

Studying the Impact of Morbid Obesity and Massive Weight Loss on Skin Quality

Thesis

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INTRODUCTION

In developed western culture, a lean body is considered to reflect good self-control, and is regarded as attractive, healthy and socially acceptable. Obesity is a stigmatizing disorder, especially among women, which may explain why women predominate in seeking treatment as they usually disrespect their body shape(**Chandawarkar, 2006**).

Obesity was not until the 20th century that it became common, so much so that in 1997 the World Health Organization (WHO) formally recognized obesity as a global epidemic (**Caballero, 2007**).

It is a chronic disease consisting of excessive fat accumulation in adipose tissue and presents a growing incidence not only in the industrial nations but also in developing nations. And the most serious forms of obesity, morbid obesity ($\text{BMI} \geq 40 \text{ kg/m}^2$), is increasing at a faster rate than other categories of obesity (**Wolf&Kuhlmann,2007**) this morbid obesity is defined as a BMI over 40 or a BMI over 35 in combination with comorbidities(**Caballero,2007**).

About 300 million people around the world are obese ($\text{BMI} > 30$). In the USA nearly two thirds of the population can be classified as overweight or obese (**Fontaine et al., 2003**).While in

most European countries obesity rates tripled over the past two decades. The prevalence of obesity varies significantly among countries, with France and Switzerland showing the lowest obesity prevalence, and Slovenia, Croatia, and Greece the highest prevalence (**de Kervileret al., 2009**). The only remaining region of the world where obesity is not common is the sub-Saharan Africa (**Peter et al., 2005**).

Macromolecular components of connective tissue are dependent on age, gender, nutritional imbalances, hormones including those of pregnancy, use of certain drugs and chemicals, and assorted genetic and environmental factors, including prolonged exposure to sunlight and other forms of radiating energy. Obesity triggers quite a few dermatologic aberrations, affecting skin itself (barrier function), adnexae (sweat and sebaceous glands, hair follicles), vascularitylymphatics, microcirculation, and response to trauma (wound healing). Recurrent infectious troubles, hyperpigmentation, edema and ulcerations, striae, cellulitis, and other disorders are not uncommon either. (**Yosipovitch et al., 2007**)

The weight loss following bariatric surgery or diet is usually accompanied by an improvement in body image, whatever the initial psychological conditions, degree of obesity and eating behavior were, and in the majority of operated subjects

these feelings remain at a stable level in the long-term(**Chandawarkar, 2006**).

In clinical practice, however, in spite of highly satisfactory weight loss and maintenance, some individuals still disrespect their body image and seek body contouring procedures in order to improve their physical appearance. Skin and soft tissue redundancy of the trunk, buttocks, breasts, upper arms, and thighs following massive weight loss is considered unsightly. Some contraction of the skin envelop canbe expected for about 1year after a stable weight isreached, but little change is likely to occur thereafter andplastic surgical procedures may be necessary to satisfy patients' needs and restore body image (**Eisenberg et al.,2006**).

According to the American Society of Plastic Surgeons, over 68,000 body contouring procedures were performed for massive-weight-loss patients in 2005 (**de Kervileret al.,2009**).

The etiology of skin laxity after rapid weight loss is inadequately understood. It usually occurs due to damage of collagen and elastin which allows for no skin retraction after weight loss (**Aly et al., 2003**).

Collapsed skin folds after massive weight loss are often managed by plastic surgery procedures, but changes in dermal

composition and architecture have rarely been documented. Although skin collapse is basically a mechanical phenomenon resulting from adipose tissue resorption and remodeling, skin strength and elasticity may be a factor underlying both immediate and long-term results, especially dermal content of collagen and elastin. Nevertheless, very few studies have addressed this, and available information is both scant and somewhat conflicting (**Orpheu et al., 2010**).

There is a common universal belief among plastic surgeons that skin quality is impaired in massive weight loss patients or patients who have lost more than 50% of their original body weight. However, this hypothesis has yet to be tested (**Orpheu et al., 2010**).

While previous studies have reported massive weight loss patients who undergo contouring procedures are at an increased risk for healing complications such as hematomas and seromas, another undesirable outcome which cannot be easily measured is skin relaxation after contouring procedures for massive weight loss patients. The untested belief is that obesity and massive weight loss result in skin laxity after surgery due to damaged skin, those massive weight loss patients have skin with miserable elasticity, thinned strength layers like the dermis and epidermis, and poor collagen quality (**Choo et al., 2010**)

Obesity and massive weight loss represent a scientific new field for investigation. While studies have investigated clinical outcomes in bariatric and post bariatric body-contouring populations, it is time to look on a more microscopic level to understand the effects of both obesity and massive weight loss on skin and wound healing, and to combine this with a study to the skin tensile strength to assess the possible causes of skin laxity following contouring procedures.

AIM OF THE WORK

The aim of this study is to assess skin quality in morbid obese and massive weight loss patients undergoing cosmetic contouring procedures regarding the collagen and elastin content (histological study) and biomechanical properties of the skin (biomechanical study) to address the causes of skin relaxation following contouring procedures.

ANATOMY & HISTOLOGY OF HUMAN SKIN

Components of normal human skin

Skin is the largest organ in the body. In a 70-kg individual, the skin weighs over 5 kg and covers a surface area approaching 2 m². Human skin consists of a stratified, cellular epidermis and an underlying dermis of connective tissue (**Fig. 1**). Beneath the dermis is a layer of subcutaneous fat, which is separated from the rest of the body by a layer of striated muscle (**McGrath& Uitto, 2010**).

The epidermis is mainly composed of keratinocytes and is typically 0.05–0.1 mm in thickness. It is formed by division of cells in the basal layer which give rise to the spinous layer. This layer contains cells that move outwards and progressively differentiate, forming the granular layer and the stratum corneum. The cellular progression from the basal layer to the skin surface takes about 30 days but is accelerated in diseases such as psoriasis. The ‘bricklike’ shape of keratinocytes is provided by a cytoskeleton made of keratin intermediate filaments. As the epidermis differentiates, the keratinocytes become flattened. This process involves the filament aggregating protein, filaggrin, a protein component of keratohyalin granules. Indeed, keratin and

filaggrin comprise 80–90% of the mass of the epidermis (**Houben et al., 2007**).

The outermost layer of the epidermis is the stratum corneum, where cells (now called corneocytes) have lost nuclei and cytoplasmic organelles. The corneocyte has a highly insoluble, cornified envelope within the plasma membrane, formed by cross-linking of soluble protein precursors, including involucrin and loricrin, the latter contributing 70–85% to the mass of the cornified cell envelope; it also contains several lipids (fatty acids, sterols and ceramides) released from lamellar bodies within the upper, living epidermis. Other cells in the epidermis are the melanocytes, Langerhans' cells and Merkel cells (**Houben et al., 2007**).

Melanocytes are dendritic cells that distribute packages of melanin pigment in melanosomes to surrounding keratinocytes to give skin its colour. The number of melanocytes does not differ much between white and black skin. Rather it is the nature of the melanin and the size of the melanosomes that account for the different appearances (**McGrath & Uitto, 2010**).

The Langerhans' cells are also dendritic in nature, although these are of mesenchymal origin and originate from bone marrow. Langerhans' cells are antigen-presenting cells and process antigens encountered by the skin to local lymph nodes and thus

have a key role in adaptive immune responses in the skin. Merkel cells are probably derived from keratinocytes. They have a role as mechanosensory receptors in response to touch (**McGrath& Uitto, 2010**).

Human skin contains pilosebaceous follicles and sweat glands. The hair follicles comprise pockets of epithelium that are continuous with the superficial epidermis but which also envelop a small papilla of dermis at their base. A bundle of smooth muscle, the erector pili, extends at an angle between the surface of the dermis and a point in the follicle wall. Above the insertion, there are holocrine sebaceous glands which open into the pilary canal. In some sites, such as the axillae, the follicles may be associated with apocrine glands. Also derived from the epidermis and opening directly to the skin surface are the eccrine sweat glands (**Schlake, 2007**).

The epidermis is attached to the dermis via a complex network of proteins and glycoproteins that extend from inside basal keratinocytes into the superficial dermis. Besides adhesion, the dermal–epidermal junction components also contribute to cell migration (for example during wound healing) as well as epithelial– mesenchymal signaling events. Over 30 different macromolecules (collagens, laminins, integrins) interact within a

basement membrane zone that is less than 200µm across (Ghohestani et al., 2001).

The dermis is a supporting matrix or ground substance in which polysaccharides and proteins are linked to produce macromolecules that have a remarkable capacity for retaining water. The thickness of the dermis varies from less than 0.5 mm to more than 5 mm. There are two principal types of protein fiber: collagen and elastic tissue. Collagen is the major extracellular matrix protein comprising 80–85% of the dry weight of the dermis. Twenty-nine different collagens have been identified in vertebrate tissue (depicted by Roman numerals in the order of their discovery, from I to XXIX), of which at least 12 are expressed in skin. The main interstitial dermal collagens are types I and III whereas the principal basement membrane collagen (at the dermal–epidermal junction and around dermal blood vessels, nerves and appendages) is type IV collagen. Triple-helical collagen monomers polymerize into fibrils and fibres, which then become stabilized by the complex formation of both intra- and intermolecular crosslinks. Collagen fibres are extremely tough and provide skin with its tensile strength(Frederick et al., 2001).

Elastic fibres account for no more than 2–4% of the extracellular matrix in the dermis and consist of two components, elastin and elastin-associated microfibrils, which together give

skin its elasticity and resilience. Elastic microfibrils are composed of several proteins, including fibrillin, which surround the elastin and which can extend throughout the dermis in a web-like configuration to the junction between the dermis and the epidermis (**Kielty, 2006**).

The dermis also contains a number of non-collagenous glycoproteins including fibronectins, fibulins and integrins. These extracellular matrix components facilitate cell adhesion and cell motility. Between the dermal collagen and elastic tissue is the ground substance made up of glycosaminoglycan/proteoglycan macromolecules. These contribute only 0.1–0.3% of the total dry weight of the dermis but provide a vital role by maintaining hydration, mostly due to the high water-binding capacity of hyaluronic acid. About 60% of the total weight of the dermis is water (**McGrath& Uitto, 2010**).

The dermis has a very rich blood supply, although no vessels pass through the dermal–epidermal junction. There is a superficial and a deep vascular plexus. The motor innervation of the skin is autonomic, and includes a cholinergic component to the eccrine sweat glands and adrenergic components to both the eccrine and apocrine glands, to the smooth muscle and the arterioles and to the erector pili muscle. The sensory nerve endings are of several kinds; some are free, some terminate in hair

follicles and others have expanded tips (**Braverman& Yen, 1977**).

Functions of skin

A key role of skin is to provide a mechanical barrier against the external environment. The cornified cell envelope and the stratum corneum restrict water loss from the skin while keratinocyte derived endogenous antibiotics (defensins and cathelicidins) provide an innate immune defense against bacteria, viruses and fungi. The epidermis also contains a network of about 2×10^9 Langerhans' cells, which serve as sentinel cells whose prime function is to survey the epidermal environment and to initiate an immune response against microbial threats, although they may also contribute to immune tolerance in the skin. Melanin, which is mostly found in basal keratinocytes, also provides some protection against DNA damage from ultraviolet radiation (**McGrath&Uitto, 2010**).

An important function of skin is thermoregulation. Vasodilatation or vasoconstriction of the blood vessels in the deep or superficial plexuses helps regulate heat loss. Eccrine sweat glands are found at all skin sites and are present in densities of 100–600/cm²; they play a role in heat control and produce approximately 1 litre of sweat per hour during moderate exercise.

Secretions from apocrine sweat glands contribute to body odour (pheromones). Skin lubrication and water proofing is provided by sebum secreted from sebaceous glands (**McGrath & Uitto, 2010**).

Subcutaneous fat has important roles in cushioning trauma as well as providing insulation and a calorie reserve. In non-obese subjects, about 80% of the body's total fat is found in subcutaneous tissue. Fat also has an endocrine function, releasing the hormone leptin, which acts on the hypothalamus to regulate hunger and energy metabolism (**McGrath & Uitto, 2010**).

Regional skin variation

There are two main kinds of human skin: glabrous skin (non-hairy skin) and hair-bearing skin. Glabrous skin is found on the palms and soles and has a grooved surface with alternating ridges and sulci giving rise to the dermatoglyphics (fingerprints) (**McGrath & Uitto, 2010**).

Glabrous skin has a compact stratum corneum which may be up to 10 times thicker compared to other body sites such as the flexures, where the epidermis is at its thinnest. Glabrous skin also contains encapsulated sense organs within the dermis, as well as a lack of hair follicles and sebaceous glands (**McGrath & Uitto, 2010**).