

The Menopause and how it might affect cell-IQ™ treatments

Introduction

The Menopause, or the 'change of life', is the body's natural transition out of childbearing years. For women it is the reverse of puberty. During this time fertility decreases and the levels of hormones required to facilitate fertility and support pregnancy begin to naturally decrease.

Symptoms of the menopause such as hot flushes and irregular periods can begin for women in their late 30's or early 40's. This transition period before full menopause is known as peri-menopause which can typically last several years (sometimes as many as 10 years) and is when ovarian hormone production begins to decline and fluctuate. In the western world the most typical age range for menopause is between 40 and 55 with the average age for last menstruation at 51 years. On average, women who smoke cigarettes experience menopause significantly earlier than non-smokers. In rare cases a woman's ovaries might cease to function earlier, a condition known as premature ovarian failure (POF), although this is not considered to be due to the effects of aging, rather can be related to other medical complications such as autoimmune disorders, thyroid disease, diabetes mellitus (Type 1 IDDM) or due to chemo- or radio-therapy.

There are many sources of further information on the symptoms of menopause available from the internet for example, the main focus of this document is on how the process leading to and during menopause might affect expected treatment outcome from cell-IQ™.

During the peri-menopause and menopause years the amount of female hormones, oestrogen and progesterone produced by the body decreases and the 'male' hormones, androgen and testosterone, are able to have more of an effect. The net results of these hormonal balance fluctuations is an unavoidable trend towards gaining weight (typically 1-2lb per year) and for this weight to concentrate its placement around the abdominal area. Other influencing factors can compound these effects

Since the cell-IQ™ treatment is not a destructive treatment but rather relies on stimulating the body's own fat mobilisation pathways to naturally release stored triglycerides in a pre-determined location, the effects of the body's hormones and signalling pathways during menopause can affect how individual adipose cells might respond to the cell-IQ™.

Hormones and their effects on cell-IQ™

Oestrogen

This is the female sex hormone that causes monthly ovulation. As women get older and enter into the peri-menopause years the levels of oestrogen, and hence their fertility, decreases. Oestrogen decreases the body's ability to process and metabolise fatty acids after a meal and these are more likely to be deposited as fat stores. With this in mind, a decrease in this hormone would not appear to be a bad thing, but unfortunately it is the balance of oestrogen to progesterone that is the most important influence on fat storage. As women age, the amount of progesterone decrease approximately 120 times faster than levels of oestrogen and so, while oestrogen levels are actually decreasing, the balance of hormones is oestrogen dominant and hence the hormone is able to exert its influence (similarly, men in their middle age will experience a drop in progesterone which decreases levels of testosterone allowing even the relatively small amounts of oestrogen in their bodies to exert their dominance and cause middle age spread).

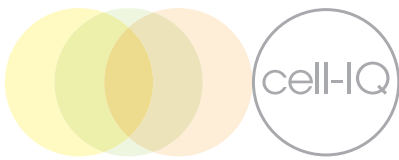
Increases in oestrogen dominance also affects the thyroid gland (which controls metabolism) causing sluggishness and compounding weight gain and increases insulin levels in the body which, as explained in more detail later in this bulletin, prohibits the cells of the body from using fat stored in adipose tissue as an energy source.

In the latter stages of peri-menopause and during the actual menopause years the oestrogen dominance decreases as the levels of the hormone start to rapidly decline. Now the lack of oestrogen being produced causing the body to look elsewhere for a supply of this hormone. Unfortunately, fat cells can produce oestrogen and so the body works harder to convert any calories to fat to increase its ability to match the shortfall of the hormone.

There has also been much concern in the medical community regarding the influence of environmental oestrogens found in food, water, pesticides, plastics and other materials. These xeno-oestrogens mimic the effect of oestrogen on the body and are fat soluble so are capable of being stored long term. There is a huge amount of studies being conducted at the moment into the contributing factors of these xeno-oestrogens with the prevalence of obesity in the western/developed world.

Progesterone

Progesterone is from steroid family of hormones and is produced in women in the ovaries, the adrenal glands, and during pregnancy in the placenta. Its role is to support the menstrual cycle and pregnancy. Decreases in levels of this hormone are typically related to increases in sodium retention and thus more extra-cellular fluid



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volume manifesting in water retention and 'bloating' which may affect measurements during fat reduction treatments.

Androgens

The generic term for any natural or synthetic compound, usually a steroid hormone, that stimulates or controls the development and maintenance of masculine characteristics. The primary and most well-known androgen is testosterone.

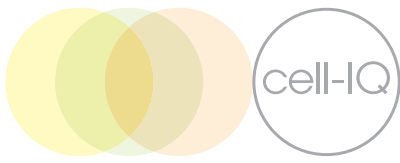
Androgens inhibit the ability of some fat cells to store lipids by blocking the normal signalling pathways that support adipocyte function, hence why males typically have less adipose tissue than females.

While the entire role of testosterone in women is not fully understood by the medical community it is known to be linked to the metabolism as it helps the body create lean muscle mass out of the calories ingested. Muscles burn more calories than fat cells, so increasing muscle mass increases the base metabolic rate. As with the other sex hormones, testosterone levels decrease after the 20's and the muscle mass reduces, to be replaced by fat tissue. This reduces metabolic rate and can increase the speed of fat storage.

Insulin resistance

Insulin is an anabolic hormone whose primary role is to regulate the blood glucose levels. When we eat a meal, within 15 minutes our body absorbs the majority of the glucose from the meal through our intestines and into our blood. The increase in blood sugar levels prompts the pancreas to release insulin which binds to insulin receptors in the liver and muscle cells telling them to increase the uptake of amino acids and glucose from the blood and activates glycogen and triglyceride synthesis. This increase in insulin also prevents the body breaking down any stored triglycerides in adipose tissue or gluconeogenesis in the liver at that time whilst the metabolites from the meal are dealt with. Once the blood sugar levels are reduced the secretion of insulin stops. A lack of insulin binding to the insulin receptors can then stimulate the cells to mobilise stored triglycerides into the blood as free fatty acids which in turn increases blood glucose levels and provides a source of energy when external calories are not available.

A woman's body becomes less sensitive to the actions of insulin after about 40. Increasing fat stores down-regulate insulin receptors so that the cells no longer respond to the normal levels of blood insulin. This failure of the cells to respond normally leaves sugars, fats and proteins in the bloodstream so that they are unable to be used as energy/nourishment. So, despite eating an ample diet, the body in effect becomes starved and stores fat in desperation, particularly in the abdominal cavity (around the liver, bowels and pancreas). This fat then further impairs the action of insulin creating a vicious cycle. Insulin resistance along with increased fat



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storage around the abdomen area is often an early sign of the development of Type 2 non-insulin dependant diabetes (NIDDM).

The net action of this resistance is that prior to menopause, whereas a woman might eat 1000 calories, burn 700 and store 300 as fat, after starting menopause for that same 1000 calories she might burn off 300 and store 700 as fat - primarily around the abdomen as directed by her androgen levels!

Conclusion

There is no escaping the menopause or its effects, including the natural tendency for 'middle-aged spread'. When performing *cell-IQ*™ treatment on women from late 30's and older, especially those patients seeking treatment on a 'pot belly that was never there 5 years ago!', we must consider the early effects of hormonal fluctuations that may already be working against us in fat reduction and provide realistic expectations to the patient for treatment. Depending on the strength of these hormonal effects on an individual patient, we may find that a particular patient is failing to respond *as well* as we might expect immediately after a treatment, or is responding but relapsing back to pre-treatment weight/measurements more quickly than anticipated compared to similarly shaped patients in the age range of 18-35.

The closer a patient is to menopause, or during and after menopause, the harder it is for *cell-IQ*™ to make its message to release its stored fat be 'heard' by the adipose cells, in fact the body is actively doing its best to turn any excess calories taken in from the diet into extra fat.

Hormone replacement therapies (HRT) go some way to manage some of the symptoms of menopause but cannot fully reverse the effects on the body, in particular weight gain. Many women on HRT say that they gain more weight once commencing the therapy but this should be temporary and is mainly due to the development of fluid retention that settles after 4-6 weeks. Prolonged weight gain on HRT is mostly attributed to other factors such as comfort eating. *cell-IQ*™ patients who are on HRT may still not experience the expected treatment results just because their therapy is managing their other symptoms of menopause.

Not to be disheartened and immediately contraindicate anyone over the age of 35 to *cell-IQ*™ treatment, clinical feedback indicate mostly very satisfying results in their older patients, but these considerations might provide the starting point for answer why the occasional one might fail to meet expectations.

As a final note to take into consideration based on the average patient's lifestyles nowadays we must also bear in mind the effects of stress on fat storage. High or



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long duration stress levels release corticosteroid (cortisol) hormones which are responsible for signalling the body to go into fat storage mode. These hormones make the body believe it is in a period of famine and, convinced that it might not get normal levels of food intake over the coming weeks/months, the body turns all available calories into fat to ensure a source of energy during this time. So while we are busy telling individual cells to release their contents with the cell-IQ™ the body as a whole is busy trying to increase fat levels in direct competition to the laser lipolysis treatment.