

All about ageing

- 1.1. Why do we age?
- 1.2. Is it in our glands?
- 1.3. What is happening when the skin ages?
 - 1.3.1. The outer layer
 - 1.3.2. The inner layer
- 1.4. Attitudes towards ageing
 - 1.4.1. The colour of ageing

Understanding the causes of ageing

2. The sun: ageing's main culprit

- 2.1. Sunburn and suntan
 - 2.1.1. The longer-term effects of sunburns and suntans
- 2.2. Epidermal agers versus dermal agers

3. The other major causes of skin ageing

- 3.1. Expression
- 3.2. Elocution
- 3.3. Tension
- 3.4. Glare
- 3.5. Sleep
- 3.6. Gravity
- 3.7. Body fat changes
- 3.8. Smoking
- 3.9. Diet
- 3.10. Acne
- 3.11. Heredity

Introduction

‘Age is a very high price to pay for maturity.’ - Paulo Coelho

The overall process of ageing has long been viewed with a host of mixed feelings. At its best, it can bring us maturity and the wisdom of experience. Recent demographic changes – later marriages, declining birth rates and longer life expectancy – have shifted the population profile towards old age. We now expect a long life and are seeking to live that long life relatively free of the burden of ageing and disease. This is no trivial matter for people and governments worldwide as the cost of looking after the elderly and diseased in old age is escalating.

Interestingly, it may be that physical appearance has more to do with your state of health and well-being than how old you are or how young you feel. A study of more than a thousand similarly aged elderly men has shown that those who look old for their years are in fact showing their age in their other organ systems and, by every measurement, their health is inferior to their better looking counterparts. The better looking men actually live longer.

Many things can be done nowadays to delay the clock, but it is essential that sanity prevails if one is to cope with the remaining years. It is important not to go too far and attempt at 50 to look 25. However, if you are 50 it is more than reasonable to be a most attractive and desirable 50-year-old. The best cosmetic techniques are those where the patient is asked perpetually if they have been away on a recent vacation or told they look well.

For the defenceless public, it is especially difficult to keep realistic expectations in the face of advertising that saturates with false promises and useless products.

There exists a rare group of people who are truly prematurely aged. There are some classic, albeit rare syndromes where these individuals die of old age in their first or second decade of life. However, far more common is the more subtle, inherited early ageing process which renders people looking one or two decades older than their chronological peers. There is no doubt that some people are dealt a cruel blow by their genetic makeup. We all know many people who look 50 when they have 30 printed on their birth certificates. These patients may have a justifiable call on medical services to improve their lot. More commonly (and probably even universally) we see a specific region or problem area in an individual which appears to be ‘letting down’ their overall appearance. This may be a pronounced frown, a downturned mouth, gaunt cheeks, excessively wrinkled cheeks, a drooping set of eyelids or various other issues.

All about ageing

1.1. Why do we age?

For many years, little active research was performed in the area of ageing, nowadays the area is being investigated with zeal. Despite this, there is no universally accepted theory of ageing.

Our body is made up of small building blocks or cells, somewhat like a house. Houses look great when they are first designed and built, but over time they inevitably deteriorate and begin looking a bit worse for wear. The human body in general, and the skin in particular, is like an automatically regenerating house. It replaces the tired old bricks or cells with new ones all the time. But these new cells are never quite as good as the ones that preceded them. The question of why these new cells are not as good is the central question of ageing research. As we age, it becomes necessary for us to begin making our own repairs, perhaps by putting a few fillers, lasers, muscle relaxing injections or other similarly low risk options here and there. More radical options - maybe even a complete re-working – might be considered if the issues are more severe.

Some believe that the cells that make up our bodies each have a predetermined life span and a maximum number of times that they can divide to replace themselves before they get too old (the programmatic theory). This means that from the time you are born, your chips are down and there is nothing you can do to change that upper limit of survival.

A number of other theories are suggested to explain this inbuilt phenomenon of ageing. One such theory is that there are longevity genes in some long-lived subjects and another, possibly more important, phenomenon concerning the role of 'telomeres'.

Each time a cell divides, its genetic material (set of chromosomes) also divides. Telomeres are interesting anti-ageing structures set on the end of these chromosomes that allow structures to reproduce themselves. They are progressively shortened each time the cell divides until such a time that they are too short to allow cell division and reproduction to occur. The cell is the last of its line and ceases to exist at the end of its finite lifetime. An example of this would be when a fibroblast cell that normally produces collagen in the second layer of the skin ends its useful life and ceases to produce any more collagen. This induces a laxity of the skin commonly seen in the elderly.

Others believe in a less fatalistic or pessimistic approach. They feel that the cells start life with enormous capacity but they are continually being bombarded with a huge variety of environmental stresses producing toxic, poisonous substances (stochastic or free radical theory). These nasty stresses may be presented by diet, smoking or exposure to ultraviolet, X-ray or other wavelength radiation. These in turn lead to wear and tear, denaturing the cells and stiffening the fibres of the body which eventually leads to the ageing and death of the individual.

Cells have protective mechanisms against these repeated insults. One of these defences involves a group of substances called antioxidants, the supply of which is exhaustible. The diet and natural pathways in the body are responsible for the supply of these substances to the cells to maximise their protection against environmental stresses. According to the wear and tear theory, it is when the cells run out of these antioxidants that they can no longer adequately protect themselves against the mass of life's daily insults. Once the supply of these antioxidants in the diet and from other means is exhausted, the cells are prone to sustain irreversible damage. This has led to the popularity of taking Selenium, vitamins C and E, and other newer established antioxidants which theoretically add to the stock of the body's own reserve. In theory this will decrease our natural

ageing by prolonging the life of the cells. Vitamin C as well as being an antioxidant is also important in the direct production of collagen. Its supply is important in limiting the stiffening and loss of collagen bundles that occurs with age.

Unfortunately, ageing theories such as these are very difficult to prove or disprove in human beings because of the long lead-time between a change in the diet or environment and the resultant change in the life span of that person. However, models involving growing cells from the upper and lower layers of the skin may, in the future allow for a simpler understanding of the ageing skin.

1.2. Is it in our glands?

Gland function changes with increasing years and may be an important factor in ageing. In females, there is a decline in hormones from the ovary such as oestrogen in the years preceding menopause. The role oestrogens play in ageing of human skin is also very uncertain. What is known is that oestrogen affects skin oil production and that it causes some alteration in collagen and in the thickness of the epidermis. There are also side-effects of oestrogen in the skin including the brown staining of chloasma and increased blood vessels in the skin: both spider naevi ('spiders') and telangiectasia ('broken blood vessels'). Whether these female hormones should be replaced as they begin to wane at menopause is as controversial as any issue in medicine.

Males also have a loss of androgens, but the male menopause (or men-pause) is more drawn out than its female counterpart. Male hormones levels slowly subside with advancing years. Testosterone and the other male hormones, as all body building devotees and weightlifters will attest, are also important anabolic hormones. Anabolic hormones are those that build up tissue. When they are reduced as the individual ages, muscle and bone mass decline. The number of red blood cells also diminishes and their ability to carry oxygen to the tissues becomes less efficient and so may the function of these tissues. This male menopause may be a little more subtle than the female counterpart but still has ageing effects that are significant.

The ageing process is associated with other gland abnormalities with reduced activity being the order of the day. The thyroid gland is largely responsible for our energy levels and its metabolism changes in old age. Less is produced but less is also broken down. Interestingly enough, many of the signs we normally associate with old age such as dry skin, poor skin tone and healing, loss of hair and a heavy, sagging, expressionless face are routinely seen with lower-than-normal thyroid gland function. However, there is little evidence that the normal changes in thyroid gland function seen with ageing, relate to ageing of the skin, nor that this hormone is truly deficient in the elderly.

The immune system declines with age, possibly explaining the increased incidence of cancer seen with advancing age. The immune system conducts surveillance throughout life discarding precancerous and cancerous cells on a daily basis. When this vigilance wanes, cancerous cells both in the skin and elsewhere escape and become invasive and dangerous.

1.3. What is happening when the skin ages?

The skin is composed of two main layers and ageing manifests differently in each of them.

1.3.1. The outer layer

The outer, or upper layer is called the epidermis. It is very thin, only 0.1mm deep, and forms the waterproof outer covering to the skin. It is made up of a number of cell layers and different cell types. These cells fit together like roof shingles. For the top layer of skin to work properly it has to work as a unit.

The basal cells of the epidermis develop at the bottom of this layer and their progeny drift slowly towards the surface. Early in life, these basal cells rapidly divide, but as the skin ages their reproduction slows down and the thickness of the skin correspondingly declines. As further consequences of this slower replacement and turnover, wound healing is slower and there is a slower clearance of chemicals from the skin.

These cells, like all cells that are rapidly dividing in the body, are easily damaged by radiation. In Australia, ultraviolet radiation from the sun is the most damaging type although the longer wave radiation types, such as visible and infrared, seem to cause more damage than previously thought. Cells injured by this radiation produce a deformed group of cells. This causes a dry, scaling, wrinkled outer skin layer, rough to the touch and unpleasant to the eye.

The outer skin layer, the epidermis, is folded heavily to fit into the second layer, the dermis, like dovetailing in carpentry. As the skin ages, this junction flattens and allows the layers to shear a lot more easily. At this stage the skin is far more fragile than it has been before.

The other cells of great importance in this outer layer are the melanocytes. These cells give us our innate colour and are responsible for providing a blanket overlying and protecting the fragile basal cells from the harsh effects of the Australian sun. This innate skin pigmentation provides our only inbuilt protection from the sun.

The pigment cells are fairly constant in number until the fourth decade of life. Thereafter, there is a decline in the number of these cells by about 10–20% per decade. As a result, it becomes impossible for a person to gain the same tan as they could in their youth. Collections of moles, which are local accumulations of these melanocytes, also start to decline in colour, size and eventually number. Melanocytes are relatively fragile cells and repeated sunburns would appear to be the trigger for the relatively common and potentially nasty melanoma: the cancer arising from these cells.

1.3.2. The inner layer

The layer of skin beneath the epidermis is called the dermis and it's a busy place. It's full of blood vessels, elastin and bundles of collagen all embedded in a jelly-type sea of glycosaminoglycans. The cells that produce collagen, elastin and glycosaminoglycans are called fibroblasts and are very important in the ageing of this layer. It is felt that radiation and ageing of these cells leads them to produce fibres and glycosaminoglycans with a damaged and dishevelled architecture.

Collagen and elastin are large fibres found deep inside the skin and are responsible for the elasticity and supple nature of the skin. When they are damaged the changes are seen in the way the skin wrinkles, folds and sags. Protection of this layer is essential if one wishes to retain a youthful skin for a maximum period of time.

1.4. Attitudes towards ageing

There is little doubt that in western society we have a contrasting view to many eastern and indigenous philosophies where the signs of old age are revered as the natural accompaniment of years of learning and the gaining of wisdom. In the west and increasingly in the eastern regions, attitudes to ageing demand an attempt be made to look one's best throughout life and to delay the more obvious signs of physical ageing.

Lots of statistics

In a study recently performed, amongst 1203 Australian adults aged between 18 and 64 some interesting and some disturbing findings were noted. 76% of respondents said they were satisfied with their facial appearance, yet 67% said they would like to change something about that same facial appearance. Also interestingly 94% of people who were married or in a long-term relationship said they were satisfied with their partners facial appearance and yet 35% of the same respondents suggested they would like to change something about that partners facial appearance.

More specifically looking at their perceptions and concern regarding ageing, 80% said they were worried that their looks made them look older than their years and 50% said they also worried about what effects growing older would have on their facial appearance. Roughly 80% suggested that society places a lower pressure on people to look young and that same percentage feels that society tends to perceive women and men differently as they age. Women are only considered to look more distinguished as they age by 43% of the population whereas men are considered more distinguished by 73%. Men tended to look more in control, more intelligent, have more character and seem more experienced than women as they grew older according to the survey, whereas women tended to look more haggard, more stressed, more worried, to appear as though they've had a hard life and spent too much time in the sun. 61% suggested if they could slow down the ageing process in relation to their facial appearance, they would. Almost two thirds of respondents suggested they would consider having cosmetic treatments or surgical procedures.

1.4.1. The colour of ageing

It would seem that the skin in all its complexity is really only made up of three colours that we perceive. The first colour is the brown colour of melanin in the top layer (and sometimes lesions in deeper layers) of the skin which is evenly toned through our early years but is progressively marred by the advent of freckles, sun induced keratoses, lentigines (brown sun spots) and age-induced flat and warty lesions. There are also a number of hormonal and sun-induced smudgy rashes that affect the skin, most often in females occurring during the twenties and thirties, which may negatively impact on the individual's tone and appearance.

The second is the red colour of haemoglobin bouncing back at us from light striking blood vessels in the second layer of the skin. These blood vessels are uniform in youth but become denatured by sun and disease producing an irregularity that is perceived by us as associated with an ageing individual and negatively associated with the beauty of that individual.

The third colour we perceive is the reflection from the collagen in the second layer of the skin (dermis). This should be even and milky, luminescent and youthful. It gives the skin its glow, its vibrancy, its vitality. With ageing, and especially sun-induced ageing, this is progressively lost. The collagen becomes dishevelled and disorganised, not allowing the light to reflect evenly. This leads to a scattered and dull appearance. It is this lustre that anti-ageing strategies strive to restore by the process of collagen remodeling.

Understanding the causes of ageing

2. The sun: ageing's main culprit

'Truth is like the sun. You can shut it out for a time, but it ain't goin' away.' - Elvis Presley

Skin ages under the influence of a number of factors besides natural programming. The most important external factor is without doubt sunshine.

2.1. Sunburn and suntan

Before exploring how the sun ages the skin, it is useful to go back one step and find out how the sun's rays interact with the skin on a day to day basis. Most of us are familiar with at least two ways the sun impacts on the skin: the suntan and sunburn reaction.

Sunlight, as we all know, is made up of a rainbow of visible colours but it also contains many more types of rays that are invisible to our eyes. These include some we are familiar with such as the shorter X-rays and UV rays, and the longer infrared rays and radio waves.

The shorter the ray's wavelength, the more energetic and damaging it is but the less likely it is to penetrate deeply into tissues. Longer wavelengths, though less harmful, penetrate further. Skin-damaging ultraviolet light is composed of three groupings of wavelengths: UVA, UVB and UVC.

UVC is the shortest and most damaging, but fortunately is completely filtered out by the ozone layer. Similarly 90% of the UVB, the next-shortest group, is filtered by the same ozone layer.

When sunlight falls on unprotected skin it impacts directly on the delicate basal cells: the cells sitting at the junction of the first layer of the skin (epidermis) and the second layer of the skin (dermis) which produce the bulk of the cells of the epidermis. The sun's rays also land directly on the melanocytes: the cells that produce the protective pigment of the skin (melanin). Some of the sunlight also gets through to the blood vessels high up in the dermis.

Normal sun exposure produces some damage to all these structures but in the case of sunburn, injury is extreme. With sunburn many basal cells and melanocytes are destroyed and blood vessels become swollen and leak all sorts of chemicals. These chemicals rise up into the epidermis and further injure this top layer. This layer may be so severely injured that it lifts off the dermis producing the all-too-common event of a blistering sunburn.

Blistering is an important sign of severe damage to the skin. If this blistering happens often in the maturing skin of childhood and adolescence, then the damage to the skin cells is thought to be sufficient to produce future skin cancers and melanomas.

It must be remembered that the only inbuilt protection is the natural inherited level of pigment in the skin, known to dermatologists as melanin. Melanin forms a protective barrier like a blanket over the top of the epidermal cells. This inbuilt layer of protection is obviously highest in those of darker complexion and lowest in those of palest complexion. Acquiring this layer by sunbaking involves initially damaging the epidermis before the defence systems of the skin respond by increasing the protective melanin blanket (or suntan) to prevent further damage. Every tanning reaction is a sign of distress of the skin. There is no such thing as a safe tan.

2.1.1. The longer-term effects of sunburns and suntans

It was initially felt that the sun was therapeutically good for the skin and certainly not something that would age you but rather give and maintain a youthful, virile appearance. We now understand that this is not correct and that the sun is responsible for the multitude of skin cancers seen today. We also know that the sun is responsible for prematurely ageing the skin.

Recently it has been shown that sunshine does not accelerate the natural ageing process, it produces a totally different type of ageing. Photoageing, as it is called, is a process best thought of as a thickening or coarsening of the skin versus the delicate and cosmetically more acceptable thinning soft skin of true 'chronological' or normal ageing.

Normal ageing manifests as slightly thinner skin with diminished sweating and oil production. It is smooth and unblemished with some loss of elasticity. Skin surface markings and expression lines are deepened and broadened. All this happens in a fairly linear, gentle decline from the cradle to the grave.

With photoageing there is nothing gentle about the changes. Roughness and coarseness are the order of the day. The outer layer is rough and dry with irregular and blotchy pigmentation, studded with a myriad of benign and malignant barnacles.

With ultraviolet radiation continually streaming down on a sun-worshipping public, the dermis can become severely damaged. The elastic tissues in the skin become a gnarled dishevelled mess instead of pliable and delicate. Collagen is decreased with sun exposure over time. The blood vessels are also impacted by sun exposure becoming dilated and tortuous. This shows itself clinically as telangiectasia or 'broken blood vessels' on the face and upper body.

In the final stages of sun degeneration, the skin appears as thick, coarse, yellow, heavily lined and leathery. In sun-protected skin, these changes in the blood vessels and dermal fibres are not seen.

2.2. Epidermal agers versus dermal agers

There are two main types, or wavelengths, of ultraviolet light that create destruction to the delicate skin layers.

Sunburn is largely induced by UVB and is a sign of damage to the epidermis. UVB is the wavelength produced by the midday sun and this is the spectrum of sunlight that most of our skin cancer avoidance programs highlight. It is also the main type of sunlight that sunscreens try to block out and SPF numbers on our sunscreens refer only to this particular wavelength (e.g. 15+ or 30+).

UVB does not penetrate the skin very deeply and does not really damage the deeper dermis but is more destructive to the epidermis and upper dermis, destroying these outer layers to form dry, blotchy, discoloured skin and induce sunspots and skin cancers. This type of damage is classically seen in patients who are recurrently sunburnt and is particularly severe in those with a Celtic complexion of blonde or red hair, blue eyes and fair skin.

The other wavelength which is as important from an ageing viewpoint is the longer UVA wavelength. UVA is present all day and all year round whereas UVB is present mainly in summer and the middle

of the day. There is 1000 times more UVA than UVB in summer but it is a much less destructive wavelength as a prolonged period of exposure is required. It penetrates deeply into the dermis and destroys this layer, especially the elastin fibres and blood vessels. UVA also is very suppressive of the local skin immune system, inhibiting defence systems and allowing skin cancers to evolve unchallenged. There is also accumulating evidence that infrared radiation (the warming rays of sunshine) may produce sunspots and severe changes in elastin, similar to that seen in UV damage. About one third of the solar energy reaching human skin is infrared.

It appears that topically applied antioxidants in skin care creams may protect against this effect and we may see them added to sunscreens in the future.

People at risk of problems with these longer wavelengths are those who have not experienced recurrent sunburns as a warning. They may even be particularly 'good tanners'. They tend to spend a lot of time in the sun allowing the dose of weaker, longer, more penetrating rays to add up and gradually destroy the elastic tissues and collagen of their dermis.

We are at great risk of this latter type of ageing becoming more rather than less common with our emphasis on sunscreens and skin cancer prevention. How can this be so? The problem we face is that we have a society that still believes that if you are white, you should aim to be brown. We are not yet used to the concept that no tan is healthy. This means that most people who use sunscreens know that they should do this to prevent skin cancer. However, they commonly have two misconceptions.

The first of the misconceptions is a false belief that they are totally immune to the effects of the sun by applying sunscreen every two hours and are therefore safe to stay outside from dawn to dusk. If a person who is able to tolerate fifteen minutes of midday sun before sunburn occurs applies sunscreen with an SPF of 15, they are able to tolerate 15 multiplied by 15 minutes or a total of 225 minutes of sun. That is the maximum daily sun protection attainable from that sunscreen. Reapplication of further sunscreen will not provide further protection.

Secondly, whilst a person is blocking the warning sign of sunburn, they may not be blocking the other damaging wavelengths of sunlight. Instead of getting out of the sun after 15–20 minutes, they tend to stay outdoors longer and incur large doses of UVA and other radiation types. This is far less of a problem if the newer 'broad-spectrum' sunscreens are used, which protect from UVA and other rays.

There is data to suggest that the correct use of these broad-spectrum sunscreens is associated with a significant decrease in skin cancer risk – the foregoing discussion should not be construed as an argument against the routine use of sunscreens. It is instead an argument against using these agents incorrectly, using them to stay for an ever-increasing time in the sun as some sort of aid in getting a tan. They do not produce a 'safe tan' but instead protect your skin when you are unavoidably in sunshine.

Damaging UVA and UVB are at their highest levels between 11am and 1pm. Although lower, damaging levels continue between 9am and 3pm. This is good reason to recommend placing outdoor summer activities either prior to 9am or after 3pm. However, it is good to remember that UVA, the deeply damaging wavelength is present all day and in every season to some extent and will not be affected by clouds, fog or window glass.

In temperate climates such as the major population centres in Australia, seasonal variability in ultraviolet light is high with roughly three times more UVB exposure in summer than in winter.

With cloud cover there is a danger of over-exposure to UVA and UVB. The heating effect of infrared light is effectively filtered by even light cloud cover giving the well known sense of relief from the searing sun. However, the same is certainly not the case with respect to UV light. Light cloud virtually does nothing to influence the amount of this wavelength reaching earth. Very heavy cloud cover is necessary to effectively block ultraviolet light.

Windburn is not an entity; it merely reflects that wind will cool the skin through more efficient evaporation of sweat from our bodies. With the wind and cloud's obliteration of the solar warning that heat imparts, people are likely to stay out in the sun longer than is safe, leading to what is mistakenly referred to as 'windburn'.

Added to this is the possibility of surface reflection, which again may produce an unexpected dose of ultraviolet exposure. At the beach there are some interesting sources of UV to consider. About a quarter of UV light reflects off sand and only about 5% reflects off water. The other side of this is that most UV penetrates easily through water.

Snow reflects most UV light, producing burns in areas that are usually protected such as the top lip, under the nose, eyes and eyelids. Sunscreens are therefore essential.

The quality and thickness of clothing is an important factor in its protective value, and light scattering and reflection are important to remember with respect to shade. Wet T-shirts, nylon stockings, and loosely woven clothes may allow 20–30% of the ultraviolet rays to reach the skin¹. Tightly woven clothing, on the other hand, is effective in stopping virtually all UVB. In contrast to sunscreens, it is also equally UVA blocking.

Irrespective of the grave risks, accomplishing a change of someone's attitude towards the sun and ageing is more difficult than simply warning of the skin cancer risk. Most teenagers cannot foresee themselves ever growing old, yet it is the damage they invoke in their formative years that sets them up for premature, sun-induced skin ageing.

An improved array of spray tans and artificial tanning preparations are currently available. These are now far more natural than those proceeding in colour and evenness of application. An implant may soon become available that will allow a natural, sun less tan but this is currently still in the research stage. These products all have their good and bad points. Their success certainly shows an understanding by the community of the danger of the sun's rays.

Solarium booths deliver high UVA doses and are thought to increase the chances of skin cancers produced by UVB as well as suppressing the individual's immune system. Their most important failing from the ageing viewpoint is the massive dose of long-range ultraviolet light that is necessary to maintain a tan all year round.

The last thing to touch on is the Vitamin D debate. Vitamin D is essential to decrease the risks of some internal cancers, cardiovascular and bone diseases. It can be ingested in foods and tablets or is produced in the skin in response to sun exposure, specifically unprotected UVB exposure. It has been suggested that we should relax our recommendations on sun exposure to ensure sufficient Vitamin D production in the skin. The current recommendations are 20 minutes of unprotected sun exposure to face and forearms three times a week, which seems reasonable.

So what recommendations would I make to the outdoors-loving public?

1. Stay inside between 11am and 1pm in summer – have lunch, feed the kids, have a siesta, reorientate your summer lunch break The long school lunch-break should be seriously questioned. It makes no sense health-wise for Australian schools to schedule lunch as the longest break in the middle of the day.
2. Don't abuse sunscreens by using them to overstay your welcome in the sun.
3. At all times seek and rely on the shade of adequate clothing, umbrellas, trees and hats.
4. Don't be fooled by cloudy days or cooling winds as UV rays are still present.

3. The other major causes of skin ageing

'The tragedy of old age is not that one is old, but that one is young.' - Oscar Wilde

There are several other causes of skin ageing (some avoidable, some less so) that are also significant. These include: expression, elocution, tension, glare, sleep, gravity and facial volume loss, body fat changes, smoking, diet, acne and heredity.

3.1. Expression

Expression lines are the result of underlying muscle movement. Facial muscles are the most energetic muscles we possess and are constantly active throughout life in expressing our myriad of daily emotions.

Facial expressions may occur, such as annoyance where the nostrils may flare, an eyelid rise or the forehead set in a classic frown. These expressions are also essential in verbal and non-verbal communication. A disbelieving look, a glare, a glint of recognition and a warm smile are all examples of the face evoking non-verbal communication. They are essential for speech, producing the enormous variety of accents and speaking styles, which are probably as individual as fingerprints. All these facial movements crease the skin recurrently throughout life. This is relatively better handled when one's face is ten years old and bouncy with normal suppleness and elasticity. As the skin becomes less elastic with age it will often form permanent wrinkles and expression lines induced by these facial expressions.

In sun-damaged skin, the plastic nature of the skin is lost prematurely and this wrinkling process progresses much more rapidly.

3.2. Elocution

Elocution also leads to a variety of problems. Some people enunciate too well, pursing their lips, wrinkling their eyes, raising their eyebrows, or frowning every time they speak. This is often responsible for early vertical wrinkles around the upper lip, horizontal forehead lines, crow's feet around the eyes or deep vertical lines between the eyebrows.

Training may be necessary to suppress natural tendencies in the most articulate, especially with lip pursing. Studying one's diction in the mirror to perceive any habitual movements is sometimes useful and is often easiest to do whilst speaking on the telephone. Correction of these tendencies is very difficult as they represent a reversal of a lifetime of learning.

3.3. Tension

Horizontal forehead wrinkling is a very common problem affecting virtually all of us. It is formed under the action of three main factors: tension, attempted correction of drooping eyebrows which develop with increasing age and habits developed in speech.

Tension or anxiety gives many perpetually worried people a perpetually worried-looking brow. The muscle underlying the forehead skin is thrown into spasm for most of the waking hours and this may continue during fitful sleeping patterns at night.

Sometimes drooping of the eyebrows is bequeathed as a family heirloom and may make its appearance during the third and fourth decades. This may lead to a necessary elevation of the eyebrows in order to obtain adequate vision. People often perceive that it is their eyelids getting in the way and seek opinions about eyelid reductions but occasionally only a brow lift, a surgical procedure to raise the eyebrows, will rectify this problem.

The last cause is the raising of the eyebrows in everyday speech by someone for whom this is a part of their facial expression and tends to occur when making or stressing a point. Taping one's forehead to bring attention to those actions producing horizontal forehead wrinkling looks rather silly but may be useful at home in inducing behaviour modification and minimising future damage but most often now this would be treated with muscle relaxing injections, with or without dermal fillers.

3.4. Glare

Glare from sunlight is a major cause of eye disease, producing growths on the surface of the eye and cataracts. It also leads to squinting as the eyes attempt to protect themselves, thus causing eyelid wrinkling and crow's feet. A simple and stylish measure to prevent this is the wearing of sunglasses with good protective, and preferably wrap-around lenses when in sunshine.

3.5. Sleep

Facial muscles are not the only line-creating force; even the humble act of sleeping is not without its risks, producing asymmetrical creases favouring the side that is preferentially slept on. Usually these appear as vertically or diagonally orientated lines on the side of the forehead or cheek. Sometimes a rayon or silk pillow case can be used to prevent the face 'sticking' to the pillow at night.

3.6. Gravity and loss of volume

Gravity is an overrated cause for sagging and heavy wrinkling occurring with advancing years. Blaming gravity itself is just like shooting the messenger. Prominent cheek jowls, deep folds at the corners of the mouth and nose, drooping eyebrows and a double chin contribute to a tired, haggard visage.

Unfortunately, we cannot influence the effects of gravity on the skin in a direct, preventative fashion. However, these effects are exaggerated in damaged skin where the elasticity of the dermis is lost for whatever reason. The prototype for this is the disease of cutis laxa where the elastic tissue is lost and the skin hangs like a hound-dog: loosely and pendulously under the influence of gravity. This is usually hereditary although it may rarely occur following drug allergies and certain diseases. Anything that destroys the elastic tissues in the skin magnifies the effect of gravity, exaggerating the consequent sagging and heavy wrinkling. The sun, especially ultraviolet light, preferentially affects

this elastic component of the skin and is the most important avoidable cause of this problem. Severe post-acne scarring will also destroy the elastic nature of the skin and increase gravitational effects.

As we age, bone resorption is especially obvious in the cheek and jaw line as is the loss of fat in the cheeks, around the eyes and temples which produce a gaunt, drawn look. This fat may be impolitely redistributed to form the jowls, neck wattles and double chin. This loss of fat and bone resorption also decreases the frame on which the facial skin is draped. As is the case in completed pregnancy, the skin should remould to the new body shape, but if this skin has lost its tone due to sun damage or ageing of the elastic fibres, it no longer retains the ability to conform or shrink around this altered frame. Thus, instead it starts to sag and hang as the years pass.

A regional look at the process of gravity may be useful. The upper third of the face is dominated by the position of the brow relative to the surrounding landmarks. Deviation of the eyebrows from the normal position has profound aesthetic and functional effects. As the brows drop with age, they no longer sit on or above the bony ridge above the eye and produce a closed looking eye. This may continue to drop over the years causing the eyelids to eventually hang over the eyelashes and interfere with vision. As mentioned above, compensation may occur by wrinkling the forehead in an effort to raise these sagging eyebrows. These muscle actions are not as effective at raising the outer parts of the eyebrows and lead to folds of skin being accentuated at the temples with exaggerated crow's feet radiating outwards from the eyes.

Between the eyebrows, is the glabella region which may also attempt to raise the forehead resulting in vertical frown lines, leading to a permanent seriousness of the face with a stern and unpleasant expression.

The eyelids are an early-ageing feature, often sagging in the fourth decade and steadily worsening over the next twenty years. The upper eyelids develop excess lax skin which may hood over the eyes, again obstructing vision. There is a plate inside the eyelid which provides its shape and substance. Behind this plate are localised fat deposits. As the plate ages, it becomes thin and the fat may protrude, producing bulges that make the upper eyelids sag even more.

The lower eyelids may have protruding fat pads but also have other problems such as lax supporting muscles and ligaments which add to baggy, saggy lower lids. Operations on the upper eyelids concentrate on the removal of skin and occasionally also the fat, whilst the lower eyelid operations concentrate mainly on the removal of fat. Removal of skin here may lead to drooping of the lid margin and a hound-dog appearance. The current tendency is to limit fat removal unless required and supplement the volume under the eyes with fat or other filling agents.

The cheeks usually lose fat in the 30s giving a hollow, sunken appearance to this portion of the face. The skin draping the cheeks starts to sag and hang. This adds to the depth of the nasolabial folds, those lines from the sides of the nose to the corners of the mouth. It also induces secondary creasing in the mid-portion of the cheek, often parallel to the major expression lines.

The nose initially straightens and then starts to droop with age, so the upturned nose of youth gives way to the straighter nose of adult life and then finally, under the action of gravity and other age related changes, its tip descends towards the upper lip.

The neck and chin lines also tend to be affected by gravity induced changes. Oblique creases from the corners of the lips to the sides of the chin are a result of the cheeks beginning to sag. Jowls also develop when skin hangs over the jawline. Slack neck muscles and excessive fat accumulation below the chin produce the familiar double chin, turkey goblet and wattle neck deformities.

3.7. Body fat changes

Fat is redistributed as the years roll by. Facial fat is lost in some areas and accumulated in others as described above. The body also develops preferential sites of fat deposition. In females these areas tend to produce bulges on the inside of the thighs, knees, upper arms and abdomen. Fat in males tends toward the sides and front of the abdomen and top of the hips. The location and extent of these fatty areas tend to run in families but these sites usually do not become a problem cosmetically until the twenties. After this they steadily increase in disfigurement. Some areas occur later such as the lower tummy which tends to be a post-pregnancy phenomenon in females and a middle-age pot in males.

3.8. Smoking

Smoking seems to produce ageing changes through a number of different mechanisms. Of these, the most obvious cause of wrinkling around the mouth is the holding of the cigarette and perpetual sucking action. Smoking may also accentuate crow's feet around the eyes from wrinkling of the eyes whilst inhaling and trying to protect the eyes from smoke.

Smoking appears to produce both shallow and deeper lines on the cheeks. The hollow look of the cheeks from the sucking action of inhaling cigarette smoke and the leathery, grey look of smokers may be due to the constrictive effect of nicotine on the small facial blood vessels.

3.9. Diet

Dietary aspects of ageing skin are not well-defined. As mentioned above, the main dietary suggestions revolve around the anti-oxidant theory of the ageing of cells. Here, selenium and vitamins C and E are proposed as being important dietary aids in preventing damaged cells. Vitamin E at 400 IU daily (d-alpha tocopheryl acetate or d-alpha tocopheryl succinate) and selenium at 100 micrograms daily are the suggested doses to help the skin decrease damage from the sun. The dose of vitamin C is not defined. Certainly these theories, while superficially attractive, should not immediately be taken to mean that stuffing a handful or two of megavitamins and selenium is going to make one dance in the fountain of youth. However, encouraging research has recently pointed to the role of antioxidants (polyphenols) in diet being useful for protection from sun damage. It appears that ingesting 2 apples, 2 oranges and 2 cups of green tea a day is protective against sun damage (Kreutman ACD 2008)

3.10. Acne

Acne disrupts and damages the organisation of the skin producing scarring of the face and destroying the confidence of many impressionable teenagers in the process. As if this is not enough, this damage severely impairs collagen and elastin in the skin, leading to premature ageing. It is essential that acne is treated early and correctly to avoid these devastating long term complications. Today we have many excellent methods for treating this disease including antibiotics, hormonal therapies, isotretinoin and light treatments.

Ideally treatment should not be delayed too long so that the cure produced by these agents doesn't come too late to prevent disfiguring scarring. It is essential that parents do not take the attitude that this disease will settle on its own and is therefore not worth treating. Nor should one obsess about ritualistic dietary or facial cleansing measures, which are not critical in acne control and may delay

reasonable treatment being instituted. The result of inadequate treatment is otherwise avoidable, life-long scarring.

3.11. Heredity

Heredity is extremely important and to a certain extent, it is impossible to defeat your genes. They affect us all and no one has a perfect set. Skin type and its ability to withstand the sun and other environmental insults is largely hereditary. The elasticity of skin appears to run in families with some unfortunate pedigrees showing poor skin suppleness at a very early age. Fat deposits, including those occurring under the neck as well as early jowls all seem to be familial, as is the bony frame on which the skin drapes, e.g. sharp cheek bones or an angular jaw.