

*The Difference of Body Composition in Type
2 Diabetics and Obese Non Diabetics Using
Bioelectrical Impedence Analysis*

**A thesis submitted for partial fulfillment of the MSc
degree in Internal Medicine**

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Abstract

Body Composition is the technical term used to describe the different components that, when taken together, make up a person's body weight. Now one must keep in mind that body composition and body weight are two entirely different concepts, and they are not interchangeable.

The human body is composed of a variety of different tissue types. The so-called 'lean' tissues, such as muscle, bone, and organs are metabolically active, while adipose, or fat tissue, is not.

Adipose tissue is divided into three different categories:

1. Essential fat, which supports life, and is extremely important to normal bodily function.
2. Storage fat, which protects internal organs and supplies some energy requirements.
3. Non-essential fat, serves no real purpose, and may, in fact, be detrimental to health.

The difference in these tissues is not readily distinguishable by stepping on a scale. A scale simply takes the sum of *everything* (fat, muscle, water, hair...etc), and gives an absolute weight measurement. Scales can't determine the lean-to-fat ratio of that weight. An individual can be "over-weight" and not "over-fat." A bodybuilder, for example, may be 8% body fat, yet may be considered "over-weight" by a typical height-weight chart. Therefore, these charts are not a good indication of a person's ideal body weight for optimal health, much less for athletic performance.

In order to identify these tissues, several different methods of assessing the percent of fat vs. lean mass of an individual have been developed. These methods are referred to as Body Composition Analysis.

Key words : obesity , body composition, bioelectrical impedance, diabetes

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List of Abbreviations

AACE : American Association of Clinical Endocrinologists
ACE : American College of Endocrinology
ACE-Is : Angiotensin Converting Enzyme-Inhibitors
ADA : American Diabetes Association
AI : Angiotensin I
RB : Angiotensin II Receptor Blocker**IIA**
ALT : Alanine aminotransferase
ASCVD : Atherosclerotic Cardiovascular Disease
AST : Aspartate aminotransferase
ATP : Adenosine triphosphate
BF : Body Fat
BIA : Bioelectrical impedance analysis
BMI :Body Mass Index
BV : Body Volume
CAD : Coronary Artery Disease
CALM : Candesartan and Lisinopril Microalbuminuria
CHD : Coronary Heart Disease
CRP : c-Reactive Protein
CT : Computed tomography
CVD : Cardiovascular disease
DM : Diabetes Mellitus
DXA : Dual-energy x-ray absorptiometry
ECG :electrocardiogram
ESRD : End-Stage Renal Disease
FDA : Food and Drug Administration
FFA : Free Fatty Acids
FFM: Free Fat Mass
FPG : Fasting Plasma Glucose
GLP-1 : Glucagon-Like Peptide -1
HDL-c : High-density Lipoprotein cholesterol
IDF: *International Diabetes Federation*
IFG : Impaired Fasting Glucose
IGT : Impaired Glucose Tolerance
IIH : Idiopathic Intracranial Hypertension
IR : Insulin Resistance

IRS-1: Insulin receptor substrate 1
LCD : Low Calorie Diet
LDL-c : Low-density Lipoprotein cholesterol
LVSD : Left Ventricular Systolic Dysfunction
MetS : Metabolic Syndrome
MI : Myocardial Infarction
MR : Magnetic resonance
NAFLD : Non-Alcoholic Fatty Liver Disease
NASH :Non-Alcoholic Steatohepatitis
NCEP-ATP III: *National Cholesterol Education Project Adult Treatment Panel*
NGT : Normal Glucose Tolerance
NHANES :National Health and Nutrition Examination Survey
NHLBI : National Heart, Lung, and Blood Institute
NIH : National Institute of Health
NMR : Nuclear Magnetic Resonance
NO : Nitric Oxide
OADs :Oral Anti-diabetics
OGTT : Oral Glucose Tolerance Test
RAS : Renin-Angiotensin System
rKOA : rheumatic Knee Osteo-Arthritis
RV : Residual lung Volume
SKF : skinfold
SPECT : Single Photon Emission Computed Tomography
TBW : Total Body Water
UKPDS : United Kingdom Prospective Diabetes Study
US : Ultrasound
WHO :World Health Organization

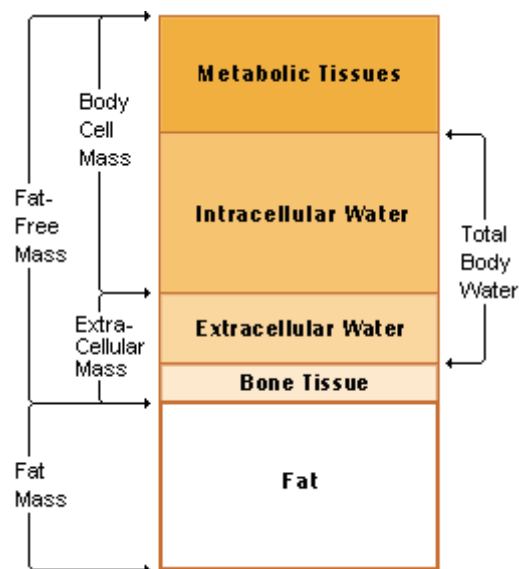
Introduction and aim of work

Introduction

Body Composition is the technical term used to describe the different components that, when taken together, make up a person's body weight. Now one must keep in mind that body composition and body weight are two entirely different concepts, and they are not interchangeable.

Body Tissues

The human body is composed of a variety of different tissue types. The so-called 'lean' tissues, such as muscle, bone, and organs are metabolically active, while adipose, or fat tissue, is not.



FUNCTIONS OF FAT:

- insulation/cushioning
- stored energy (greatest capacity)
- structural (cell membrane)
- involved in nerve conduction (myelin sheath)
- critical for absorption of fat-soluble vitamins
- backbone for steroid hormones

Adipose tissue is divided into three different categories:

1. Essential fat, which supports life, and is extremely important to normal bodily function.
2. Storage fat, which protects internal organs and supplies some energy requirements.
3. Non-essential fat, serves no real purpose, and may, in fact, be detrimental to health.

The difference in these tissues is not readily distinguishable by stepping on a scale. A scale simply takes the sum of *everything* (fat, muscle, water, hair...etc), and gives an absolute weight measurement. Scales can't determine the lean-to-fat ratio of that weight. An individual can be "over-weight" and not "over-fat." A bodybuilder, for example, may be 8% body fat, yet may be considered "over-weight" by a typical height-weight chart. Therefore, these charts are not a good indication of a person's ideal body weight for optimal health, much less for athletic performance.

In order to identify these tissues, several different methods of assessing the percent of fat vs. lean mass of an individual have been developed. These methods are referred to as Body Composition Analysis.

Tools and Techniques used for Measuring Body Composition (alphabetically)

- Bioelectrical Impedance Analysis (BIA)
- Bioelectrical Impedance Spectroscopy (BIS)
- Body Mass Index (BMI)
- Body Weight
- Bone Dimensions
- Circumference Measurements
- Computed Tomography (CT)
- Creatinine
- Dual-energy X-ray Absorptiometry (DXA)
- Hand-Grip Dynamometer .
- Hydrodensitometry
- Isotope Dilution
- Magnetic Resonance Imaging (MRI)
- Methylhistidine
- Near Infrared (NIR) Interactance
- Neutron Activation Analysis
- Photon Absorptiometry
- Prompt Gamma Activation Analysis
- Skinfold Calipers
- Total Body Electrical Conductivity (TOBEC)
- Ultrasound
- Waist/Hip Ratio (WHR)
- Whole Body Potassium Counting

Aim of the work:

The work aims at studying body composition in a group of type 2 diabetic patient and comparing it with body composition in obese non diabetics

1. Finding the average values of height, weight, BMI, Fat%
2. Assessment of their body composition using Bioelectrical Impedence Analysis.

Trying to answer the question is there is any difference in body composition in type 2 diabetics and obese non diabetic patients or not?

Obesity

Definition :

Obesity is defined by the World Health Organization (WHO) as a condition of excessive fat accumulation in the body to the extent that health and well-being are adversely affected (**World Health Organization, 2000**).

The WHO definition of obesity in adults is a Body Mass Index (BMI) of $>30\text{kg/m}^2$ (**World Health Organization, 2000**).

In the United States, data from the second National Health and Nutrition Examination Survey (NHANES II) were used to define obesity in adults as a BMI of 27.3 or more for women and 27.8 or more for men (**Najjar et al, 1987**).

In the year 2000, there were more than 300 million obese adults worldwide, and industrialized countries showed a prevalence of obesity of approximately 20%. Currently, more than one-half of the adult population in the U.S. is overweight or obese (**Mokdad et al, 2003**).

The prevalence of obesity in children is also dramatically increasing worldwide, and this contributes to adulthood obesity (**McTigue et al, 2002**).

Etiology :

The etiology of obesity is multifactorial and involves energy imbalance, genes and environmental, polygenic, and, monogenic causes (**Robenbaum et al, 1997**).

1. Energy balance :

Obesity is caused by an excessive intake of calories in relation to energy expenditure over a long period of time. The gastrointestinal tract has the capacity to absorb large amounts of nutrients. So large increase in body fat can result from even minor but chronic differences between energy intake and energy expenditure (**Robenbaum et al, 1997**).

2 . Genes and environment :

The epidemic of obesity is occurring on genetic backgrounds that have not changed, but it is nonetheless clear that genetics plays an important role in the development of obesity (**Snyder et al, 2004**).

Within a permissive environment, the more common genetic factors involved in obesity regulate the distribution of body fat, the metabolic rate and its response to exercise and diet, and the control of feeding and food preferences (**Vohl et al, 2004**)& (**Bouchard et al, 2004**).

Body size depends on the complex interaction between genetic background and environmental factors. In humans, genetic background explains only an estimated 40 % of variance in body mass (**Bouchard & Perusse, 1993**).

Epidemiologic studies have identified several environmental factors that contribute to the continued weight gain documented over the past several decades in westernized countries. The foremost among these factors are an increasingly sedentary lifestyle (e.g., less physical activity and more time spent watching television), and the availability of energy-dense (high-fat), (concentrated-sugar), low-fiber foods (**Gord et al, 2002**)& (**Drewnowski et al, 2004**)

In children, the increased consumption of sugar-added beverages and reduction of dairy intake have been associated with greater weight gain in prospective studies (**Ludwig et al, 2001**)& (**Pereira et al, 2002**).

Persons of certain genetic backgrounds are especially likely to gain weight and experience obesity related diseases when exposed to modern lifestyle . Over the past 50 years , the striking changes in the lifestyle of Pima Indians living in Arizona has led to an epidemic of diet (15% of energy as fat) , in addition to being in more sedentary life than rural life .

3. Monogenic causes of obesity :

Data from twin, adoption, and family studies indicate that genetic factors play a major role in the determination of the interindividual variation in fat mass within human populations (**Bouchard et al, 1990**)

Little precise information regarding genetic causes of human obesity has been identified, namely, mutations in the genes encoding leptin, (**Strobel et al, 1998**), leptin receptor (**Clement et al, 1998**), prohormone convertase(PC)-1 (**Jackson et al, 1997**), pro-opiomelanocortin (POMC)

(**Krude et al,1998**), and the melanocortin-4 receptor(MC4R) (**Yeo et al,1998**).In all of these syndromes, hyperphagia results from dysfunction of hypothalamic pathways controlling satiety.

A.Leptin gene mutation :

Leptin is a hormone that is secreted by fat cells in direct proportion to total fat mass, is transported across the blood-brain barrier, and has receptors in hypothalamic nuclei that control appetite and energy expenditure (**Schwartz et al,2000**).

Since the discovery of the adipose tissue protein leptin , much progress has been made in understanding the molecular basis of body fat regulation . Leptin has major effects on food intake; leptin deficiency is associated with hyperphagia (**Farooqi et al, 2002**) suggesting a lack of a satiety response; and partial leptin deficiency is also associated with increased body fat.

A large body of evidence indicates that leptin, along with insulin, has actions within the central nervous system to inhibit food intake and activate thermogenesis (**Schwartz et al, 2000**).

Leptin and insulin function as critical signals to the brain in the long-term regulation of energy homeostasis and body adiposity, and do so in part by interacting with short-term signals of satiety emanating from the gastrointestinal tract such as cholecystokinin (**Harvel,2001**).

The pathophysiologic relevance of leptin was established in two extremely obese cousins with hyperphagia , who belonged to a consanguineous family of Pakistani origin (**Montague et al,1997**)

These cousins were homozygous for a single nucleotide deletion at position 398 of leptin gene .The administration of low doses of leptin to individuals with genetic leptin deficiency diminishes hyperphagia and induces weight loss that consists primarily of excess body fat. In contrast, administration of long-acting forms of leptin to humans without leptin deficiency induces only modest and variable weight loss or no weight loss (**Hukshorn et al, 2000**)