the research study.
2. The ASA24 software was accessed to input the dietary recalls and obtain nutrient content. The necessary credentials were used to log in to the software.
3. Each participant was assigned a unique ASA24 ID, which was separate from their overall study ID. This ID ensured that the data entered into ASA24 could be accurately matched with its respective participant. All participant IDs, including the ASA24 ID and the corresponding study ID, were tracked and recorded in a master Excel sheet.
4. The details of the 3-day dietary recall were manually entered into ASA24. The food items, portion sizes, and additional details were entered following the prompts and instructions provided by the software.
5. The appropriate food items from the ASA24 database were selected to match the reported foods. In cases where specific items were not available, suitable alternatives were chosen or estimations were made based on similar foods or ingredients. Serving guides and reference materials were used to estimate portion sizes accurately.
6. Throughout the process, the participant's ASA24 ID was cross-referenced with the master Excel sheet to ensure that the data being entered matched the correct participant. This step helped maintain the integrity of the data and ensured that it was correctly associated with the respective participant.

7. ASA24 automatically calculated the nutrient content based on the entered data, providing nutrient values such as calories, macronutrients, vitamins, minerals, and other relevant nutrients.
8. The calculated nutrient values were used to analyze the dietary intake of the participants. 3-day averages of the various nutrients were calculated to understand the average nutrient intake over the specified period.

It should be noted that while ASA24 provided nutrient estimates based on the entered data, some limitations and potential inaccuracies in nutrient estimation may exist. However, following the standardized process and utilizing the available resources within ASA24 helped to minimize errors and provide valuable insights into the nutrient content of the three-day dietary recalls.

### ***DII Calculation***

Calculation of DII was based on the dietary intake data obtained from three-day food recalls of the study participants and processed using the ASA24 software to determine average content for each nutrient component in each participant's diet. The process employed to compute DII scores for the participants is detailed as follows:

1. The mean and standard deviation values were used as multipliers to express an individual's "exposure" to a nutrient relative to the 'standard global mean' as a Z-score. This step was done by subtracting the standard mean value from the reported amount and dividing by its standard deviation. Mean and standard deviation values for all forty-five parameters (35 were used in this study) were obtained from the table of food parameters included in the DII, inflammatory effect scores, and intake values from the global composite dataset provided by Shivappa et al. (2014).
2. To minimize the effect of 'right skewing', the Z-score was converted to a percentile score.

3. To achieve a symmetrical distribution with values centered on 0 and bounded between 21 (maximally anti-inflammatory) and 11 (maximally pro-inflammatory), each percentile score was doubled and then '1' was subtracted.
4. The centered percentile value for each food parameter for each participant was then multiplied by its respective 'overall food parameter-specific inflammatory effect score' to obtain the 'food parameter-specific DII score'.
5. All of the 'food parameter-specific DII scores' for each participant were summed to create the 'overall DII score' for each individual participant.

Values used to calculate DII scores are presented in Table 1. The steps for calculation of DII scores indicated above were used to create an Excel formula to automatize the calculation of DII scores for all participants. The obtained DII scores were added as a new variable into the master SPSS data sheet.

**Table 1***Food Parameters Used to Calculate DII*

Food parameter	Overall inflammatory effect score	Global daily mean intake (units/d)	SD
Alcohol (g)	-0.278	13.98	3.72
Vitamin B12 (µg)	0.106	5.15	2.70
Vitamin B6 (mg)	-0.365	1.47	0.74
β-Carotene (µg)	0.584	3718	1720
Caffeine (g)	-0.110	8.05	6.67
Carbohydrate (g)	0.097	272.2	40.0
Cholesterol (mg)	0.110	279.4	51.2
Energy (kcal)	0.180	2056	338
Total fat (g)	0.298	71.4	19.4
Fiber (g)	-0.663	18.8	4.9
Folic acid (µg)	-0.190	273	70.7
Fe (mg)	0.032	13.35	3.71
Mg (mg)	-0.484	310.1	139.4
MUFA (g)	-0.009	27.0	6.1
Niacin (mg)	-0.246	25.90	11.77
n-3 Fatty acids (g)	-0.436	1.06	1.06
n-6 Fatty acids (g)	-0.159	10.80	7.50
Protein (g)	0.021	79.4	13.9
PUFA (g)	-0.337	13.88	3.76
Riboflavin (mg)	-0.068	1.70	0.79
Saturated fat (g)	0.373	28.6	8.0
Se (µg)	-0.191	67.0	25.1
Thiamin (mg)	-0.098	1.70	0.66
Vitamin A (RE)	-0.401	983.9	518.6
Vitamin C (mg)	-0.424	118.2	43.46
Vitamin D (µg)	-0.446	6.26	2.21
Vitamin E (mg)	-0.419	8.73	1.49
Zn (mg)	-0.313	9.84	2.19
Anthocyanidins (mg)	-0.131	18.05	21.14

*Note.* DII scores range from -8.87 (maximally anti-inflammatory diet) to +7.98 (maximally pro-inflammatory diet), with a median score of +0.23 (Shivappa et.al, 2014).

## **Data Analysis**

To address the research questions posed to investigate the relationships between DII scores and body composition measures in first-year university students, a comprehensive data analysis plan was implemented.

### ***Demographics, Anthropometrics, and Dietary Characteristics of First-Year College Students***

Descriptive statistics were used to obtain essential characteristics of the study population, including demographics, anthropometrics, and dietary intake characteristics. Demographics, such as gender, age, race, and academic major, were summarized using frequencies and percentages to provide a comprehensive overview of the participants' background. Anthropometric measurements, including weight, BMI, waist circumference, hip circumference, total body fat percentage, legs lean mass, trunk lean mass, legs fat mass, and trunk fat mass, were analyzed using descriptive statistics, such as mean and standard deviation, to understand the central tendencies and variability in these variables. Lastly, dietary characteristics, which included average macronutrient intakes and average DII scores, were also obtained using descriptive statistics.

### ***Associations between DII and Anthropometric Parameters***

To examine the relationships between DII scores and body composition parameters, Pearson's correlation tests were performed. Correlation analysis was used to explore the linear associations between DII scores and various anthropometric measures, such as BMI, waist circumference (WC), total body fat percentage, visceral fat mass, visceral fat volume, body fat components (android, gynoid, legs, and trunk tissue percent fat), and lean mass components in the study subjects. Additionally, the correlation matrix tables (Tables 5-7) presented the correlation

coefficients (Pearson's  $r$ ) and p-values for each pair of variables, providing a comprehensive overview of the associations between DII scores and the body composition parameters.

#### ***Association between WC and VAT***

Pearson's correlation analysis was conducted to investigate the association between WC and VAT parameters, specifically VAT Fat Mass and VAT Volume. Pearson's correlation allowed to assess the strength and direction of linear relationships between the two continuous. In this study, the correlation analysis aimed to determine if there was a significant relationship between WC and the amount and volume of visceral fat in the study participants.

#### **Gender Influence on the Relationship between DII and Body Composition Parameters**

The statistical analysis process for addressing the last objective of the study involved a series of tests to examine the relationship between gender, DII, and body composition measures.

To begin, an independent samples t-test was conducted to compare DII scores and the total fat percent between male and female subjects. Next, an analysis of covariance (ANCOVA) was performed to investigate the potential interaction between DII and gender on total fat percent, a measure of body composition. To further explore this interaction effect, linear regression analyses were conducted. The linear regression aimed to examine the relationship between DII and total fat percent while controlling for gender as a confounding variable. Lastly, partial correlation analysis was performed to separate the direct relationship between DII and body composition from the influence of gender.

Throughout the process of data analysis, appropriate statistical tests and models were applied based on the nature of the variables, data distribution, and the specific research questions. Statistical significance was set at  $p < 0.05$ , and all analyses were conducted using Statistical Package for the Social Sciences (SPSS), version 28.0.1.1 (14).

## **Chapter IV: Results**

The primary purpose of this study was to investigate the association between DII scores and body composition measures in first-year university students. The DII scoring system, a comprehensive tool that assesses the inflammatory potential of an individual's diet, was used to quantify the inflammatory status of the participants' diets. Understanding the relationship between DII scores and body composition is important as it can provide insights into the potential impact of dietary inflammation on body composition changes which, in turn, can suggest overall health outcomes. By examining anthropometric parameters such as BMI, body fat percentage, waist circumference, and visceral fat levels, the aim was to determine whether higher DII scores were associated with unfavorable body composition profiles in the population of first-year college students. The findings from this study have the potential to contribute to the existing knowledge on the interplay between dietary inflammatory potential and body composition, shedding light on the impact of dietary choices on the health and well-being of college students.

### **Demographics**

Data from a total of 75 first-year students collected from the year 2018 through 2022 was analyzed in this study. There were 40 female, 34 male, and one participant who identified as a gender status of "other". The age range of the participants was 17 – 22 years, with the mean age being 18.5 years old ( $SD = 0.9$ ). The majority of the participant identified with White/non-Hispanic race (81.1%), with 18.9% of the sample identifying as Mexican-American/Hispanic, Asian, Hmong, Bi/Multi-racial, or another race. Students of non-dietetics major contributed to 81.3% of the sample, and 18.7% of the sample were dietetics students. Approximately equal

numbers of students were unemployed and working part-time, with the percentages being 50.7 and 49.3, respectively.

**Table 2**

*Demographics of the Study Subjects*

Characteristic	Frequency	%	M	SD
Female	40	53.3	-	-
Male	34	45.3	-	-
Other	1	1.3	-	-
<b>Age (y)</b>	75	-	18.5	0.9
<b>Race</b>	74	-	-	-
White/Non-Hispanic	60	81.1	-	-
Other (Mexican-American/Hispanic Asian Hmong Bi/Multi-racial)	14	18.9	-	-
<b>College Major</b>	75	-	-	-
Dietetics	14	18.7	-	-
Other	61	81.3	-	-
<b>Employment Status</b>	75	-	-	-
Not working	38	50.7	-	-
Working part-time	37	49.3	-	-

*Note.* n = 75.

**Anthropometrics of First-Year College Students**

Anthropometric characteristics of the sample of first-year college students are outlined in Table 3. The average weight and BMI of the participants was 67.9 kg (SD = 9.1) and 23.6 kg/m<sup>2</sup> (SD = 4.2), respectively. Mean waist circumference was 80.8 cm (SD = 11.3), whereas hip circumference was 99.0 cm (SD = 8.6).

Total body fat percentage of the subjects was 28.0 % (SD = 9.1). Average legs lean mass was 15.6 lbs (SD = 6.1), and the trunk lean mass was 18.8 lbs (SD = 10.4), whereas the average legs fat mass of the participants was 15.6 lbs (SD = 6.1) and the trunk fat mass was 18.8 lbs (SD

= 10.4), which constituted 31.0 % (SD = 9.3) and 27.9 % (SD = 10.6) of the total mass, respectively. The android/gynoid (A/G) ratio, which represents the distribution of fat between the android and gynoid regions and is determined as the ratio of android total mass to gynoid total mass, averaged at 0.8 (SD = 0.3).

It is important to note that DXA provides two different measurements related VAT: VAT fat mass and VAT volume. While VAT fat mass quantifies the actual amount of fat stored in the visceral area, VAT volume measures the three-dimensional size or space occupied by the visceral fat. Both measures are valuable in understanding the distribution and impact of visceral fat on health outcomes. VAT fat mass provides information on the absolute amount of fat present, which is crucial for assessing obesity-related health risks. On the other hand, VAT volume offers insights into the spatial distribution and potential mechanical effects of visceral fat on surrounding organs and (Pouliot et al., 1994). Mean visceral fat mass of the sample was 0.5 lbs (SD = 0.7), and visceral fat volume averaged around 15.2 (SD = 21.3).

Overall, these health characteristics provide an overview of the body composition and anthropometric measurements of the sample of first-year college students. Understanding these baseline characteristics is important for further investigation into the relationships between DII scores, body composition measures, and health outcomes in this population.

**Table 3**

*Descriptive Statistics of Anthropometric Characteristics and DXA parameters of First-Year College Students*

Characteristic	M	SD
BMI (kg/m <sup>2</sup> )	23.6	4.2
Weight (kg)	67.9	13.9
WC (cm)	80.8	11.3
HC (cm)	99.0	8.6
Body Fat Percent (%)	28.0	9.1
Total Lean Mass (lbs)	103.0	23.2
Total Fat Mass (lbs)	41.2	17.7
Legs Lean Mass (lbs)	35.1	8.4
Trunk Lean Mass (lbs)	48.8	10.4
Legs Fat Mass (lbs)	15.6	6.1
Trunk Fat Mass (lbs)	18.8	10.4
Legs Fat Percent (%)	31.0	9.3
Trunk Fat Percent (%)	27.9	10.6
A/G Ratio	0.8	0.3
VAT Fat Mass (lbs)	0.5	0.7
VAT Volume (in <sup>3</sup> )	15.2	21.3

*Note.* n = 75.

## Dietary Characteristics of First-Year College Students

Descriptive statistics of dietary intake of key nutrition components among first-year college students are presented in Table 4. In terms of daily caloric intake (kcal), students on average consumed  $2090 \pm 911$  kcal, as measured by three-day dietary logs that were averaged and analyzed using ASA24 software. Regarding daily protein intake, male students consumed on average 114 g of protein from both plant and animal sources per day (SD = 56 g), whereas female students reported mean daily protein intake of 65 g (SD = 31 g).

Looking at the daily total fat intake, male participants consumed on average 101 g, (SD = 49 g), whereas female participants reported a daily total fat intake of 72 g (SD = 26 g). Further breaking down the fat intake, male students consumed on average 36 g of saturated fat (SD = 18 g), which corresponds to approximately 1.45% (SD = 0.73%) of their daily calories coming from saturated fat; whereas female students had a mean saturated fat intake of 25 g (SD = 11 g), which translates to around 1.43% (SD = 0.63%) of their daily calories being derived from saturated fat. Lastly, male students on average consumed 34 g of monounsaturated fat (SD = 17 g), and 21 g of polyunsaturated fat (SD = 12 g), whereas female students consumed 24 g (SD = 10 g), and 17 g (SD = 7 g) respectively. Lastly, male students had a mean carbohydrate intake of 286 g (SD = 117 g), and female students reported consuming 207 g of carbohydrates per day (SD = 78 g).

The DII scores in the present study ranged from -31.22 to +12.49, with a mean DII score of -2.79 (SD = 8.68). There was a significant difference in DII scores between males and females, with males having a more anti-inflammatory diet (mean DII = -5.99, SD = 8.65) compared to females (mean DII = -0.06, SD = 7.66) ( $t = 3.10$ ,  $p = 0.003$ ). A more positive DII score indicates a pro-inflammatory diet, meaning that the diet is likely to promote inflammation, while a negative DII score suggests an anti-inflammatory diet, which is associated with reduced

inflammation. Therefore, the higher DII scores indicate a more pro-inflammatory dietary pattern, while lower DII scores suggest an anti-inflammatory dietary pattern among the study participants.

These descriptive statistics provided insights into the dietary intake of first-year college students, with regards to caloric intake, macronutrient intake, as well as DII of the diets consumed by male and female participants.

**Table 4**

*Daily Dietary Intake of Key Nutrition Components of First-Year College Students*

Characteristic	M	SD
Calorie (kcal)	2090	912
Male	2490	1002
Female	1747	679
Protein (g)	87	50
Male	114	56
Female	65	31
Total Fat (g)	85	40
Male	101	49
Female	72	26
Carbohydrate (g)	244	104
Male	286	117
Female	207	78
Saturated Fat (g)	30	16
Male	36	18
Female	25	11
Monounsaturated Fat (g)	28	14
Male	34	17
Female	24	10
Polyunsaturated Fat (g)	19	10
Male	21	12
Female	17	7
DII	-2.79	8.68
Male	-5.99	8.65
Female	-0.06	7.66

*Note.* M and SD represent mean and standard deviation respectively.

### **Associations between DII and Anthropometrics Parameters**

To address the research questions one through five and determine if there was a relationship between anthropometrics and DII scores, a series of correlations were performed. The correlation matrix tables 5 through 7 reveal relationships or lack of thereof between DII scores, BMI, WC, total body fat percentage, visceral fat mass and volume, body fat components, and muscle mass in first-year university students.

First, the results revealed that there were no significant linear relationships between DII and either BMI ( $r = -0.02$ ,  $p = 0.988$ ) or WC ( $r = -0.02$ ,  $p = 0.988$ ) in the study population. These findings indicate that the DII was not significantly associated with BMI or WC in the participants.

In contrast, a strong positive correlation was observed between DII and total tissue fat percent, indicating that higher DII scores were associated with increased body fat percentage ( $r = 0.47$ ,  $p < .001$ ). Additionally, android percent fat ( $r = 0.40$ ,  $p < .001$ ), gynoid percent fat ( $r = 0.46$ ,  $p < .001$ ), legs tissue percent fat ( $r = 0.46$ ,  $p < .001$ ), and trunk tissue percent fat ( $r = 0.45$ ,  $p < .001$ ) showed significant positive correlations with DII. Lastly, DII scores showed significant positive correlations with VAT Fat Mass ( $r = 0.10$ ,  $p = 0.030$ ) and VAT Volume ( $r = 0.10$ ,  $p = 0.030$ ). Altogether these findings suggest that a more pro-inflammatory diet, as reflected by higher DII scores are associated with increased body fat percent as well as increased overall adiposity of the participants.

Lastly, the results revealed significant correlations between DII and lean mass parameters. Specifically, there was a strong negative correlation between DII scores and total lean mass ( $r = -0.46$ ,  $p < .001$ ), suggesting that higher DII scores were associated with lower total lean mass in the study participants. Similarly, DII scores showed significant negative correlations with both

trunk lean mass ( $r = -0.58$ ,  $p < .001$ ) and legs lean mass ( $r = -0.54$ ,  $p < .001$ ), indicating that a more pro-inflammatory diet was associated with lower trunk and legs lean mass.

Overall, the correlation analysis provides initial evidence of a potential link between dietary inflammatory potential, as measured by the DII, and both fat and lean body mass parameters in first-year college students, where DII is positively associated with body fat parameters, and negatively associated with lean mass parameters.

**Table 5**

*Correlation Matrix of DII and Anthropometric Parameters in First-Year College Students*

		DII	BMI	WC	VAT Fat Mass	VAT Volume
DII	Pearson's r	-				
	p-value	-				
BMI	Pearson's r	-0.11	-			
	p-value	0.350	-			
WC	Pearson's r	-0.02	0.80	-		
	p-value	0.880	<.001	-		
VAT Fat Mass	Pearson's r	0.10	0.79	0.75	-	
	p-value	0.419	<.001	<.001	-	
VAT Volume	Pearson's r	0.10	0.80	0.75	1.00	-
	p-value	0.418	<.001	<.001	<.001	-

*Note.* The p-value represents the probability of obtaining results as extreme as the observed results, assuming the null hypothesis is true. Significance level of  $p < 0.05$  was used.

**Table 6***Correlation Matrix of DII and Body Fat Percent Parameters in First-Year College Students*

		DII	Total Fat %	Android Fat %	Gynoid Fat %	Legs Fat %	Trunk Fat %
DII	Pearson's r	-					
	p-value	-					
Total Fat %	Pearson's r	0.47	-				
	p-value	<.001	-				
Android Fat %	Pearson's r	0.40	0.92	-			
	p-value	<.001	<.001	-			
Gynoid Fat %	Pearson's r	0.46	0.92	0.78	-		
	p-value	<.001	<.001	<.001	-		
Legs Fat %	Pearson's r	0.46	0.93	0.76	0.96	-	
	p-value	<.001	<.001	<.001	<.001	-	
Trunk Fat %	Pearson's r	0.45	0.96	0.99	0.83	0.82	-
	p-value	<.001	<.001	<.001	<.001	<.001	-

Note. The p-value represents the probability of obtaining results as extreme as the observed

results, assuming the null hypothesis is true. Significance level of  $p < 0.05$  was used.

**Table 7***Correlation Matrix of DII and Lean Body Mass Parameters in First-Year College Students*

		DII	Total Lean Mass	Android Lean Mass	Trunk Lean Mass	Legs Lean Mass
DII	Pearson's r	-				
	p-value	-				
Total Lean Mass	Pearson's r	-0.46	-			
	p-value	<.001	-			
Android Lean Mass	Pearson's r	-0.47	0.96	-		
	p-value	<.001	<.001	-		
Trunk Lean Mass	Pearson's r	-0.46	0.98	0.98	-	
	p-value	<.001	<.001	<.001	-	
Legs Lean Mass	Pearson's r	-0.43	0.98	0.92	0.93	-
	p-value	<.001	<.001	<.001	<.001	-

Note. The p-value (p) represents the probability of obtaining results as extreme as the observed

results, assuming the null hypothesis is true. Significance level of  $p < 0.05$  was used.

### Association between WC and VAT

To address the second primary objective of the study aimed to determine the strength of the association between WC and VAT, Pearson's correlation analysis was performed. The correlation matrix displayed the associations between WC, VAT Fat Mass, VAT Volume in the study population (Table 8). The correlation coefficient between WC and VAT Fat Mass was found to be strong and positive ( $r = 0.75$ ,  $p < .001$ ), indicating that as waist circumference increases, there is a significant tendency for VAT Fat Mass to increase as well. Similarly, WC showed a strong positive correlation with VAT Volume ( $r = 0.75$ ,  $p < .001$ ), indicating that as waist circumference increases, there is a significant tendency for VAT Volume to increase. Moreover, VAT Fat Mass and VAT Volume exhibited a perfect positive correlation ( $r = 1.00$ ,  $p < .001$ ), signifying that these two variables vary in direct proportion, increasing or decreasing together. These findings highlight a meaningful relationship between WC and visceral adipose tissue characteristics in our study participants, suggesting that changes in WC are associated with corresponding changes in both VAT Fat Mass and VAT Volume.

**Table 8**

*Correlation Matrix between WC and VAT Parameters*

		WC	VAT Fat Mass	VAT Volume
WC	Pearson's r	-		
	p-value	-		
VAT Fat Mass	Pearson's r	0.75	-	
	p-value	<.001	-	
VAT Volume	Pearson's r	0.75	1.00	-
	p-value	<.001	<.001	-

*Note.* The p-value represents the probability of obtaining results as extreme as the observed results, assuming the null hypothesis is true. Significance level of  $p < 0.05$  was used.

### **Gender Influence on the Relationship between DII and Body Composition**

Addressing the last objective of the study, a series of statistical analyses were conducted. First, independent samples t-test was conducted to examine the relationship between gender and both the Dietary Inflammatory Index (DII) and Total Percent Fat. One participant was excluded due to their gender being categorized as "other," resulting in a sample size with two gender levels (male and female).

As previously reported, the results revealed a statistically significant difference in DII scores between males ( $M = -5.99$ ,  $SD = 8.65$ ) and females ( $M = -0.06$ ,  $SD = 7.66$ ) ( $t = 3.10$ ,  $df = 72.00$ ,  $p = 0.003$ ). On average, females had significantly higher DII scores compared to males, suggesting females eat more inflammatory diet. Similarly, the t-test showed a significant difference in total fat percentage between females ( $M = 33.0$ ,  $SD = 7.6$ ) and males ( $M = 22.1$ ,  $SD = 7.1$ ) ( $t = 6.32$ ,  $df = 72.00$ ,  $p < .001$ ). Thus, as expected, females had significantly higher total percent fat compared to males.

To answer the question whether the relationship between DII and body composition, particularly body fat percent, differs between the genders, an ANCOVA was conducted to examine the potential interaction between the DII and gender on total percent fat, a measure of body composition. The results revealed a significant interaction effect between DII and gender on total fat percent,  $F(1, 71) = 27.24$ ,  $p < .001$ . This significant interaction indicates that the relationship between DII and body fat percent differs between genders.

To further explore the nature and direction of this interaction effect, post-hoc analyses in form of linear regressions were performed. The linear regression analysis aimed to examine the relationship between the DII and total fat percent while controlling for gender as a potential confounding variable. The model demonstrated a reasonable fit to the data ( $R = 0.66$ ), and

approximately 44% of the variance in total fat percent could be accounted for by DII and gender. After controlling for gender, DII showed a positive and significant association with the total fat percent ( $\beta = 0.31$ ,  $SE = 0.10$ ,  $t = 3.15$ ,  $p = 0.002$ ), suggesting that for each one-unit increase in DII, there is an associated increase of approximately 0.31 units in body fat percent, after controlling for gender and other potential confounding variables. Additionally, gender was found to be significantly related to the total fat percent. Specifically, males had lower total percent fat ( $\beta = -8.95$ ,  $SE = 1.72$ ,  $t = -5.22$ ,  $p < 0.001$ ) compared to females, suggesting that being male is associated with a decrease of approximately 8.95 units in fat percentage compared to being female, after adjusting for DII and other potential confounding variables. Overall, the results indicate that while both DII and gender significantly contribute to the prediction of total fat percent, in terms of the relative magnitude of the coefficients in this analysis, gender appears to have a stronger influence on total body fat percentage than DII.

Lastly, to separate the direct relationship between the DII and the body composition variables from any influence that gender might have on this relationship, partial correlation analysis was conducted. The correlation coefficients between DII and anthropometric parameters while controlling for the influence of gender were computed and the results were presented in Table 9. The results revealed that there was a significant positive correlation between DII and total fat percent ( $r = 0.35$ ,  $p = 0.002$ ), suggesting that a more pro-inflammatory diet was associated with higher levels of total body fat, even after accounting for gender. Additionally, there was a significant positive correlation between DII and trunk tissue fat percent ( $r = 0.36$ ,  $p = 0.002$ ) and legs tissue fat percent ( $r = 0.33$ ,  $p = 0.004$ ). However, there were no significant correlations between DII and BMI ( $r = -0.07$ ,  $p = 0.551$ ) or VAT Fat Mass ( $r = 0.11$ ,  $p = 0.337$ ) after controlling for gender.

Overall, the partial correlation results suggest that the association between DII and body composition, particularly total body fat, trunk tissue fat, and legs tissue fat, remains significant even when considering the potential confounding effect of gender. However, DII does not show a significant relationship with BMI or VAT Fat Mass once gender is taken into account.

**Table 9**

*Partial Correlation Matrix between DII and Anthropometrics*

		DII	BMI	VAT Fat Mass	Total Fat %	Trunk Fat %	Legs Fat %
DII	Pearson's r	-					
	p-value	-					
BMI	Pearson's r	-0.07	-				
	p-value	0.551	-				
VAT Fat Mass	Pearson's r	0.11	0.80	-			
	p-value	0.337	<.001	-			
Total Fat %	Pearson's r	0.35	0.67	0.70	-		
	p-value	0.002	<.001	<.001	-		
Trunk Fat %	Pearson's r	0.36	0.70	0.76	0.96	-	
	p-value	0.002	<.001	<.001	<.001	-	
Legs Fat %	Pearson's r	0.33	0.48	0.49	0.91	0.82	-
	p-value	0.004	<.001	<.001	<.001	<.001	-

*Note.* Controlling for gender.

The p-value (p) represents the probability of obtaining results as extreme as the observed results, assuming the null hypothesis is true. Significance level of  $p < 0.05$  was used.

## Chapter V: Discussion and Conclusions

The present study had three main objectives:

- Determine the correlation between DII values and BMI, waist circumference, body fat percent, and visceral fat in university students.
- Assess the strength of the relationship between visceral fat measured by DXA and manually measured waist circumference, investigating whether waist circumference can serve as an outcome variable for diet-related modifications.
- Explore potential gender-related differences in the impact of DII on body composition among first-year university students. In addressing these objectives, a total of eight research questions were addressed.

The population sample consisted of 75 first-year college students, with the age range of 17 to 22 years, and the majority identified as White/non-Hispanic. The average weight and BMI of the participants fell within the normal range. Notably, the average total body fat percentage for both males and females was relatively high ( $M = 28.0$ ,  $SD = 9.1$ ), indicating a potential concern for excess adiposity among the study participants. Body fat percentage can vary widely among individuals based on various factors, including age, gender, and fitness level (Ortega et al., 2016). For males, a healthy body fat percentage typically ranges from 10% to 20%, while for females, it usually falls within the range of 20% to 30% (Kuriyan, 2018). However, these ranges can be influenced by individual differences and may not be applicable to all populations (Kuriyan, 2018).

The DXA scan allowed for the examination of VAT characteristics, distinguishing between VAT fat mass and VAT volume, which are both critical for understanding the health implications of visceral fat accumulation. Three-day diet records filled out by the participants

revealed variations in daily caloric intake, protein, fat, and carbohydrate consumption between male and female students. Additionally, the participants' DII scores showed a wide range, with female students having significantly higher DII scores ( $M = -0.06$ ,  $SD = 7.66$ ) compared to male students ( $M = -5.99$ ,  $SD = 8.65$ ), indicating a more pro-inflammatory dietary patterns in females, compared to the male subjects. While this observation was consistent with some previous research, it also introduced discrepancies with other studies. For instance, a study conducted by DiNatale et al. (2023) reported similar results, where significantly more females were found in the higher quartiles of DII scores, indicating a more pro-inflammatory diet, compared to males, supporting the results of the present investigation (DiNatale et al., 2023). However, contrasting findings were reported by Strath et al. (2022), where the distribution of DII scores exhibited no significant gender differences. The study investigated the relationship between DII and movement-evoked pain severity in individuals with chronic low back pain and, among other finding, observed no significant differences in DII scores between males and females or between non-Hispanic Black and non-Hispanic White participants. Surprisingly, while no differences in DII between the genders was observed, the gender was found to significantly modify the relationship between DII and movement-evoked pain severity, with movement-evoked pain severity being significantly impacted by DII scores in females but not males (Strath et al., 2022).

The main findings of the present study provided several insights into the relationship between DII, anthropometric parameters, and gender among first-year college students. Firstly, the results revealed that there were no significant linear relationships between the DII and either BMI or WC. This observation was consistent with some previous studies (Ashwell et al., 2012; Corrêa et al., 2022; Lee et al., 2008) that suggest that BMI may not always be the most relevant indicator of the body's condition, especially when considering the impact of dietary

inflammation. These results highlight the complexity of the relationship between diet, body composition, and anthropometric measurements, and they remind of the need for a comprehensive understanding of various factors that influence the body fat distribution.

On the other hand, a strong positive correlation was observed between DII and total tissue fat percent, as well as other body fat components such as android, gynoid, legs, and trunk tissue fat. These findings indicate that a more pro-inflammatory diet is associated with increased body fat percentage and overall adiposity in the study participants. These results were consistent with existing literature (Ren et al., 2018; Sokol et al., 2016) that suggested a potential link between DII and body adiposity. It is important to note that the accumulation of excess body fat, particularly visceral fat, has been linked to adverse cardiometabolic health outcomes (Hetherington-Rauth et al., 2018; Neeland et al., 2012), therefore, the significant positive correlations between DII and the body adiposity highlight the potential role of dietary inflammation in influencing adiposity and its implications for health.

Interestingly, the study also revealed significant negative correlations between DII scores and lean mass parameters, which included total lean mass, trunk lean mass, and legs lean mass. These findings suggest that a more pro-inflammatory diet is associated with lower lean muscle mass in the study participants. This observation is consistent with previous research (Gojanovic et al., 2021) that has indicated a potential negative impact of dietary inflammation on muscle mass. Maintaining lean muscle mass is crucial for overall health, as it plays a vital role in metabolic health and physical function (Wolfe, 2006). The negative correlations between DII and lean mass parameters suggest the importance of considering not only body fat but also lean muscle mass when assessing the impact of DII on body composition.

Additionally, the study aimed to investigate the significant relationship between WC and VAT among first-year university students and demonstrated a strong positive correlation between WC and both VAT Fat Mass and VAT Volume, indicating that as WC increases, there is a significant tendency for the visceral fat to increase as well. VAT accumulation has been associated with adverse health outcomes, making it a strong indicator of metabolic health (Jung et al., 2016). The findings of the present study suggested that measuring WC may serve as a practical and accessible method to assess visceral fat levels and potentially identify individuals at risk for obesity-related health issues. This finding was consistent with some investigations but not with others, depending on the specifics of the populations participating in the studies. For example, while the present study, which focused on first-year university students, observed a significant relationship between WC and VAT, indicating that WC can be used as a method to assess VAT levels in this specific population, other studies examining diverse racial backgrounds and populations showed variations in the WC-VAT relationship (Sumner et al., 2012).

For instance, a study on premenopausal women from various racial backgrounds found that while the relationship between WC and BMI did not differ significantly by race, the WC-VAT relationship varied significantly between black and white women (Sumner et al., 2012). In this study, data from five different cohorts of women were combined, including white South Africans, African-Americans, black South Africans, West Africans, and black Africans living in the United States. The researchers measured BMI, WC, and VAT in these women and compared the WC-BMI and WC-VAT relationships across racial groups. The results showed that while BMI and WC did not differ significantly between blacks and whites, there were substantial differences in VAT levels. Specifically, blacks had lower VAT ( $64 \pm 42 \text{ cm}^2$ ) compared to whites ( $101 \pm 59 \text{ cm}^2$ ), despite having a higher BMI ( $29.6 \pm 7.6 \text{ kg/m}^2$  vs.  $27.6 \pm 6.6 \text{ kg/m}^2$ ) ( $P <$

0.001). Furthermore, the study revealed that the WC-VAT relationship differed significantly between the two racial groups. The relationship between WC and VAT in blacks ( $\beta$  (s.e.) WC = 1.38 (0.11)) was notably different from that in whites ( $\beta$  (s.e.) WC = 3.18 (0.21)) ( $P < 0.001$ ). This observation suggested that white women had a greater increase in VAT per unit increase in WC compared to black women, suggesting potential racial-specific differences in how WC reflects VAT levels (Sumner et al., 2011).

The discrepancies in the association between WC and VAT observed by Sumner et al. may be attributed to various factors, such as genetic differences, lifestyle, dietary patterns, and body composition variations among different populations (Raji et al., 2001). Differences in body fat distribution patterns and VAT accumulation have been well-documented among racial and ethnic groups, which could contribute to the observed variations in the relationship between WC and VAT in different studies (Raji et al., 2001).

Despite the discrepancies, the present study described the relationship between WC and VAT within the specific population of first-year college students. The finding reinforced the need to consider WC as more than just a marker of general body fat but also as an indicator of VAT accumulation in this age group, which can be particularly relevant given the increased risk of obesity-related health issues during early adulthood (Singh et al., 2008) and the relatively high average total body fat percentage of the present study sample ( $M = 28.0$ ,  $SD = 9.1$ ). Moreover, the racial differences observed in the WC-VAT relationship in diverse populations underscore the importance of considering ethnic and racial diversity when interpreting anthropometric measurements for health risk assessments (Sumner et al., 2011).

Adding to the complexity of the observed correlations, the study revealed that DII scores showed significant positive correlations with both VAT Fat Mass and VAT Volume, but did not

correlate significantly with WC. The discrepancy in the correlations between DII and WC compared to DII and VAT suggests that the relationship between the DII and the distribution of body fat may not be consistent across different adipose tissue depots throughout the body. Interestingly that while VAT, a specific measure of adiposity with known health implications, is associated with DII, the general indicator of dietary inflammation, the same relationship is not evident between the DII and WC, the method of VAT levels assessment. One possible explanation for this discrepancy is that other factors, such as the levels of physical activity and stress, as well as individual metabolic responses to specific dietary components, could contribute to the variability in WC and overshadow the impact of dietary inflammation on this measure. These results emphasize the need for further research to better understand the complex interplay between dietary inflammation and body fat distribution and the potential influence of other confounding variables.

Lastly, a series of statistical analyses were conducted to explore potential gender-related differences in the impact of DII on body composition among the study participants. First, a significant interaction effect was revealed between DII and gender on total body fat percent. The subsequent linear regression analysis indicated that while both DII and gender significantly contributed to the prediction of total fat percent, gender appeared to have a stronger influence on the total body fat percentage than DII. The findings indicate that both DII and gender play significant roles in influencing body fat distribution in response to dietary inflammation. The significant interaction effect between DII and gender on total body fat percentage highlights the importance of considering both factors when assessing body composition.

Various gender-related factors, such as hormonal differences, different metabolic rates, and differences in body composition, are the established contributors to variations in body fat

distribution between males and females (Lovejoy et al., 2008). The influence of sex hormones, for instance, particularly estrogen and testosterone, plays a significant role in shaping body fat distribution and the response to inflammation (Palmer, & Clegg, 2015). That is, estrogen has been associated with differences in fat accumulation patterns in women, especially during reproductive life stages. On the other hand, testosterone affects body fat distribution patterns in men (Palmer, & Clegg, 2015). Additionally, sex hormones can influence inflammatory responses, potentially contributing to sex-specific variations in how the body responds to dietary inflammatory stimuli (Connelly et al., 2021). Mechanistically, sex-specific differences in adipose tissue physiology may underlie variations in the relationship between dietary inflammation and body composition (Karastergiou et al., 2012). For example, adipose tissue in women and men differs in terms of cellular composition, gene expression, and lipid metabolism (Arner, 2005), which could impact inflammation and fat distribution patterns. Lastly, sex-specific differences in immune cell populations, cytokine profiles, and immune response pathways may influence the link between diet-induced inflammation and body fat distribution (Giefing-Kröll, 2015).

The factors addressed above can potentially explain the stronger influence of gender on total body fat percentage observed in the study. It is expected that gender-related differences in body composition would be a major contributor to overall adiposity (Lovejoy et al., 2008). However, in the present study, there was a significant independent influence of DII on body fat percentage, beyond the impact of gender-related factors, the finding which suggests that dietary inflammation has a substantial impact on body adiposity, irrespective of gender. It indicates that the quality and inflammatory potential of the diet can be one of the determinants of body fat distribution and overall adiposity. Considering the complexity of body fat regulation (Guyenet & Schwartz, 2012), it is not surprising that both gender-related factors and dietary inflammation

contribute to the variation in body fat percentage. The present study highlights the importance of understanding the role of dietary inflammation in shaping body composition, specifically in the context of gender differences. It emphasizes that while gender-related factors are significant, dietary patterns and inflammation should also be taken into account when assessing and managing body adiposity.

Despite the limited explicit research on gender-related factors in context of body fat distribution and DII, with one possible explanation for the scarcity of literature being that the research on sex differences and gender-related factors in health outcomes is a relatively recent area of investigation (Tannenbaum et al., 2016), several studies discussed the importance of considering sex-specific factors in health and disease (Mauvais-Jarvis et al., 2020; Regitz-Zagrosek, 2012). Overall, investigating sex differences in the context of body composition and inflammation may lead to more personalized interventions and a deeper understanding of metabolic health.

### **Limitations**

The study has several limitations that should be considered when interpreting the findings. They are:

1. The study used a cross-sectional design, which limits the ability to establish causal relationships between DII scores and body composition measures.
2. The present study focused solely on the first-year enrollment of participants, providing a snapshot of their dietary habits and body composition during this specific period. As such, the long-term effects and changes in DII scores and body composition over subsequent years were not captured.

3. The study relied on self-reported dietary information to calculate DII scores, which may introduce recall bias and inaccuracies in dietary assessment.
4. The study's sample size was dependent on voluntary participation of the students, which could introduce selection bias.
5. The DII, while a widely used index, is still a surrogate measure for actual dietary intake. It is based on self-reported dietary data, and some aspects of the diet that influence inflammation might not be fully captured by this index.

### **Further Directions**

The findings of this study provide a foundation for further research in several areas related to the association between inflammation and body composition in university students. Future investigations can build upon these findings to deepen our understanding and address the limitations of the current study.

Firstly, carrying out a longitudinal study will allow for the examination of changes in DII scores and body composition measures over time. By continuing to collect data at multiple time points over the four years of students' college enrollment for the *Factors Influencing Body Composition in College Students Longitudinal Study*, researchers can investigate the trajectory of dietary inflammatory potential and its impact on body composition changes.

Additionally, interventional trials can be conducted to explore the causal relationships between dietary inflammatory potential and changes in body composition. Randomized controlled trials could be designed to assess the effects of dietary interventions aimed at reducing inflammation on body composition outcomes. Such interventions may involve promoting anti-inflammatory dietary patterns and evaluating their impact on body fat percentage, body fat distribution, and other body composition measures.

Incorporating biochemical markers of inflammation, such as CRPs, interleukins, and adipokines, could provide a more comprehensive assessment of the students' inflammatory status. By measuring these biomarkers alongside obtaining DII scores and body composition measures, researchers can establish a direct link between dietary inflammatory potential, systemic inflammation, and changes in body composition.

Extending the research to include diverse college student populations, by potentially collaborating with other colleges in the area, would enhance the generalizability of the findings. Considering factors such as ethnicity, socioeconomic status, and cultural background can provide a more comprehensive understanding of how DII scores and body composition measures interact across different student groups.

Lastly, further research could investigate additional factors beyond the gender that may influence the association between DII scores and body composition measures in college students. Factors such as physical activity levels, sleep quality, stress levels, and psychosocial factors could be examined to better understand their interactions with DII, body composition, and health outcomes.

## **Conclusion**

In conclusion, the study provided insights into the complex relationship between dietary inflammation, body composition, and gender-related factors among first-year university students. The results revealed significant correlations between the DII and total body fat percentage, as well as various body fat components, suggesting that a more pro-inflammatory diet is associated with increased adiposity. Furthermore, the study observed significant gender-related differences in body fat distribution, which can be attributed to established contributors such as hormonal differences, different metabolic rates, and variations in body composition between males and

females. These gender-related factors appeared to have a stronger influence on total body fat percentage compared to DII. Despite this, the study also identified significant independent influence of DII on body fat percentage, beyond the impact of gender-related factors. This finding highlights the substantial impact of dietary inflammation on body adiposity, independently of gender. Therefore, understanding the inflammatory potential of the diet can be one of the critical determinants of body fat distribution and overall adiposity.

The study's exploration of the relationship between WC and VAT provided valuable insights into the potential use of WC as a practical and accessible method to assess visceral fat levels. While some variability was observed in the WC-VAT relationship across different populations, the present study identified a strong positive correlation between WC and VAT in the specific population of first-year college students.

Overall, this study addressed the importance of considering both gender-related factors and dietary inflammation when assessing and managing body composition. The findings highlight the need for a comprehensive understanding of the multiple factors that influence body fat distribution and the complex interplay between dietary inflammation and body composition.

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**Appendix A**  
**IRB Approval**

**Re:** Initial - IRB-FY2023-8

*Factors influencing body composition in college students*

Dear Lindsay Heidelberger,

In accordance with Federal Regulations, your project, *Factors influencing body composition in college students* was reviewed by the full University of Wisconsin Institutional Review Board on September 12, 2022, and was approved through Full Board Review through September 12, 2023 in accordance with Federal Policy for the Protection of Human Subjects (45 CFR 46). If a renewal is needed, it is to be submitted at least 10 working days prior to the expiration date.

**Please address the following areas prior to data collection:** Please update the consent form to match the current template prior to data collection (i.e., IRB contact information and location).

Responsibilities for Principal Investigators of IRB-approved research:

1. No subjects may be involved in any study procedure prior to the IRB approval date or after the expiration date.
2. All unanticipated or serious adverse events must be reported to the IRB.
3. All protocol modifications must be approved prior to implementation unless they are intended to reduce risk.
4. All protocol deviations must be reported to the IRB.
5. All recruitment materials and methods must be approved by the IRB prior to being used.
6. Research which involves financial compensation to participants must follow appropriate UW-Stout payment procedures.

7. Consent forms must adhere to UW-Stout IRB standards and indicate that the research has been approved by the UW-Stout IRB as required by federal regulations (see UW-Stout IRB consent form templates for more details).
8. Federal regulations require IRB review of ongoing projects on an annual basis.
9. Any modifications to the approved study must be submitted for review through Cayuse IRB. All approval letters and study documents are located within the Study Details in Cayuse IRB.

Thank you for your cooperation with the IRB and best wishes with your project. Should you have any questions regarding this letter or need further assistance, please contact the IRB office at 715-232-4042 or email [irb@uwstout.edu](mailto:irb@uwstout.edu).

Sincerely,

A handwritten signature in black ink that reads "Mike Meni". The signature is written in a cursive style with a long, sweeping tail on the final letter.

Michael Mensink, Ph.D.; IRB Chair

University of Wisconsin-Stout Institutional Review Board

## Appendix B

### Consent to Participate In UW-Stout Approved Research

**Title:** Factors influencing body composition in college students

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**Investigators:**

Kerry Peterson, PhD, RD, a faculty member at UW-Stout. Department of Food and Nutrition, 205 Heritage Hall, 415-10th Avenue, Menomonie, WI 54751. Phone: 715.232.2545. Email: [petersonke@uwstout.edu](mailto:petersonke@uwstout.edu)

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Karen Ostenso, PhD, RD, CD, a faculty member at UW-Stout. Department of Food and Nutrition, 225 Heritage Hall, 415-10th Avenue, Menomonie, WI 54751. Phone: 715.232.2394. Email: [ostensok@uwstout.edu](mailto:ostensok@uwstout.edu)

**Description:**

You are consenting to voluntarily participate in an on-going study aimed to determine the relationship between dietary factors and changes in the composition of your body during your 4 years at UW-Stout. As part of this study, you will have a Dual X-ray Absorptiometry (DXA) scan to obtain an in-depth analysis of your fat tissue and your muscle mass. You will also be asked to track the foods and beverages you consume, answer a variety of questions about yourself, the foods you eat and your food supply, social support, where you purchase foods, and your beliefs about diet and health. This form will explain the surveys/measurements that will be performed, any risks to you that may arise from participating in this research, the benefits to you if you agree to participate, and the voluntary nature of the test.

**Procedures**

If you choose to participate in this study, the following will happen each of the 4 years you are enrolled at UW-Stout. You will be asked to complete the surveys electronically at home and will report to the Human Performance Lab, room 423 Heritage Hall only for the body scan. Please bring your University-issued laptop as some of the surveys are taken electronically.

**Prior to your arrival to the Human Performance Lab**

Participants must self-screen for COVID-19 symptoms prior to arriving at the Human Performance Lab for the DXA scan. You must attest that you have not recently experienced any of the following symptoms:

- Fever or chills
- Mild or moderate difficulty breathing
- New or worsening cough

- Sustained loss of smell, taste, or appetite
- Sore throat
- Vomiting or diarrhea
- Aching throughout the body

You must wear a mask at all times upon your arrival at the Human Performance Lab.

If you do not have a mask, one will be provided to you.

### Surveys

- The first survey you will be asked to complete is a brief demographic survey that will ask you questions about yourself, including year in college, major, ethnicity/race, gender, financial status, and living arrangement. This survey should take no more than 5 minutes to complete and is taken electronically.
- The Food Frequency Questionnaire (FFQ) is used to determine how you eat over the course of a year. This survey should take approximately 45 minutes to complete and is taken online.
- Eating Attitudes Test-26 and the ORTO questionnaire are intended to assess risk for eating disorders and disordered eating behaviors. These questions should take no more than 5 minutes to complete and are taken electronically.
- U.S. Food Security Module is used to determine barriers to obtaining safe and healthy foods. These questions should take no longer than 5 minutes to complete and is taken electronically.

- You will also be asked to record the food and beverages you consume for 3 days and return your intake record to the study staff. We will have you write down what you eat and drink on a form that is provided to you.

### **Dual X-ray Absorptiometry (DXA) scan**

Traditionally used for assessment of bone density, a DXA scan is a total body X-ray and the gold standard for body composition analysis. The DXA is a machine with an “arm” that branches out over the table that you lay on. While having a DXA scan performed you will be asked to lie still on a padded table for approximately 10 minutes with strap velcroed around your legs to stabilize them. The researcher may need to help you position your body correctly on the table by moving your legs or asking you to adjust your head. During this time an x-ray beam that emits a small amount of radiation will pass over your body to measure your body fat, the amount of muscle in your body, and the density of your bones. This scan will only provide information about your fat mass and your muscle mass. If you choose to participate in this study, a DXA scan will be performed in your 1, 2, 3, and 4<sup>th</sup> year in college. At completion of the study, you will be given a multi-paged print out where you will see percentages, mass, and images accounting for the various data obtained.

### **How to prepare for the DXA scan**

The DXA scan requires minimal preparation. You should remove all metal prior to having the DXA scan. This includes zippers and clasps on clothing and jewelry. If you have metal plates or screws from prior surgeries or jewelry that cannot be removed, this does not exclude you from a DXA scan, however, your results will be less accurate. Please inform the assistant of any non-removable metal prior to your scan.

*For Women Subjects:* All females regardless of reproductive age or status will be asked to verbally confirm they are not currently pregnant or think they may be pregnant.

### **Risks**

The risks of completing the study surveys is minimal. However, there is the risk that you may find some of the questions to be sensitive and there is the risk that some questions may cause emotional discomfort. Additionally, for female subjects there is the risk that questions pertaining to pregnancy status may cause emotional discomfort. The University of Wisconsin-Stout Counseling Center (715.232.2468) provides support for eating disorders or any other mental health concerns that you may have. Counseling services on campus are confidential and free of cost for enrolled students.

The DXA scan will be performed by trained personnel. All attempts will be made to minimize the risks associated with your participation in this test.

- *Radiation.* The DXA scan is a full body x-ray. Therefore, you will be exposed to a small amount of radiation during the test. It is estimated that the average person receives a dose of radiation from naturally occurring sources of about 3 mSv per year. The amount of radiation you will be exposed to during a DXA scan is approximately 0.001 mSv. This equates to about 3h of natural background radiation. While the amount of radiation received from a DXA scan is minimal, exposure to any amount of radiation can cause changes to genes or damage body tissues. The more radiation you receive or are exposed to, the greater your likelihood of risk or complications related to radiation, such as cancer and/or tumors. Should you have additional questions regarding radiation dose please discuss this with the researchers at this time. You may also consult with your physician and/or radiologist. Additional information and resources are available to you at

<https://www.radiologyinfo.org>; <https://www.epa.gov/radiation/radiation-sources-and-doses>; and <https://www.nrc.gov/about-nrc/radiation/around-us/doses-daily-lives.html>

During the COVID-19 pandemic, any face-to-face interaction poses additional risk to your health. All attempts will be made to minimize the risks associated with your face-to-face participation including, proper sanitation procedures, following safety protocols, and requiring all study personnel and research participants to wear appropriate personal protective equipment. If you have additional concerns or questions regarding COVID-19 policies or procedures, please visit the UW-Stout COVID-19 Planning and Preparedness webpage <https://www.uwstout.edu/covid-19-coronavirus-planning-and-preparedness>

### **Benefits**

You will receive no direct benefits from participating in this research study. However, your responses may help us learn more about factors influencing changes in weight status and body composition while in college. Understanding these factors will help us identify the dietary-related challenges faced by college students and to determine how better meet the needs of our students.

### **Confidentiality**

All of your information will be kept confidential during and after collection of data. Only the University of Wisconsin Stout researchers will have access to your information. The information collected will be stored in locked file cabinets and on a secure computer, which is password protected. The data will not be used for any purposes other than for the purposes stated above, i.e. measuring dietary factors, your percent body fat, lean mass, and any changes that may have occurred since a previous test.

### **Right to Withdraw**

Your participation in this study is entirely voluntary. You may choose not to participate without any adverse consequences to you. Should you choose to participate and later wish to withdraw from the study, you may discontinue your participation at this time without incurring adverse consequences.

### **Time Commitment and Payment**

This is a 4-year follow-up study and all testing will occur each year you are enrolled at UW-Stout. Total time to complete the surveys and the DXA scan is approximately 1.5 hours or less at each visit, for a total time commitment of no more than 6 hours. You will be compensated for your time after each visit (\$25). If you participate in all 4 visits, you will receive \$50 at your final visit.

### **IRB Approval:**

This study has been reviewed and approved by The University of Wisconsin-Stout's Institutional Review Board (IRB). The IRB has determined that this study meets the ethical obligations required by federal law and University policies. If you have questions or concerns regarding this study please contact the Investigator. If you have any questions, concerns, or reports regarding your rights as a research subject, please contact the IRB Administrator.

**Investigator:** Lindsay Heidelberg, PhD, RDN

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Programs.

**IRB Administrator**

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**Statement of Consent:**

I have read the above and understand the procedure benefits and risks associated with participation in this test. I understand that participation is voluntary and that my refusal to participate will not result in adverse consequences to me, and that I may discontinue participation at any time without penalty or loss of benefits to which I am otherwise entitled.

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Printed Name (first and last)

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Signature

Date

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UW-Stout Witness (print and sign)

Date