Australasian Society of Cosmetic Dermatologists

IN THIS ISSUE

OPINIONS AND PROGRESS IN

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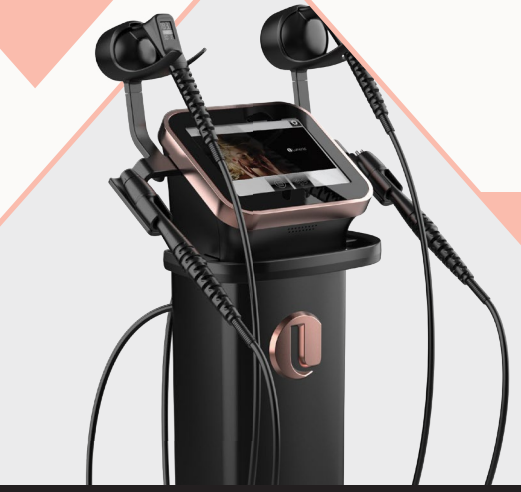
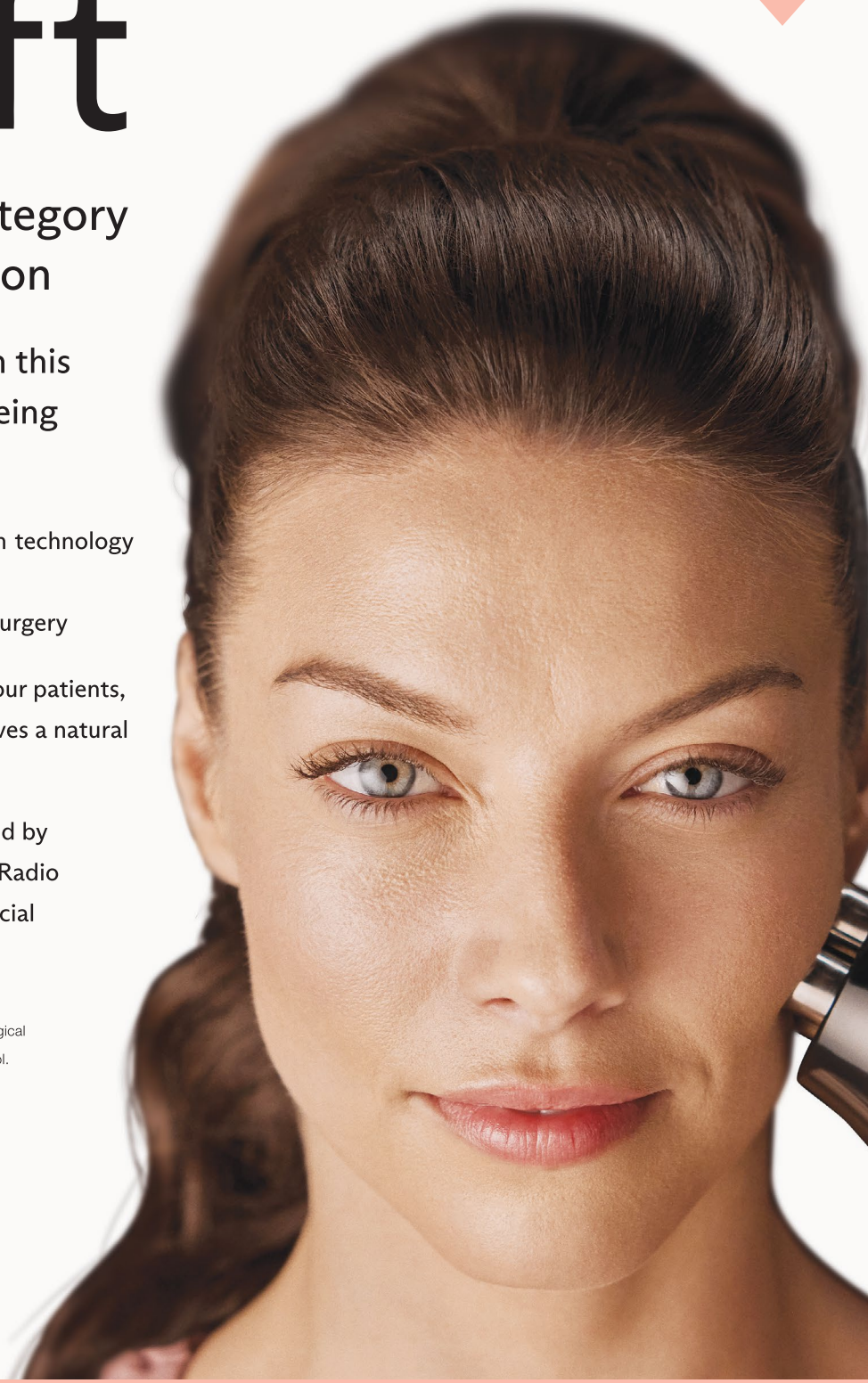
*References: Kaplan H., Gat A., Journal of Cosmetic and Laser Therapy.| Potekaev N, Zhukova O., Journal of Cosmetics, Dermatological Sciences and Applications..| Boisnic S, Branchet MC., Eur J Dermatol.





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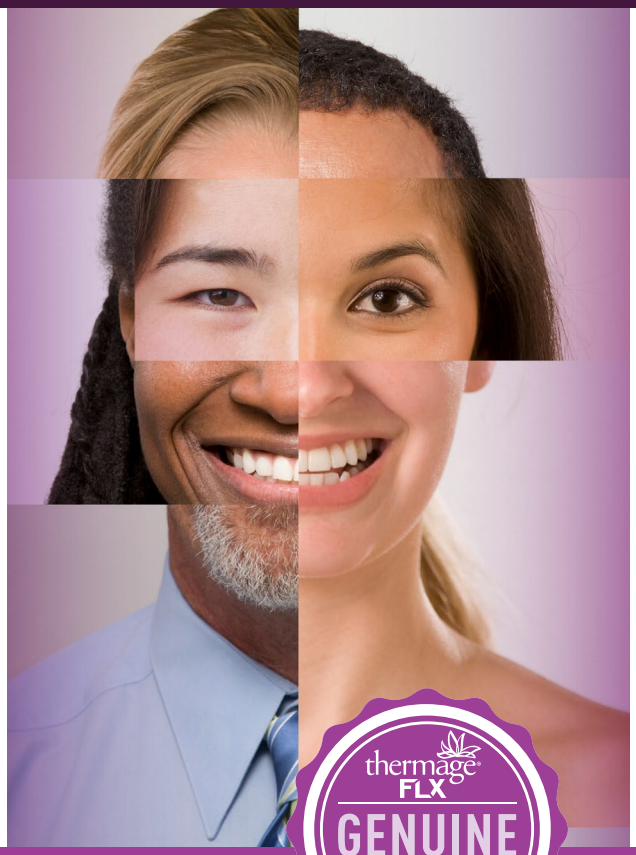

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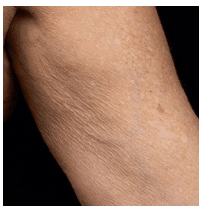

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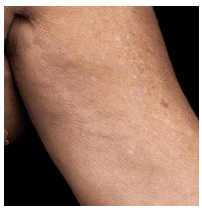

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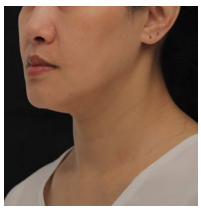

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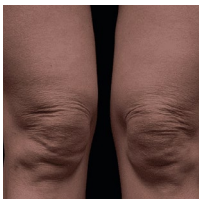

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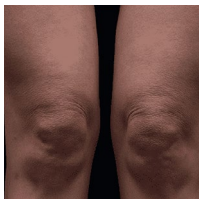

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FACIAL AGEING 1

Welcome to
Facial Ageing 1: The first of a 2-part facial ageing series focusing on anatomy, ageing mechanisms, and corrective options.

Our esteemed Guest Editors for this issue are Associate Professor Deshan Sebaratnam and Dr Naveen Somia. They have seamlessly collaborated to bring you the best in Facial Ageing topics, with the joint perspective of cosmetic dermatology and plastic surgery, respectively.

The recent ASCD Symposium in Melbourne on "Challenges" was a great success and very well received by both speakers and attendees. We are excited to introduce the ASCD Coffee Chat sessions featuring experts and friends candidly discussing topics of interest, sharing tips and tricks, and showcasing similarities and differences in treatment approaches. We have launched the Coffee Chat sessions, starting with "Pigmentary Concerns" followed by "Acne Scarring", which will soon be accessible from the ASCD website, with more topics to follow.

This issue features "TCA and the treatment of atrophic acne scars" by Sun and Lim and Industry submissions on dynamic muscle stimulation for facial sagging. Additional peer-reviewed submissions have been slated for future issues. Further, the OPCD journal is now receiving free papers for peer-reviewed publication.

It is a great privilege to be a part of your continuing education activities, and as always, we welcome any feedback to improve the journal. We hope you enjoy this issue.

Co-Editors in Chief
Dr Adrian Lim
Clinical Professor Saxon D Smith

Contents

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Guest Editorial — Facial Ageing 1

Guest Editors: Deshan Sebaratnam and Naveen Somia

Correspondence: Deshan Sebaratnam deshan.sebaratnam@health.nsw.gov.au

ASCD References to come...

This edition is the first of a two-part series on Facial Ageing.

Ageing is an inevitable part of the human condition. We all accumulate tell-tale signs of the passage of time as our skin, muscle, fat, bones, and physiology change. Dr Xin Lin Wong discusses the structural changes contributing to the appearance of an aged face, reviewing intrinsic and extrinsic factors and broad approaches to amelioration. Professor Greg Goodman's study complements Dr Wong's article as it explores social determinants of ageing, such as alcohol and tobacco consumption, and the use of deoxycholate as a treatment for age-related jowling.

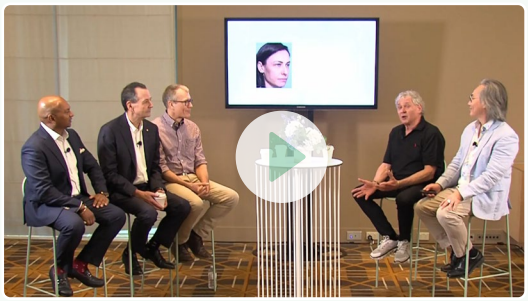
We all want to present the best version of ourselves at any age, and wanting to appear younger is a common request for any cosmetic practitioner. Dr Kelvin Truong discusses approaches to patient communication during a cosmetic consultation and screening approaches for body dysmorphic disorder.

This edition also introduces the concept of transcutaneous electrical muscle stimulation and explores the anti-ageing potential of this intervention.

We hope this issue provides insights into the mechanisms of ageing and approaches to cosmetic consultation regarding holistic rejuvenation, which the next edition on Facial Ageing will explore in detail.

Coffee talks

— experts spill secrets, tips and tricks that you can't get anywhere else



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All Things Pigment

Hosted by Dr Michael Rich and Dr Adrian Lim. Our expert panelists include Professor Greg Goodman, Dr Shobhan Manoharan and Dr John Sullivan.

The panelists cover commonly encountered pigmentary conditions such as melasma and solar lentigines in the setting of diverse skin types. Listen to the panelists candidly discuss how they actually treat these pigmentary issues.

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Acne Scar Repair

Our expert panelist include Greg Goodman, Shobhan Manoharan and Anita Patel.

We cover treatment of typical acne scar cases and discuss energy devices from fractional laser to radiofrequency.

Listen to the panelists candidly discuss how they actually treat acne scarring including device selection and use of specialised scar repair techniques such as TCA CROSS and fillers.

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Predictors of ageing: An analysis of two cross sectional studies illustrating the effects of smoking and alcohol and how Australian women age when compared to counterparts in three other countries

Greg J Goodman[1,2]

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2. University College of London, London, UK

Correspondence: Greg Goodman gg@div.net.au

Disclosures: Greg Goodman is a speaker, investigator and consultant at Abbvie, and Allergan Inc.

BACKGROUND: Cutaneous ageing is the result of many factors and is commonly divided into intrinsic or unavoidable ageing and extrinsic or environmental ageing. Alcohol and tobacco consumption and photodamage are all causes of accelerated ageing. Comparisons of environmental inter-country ageing may help elucidate factors involved in the ageing process. Two studies have been performed in survey format and will be examined.

OBJECTIVE: To study and particularly to compare and contrast effects of the heavy use of social habits (smoking and drinking) on aspects of facial ageing and to compare ageing characteristics after controlling for tobacco and alcohol consumption of Australian women versus those of the UK, USA and Canada.

METHODS: A large multinational You Gov survey was utilised to perform two cross-sectional studies of 18-75-year-old women using photonumeric scales and self-examination to assess the severity of facial age changes. Eleven facial characteristics were examined in comparing alcohol and tobacco and in the national comparison study, eight scales were used.

RESULTS: 3267 women were available for analysis. Both heavy smoking and drinking were associated with upper facial lines. Smoking was also associated with perioral lines and volume changes (under eye puffiness, tear trough severity, depth of nasolabial folds and oral commissures, and decreased lip fullness; $P<0.025\%$). Heavy alcohol use (≥8 drinks/week) was also associated with volume changes of under eye puffiness and depth of oral commissures and midface volume loss, and increased appearance of blood vessels ($P\leq0.042$). In the inter-country ageing study increased age was associated with severity of all facial characteristics, with Australian women self-reporting severe ageing changes occurring earlier than those of the UK, USA and Canada. When compared to USA women, Australian women aged 10-20 years earlier than USA women.

DISCUSSION: Two external social habits (smoking and drinking) when isolated appear to induce ageing in overlapping but different ways. It may be that some aspects of ageing prevention such as antioxidant exhaustion are shared but other mechanisms are probably specific to each agent. Australians seem to age very much earlier and more severely than the three other countries studied in all characteristics examined. When controlled for other factors, lines and volume ageing were all affected earlier in Australians. Australians living in temperate, and sub-tropical coastal cities are able to be outside all year from an early age. It is felt that the more deeply penetrating UVA, visible and infrared radiation affect deeper tissues altering facial volume and enhancing ageing.

KEYWORDS: facial ageing, facial lines, facial volume loss

ASCD References to come...

Introduction

It is well known that environmental exposure and dietary indiscretion are related to the appearance of health and the ageing process. Poor dietary control with the ingestion of fast foods having a high glycaemic index has a deleterious effect by the induction of oxidative stress, inflammation and glycation end products.[1] This over time probably takes its toll on our skin and bodies.

Air pollution is also deleterious to the skin ageing process with particulate matter being responsible for increases in pigment spots and wrinkles.[2] Particulate matter may not only induce reactive oxygen species (ROS)[3] but actually act as carriers for toxic compounds into the subcellular environment to bring ROS directly to the mitochondria.[4]

However, this article specifically interrogates the findings of two previous articles on certain environmental exposures and ageing. One of these articles addressed the effects of alcohol and smoking on facial ageing,[5] and the other examined the appearance of facial ageing between countries and then interrogated the environmental sun exposure of the participants.[6]

Unsurprisingly, each habit induced severe changes in proportion to the quantity of environmental exposure or habit consumed. Surprisingly, however, each insult appeared to have had different effects on the appearance of facial ageing and these will be discussed and explored.

Methods

Both studies were large multinational studies with analysis of 18-75-year-old women across Australia, Canada, the USA and the UK. Women were voluntarily recruited into a proprietary opt-in survey panel and completed an internet-based questionnaire about their facial ageing. All participants in these internet, cross sectional surveys were asked to use a mirror and self-analyse the specific feature against photonumeric scales. In the smoking and alcohol studies, 11 scales were used whilst in the one looking at age and sun exposure habits eight scales were used.

In the smoking and alcohol study linear regressions were used to assess facial feature status and severity against smoking status (never vs current and former) and smoking quantity by pack years (0 versus 1-10, 11-20 and greater than 20 years).[5] Similarly for alcohol, alcohol use (none versus moderate or heavy) and beverage type was assessed. Results were controlled for body mass, race, country, and age. The 11 facial features studied were static forehead, glabella, crow's feet and perioral lines, under eye puffiness, tear troughs, mid facial volume loss, nasolabial folds, marionettes, lip fullness and facial telangiectasia.

In the second study, a comparison of facial ageing across the four countries was performed.[6] The eight aspects of facial ageing comprised forehead, glabella, crow's feet and perioral lines, tear troughs,

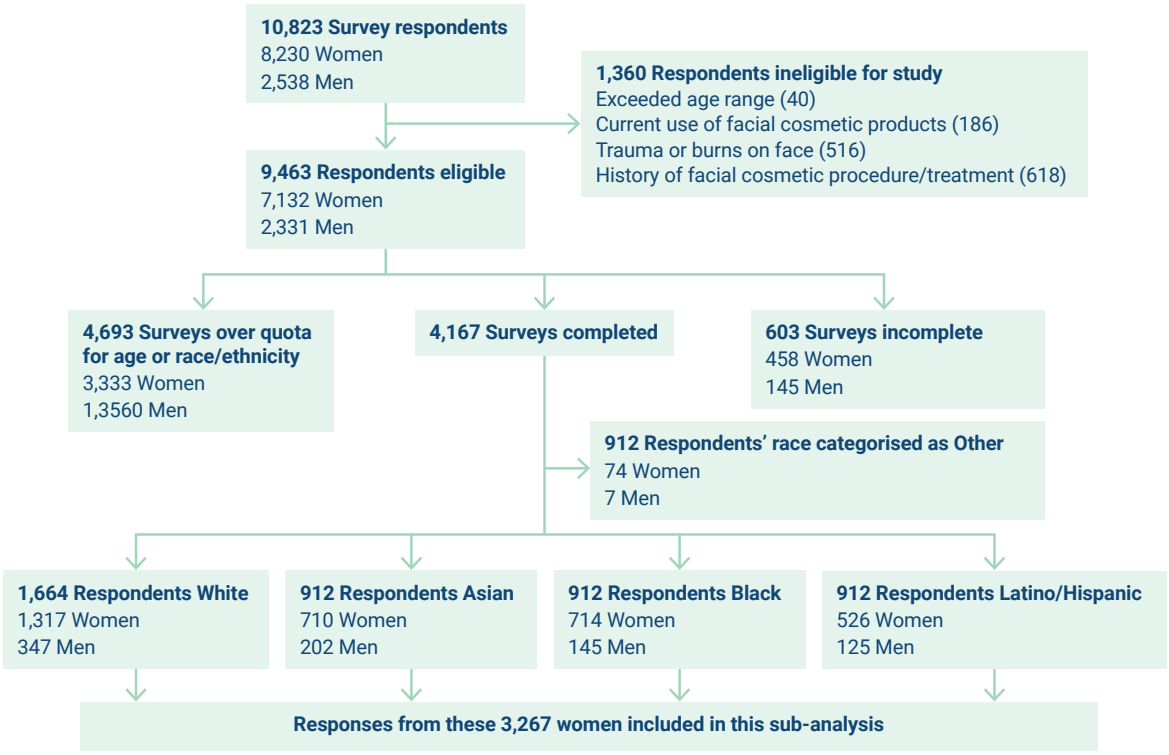

Figure 1: *Selection flow for respondents to derive sub analysis for investigation. From Goodman et al. 2019[5]*

midface volume, nasolabial folds and grooves and oral commissures. This was studied for Caucasian and Asian women of Fitzpatrick skin types 1-3, analysed after controlling for age and race by linear regression for the impact of each country. Australian women were compared to those from the USA, UK, and Canada.

Results

Study population for smoking and alcohol survey respondents

10,823 people responded to the survey invitation over a 3-month period in late 2013 to early 2014 and a sub analysis conducted in 2016 and 2017 of 3267 women (Figure 1).

Of this group 1,569 (48.0%) were from the USA, 591 (18.1%) from Canada, 588 (18.0%) from Australia, and 519 (15.9%) from the UK. The relative proportions of survey respondents in each age range were similar. Fitzpatrick skin type distribution were also similar across all age groups.

Overall, 1,166 women (35.7%) were current or former smokers mostly of cigarettes (1,144/1,166; 98.1%).

Just over half of respondents (1,727; 52.9%) reported that they drank alcohol. Of these, 226 women (13.1% of the drinkers) consumed eight or more drinks per week (defined as "heavy drinking"[7]). Women aged 18 to 29 years composed the highest proportion of alcohol drinkers, but the greatest proportion of heavy drinkers was aged 50 to 69 years. Wine or champagne were the most consumed alcoholic beverages by women of all age groups.

Main findings – smoking and alcohol consumption

Smoking was associated with an increased facial line severity (forehead, crow's feet, glabellar and perioral lines), volume changes (under eye puffiness, tear trough severity, depth of nasolabial folds, oral commissures and decreased lip fullness) (P≤0.025). Smoking did not appear to statistically influence midface volume or visible blood vessels (Figure 2).

Heavy alcohol use (≥8 drinks/week) was also associated with increased upper facial lines (forehead, crow's feet and glabella lines), and volume changes of under eye puffiness and depth of oral commissures but also was associated with midface volume loss, and increased appearance of blood vessels (P≤0.042) (Figure 3).

Smoking pack years

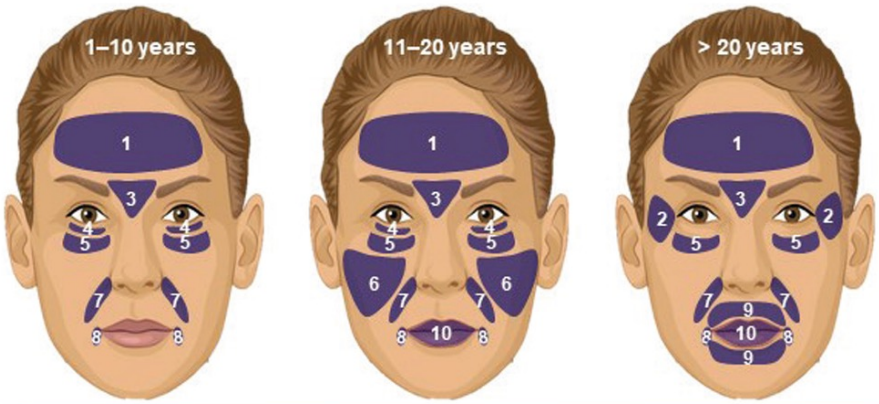

Figure 2. *The effect of smoking pack years on facial ageing*

Long term smoking (>20 years) is associated with all lines – forehead (1), crow's feet (2) glabella (3) and perioral (9) being increased. Volume areas – tear troughs (5), nasolabial folds (7), marionettes (8) and lip fullness (10) are affected with prolonged smoking. Under eye puffiness was seen in the 1-10 and 11-20 groups. Adapted from Goodman et al. 2019[5]

Alcohol consumption level

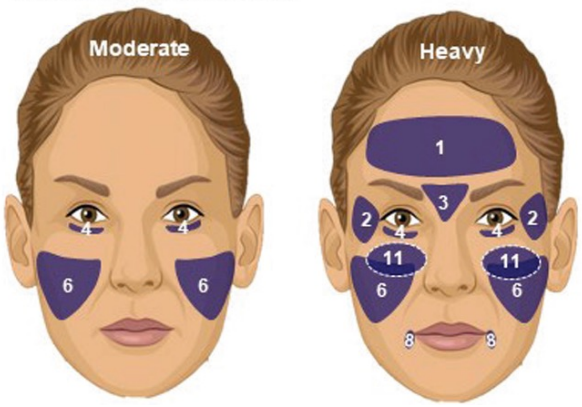

Figure 3. *The effect of alcohol consumption on facial ageing*

Findings suggest that heavy drinkers (≥8 drinks/week) have associated increases in upper facial lines – forehead (1), crow's feet (2), glabella (3) but not perioral lines. They also had volume changes of under eye puffiness (4), but not nasolabial fold, marionette or lip fullness issues, but additionally had mid facial volume loss (11) and visible telangiectases (6). Adapted from Goodman et al. 2019[5]

Although there was some overlap, there were substantial differences between social habits in their effect when consumed to a high degree or for many years (Table 1). Upper facial lines are significantly represented in both groups, but volume shifts with age differed as did the relationship to telangiectases.

Table 1. *Comparison of features affected by heavy consumption of alcohol and smoking. Only upper facial lines are present in both groups – forehead, crow's feet and glabella lines (**X**). Other features tend to occur more with one habit more than the other (X).*

Number	Facial feature	Heavy smoking	Heavy alcohol consumption
1	Forehead lines	X	X
2	Crow's feet	X	X
3	Glabella lines	X	X
4	Under eye puffiness		X
5	Tear troughs	X	
6	Mid facial volume loss		X
7	Nasolabial folds	X	
8	Oral commissures	X	
9	Perioral lines	X	
10	Lip fullness	X	
11	Visible blood vessels		X

Main findings – environmental (sun) exposure and facial ageing

The same group of participants (n=3267) was used for the initial study group (Figure 1) and a sub analysis of 1472 women from Australia, Canada, the USA and UK performed. This was limited to Caucasian (75%) and Asian females (25%) of Fitzpatrick skin types 1-3 so that ethnicities between countries could be best compared.

Some interesting demographic characteristics uncovered included that Australians were the most obese of the four countries with Australians more likely to wear hats, and with Canadians more likely to get sunburnt.

Increasing age was directly correlated with severity of all facial ageing features (P<0.0001) and having Asian ethnicity protective for all ageing features (P<0.05) except nasolabial folds.

Comparison results of Australian vs USA, UK, and Canadian women

Australian women described more severe facial lines at an earlier age and had higher rates of change with age than women from the other countries. This was particularly true when compared to the USA. Australian women had more severe facial lines for all line types (P≤ 0.04) except for static forehead lines compared to Canadian women, where the differences were not statistically significant (Figure 4). As can be visualised

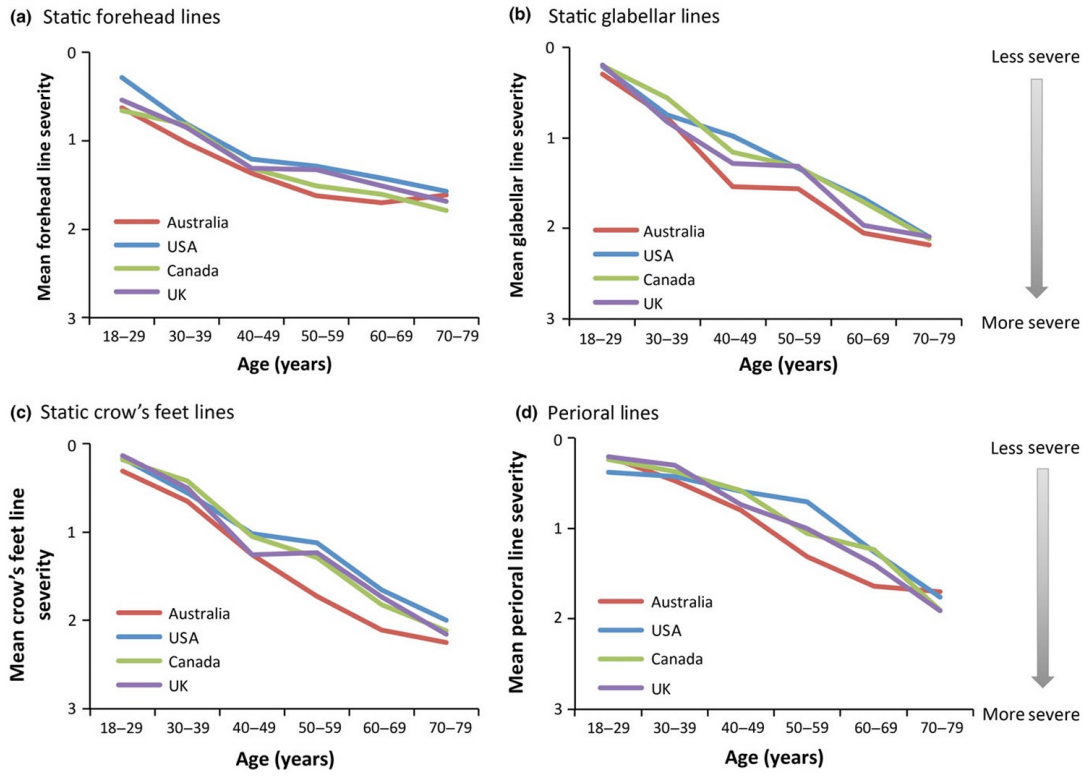

Figure 4. *Australian women (red line) self-reported changes by age compared to women from the USA, UK and Canada. This shows increasing severity for Australian women of all lines (forehead, glabella, crow's feet and perioral) at virtually every age (until the 70s where severity of lines are similar). From Goodman et al. 2018*[6]

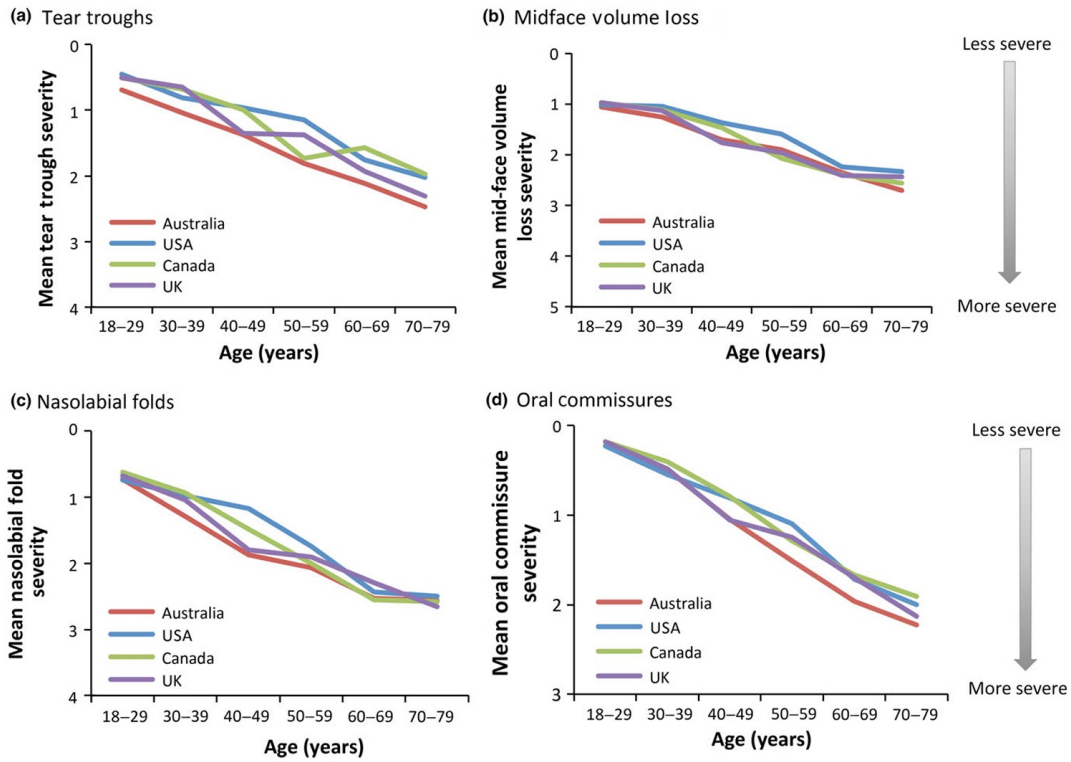

Figure 5. *Australian women (red line) self-reported changes by age compared to women from the US, UK and Canada. This shows a trend to increased severity of Australian women of volume deficiency. From Goodman et al. 2018[6]*

in Figure 4, Australian women (red line) have more severe ageing at almost every age group with rapid changes in severity especially seen.

Regarding volume-related facial features, even though the mean BMI of Australian women was greater than those of women from the other countries they still illustrated greater severity and higher rates of change for tear troughs and nasolabial folds compared to the USA, UK and Canada (P≤0.033; Figure 5).

Oral commissures were also more severe in Australian women, but only statistically significant against Canadian women (P=0.002). Mean tear trough severity was greater among the youngest Australians (18-29 years) than the other countries. Older Australian women also reported the greatest severity of tear troughs, midface volume loss and oral commissures (Figure 5).

Another method used to study ageing between nationalities was to specifically look at the age when greater or equal to 30% of women first reported moderate to severe ageing of each facial feature. The greatest differences using this method were seen between Australian and USA women.

Over 30% of Australian women reported moderate or severe signs of facial ageing for all features from the ages of 30-59 years but this proportion of USA women did not report this level of severity until the ages of 40-69 years (Figure 6).

With regard to the course of facial ageing, nasolabial folds preceded other features in Australians, with advanced severity reported by ≥30% of those aged 30-39 years (Figure 6). This level of nasolabial fold severity was reported two decades later by the same proportion of USA women.

By the age of 40-49 years, ≥30% of Australians reported advanced ageing changes for all upper face lines and tear troughs (Figure 6). In contrast, USA women reported moderate to severe static forehead lines in their forties, glabellar lines in their fifties and crow's feet, perioral lines, tear troughs, midface volume loss and oral commissures in their sixties.

Discussion

The skin and its underlying structures are damaged throughout life by many influences - some environmental such as the sun, whilst some impinged on it by social habits such as excess smoking and heavy drinking.

Excess alcohol and heavy smoking are both associated with severity of upper facial lines.

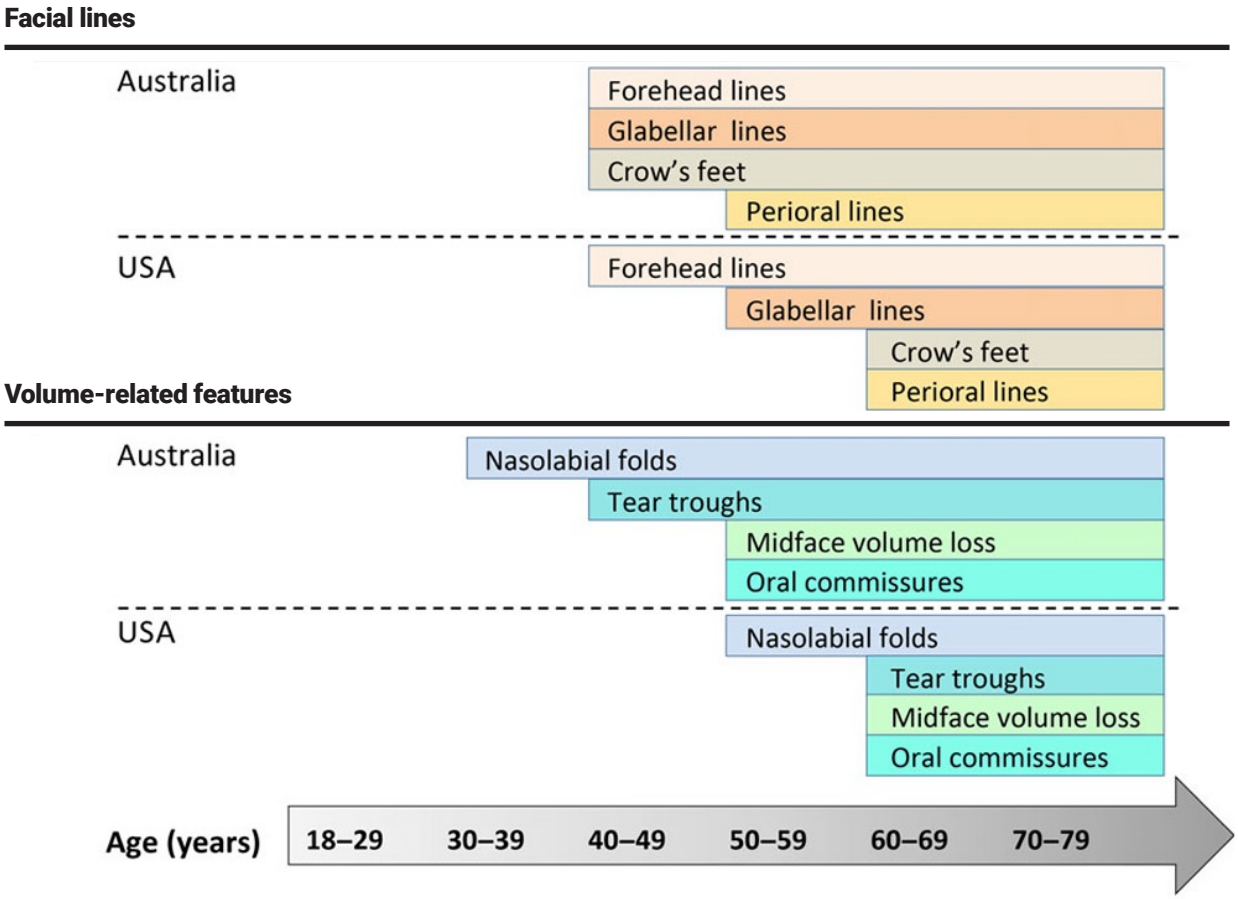

Figure 6. *The decade where 30% of respondents first noticed severe changes of ageing of any of 8 facial features is compared between Australian and USA women. For crow's feet, nasolabial folds and tear troughs Australian women noticed these ageing changes 20 years before their USA counterparts. For all other features except forehead lines, Australian women were 10 years ahead noting these ageing changes. From Goodman et al. 2018*[6]

However, alcohol excess is also associated with under eye puffiness and mid facial volume loss and visible blood vessels but not tear troughs, nasolabial folds, oral commissures nor perioral lines. Smoking is associated with depth of tear troughs, nasolabial folds, oral commissures and perioral lines but not under eye puffiness, mid facial volume loss and visible blood vessels.

The question remains as to why there appears to be such differences between these two insults. Differences may be mechanical - by recurrent facial movements whilst smoking - inducing facial lines, but volume changes are more difficult to explain. However, antioxidant exhaustion is also a potent issue with smokers depleting these important defences leaving these agents unavailable to help prevent photoageing. Cigarette smoking has been positively correlated with photodamage,[8] possibly relating to a downregulation of the aryl hydrocarbon receptor, a transcription factor that mediates the toxicity of ultraviolet B-generated photoproducts in the body.[9]

Like smoking, alcohol has a detrimental effect on antioxidant defence making the drinker open to photodamage.[10] Excessive alcohol consumption impacts the body in several other ways such as inducing vitamin deficiencies, inducing inflammatory changes and diminishing the capacity of skin fibroblasts to produce collagen.[11] The fat loss has been reported before[12] and we have helped confirm that a sagging volume depleted midface and lower eyelid area are part of the legacy faced by the heavy drinker.

Comparing the ageing changes in Australians as against other countries and particularly as against women from the USA it becomes evident from this large cross-sectional study that Australian women fare badly in eight facial ageing features studied. Australian women report more severe ageing changes at an earlier age than their Canadian, UK and USA counterparts. Looking at facial lines and volume changes particularly against their USA counterparts, Australian women noted severe changes of facial ageing occurring in 30% of respondents at a much earlier age. Crow's feet, nasolabial folds and tear troughs reached severity 20 years ahead of the USA cohort, whilst only forehead lines were similar in ageing. The other five facial features (midface volume, oral commissures, glabella lines and perioral lines) aged 10 years faster in Australian women.

Figure 7. *Australian major cities and population tends to be very coastal. This map of the country distribution of You Gov survey respondents shows the differences in location and may impact on the ability of inhabitants to be exposed to chronic sun exposure. Coastal living in Australia is temperate or subtropical allowing full year outdoor activities.*

Why do Australian women fare so badly? Australia is a sun-drenched country and photoageing is known to be disproportionately harsh on the largely fair skin inhabitants. This may explain the typical photodamage signs of excess lines,[13,14] but does not readily explain the pronounced volume changes seen here. This study[6] controlled for race as well as smoking and drinking habits, and showed volume depletion issues associated with alcohol excess.

It is probable that the lifetime sun exposure endured by Australians has something to do with the largely coastal living population and the relatively temperate climate this produces (Figure 7).

Unlike the USA, UK and Canada, Australians can and do enjoy the benefits of a climate allowing year-round outdoor activity from early childhood. Yet how does this influence volume?

One answer may relate to changes in tissue support. Greater photodamage during childhood and adolescence results in less mechanical support for the underlying tissue structure, so that it reveals tissue sagging and fat loss in the form of nasolabial folds and tear troughs from an earlier age. But a more interesting possibility is that photodamaging wavelengths of solar radiation may not only be limited to longer range UV but may also include visible, infrared and longer wavelengths. Given that low level light and many longer range wavelengths are now used for fat removal,[15,16] possibly, chronic low-dose exposure to these wavelengths throughout life may contribute to the volume changes observed.

Strengths of these studies include the large sample size of the You Gov database. Respondents are used to filling in surveys and the collection system is robust. However, limitations for both these studies are that some data collection relies on memory and accuracy of estimation of daily habits. The self-grading using a mirror also has its limitations and the photonumeric scales may also pose difficulties in self-examination.

Conclusions

Two large cross-sectional studies have been explored. Alcohol and smoking appear to both age the skin but in different ways. All the mechanisms are not fully elucidated and although this study controlled for smoking and drinking respectively, many would be expected to do both and have the combined effects of these habits.

When a subset analysis of Fitzpatrick skin type 1-3 Caucasian and Asian respondents were compared, Australian women aged earlier and more severely both in terms of all facial lines and volume characteristics studied.

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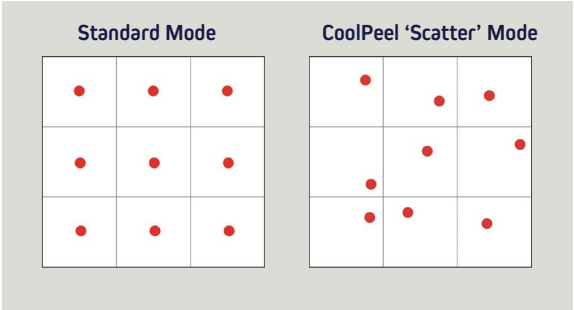

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Patient consultation and communication

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OUTLINE: Communication skills are not well taught in medical school and poor communication is correlated with suboptimal outcomes for the patient and clinician. We overview key aspects of communication skills, clinician behavioural adaptability and how these relate to the cosmetic consultation and body dysmorphic disorder in the cosmetic setting.

KEYWORDS: communication, paternalism, patient-centred, cosmetic consultation, body dysmorphic disorder

ASCD References to come...

From paternalism to patient-centred care

Medical consultations have largely transitioned from a paternalistic and doctor-centred practice to a more patient-centred, partnership-style approach.[1,2] A paternalistic approach remains relevant in some consultation contexts, such as in the care of children or severely ill patients, but it has been criticised as being insensitive to patient needs and values by prioritising beneficence over autonomy and the clinician's view over the patient's.[3,4]

In tandem with the shift towards patient-centred care, a parallel transition from a biomedical to a biopsychosocial model of healthcare occurred. The biomedical model is characterised by a reductionist approach that attributes illness to a single physiological locus in the human body.[5,6] This model tied in well with the paternalistic practice of medicine where doctors who had extensive knowledge in healthcare focused only on the disease and its management without considering the individual or their values.[7] Limitations of the biomedical model are apparent; patients were treated as conditions and not holistically as individuals.[5,6] To address these concerns, the biopsychosocial model incorporated biological, psychological, and social factors into a person's health.[6,8]

The effectiveness of patient participation and shared decision making in patient-centred care is backed by evidence. In one observational study, patients were more likely to report that they were satisfied with their medical consultation if their ideas and concerns were heard and addressed.[9] Patients who were involved in shared-decision making were twice as effective in eliciting information from the clinician and reported fewer adverse outcomes when compared to the control arm in a randomised controlled trial on diabetic patients.[10] In another study investigating prescribing decisions and patient medication adherence, the authors found the source of misunderstandings stemmed from a lack of patient involvement in the medical consultation; patients were unable to express their expectations and preferences and unable to provide feedback on the clinician's decisions.[11]

Key consultation elements

Patients seeking cosmetic procedures may carry complex emotions because of their perceived imperfections, which may lead to unreasonable expectations from their procedure.[12,13] The initial consultation with the patient should be holistic, and include a thorough evaluation of the patient's medical,

cosmetic, social, and psychological history, alongside a focussed physical examination.[12,13] A thorough cosmetic consultation not only improves patient satisfaction, promotes mutual understanding and a long-term therapeutic relationship, but is an opportunity for the clinician to identify unsuitable candidates for cosmetic procedures, such as patients with body dysmorphic disorder (BDD).[13,14]

The 'Empathic Communication Funnel' proposed by McDonald and Heydenrych provides guidance on how to communicate effectively and empathetically (Figure 1).[13] The initial stage involves asking open questions and listening attentively to help build a connection with the patient. It is also during this stage that clinicians can begin to tease out underlying motivations for the visit. The second stage involves the use of unbiased and non-judgemental probing, reflective, and hypothetical questions to obtain more information and build rapport with the patient. The third and final stage aims to assess the feasibility of the patient's request and utilise shared-decision making.[13,14]

Throughout all stages of the consultation, the clinician builds rapport with the patient through verbal and non-verbal means of communication.[13] This is important in ensuring that the patient comprehends the information exchange, as well as to clarify any points of confusion. Finally, it is important to remember that an honest clinician builds trust with the patient by frequently clarifying that they understand the patient's perspective and requests.[14]

Communication skills

Patients are at a higher risk of developing psychological issues, such as depression and anxiety, if their concerns and need for information are not being met.[15,16] This is concerning as only half of patient concerns and complaints are elicited in some medical consultations.[17] Furthermore, the psychological morbidity of certain medical conditions is only recognised in less than half of those who suffer it.[18] These factors contribute to suboptimal treatment adherence, patient satisfaction and outcomes, and underscore the importance of effective communication skills.[19]

Clinicians attribute lack of training in communication skills as the primary reason for high emotional exhaustion, depersonalisation, and lack of job satisfaction.[20] The regimented manner in which clinicians deliver information may impede on the patient's ability to understand their diagnosis and acts as a barrier to effective communication. One study demonstrated that too little or too much information may lead to psychological issues such as anxiety and depression.[21] Interestingly, premature reassurance of a patient's presenting complaint may cause the patient to limit the expression of their concerns and disrupt future opportunities for the patient to discuss these issues.[22]

Clinicians should be aware that some barriers to effective communication may be patient related. Patients who choose to keep their social and psychological issues to themselves often do so under the assumption that it may take away time from discussing their medical treatment and management. However, this can result in increased psychosocial burden, which has a negative effect on the patient's overall experience.[23]

A myriad of studies have demonstrated the importance of communication skills in building patient trust and patient satisfaction.[24,25] Price et al. in their literature review showed that improved patient satisfaction was associated with increased levels of treatment adherence, improved clinical outcomes, better patient safety within hospitals, and less health care utilisation.[26] Good communication skills are symbiotic; patients are more satisfied with their care and clinicians achieve greater satisfaction and less work-related burnout.

The corollary of this is the importance of addressing both the patient's physical and psychological concerns at any opportunity with effective communication skills. Key concepts for effective consultation and communication are summarised in Table 1.[17,27]

Table 1. *Key concepts for effective consultation and communication*

Determining the patient's main presenting complaint
Determine the patient's perception of their main presenting complaint
Ascertaining the physical and emotional aspects of their presenting complaint
Ascertaining the social impact of the patients presenting complaint
Determining how much the patient wants to participate in decision making
Tailoring communication style (clinician behavioural adaptability is key) to the medical consultation
Checking the patient's understanding throughout the medical consultation
Where possible, discussing non-pharmacological and pharmacological treatment options with the patient

Patient-centred care and clinician behavioural adaptability

Studies have shown that a successful medical consultation depends on adapting communication to meet the patient's needs.[28–30] In place of traditional paternalistic methods of communication, in which the

patient does little in the way of participation, the modern clinician utilises a patient-centred approach. This involves the clinician understanding the patient's values, information requirements, emotional needs, and working with the patient as partners to address their medical concerns.[8,31,32] The evidence is clear on the benefit of a patient-centred medical consultation; clinicians experience less work-related burnout and patients are more likely to be satisfied with the consultation, trust the clinician, adhere to medication recommendation, and are less likely to sue for malpractice.[33-35]

However, a patient-centred communication style does not suit all patients. For example, one study found that patients with a higher level of baseline anxiety experienced more anxiety when participating in a medical consultation where the clinician utilised a patient-centred communication approach.[36] This has naturally led to a drive for a communication style such as "behavioural adaptability", that can potentially be tailored to suit a greater number of patients.

Clinician behavioural adaptability refers to a clinician's ability to adjust their verbal and non-verbal approach to best suit the needs of each individual patient.[28] For example, a clinician may adopt a more dominant demeanour with patients who prefer a paternalistic interaction style, while utilising an egalitarian partnership approach with assertive individuals.[28,37] When the clinician's communication style aligns with the patient's preference for information giving, participation and interaction style, patient satisfaction and clinical outcomes improve.[38-41]

Both verbal and non-verbal communication skills are integral in a clinician's behavioural adaptability. Carrard et al. showed that when clinicians use a dominant body language with patients who prefer a paternalistic communication style, it leads to positive outcomes.[29] Patients are more satisfied, trust the clinician more, and rate the clinician as more competent.[29] However, in a larger follow up study by Carrard et al. involving 61 clinicians and 244 participating patients, they were not able to consistently replicate the findings of the earlier study.[30] The study demonstrated that although both male and female clinicians were able to effectively demonstrate behavioural adaptability, the link between behavioural adaptability and patient outcomes was confirmed for female clinicians but not male clinicians, underscoring the complexity of this area of study.

On reflection, it is possible that behavioural adaptability is only one aspect of effective communication, and on its own, may not always correlate with a positive patient experience. Conceivably, clinician behavioural adaptability in the extreme, may come at the expense of perceived clinician authenticity (behavioural faking), and judged negatively by patients. It could also be that patients may harbour internalised paternalism and judge male clinicians who display less "caring and sharing" more favourably than female clinicians with the same behaviour. Perhaps there could be some merit in the suggestion that equally competent clinicians should simply "be themselves" and "find their own tribe" (or allow receptive patients to find them).

The cosmetic consultation

The principles of patient-centred, collaborative consultation are similarly relevant and applicable to the typical cosmetic consultation. Key aspects of a cosmetic consultation include: a "non-illness" model; managing progressive ageing as opposed to episodic illness; understanding the motivation for procedures; managing patient expectations; identifying BDD (higher prevalence in cosmetic patients); navigating the ethics of marketing and promoting procedures; and a need for treatment planning.

Cosmetic patients are diverse in their age, personality, attitude, beauty aspirations, media conditioning, and motivation. However, the three most cited reasons for having cosmetic work done are: "looking better for my age"; "feeling more confident"; "appearing more attractive".[42] It is also not uncommon for novice cosmetic patients to say: "I don't want to look to different" or "I'm afraid of people knowing I've had something done". The clinician must therefore gain an understanding of the patient's motivation and expectations. On the other hand, red flags for BDD must not be missed as these patients are invariably never well-served by cosmetic procedures.[43]

It has been demonstrated that clinicians prevented patients from completing their opening statement in 72% of visits by interrupting the patient after a mean duration of 23 seconds. The study also found that patients typically needed 29 seconds – a further 6 seconds longer than those who were interrupted - to complete their opening statement of concern, which should therefore not significantly prolong the consultation.[44]

After attentively listening to the patient's presenting concern, a relevant history and examination is undertaken, followed by formulation of an assessment and corresponding treatment plan. It is useful to list the patient's concern in order of importance along with a severity rating, for example: "solar lentigines (mild), crow's feet (moderate) and marionette folds (marked)". Standardised baseline photographs can greatly assist with assessment, collaborative treatment planning, as well as record keeping. As part of the initial discussion, it is useful for the clinician to offer a forward projection of the likely chronological ageing issues that the patient may encounter. A re-evaluation and revised treatment plan should be offered and undertaken periodically.

Treatment planning

At the initial consultation, a treatment plan is formulated after the assessment stage where the clinician and patient should jointly agree on the areas of main concern. For each cosmetic issue, the patient needs to be educated on the treatment options, including a discussion of realistic outcomes, number of treatment sessions needed, downtime, after-care, financial consent and longevity of results. The treatment plan will need to factor in patient budgetary and downtime constraints.

For a given cosmetic procedure, the consent process should cover the above discussion points as well as a meaningful discussion of risks and complications that are both general and specific to the patient – for example the increased risk of post-inflammatory hyperpigmentation in a patient of colour or infection in an immunosuppressed patient.

The patient's main concerns should generally be addressed first but apprehensive patients may be better off having another simpler, less invasive procedure to build trust and rapport – for example neurotoxin and filler treatment before going on to high intensity skin resurfacing – "planning big but starting small". When there is a discordance between clinician and patient perspective on treatment priorities, it would be reasonable to start with the patient's main concern, for example, prioritising lip volume over cheeks, and progressively introduce the clinician perspective over time. The clinician should be wary of the need to educate but not to oversell on any particular procedure.

Body dysmorphic disorder (BDD)

Narcissistic, histrionic personality, and BDD (quoted as obsessive-compulsive personality in Napoleon's paper) are the most common psychiatric conditions amongst patients seeking cosmetic surgery.[45] Patients with narcissistic or histrionic personality disorder respond well to reassurance and report high levels of satisfaction after their cosmetic procedure.[45] In contrast, patients with BDD are not receptive of reassurance, and are often dissatisfied with the outcome of their surgical procedures – factors which may attribute to a higher rate of medical litigation.[46] Clinicians should also be aware that BDD patients may become violent – BDD patients have threatened, and on occasion gone through with, violence against their clinician when treatment has been subjectively ineffective.[47]

BDD often starts in youth or teenage years and the onset of symptoms tend to occur later for men (at 36 years old) compared to women (at 31 years old).[48,49] Men with BDD are more likely to be single, live alone, have functional impairments, and be unemployed.[48,49]

The incidence of BDD in the general population is between 1-6%, and higher amongst patients seeking cosmetic surgery (16%).[50-52] In the past, patients were often labelled with delusions of dysmorphosis, dermatologic hypochondriasis, insatiable surgery patients, or polysurgery addicts.[43] In retrospect, these patients are better recognised as having BDD. The high prevalence of BDD amongst patients seeking cosmetic surgery places an onus on the clinician to better understand the BDD patient.

The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, defines BDD as a "preoccupation with one or more perceived defects or flaws in physical appearance that are not observable or appear slight to others".[53] Typical flaws involve subjective worries about facial lines, thinning of scalp hair, nose anatomy, or body shape.[43] Negative self-evaluation drives individuals with BDD towards cosmetic procedures.[54]

Skin picking is a BDD-related behaviour of particular relevance to dermatologists and cosmetic surgeons. Approximately one-third of these patients engage in compulsive skin picking in an attempt to remove perceived blemishes or improve their skin's appearance.[55] Patients with BDD may spend up to eight hours a day critically analysing their perceived defects.[56] This is associated with mood disorders, poor quality of life, and functional impairment; where patients miss work, avoid leaving their house, and avoid social interactions.[57] One study found that the mental health-related quality of life of these patients was worse than that of patients with type 2 diabetes, a recent heart attack, or severe mental illnesses such as depression.[56]

Due to low satisfaction rates and a persistent preoccupation with perceived flaws after a cosmetic procedure, it is crucial clinicians identify patients with BDD. To do so, clinicians should implement pre-procedure screening for potential BDD using a simple psychological questionnaire (Figure 2). This will help detect BDD among those seeking aesthetic procedures. The BDD screening tools that have been validated to be used specifically in dermatology settings are the Body Dysmorphic Disorder Questionnaire (BDDQ), Body Dysmorphic Disorder Symptom Scale (BDD-SS), Dysmorphic Concern Questions (DCQ), and Body Dysmorphic Disorder Questionnaire – Dermatology version (BDDQ-DV).[58] The BDDQ-DV questionnaire is used most frequently.[58]

For patients with BDD, cosmetic procedures are not suggested because they are unlikely to result in high post-procedure satisfaction. Effective management of these patients requires a focus on psychoeducation. In dealing with BDD patients, a sincere acknowledgement of their suffering is helpful but any argument over the validity of their perceived defects is counterproductive.

The key messages to get across BDD patients are summarised in Table 2. Referring them to a psychiatrist or psychologist with expertise in this area for cognitive behavioural therapy and/or pharmacotherapy is crucial.[45] However, as BDD patients do not usually recognise that they have a mental disorder, they may resist being referred for psychiatric or psychological treatment.

Summary

The biopsychosocial model of healthcare incorporates the patient's social, psychological, and biological factors in their care. Clinicians may choose to adopt the 'Empathic Communication Funnel' to guide their consultation. Good and effective communication skills are integral to optimal patient outcomes as well as reducing clinician burnout. A thorough cosmetic consultation not only improves patient satisfaction, mutual understanding, and helps develop a long-term therapeutic relationship but is an opportunity for the clinician to identify unsuitable candidates for cosmetic procedures, such as patients with BDD.

Table 2. *Key messages to convey to patients with body dysmorphic disorder (BDD) during a consultation. Adapted from Sun et al.[60]*

Message	Example
The possibility of BDD	I am concerned that you might have a common body image condition called body dysmorphic disorder
A discrepancy between others' perceptions and patient's view of themselves	People suffering from BDD see themselves as deformed, but they appear objectively normal and attractive to others
Excessive time and money spent	People with BDD may not feel understood by their friends, family, or doctors. They may spend lots of time and money trying to hide or fix their perceived deformity
Acknowledgement of suffering	People with BDD suffer from poor quality of life, depression, and sometimes suicidal thoughts and behaviours
Lack of response of BDD to cosmetic procedures	BDD does not respond to cosmetic dermatologic or plastic surgery treatments
Availability of effective treatments	BDD is a treatable condition, but requires the use of pharmacological therapy as well as cognitive behavioural therapy

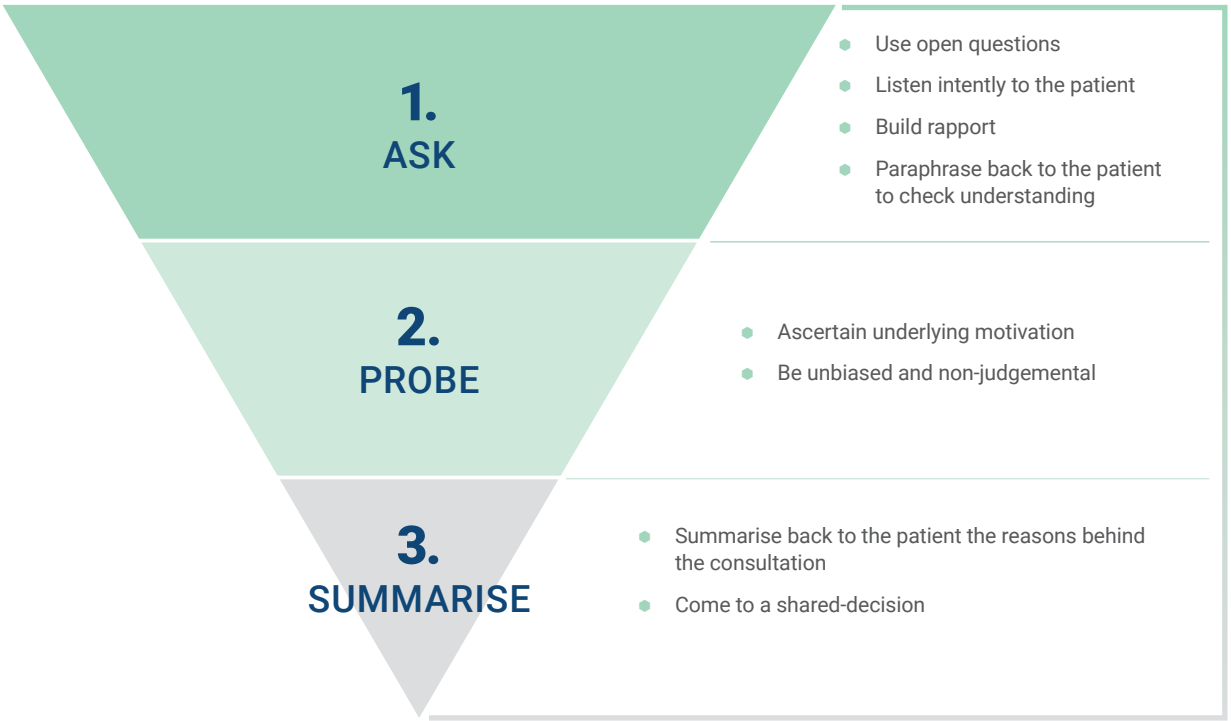

Figure 1. *The 'Empathic Communication Funnel' adapted from McDonald and Heydenrych.[13] The first stage focusses on rapport development and gathering pertinent information through the use of open-ended questions. The second stage involves delving into the underlying motivations by probing and use of reflective and hypothetical questions. The third stage is aimed at coming to a shared decision with the patient. Listening is key to all three stages.*

Are you very concerned about the appearance of some part of your body, which you consider especially unattractive?

YES ☐ NO ☐

If no, thank you for your time and attention. You are finished with this questionnaire.

If yes, do these concerns preoccupy you? That is, you think about them a lot and they're hard to stop thinking about?

YES ☐ NO ☐

What are these concerns? What specifically bothers you about the appearance of these body parts?

What effect has your preoccupation with your appearance had on your life?

Has you defect often cause d a lot of distress, torment, or pain? How much? (*circle the best answer)*

1	2	3	4	5
No distress	Mild, and not too disturbing	Moderate and disturbing but still manageable	Severe, and very disturbing	Extreme, and disabling

Has your defect caused you impairment in social, occupational, or other important areas of functioning? How much? *(circle the best answer)*

1	2	3	4	5
No limitation	Mild interference but overall performance not impaired	Moderate, definite interference, but still manageable	Severe, causes substantial impairment	Extreme, incapacitating

Has your defect often significantly interfered with your social life?

YES ☐ NO ☐

If yes, how?

Has your defect often significantly interfered with your school work, your job, or your ability to function in your role?

YES ☐ NO ☐

Are there things you avoid because of your defect?

Figure 2. *Body Dysmorphic Disorder Questionnaire-Dermatology Version (adapted from Dufresne).[59] To screen positive for BDD, patients must report the presence of reoccupation as well as at least moderate (score of 3 or higher) distress or impairment in functioning.[58]*

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Facial ageing

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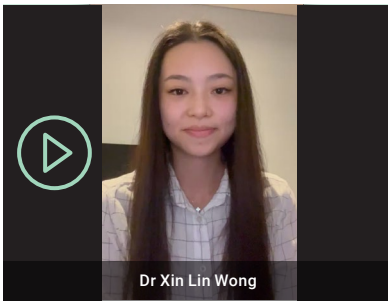

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OUTLINE: Facial ageing is a multifaceted process that affects all facial tissues, including the anatomically distinct facial layers. It is important to have a thorough understanding of the anatomy and biomechanics that exist between different tissue compartments to accurately treat facial ageing. This article provides a framework for understanding the ageing process in each layer and describes the characteristic signs of ageing by breaking down the face into superior, middle, and inferior thirds. It also discusses the most effective corrective options available to mitigate and counteract the effects of facial ageing.

KEYWORDS: fat compartments, SMAS, facial ligaments, bone resorption, facial thirds

ASCD References to come...

Introduction

The face is the most cosmetically sensitive region to age-related changes. The process of facial ageing is a continuous and intricate phenomenon that involves all facial tissues including skin, fat, ligaments, fascia, and bone structure, which are all interconnected and interdependent.[1] Senescence affects all populations and is visually evident due to a loss of harmony, symmetry, and balance of physical features. From a psychosocial standpoint, ageing can negatively impact the individual's emotional state and can potentially alter self-perception by reducing confidence and self-esteem. It can also affect how individuals are perceived by others as their projected emotions can be misconstrued.[2] The concept of ageing gracefully is closely associated with maintaining balanced and proportionate facial features, having smooth facial silhouettes and minimising the presence of blemishes and hollows.[2] Careful evaluation of the interplay between the distinct layers of the face, as well as regional changes within the face, are needed to achieve safe and sustainable modulation with modern cosmetic therapies.[3]

Ageing in layers

The complex topic of facial ageing can be delineated into layers and their interactions with each other. In the infraorbital region there is as little as three layers but this increases to nine layers in the temporal region.[3]

Broadly, the face can be divided into five distinct layers: skin (layer 1); superficial subcutaneous fat (layer 2); the superficial musculo-aponeurotic system (layer 3); loose connective tissue composed of ligaments and deep fat compartments (layer 4); and deep fascia and periosteum (layer 5) (Figure 1).[3] As deeper compartments deflate (layer 4 and 5) there is a decline in structural support for the overlying layers. This is further exacerbated by age-related elastosis of the skin (layer 1) and weakening of the fibrous attachments between each layer resulting in anatomical descension and soft tissue sagging.[4] Age-related change is seen in all levels with specific structures in each layer that contribute to the appearance of the ageing face.[3]

Skin

The appearance of healthy youthful skin relies on the maintenance of skin hydration with a intact skin barrier, structural support from the epidermis, dermis and hypodermis as well as a balanced extracellular matrix consisting of collagen and elastin.[5] There are countless molecular and cellular pathways that underpin the biological process of facial ageing.[5] Both intrinsic and extrinsic factors as well as their interaction will contribute to the appearance of aged skin. Changes to the structure of the skin leads to formation of wrinkles, increased laxity, poor skin texture and thinning of the underlying tissues.[5]

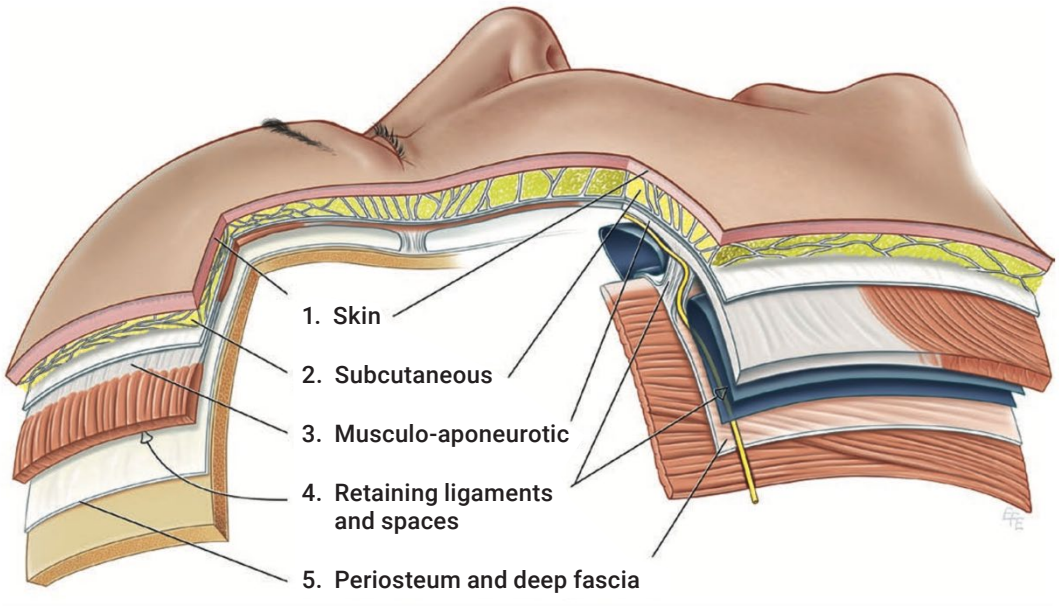

Figure 1. *Key facial layers consisting of skin, subcutaneous fat, superficial musculoaponeurotic layer, retaining ligaments and periosteum. Source: https://bmendelson.com.au*

Intrinsic factors

As we age, both the proliferative and metabolic capabilities of fibroblasts are diminished leading to a reduction in collagen and elastin.[5,6] The collagen fibres produced are not only thinner but reduced in density.[5,6] It is therefore unable to provide adequate structural support for the extracellular matrix which contributes to the appearance of aged skin.[5,6] Elastin fibres are prone to degeneration from calcification leading to breakdown of the elastic fibre network and a loss of elasticity from the skin.[5] With increasing age there is also an accumulation of gene mutations caused by free radicals leading to deoxyribonucleic acid (DNA) instability, alterations in mitochondrial function and compromised cellular metabolism.[5] Free radicals and reactive oxygen species (ROS) are highly reactive and cause damage to critical cell structures. Repeated insults over time reduce the repair capacity of DNA resulting in mutations or apoptosis if damage is irreparable. Declining sex hormones in combination with decreased levels of melatonin, cortisol, thyroxine, growth hormone, insulin-like growth factor and vitamin D 1-25 hydroxy have also been found to be significant factors in the acceleration of biological ageing.[5]

Extrinsic factors

Amongst extrinsic causes, ultraviolet radiation (UV) is the leading contributor towards premature ageing.[5] UV radiation greatly accelerates the ageing process through production of ROS, increased activation of matrix metalloproteases (MMP) and formation of thymine dimers.[5] MMP induces collagen and elastin degradation causing an accumulation of abnormal disordered elastic and collagen fibres.[7] Elastic fibres become disordered both in number and thickness leading to the degradation of the elastic fibre network.[2,5,7] The replacement of a healthy extracellular matrix with dysfunctional and disorganised proteins leads to the development of solar elastosis.[2] Sun-aged skin has also been found to have an inappropriate increase in glycosaminoglycans. The accumulation of these compounds is poorly organised and alters the regulation of hydration leading to the characteristic leathery appearance of photodamaged skin.[2] Other environmental factors that lead to a pathogenic accumulation of DNA mutations include air pollution, smoking and malnutrition, which interacts and contributes to the physiological process of ageing.[5]

Fat

Facial adipose tissue is organised in two parallel layers which is divided by a superficial fascial layer referred to as the superficial musculoaponeurotic system (SMAS, layer 3).[1] The two compartments are named by their relation to the SMAS termed the superficial subcutaneous fat (layer 2) or the deep fat layer (layer 4) which is continuous with the general fat present in the body.[2,3] Superficial and deep facial adipose layers can be further delineated into morphologically distinct compartments (Figure 2).[2] It is appreciated that irrespective of body mass index, there is an overall age-related reduction in facial fat mass leading to a decrease in facial soft tissue thickness.[1] This overall volume reduction leads to structural and contour changes within and across the superficial and deep fat compartments, contributing to the appearance of facial ageing.[8]

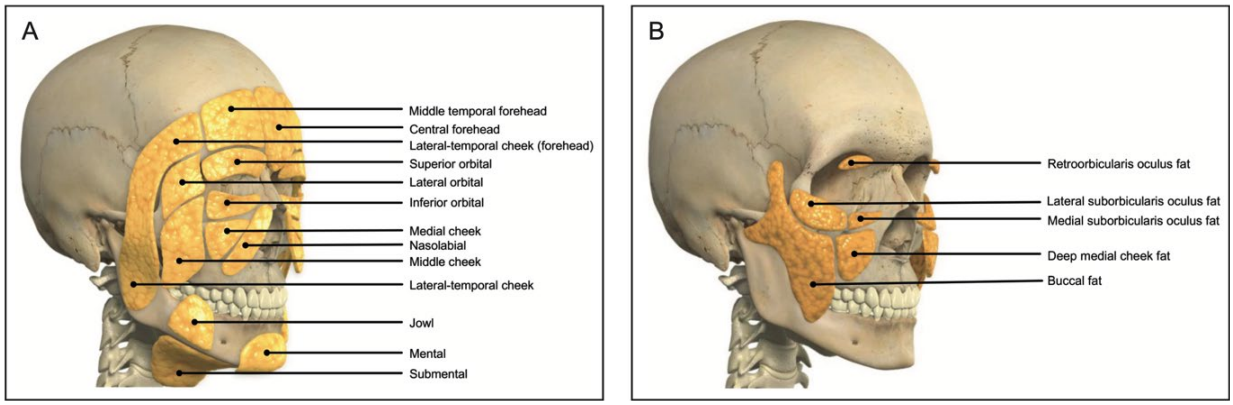

Figure 2. *Fat pads of the face can be characterised as superficial (A) or deep (B) compartments. Superficial fat pads include distinct forehead, periorbital, midface and lower-face compartments. Deep fat compartments include the retro-orbicularis oculi fat (ROOF), medial and lateral sub-orbicularis oculi fat (SOOF) as well as the deep medial cheek fat and buccal fat pads. Adapted from Swift et al.*[2]

Superficial fat

Superficial subcutaneous fat is compartmentalised by fibrous septae.[3] The fat lobules in these septae are finely lobulated, continuously distributed and arranged in a tight uniform fashion.[9] The superficial fat compartments are exposed to both resting and dynamic tension exerted by the facial mimetic muscles.[2] With increasing age, superficial facial fat is thought to redistribute due to the presence of gravitational forces and can either hypertrophy or atrophy depending on anatomical location.[2] Superficial fat pads can be broadly divided into the forehead (central, middle temporal, lateral-temporal-cheek), periorbital (supraorbital, lateral orbital, infraorbital), midface (nasolabial, medial cheek, middle cheek, lateral temporal-cheek) and lower-face region (superior jowl, inferior jowl, mental, submental).[2,4] With age, fat atrophy is most observed in the superficial fat compartments of the glabellar, temporal, and superior and lateral orbital regions.[2,5,8] Fat pad redistribution and protrusion are seen in the infraorbital, lateral nasolabial fold, labiomental crease, malar and submental jowl region due to gravitational forces and the biomechanics of the ageing face.[2,5,8]

Deep fat

Deep fat compartments include the retro-orbicularis oculi fat (ROOF), medial and lateral sub-orbicularis oculi fat (SOOF) as well as the deep medial cheek fat and buccal fat pads.[2] The deep fat compartments contain larger loosely organised adipocytes that are arranged in a disordered pattern.[9] The deep fat layer is relatively immobile due to the firm attachment to the underlying periosteum. With age, deep fat pads can deflate leading to subsequent loss of contour and inadequate support for overlying fat compartments causing a superficial fat "psuedoptosis".[2,4] Deflation of deep fat pads can contribute to facial ageing and filler injections are a popular method of targeted restoration of deep fat pad volume loss.

Adipocytokine and adipogenic activity of fat

Apart from providing structural cushion and energy storage, adipose tissue has recently been shown to be metabolically active and integral to normal skin functioning and actively supports skin health, elasticity, and appropriate response to injury and disease.[8] The effectiveness of adipose tissue in supporting both skin and metabolic health depends on both the quality and quantity of the adipocytes present.[8]

Dermal white adipose tissue is located directly inferior to the dermis and has a complex intertwined relationship with the overlying papillary and reticular dermis. Dermal white adipose tissue is involved in influencing and promoting dermal fibroblast activity through the secretion of adipocytokines.[8,10] Both dermal adipocytes and fibroblasts are derived from the same precursor cell and exhibit a high degree of plasticity allowing them to be able to trans-differentiate into each other throughout the murine hair cycle.[8,10] Adipocytes can influence insulin resistance and the inflammatory cellular milieu within the adipose tissue, which then regulates the quantity and quality of adipogenesis with flow on effects to the dermis, subcutaneous fat pads, and the appearance of facial ageing (Figure 3).

Adipose tissue plays a fundamental role in metabolic homeostasis by adjusting to biological stressors through the process of hypertrophy and hyperplasia (Figure 3). Healthy fat is predominantly hyperplastic whereas unhealthy fat is predominantly hypertrophic. Adipogenic hypertrophy occurs when there is absorption of free fatty acids into existing adipocytes whilst adipogenic hyperplasia refers to the formation of new adipocytes.[8] The balance between hypertrophy and hyperplasia determines healthy versus unhealthy adipose tissue. Poor diet, excessive UV exposure or excess inflammation can disrupt this equilibrium and create excessive hypertrophic adipocytes.

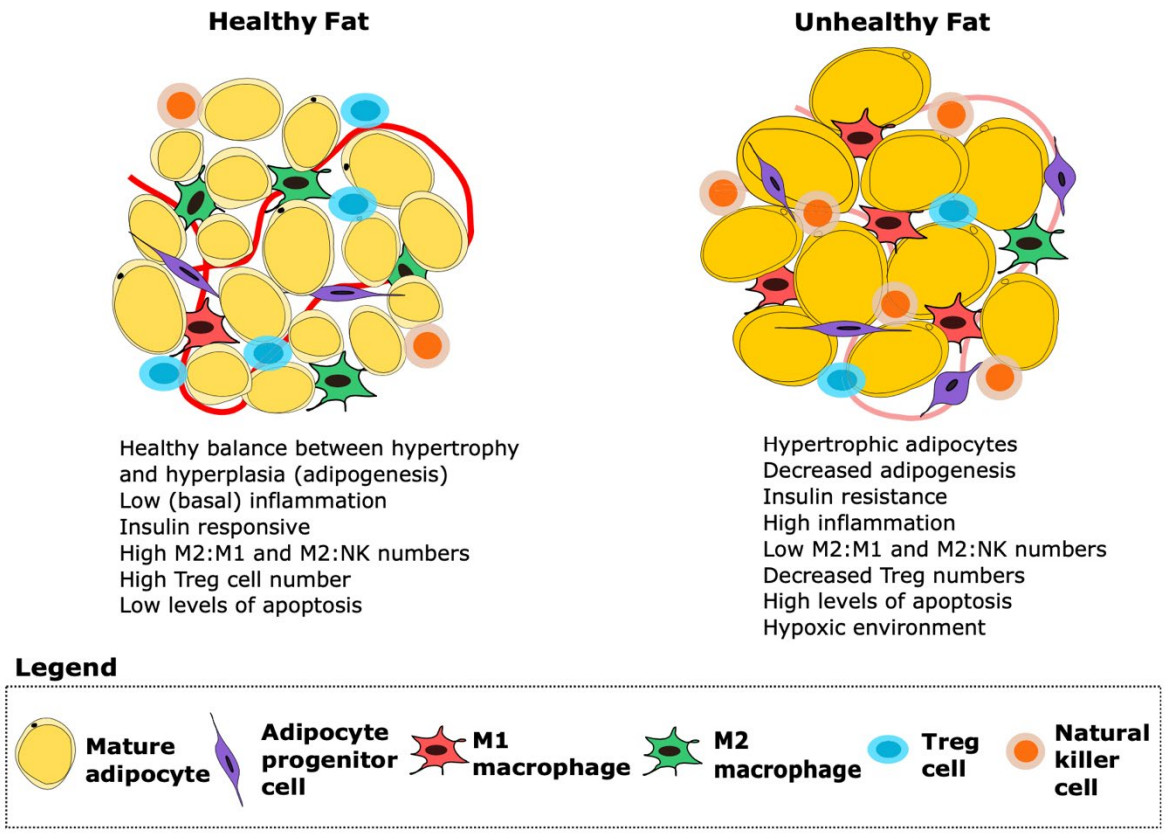

Figure 3. *Healthy versus unhealthy adipose tissue; healthy fat is predominantly hyperplastic and low inflammation whereas unhealthy fat is predominantly hypertrophic and high inflammation. Adapted from Galanin et al.*[8]

Excess hypertrophic adipocytes lead to increased secretion of MMP which can weaken the supporting extracellular matrix and produce dermal thinning. Hypertrophy of adipocytes also leads to a change in the secretome that favours a principally pro-inflammatory phenotype.[8] Hypertrophic adipose tissue is less rigid than its healthy counterparts resulting in insufficient structural support for overlying structures.[8] A disproportionate increase in facial adipose corresponds with a reciprocal loss of collagen, elastin and subsequent facial sagging.[8] Intriguingly, it is unknown whether fat modulating procedures such as cryolipolysis or deoxycholate lipolysis could have any long term effect on the subsequent metabolic and biochemical integrity of the treated adipose tissue.

Muscle

The dynamic movements of the face are dependent on the muscles of facial expression.[1] With time and repeated contraction, facial muscles lengthen and resting muscle tone is closer to that of maximum contracture.[2,3] This increase in tone exerts a greater pull on atrophied facial tissue leading to the projection of hyperdynamic caricature-like expressions such as a prominent scowl or exaggerated down-turning of the corners of the mouth.[2]

A significant indication of age is the evolution of dynamic facial lines into permanent static facial wrinkles. This occurs due to repeated muscular contraction, loss of skin elasticity and the consequent accentuation of skin creases and is particularly evident in the glabellar/scowl area due to active nature of the corrugator supercilia and procerus muscle.[1,3,5]

Bone

The facial bones play a fundamental role in the appearance of ageing. It acts as the structural foundation for the superficial soft tissue giving stability and definition in youth.[2] The skeletal bones of the face undergo lifelong remodelling characterised by cartilaginous changes and bone resorption (Figure 4).[2,3] This gradual loss of bony projection and correlated loss of structural support leads to gravitational descent and drooping of the overlying soft tissue.[1,11] Craniocaudally there is protrusion of the glabella, increase in craniofacial convexity, recession of the superomedial and inferolateral orbital rims (Figure 4), increase in length and width of the nasal aperture, increase in breadth and depth of facial dimensions, and maxillomandibular shrinking leading to a shortened facial height.[2,3,11]

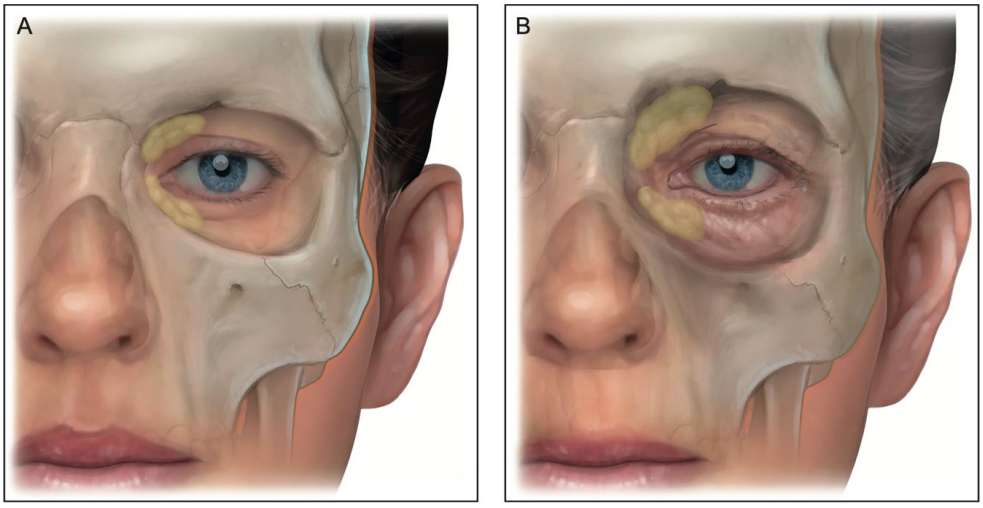

***Figure 4**. Orbital bone resorption: comparison between a young adult (A) versus older adult (B). There is progressive age-related recession of the superomedial and inferolateral orbital rims, resulting in prominence of brow and tear troughs. Adapted from Swift et al.[2]*

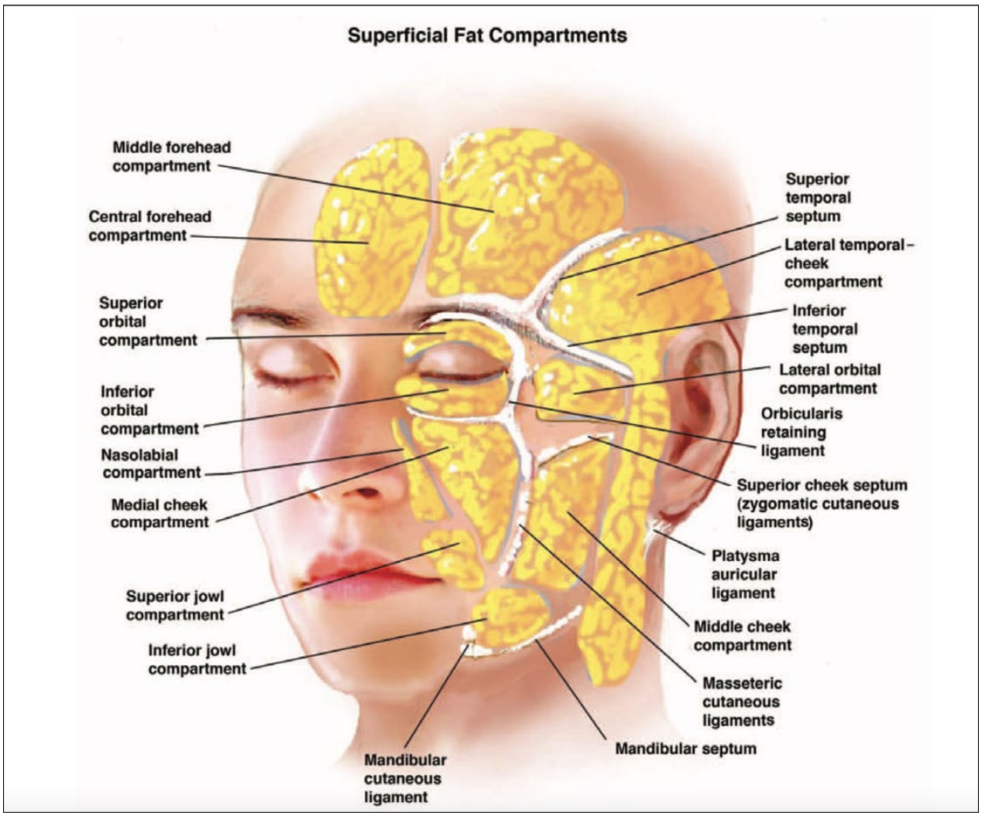

***Figure 5.** The facial retaining ligaments in relation to the superficial fat compartments. Adapted from Alghoul et al.[12]*

The most significant change occurs in the jaw where bone volume reduction causes a decrease in the maxillary angle but an increase in the mandibular angle leading to chin and jaw projection.[2,3,11] Age-related changes in the underlying bony structures of the face lead to consequent displacement of fat pads, weakening of supporting ligaments and development of skin wrinkling.[2]

Facial ligaments and biomechanics

The retaining ligaments are strong fibrous attachments, which help anchor, stabilise, and divide the face into superficial and deep compartments (Figure 5).[12] They originate from the periosteum or deep fascia and travel perpendicularly to insert onto the SMAS.[11,12] The ligaments can be further subdivided into

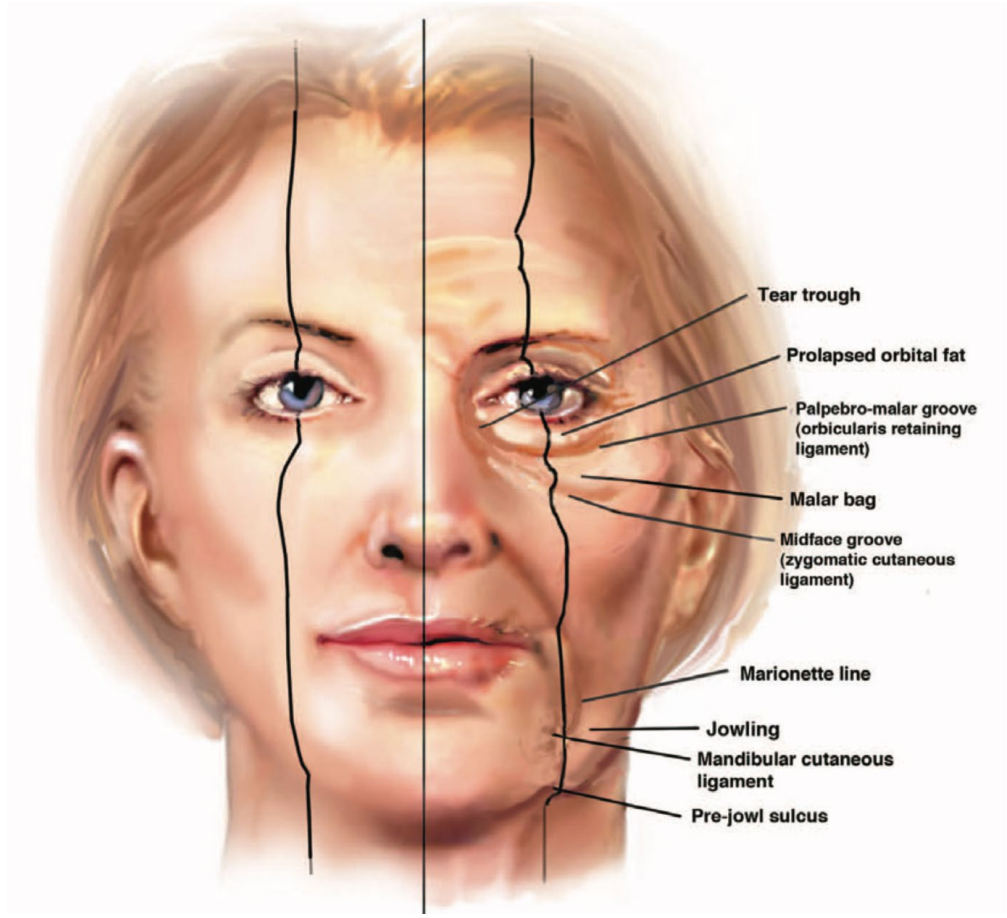

Figure 6. *Facial ageing as they relate to the retaining ligaments. The retaining ligaments form grooves and folds that become accentuated by age-related soft tissue protrusion and ptosis. Adapted from Alghoul et al.*[12]

osteocutaneous ligaments (zygomatic, mandibular cutaneous) which originate from the periosteum, and fasciocutaneous ligaments (masseteric, parotid cutaneous) which coalesce between the superficial and deep fascia of the face (Figure 5).[12] The SMAS gives rise to several finer retinacular ligament branches that traverse the subcutaneous fat layer and attach onto the dermis. These structures, termed the subcutaneous fibrous septa, are susceptible to weakening over time due to repeated facial muscle movement.[11] The facial retaining ligaments on the other hand, do not appear to undergo significant age-related change, and are responsible for the bulging and sagging seen in ageing due to the gravitational descent of unsupported soft tissue adjacent to the ligaments.[12,13]

The interchange between the medial and lateral face is delineated by the functional boundary formed by the line of ligaments.[1] This landmark also correlates to a change from oblique fascial arrangement in the medial face to a parallel arrangement laterally.[14] This difference in distribution is due to the lack of facial muscles and major facial ligaments in the lateral face. Consequently, due to the absence of supportive structures, the lateral facial soft tissue is particularly vulnerable to the effects of gravity and tends to exhibit earlier signs of age-related sagging and ptosis than the medial region.[1] Similarly, the vector for rotational or advancement flaps and lifts are typically laterally and inferiorly based.

Ageing by facial thirds

By breaking down the face into thirds, clinicians can conveniently identify the most prominent signs of ageing affecting their patient. Typically, ageing in the upper third of the face is usually seen through formation of wrinkles and changes related to the eyes. The middle third is mostly characterised by changes in the nasolabial region whilst ageing in the lower third is most noticeable around the lips. By using this approach, clinicians can better understand the specific needs of their patients and create personalised treatment plans that address their concerns.

Ageing in the upper third

In the upper third of the face, ageing can be identified by an increase in forehead height due to receding

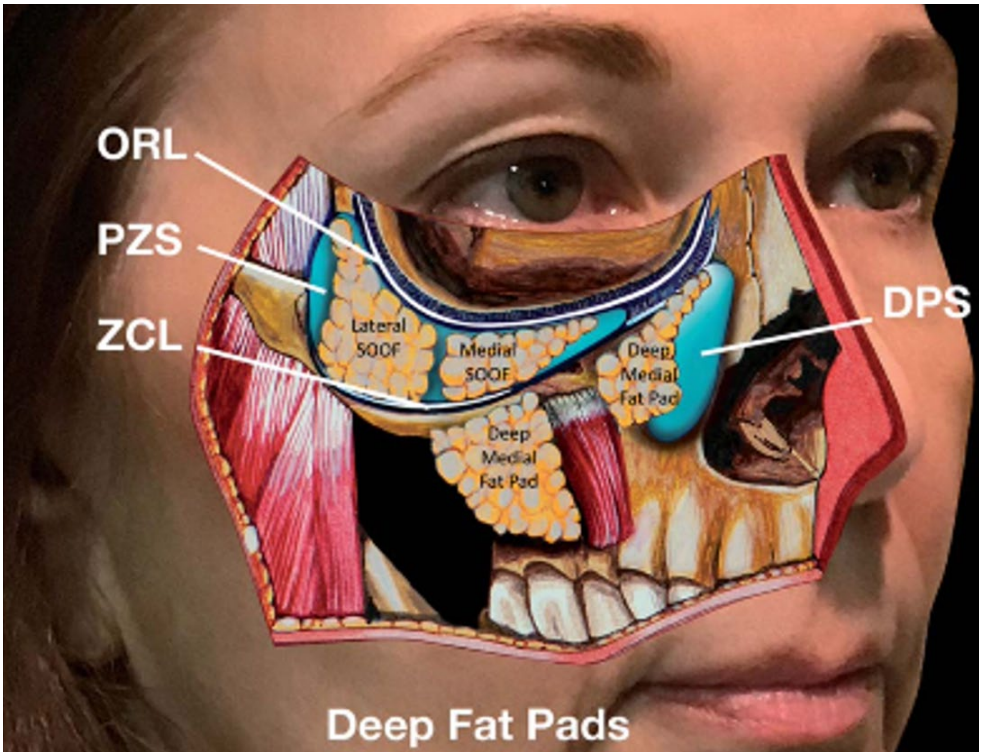

Figure 7. *The orbicularis retaining ligament (ORL) and the zygomatic cutaneous ligament (ZCL) coalesce medially to form the tear trough. Tear troughs become prominent when there is deflation of the deep fat compartments (shown) along with protrusion of the superficial infraorbital fat compartments (not shown).*
DPS, deep pyriform space; PZS, prezygomatic space.
Adapted from Lipp et al.[15]

hairline and brow ptosis. Wrinkle formation exacerbates this appearance with the development of forehead lines, glabellar rhytids and crow's feet. The lateral eyebrow begins to sag due to atrophy of the ROOF and bony resorption of the frontal bone.[2] Flattening of the medial forehead also results in blunting of the nasofrontal angle contributing to decreased projection.[2]

The formation of upper eyelid ptosis has a multifactorial contribution. In the deeper layers an increased laxity of the orbicularis retaining ligament and frontalis ligament leads to a decreased stability of the bordering orbicularis oculi muscle and drooping of the ROOF compartment.[3] In the superficial layers, there is usually atrophy of the supraorbital fat compartment and increased skin laxity. The presence of upper eyelid ptosis also causes elongation of the upper lid length and malalignment of the eyelid crease and upper eyelid margin which contributes to the appearance of ageing.[2]

Although the globe itself does not change in size with age, the development of senile enophthalmos gives a deep-seated or sunken appearance due to posterior retrusion of the eye and narrowing of the palpebral fissure.[2] Recession of the inferior orbital rim results in herniation of poorly supported superficial infraorbital fat inferiorly to create the classical appearance of eye bags.[2] Concurrently, there is pseudo-hypertrophy of the infraorbital fat which exerts traction on the lower eyelid and produces scleral show.[2] Infra-orbitally characteristic signs of ageing include deepening of the tear trough (medial orbicularis retaining ligament) and palpebromalar groove (lateral orbicularis retaining ligament) accentuated by adjacent superficial fat and soft tissue protrusion (Figure 6). Anatomically, the tear trough is formed by the merging of the orbicularis retaining ligament and the zygomatic cutaneous ligament (Figure 7).[15] This area appears hollow due to the presence of thin transparent skin overlying the orbicularis oculi muscle.[2] Tear troughs become increasingly visible with older age as the SOOF delates and there is anterior displacement of the superficial infraorbital compartment (Figures 6, 7).[2,3]

Ageing in the middle third

Ageing in the mid-face is predominantly caused by deflation of fat pads and loss of projection. As deep medial and deep buccal fat pads diminish, the superficial medial cheek fat pads and superficial temporal fat pads become ptotic in an inferomedial vector and gain adipose volume in the caudal aspect.[8] Weakening of the SMAS and retaining ligaments may also further contribute to the inferior movement of the fat pads.[2] This results in the appearance of lateral cheek atrophy, an enlarged submalar hollowing and flattening of the ogee curve.[2] The nose also shows signs of senescence

with tip ptosis which may give the visual illusion of a prominent dorsal hump or a prominent supratip.[2,16]

The development of the nasolabial sulcus is a common concern for many patients and has a multifactorial origin. Recession of the underlying facial bone (maxilla and mandible), lateral deep medial cheek fat pad atrophy, ligamentous fatigue, selective hypertrophy of nasolabial fat compartments against the traction of underlying muscles of facial expression, and laxity of overlying skin permits gravitational fat pad descent increasing its prominence.[1,2,4]

Ageing in the lower third

The lips are an important criterion of facial attractiveness and exhibit several aspects of age-related change. As the suborbicularis oris fat pad atrophies, the definition of the vermillion border and fullness of the lip is lost. The perioral region loses definition as the Cupid's bow flattens and there is formation of perioral wrinkles.[3] Additionally, due to the repeated actions of depressor anguli oris and platysma muscle there is down turning of the oral commissures and deepening of the "marionette lines" otherwise referred to as the labiomandibular folds.[3]

The jowl deformity is formed by the downward displacement of superficial and deep fat compartments against the mandibular ligament anteriorly and the masseteric cutaneous ligament posteriorly.[3]

The combination of superior and inferior jowl fat hypertrophy, mandibular mental recession, and increased skin laxity lead to formation of the anterior mandibular grooves and formation of prominent sagging pre-jowl sulci.[2,3] Additionally, the repeated contraction of the mentalis muscle may lead to a more noticeable groove or crease in the chin area, known as the mentalis creases.[2,17] As the skin ages and thins, the dermal attachments of the mentalis muscle with the SMAS may become visible in the chin resulting in a bumpy, dimpled "peau d'orange" texture.[2,17] In some individuals, static facial wrinkles such as perioral radial lip lines (smoker's lines) and medial cheek "smile lines" may develop from repeated muscular contraction and loss of skin elasticity.

Corrective options

Fundamentally, corrective options for facial ageing can be either surgical or non-surgical. Surgical options include blepharoplasty and face/neck-lifting procedures (rhytidectomy and SMAS tightening procedures). Non-surgical options include cosmeceutical, chemical peels, injectables (e.g., neurotoxin, fillers, deoxycholate) and energy-based devices (EBD). There has been a notable rise in popularity of cosmetic injectables and EBD over the last two decades to address facial ageing features (Table 1).

Target layer	Ageing features	Examples of scars
Skin surface	Colour: telangiectases, pigmentation Texture: elastosis, fine lines Laxity: rhytids, laxity	Cosmeceuticals, chemical peels Neurotoxins, fillers EBD (ablative and non-ablative) Threads Surgical rhytidectomy/lifts
Fat	Loss: hollowing, contour changes, descent, laxity Accumulation: jowls, pouches, contour changes	Fillers Autologous fat transfer Deoxycholate EBD (targeting fat e.g., cryolipolysis, HIFU, RF) Liposuction
Muscle/SMAS	Increase tone: dynamic and static furrow, grooves and bands Decrease tone: laxity, descent	Neurotoxins EBD (targeting muscle e.g., EMS) Surgical SMAS lifts
Bone	Contour changes Accentuation of all the above	Fillers Surgical implants Dental work

Table 1. *Facial ageing by layers from skin to bone and current corrective options*

EBD, energy-based device; EMS, electrical stimulation of muscle; HIFU, high intensity focused ultrasound; RF, radiofrequency; SMAS, superficial musculoaponeurotic system

Facial ageing can be categorised along cosmetic landmarks such as by facial thirds with specific reference to periorbital, midfacial and perioral regions, or along anatomical layers: skin, fat, muscle, and bone.

Skin surface ageing can manifest as colour and textural alterations. Colour changes such as blotchy erythema and dyschromia can be treated with vascular and pigment lasers/intense pulsed light, respectively. Textural changes can be targeted with injectables, medium-depth chemical peels and device-based skin resurfacing.

Age-related changes to deep and superficial fat pads are amenable to filler/autologous fat transfer, or fat dissolving (deoxycholate) injections, depending on whether the cosmetic issue is fat loss or accumulation. EBD such as cryolipolysis, high intensity focused ultrasound or radiofrequency can also focally induce fat reduction, for example, to the submental area.

More recently, electrical muscle stimulation to rehabilitate facial muscle tone and contour has been proposed as a useful therapeutic approach for facial ageing and laxity.[18]

Conclusion

Age-related changes occur in all layers of the face. Changes in the skin, SMAS, retaining ligaments, fat compartments and bone contribute to the appearance of facial ageing. A thorough understanding of the layered anatomical basis of facial ageing and the relationship between the various soft tissue compartments of the face allows for a more purposeful and targeted approach to the correction of facial ageing.

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Deoxycholate (ATX 101) in the treatment of facial and neck fat with emphasis on treatment of the jowl: A descriptive study and limited subjective survey

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BACKGROUND: Deoxycholate has been used to treat submental fat for over a decade in Australia. Concern over damaging local structures has led to the suggested on-label use.

OBJECTIVE: This paper is designed through a mixture of author's experience and a limited survey to illustrate changes that may be possible to further improve the patient outcomes with this agent.

METHODS: The method of utilising added local anaesthetic (0.2 mL per 2 mLL vial of 2% lignocaine) and feathering the injection volumes towards the edge of the fat deposit are methods that may be utilised wherever the injection is directed. Injection into the jowls is also described using the author's preferred method.

RESULTS: From a small survey of 16 patients, utilising the added local anaesthetic, 13 of these patients reported no pain or limited pain during the procedure. Adverse reactions were swelling (94%), bruising (44%) and erythema (19%). Of these patients, one treatment (38%) or two treatments (44%) were most often required. Twelve patients were primarily injected for jowls (although some were injected for both submental and jowls). Fifty-five percent of these patients rated their improvement at 55%. Of these patients, 85% would recommend this treatment to others. No nerve injury or long-term adverse reactions were noted in this survey.

DISCUSSION: Off-label use of deoxycholate extends its usefulness with the ability to decrease the pain of injection by addition of local anesthetic. Treatment of jowls seems efficacious and safe utilising the method of subcutaneous injection above and below the mandible to treat the visible jowl fat pad injecting away from the post jowl sulcus.

KEYWORDS: deoxycholate, deoxycholic acid, ATX-101, submental fat, jowls

ASCD References to come...

Introduction

The chin, neck, and jawline play an important role in facial beauty by completing the "oval of beauty" in females[1] and maximising the chiseled appearance in males. The features that seem to be important when viewing the lower face and neck are outlined in Table 1.[2]

The 'oval of beauty' dictates that all features should sit naturally on the oval outline that depicts the young attractive female face. Although there are outliers to this concept generally it holds true and the further one strays from this oval the less attractive the person may appear. Generally, it is the lower face that holds primacy in the portrayal of attractiveness, gender dimorphism and age.

With ageing, the well chiseled angular appearance of the male face or the gentle ovality of the female face is lost with changes occurring to the chin, jawline and neck. Fat in the neck and face is found in multiple discrete superficial and deep compartments. As we age these compartments behave differently to each other with some losing volume such as in the midface and often in the pre auricular zones but others gaining volume such as the jowls, nasolabial folds and the submental region.[3] Atrophy of other structures such as bone and the dermis, as well as changes in the structure and bulk of the muscle, contribute to a general softening and ptosis of the face and neck with a tendency to descend medially and inferiorly.

When viewed by decade[4,5] as in Table 2, aspects of facial and neck appearance gradually move further away from their youthful appearance.

This leads to a squaring up of the face and a heaviness of the jowls, and with weakening of the chin and medial ptosis of neck tissues, a submental heaviness. An exaggeration of muscular vertical bands and horizontal neck lines adds to this aged appearance (Table 2). The jowls and the submental heaviness are at least partially made up of fat, which is a target for lipolytic treatments.

However, treatment options available for the neck have lagged behind the face in terms of contouring and rejuvenation. Recently, there has been a growing emphasis on the neck and lower face as part of the overall assessment and treatment plan when viewing a patient's facial appearance.[6,7] Energy-based devices and cryolipolysis have been used for targeted fat reduction, however, these options require equipment purchase and often expensive consumables. Therefore, there has been a place for a simple nonsurgical treatment option to improve the contour of the chin and submental region through submental fat reduction. Options for treatment and expected age related changes by decade are explored in Table 2. Whilst there are many surgical (neck and lower face lifting, thread lifting, platysmoplasty, liposuction) and non-surgical procedures (botulinum toxin, tissue fillers, energy based and cryolipolysis devices and deoxycholate), they are best at certain ages commensurate with the relative degrees of severity in ageing of the lower face and neck. Although surgery is a possibility when the severity of the neck and lower face issues warrant this through liposuction or generally tissue lifting or plication manoeuvers, there are issues with any surgical procedure.[8] As a result, non-surgical methods are continually being explored and developed.

Table 1. *Visual features of the lower face and neck*

Region	Feature
Mandible	Distinct inferior mandibular border from mentum to angle with no jowl overhang
Anterior neck	Subhyoid depression (visually enhances the impression that the neck is thin and long)
Anterior neck	Visible thyroid cartilage bulge
Lateral neck	Visible anterior border of the sternocleidomastoid muscle, distinct in its entire course from the mastoid to the sternum (considered least important criterion)
Chin/anterior neck	Cervicomental angle between 105 and 120° (or a 90° angle between the sternocleidomastoid and submental lines). An angle greater than 120° appears as a double chin or heavy neck

Table 2. *Ageing changes by decade in jawline and neck and possible procedures (with preferred options in* ***bold****)*

Region	Age by decade	Description	Comments re treatment
Jaw line definition	25	Tight, no redundancy	**Nil**
	35	Softening of jaw line definition	Deoxycholate, energy-based and cryolipolysis devices, **tissue fillers, botulinum toxin**
	45	Some blurring of jaw line and redundancy of tissues with mild jowl formation	**Deoxycholate, tissue fillers**, botulinum toxin, **energy-based and cryolipolysis devices**
	55	Indistinct jaw line with quite obvious jowls	**Deoxycholate**, tissue fillers, botulinum toxin, energy-based and cryolipolysis devices, **tissue lifting procedures**
	65	Significant sagging obliterating jaw definition, severe jowls	Deoxycholate, tissue fillers, botulinum toxin, energy-based and cryolipolysis devices, **tissue lifting procedures**
Chin and neck loss of definition and redundancy	25	Good chin definition, no upper neck softening	**Nil**
	35	Mild loss of chin definition, upper neck softening	**Deoxycholate, tissue fillers**, botulinum toxin, tissue lifting procedures
	45	Mild loss of chin definition, moderate upper neck softening, moderate early midline redundancy	**Deoxycholate**, tissue fillers, botulinum toxin, tissue lifting procedures
	55	Blurred chin definition, significant general neck softening and fat accumulation and/or midline laxity	**Deoxycholate**, tissue fillers, botulinum toxin, **tissue lifting and surgical procedures**
	65	Blurred chin definition, severe general neck softening and fat accumulation and/or midline laxity	Deoxycholate, tissue fillers, botulinum toxin, **tissue lifting and surgical procedures**
Neck bands	25	None or mild vertical or horizontal neck bands at rest or on movement	**Botulinum toxin**
	35	Mild vertical banding on certain facial movement and/or mild horizontal bands	**Botulinum toxin (maybe microbotulinum toxin)**
	45	Moderate vertical banding on facial movement, mild at rest, often with moderate horizontal bands	**Botulinum toxin (maybe microbotulinum toxin)**
	55	Moderate to severe vertical banding on movement, moderate to severe at rest, moderate redundancy, moderate to severe horizontal banding	**Botulinum toxin, surgical procedures**
	65	Severe vertical banding on movement and at rest, severe redundancy, moderate to severe horizontal banding	**Surgical procedures**

Non-surgical fat removal has been a concept that is sought after by a large percentage of the population especially in countries such as Australia where obesity is an issue.[9] People have attended gyms, undergone radical diets or periodic fasting,[10,11] altered their exercise patterns and their lifestyles sometimes with good effect.

However, many of these attempts are restrictive or temporary, leading some to seek more permanent solutions to alter their fat deposition locally and thus energy-based devices and cryolipolysis[12] have been popularised. Concerns about safety issues with some of these technologies has also been an issue.[13] However, injectable fat removal options have become popular because of their ease of use and ability not to carry costly and space and time occupying machinery.

Mesotherapy with a mixture of phosphatidyl choline and deoxycholate was popular[14] before it was discovered that the previously considered inactive component of deoxycholate was probably the active agent and was efficacious without phosphatidyl choline.[15,16]

Trials on the use of injectable deoxycholic acid for reducing submental fat began in 2007.[17,18] This culminated in the on-label indication of reduction in submental fullness and is now available commercially in many countries under various names, such as Kybella (United States) and Belkyra (Canada, Australia, Europe, and South Korea); (Allergan Aesthetics, an AbbVie Company, Irvine, CA, USA). When injected into fat, deoxycholic acid induces destruction of adipocytes (adipocytolysis), with a local tissue response that includes (a) transient inflammation with lymphocytic and mononuclear cell infiltration, (b) macrophage clearance of cell debris and liberated lipids, and (c) longer term fibrosis, a process that together may promote tissue tightening.[19]

Off-label use of deoxycholate has been explored especially for treatment of jowls where it is particularly efficacious.[20-23] This article explores the use of deoxycholate to inject the jowl region and is discussed from the author's experience and a small survey of patient feedback.

Treatment methods

The standard on-label method of injection is well described and involves mapping out the submental area above the hyoid bone and laterally to the area ending at the pre masseteric notch but leaving an area of 1-2 finger breadth below the inferior border of the mandible but coming up to meet the chin superiorly. A grid is provided with a template sticker that guides this placement. The treatment dots are placed 1 cm apart and the area of fat mapped out and excess spots are wiped away. 0.2 mL is injected in each zone and usually 1-3 vials (2-6 mL) is used in a session with more volume and more sessions required in males than females. The injection of higher concentrations than suggested does not seem to enhance outcome.

Off-label treatment tips influencing method of injection

Treatment volumes per injection may be varied dependent on fat distribution. Often 0.1 mL may be injected towards the periphery of the deposit.

Off-label, plain 1-2% xylocaine (lidocaine, lignocaine) may be pre added to deoxycholate to lessen the pain of injection. Usually this is added at about 10% of the volume of the deoxycholate, i.e., 0.2 mL of xylocaine to 2 mL of deoxycholate. After adding the local anaesthetic to the vial, the vial is swirled before being drawn up into 1 mL syringes for injection.

Injections may be performed, again off-label, into the jowl area. Palpate the pre masseteric notch on the jawline and confirm by asking the patient to clench their teeth. This is marked as the posterior border of the injection (Video 1). This region should not be directly injected as it marks the crossing point of the facial artery and the marginal mandibular branch of the facial nerve. It usually conforms to the posterior aspect of the jowl. The jowl is marked out whilst patients are in the sitting position and usually has superior and inferior projections (Figure 1). The area is usually an oval or circular shape extending above and below the jawline, ending at the prejowl sulcus medially.

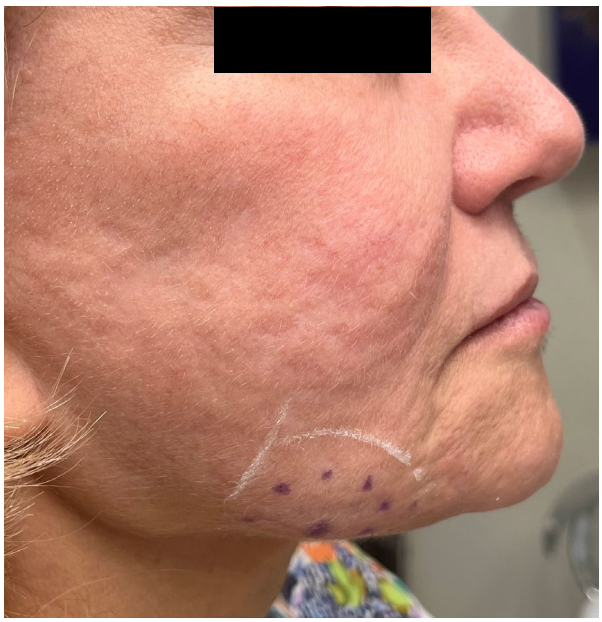


Figure 1. *The jowl above and below the mandible is outlined whilst patients are in the sitting position. The position of the post jowl sulcus is marked by a vertical line and represents the posterior limit of injection. All injections are directed medially from this point to avoid damage to the main trunk of the marginal mandibular branch of the facial nerve as it crosses the jawline.*

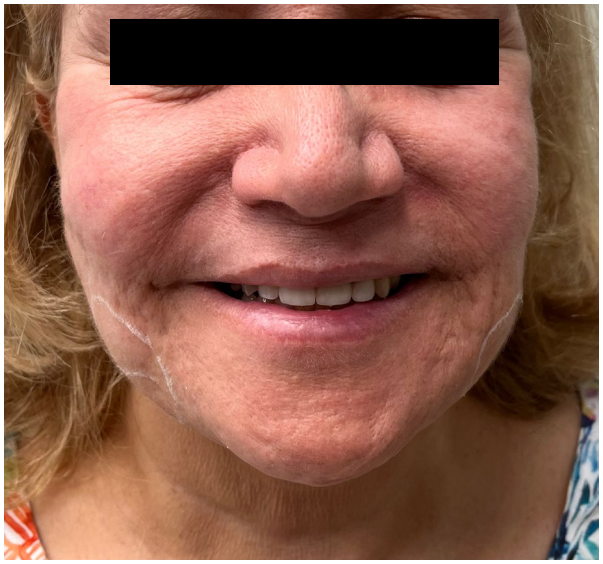


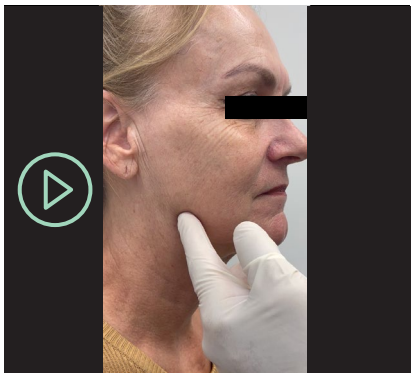
Figure 2. *Having the patient smile before the procedure allows one to note any precedent issues that may be confused with marginal mandibular nerve paresis following treatment. The patient should smile again after the procedure to see whether the deoxycholate containing the local anaesthetic has affected the smile pattern.*

The author's method of injecting these is to have the patient smile before the procedure noting any precedent issues that may be confused with marginal mandibular nerve paresis following treatment (Figure 2). This exercise of smiling is repeated after treatment. Although the nerve paresis due to deoxycholate may be delayed, the addition of lignocaine to the deoxycholate may be an early indicator of future nerve issues. If the smile is unchanged the patient is perceived to be less at risk of future issues.

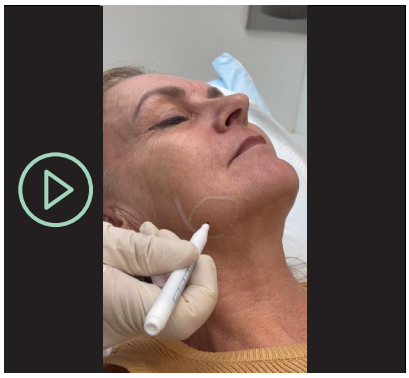
The entire jowl is marked out for injection at 1 cm intervals using a surgical marker (Figure 1, Video 2) and treatment is by usual subcutaneous injection with 0.2 mL per injection points but with 0.1 mL injection points peripherally especially posteriorly. Subcutaneous injection should be performed only, and injection should be ceased before attempting to remove the needle from its placement. Pulling the tissues towards the injector is a useful measure of ensuring the correct depth is injected. The author tends to inject at about a 45 to 60 degree angle in this region, not at 90 degrees as the fat compartment here is usually not as voluminous as the submental zone and the nerve lies deeply in the jowl region. Usually, a single vial is required per session with 5-6 injection points per side. All injections are performed pointing medially away from the post jowl sulcus. Injections are performed above and below the mandibular ramus (Video 3).

A small survey was conducted and sent by Survey Monkey to patients who were identified to have had submental and jowl treatments about their experience and satisfaction from a single clinic database and available for completion during a very limited time period (30 days) from the author's patients.

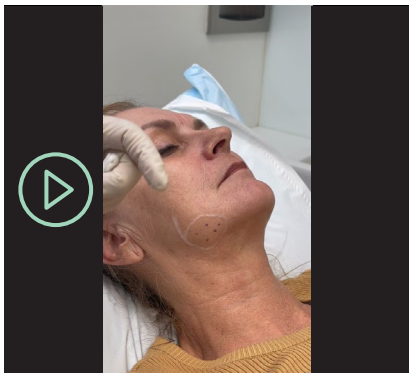
To watch video clips – click on image below to play via Vimeo.com



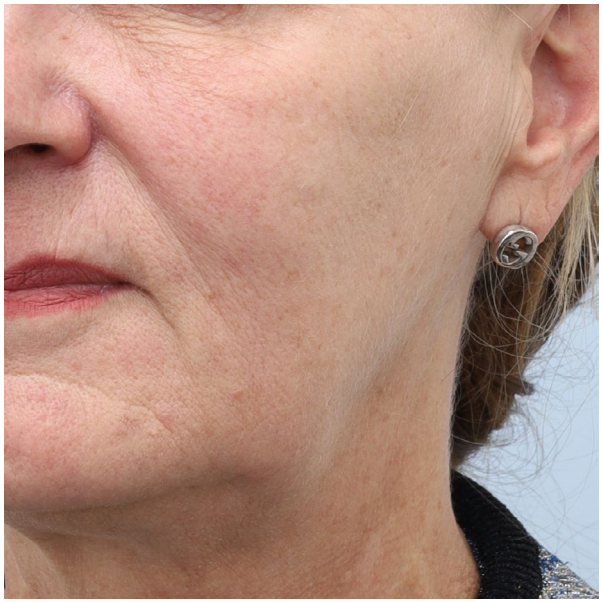
Video 1. *Palpation of the pre masseteric notch on the jawline and confirmed by asking the patient to clench their teeth. This is marked as the posterior border of the injection and the jowl region marked out for injection.*

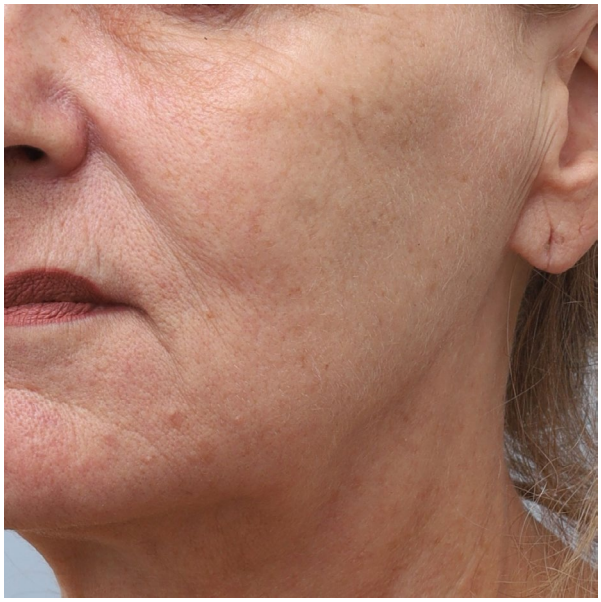


Video 2. *The area is marked approximately 1 cm apart for injection.*



Video 3. *The injections are performed about a 45 to 60° angle in the jowl (not at 90°) as the fat compartment here is usually not as voluminous as the submental zone and the arborisations of the marginal mandibular nerve lie deeply in the jowl region. Usually, a single vial is required per session with 5-6 injection points per side. All injections are performed pointing medially away from the post jowl sulcus. Injections are performed above and below the mandibular ramus.*





Figures 3a & 3b. *A typical responding patient is seen to have less jowl prominence on left oblique view.*

Results

Survey results were collated. This was a small survey outcome, limited by the short time we allowed for reply and the wish to have jowl patients included. Sixteen patients responded to the request for feedback in the time allowed (30 days).

Twelve patients (75%) were primarily interested in treatment of jowls and 4/16 (25%) in submental fat. Survey participants were asked about pain during and after the procedure. During the procedure 13/16 either had no pain (4/16, 25%) or mild pain (9/16, 56%), with the remaining patients experiencing moderate pain (3/16, 19%). This was similar to the discomfort experienced after treatment with no pain experienced in 5/16 patients (31%), mild in 8/16 (50%) and moderate in the remaining 3/16 patients (19%). It should be emphasised that lignocaine was added to deoxycholate before the procedure.

Almost all respondents experienced swelling (15/16, 94%) with 7/16 (44%) experiencing bruising and 3/16 (19%) experiencing redness.

Social downtime varied a lot between respondents with 4/16 (25%) having none, 6/16 (38%) having less than 7 days, 4/16 (25%) between 7-14 days and 2/16 (13%) over 14 days.

The number of treatments to reach the desired effect ranged from one (6/16, 38%) to two (7/16, 44%) with the remaining 3/16 (19%) requiring three treatments.

When all treatment areas were considered, 10/15 patients (67%) rated their improvement as >50%, 3/15 (20%) rated improvement as 25% and 2/15 patients (13%) saw no improvement.

The next set of questions centred on the treatment of the jowls. Satisfaction levels were sought and of the 11 patients who answered this question, 6/11 (55%) were at least 50% satisfied, whilst 5/11 (45%) were 25% satisfied or not satisfied at all. Yet when asked whether they would recommend this treatment to others, 11/13 (85%) respondents said that they would recommend. When asked specifically about symptoms associated with nerve injury such as muscle weakness in the lower face when smiling, 12 patients answered and all 12 stated that no such adverse reaction was seen.

A typical responding patient with relatively minor jowls is seen to have less jowl prominence on oblique view following treatment (Figure 3a and 3b).

Discussion

Deoxycholate is an interesting agent in its efficacy, safety, and longevity of results. It also does not appear to induce laxity and may have an actual tightening effect.[24-27]

However, concerns about the facial nerve paresis seen in the initial studies with submental fat injection of 2% in premarketing studies have led to company product information notes suggesting no treatment should be

given in the neck, jawline or lower face and that this treatment should be limited to a relatively limited area of the submentum leaving a 1-1.5 cm area below the mandible as the uppermost region for injection.[20]

The author's personal experience is devoid of this complication (marginal mandibular nerve injury) having treated many patients with this agent over more than 15 years. For much of that time the author has incorporated jowls in treatment sessions but is cautious not to inject near the post jowl sulcus where the marginal mandibular branch crosses the mandible to lie superior to as it courses towards the chin. The nerve terminates in multiple arborising branches deep to the jowl fat pad.

The jowl fat pad seems to require less treatments than submental fat often 1-2 only with transient marginal mandibular nerve palsy and alopecia being the most likely adverse reactions. Pinching the skin away from deeper structures for injection is thought to be appropriate as the marginal mandibular branch arborises deep to the jowl fat pad. Many will only inject the jowl superior to the mandible, citing a danger area just below the mandible for nerve injury.[21] However, nerve injury was still noted in 5% of patients.

After crossing the mandible, the nerve is noted to be superior to the mandible in all cases when the nerve crosses the mandible anterior to the facial artery and most often but not always when it crosses posteriorly[22] and most often exists in several branches and further arborises when coming closer to its target musculature.[23] Hence the practice of only injecting superiorly to the inferior border of the mandible may not be a required edict. The author commonly injects above and below the mandible and has not encountered issues of nerve palsy. It may be important to inject into the fat but not too deeply nor superficially. Injections should also be directed away (medially) from pre-masseteric notch where the nerve and facial artery cross the mandible.

The limitations of this paper are the small sample size of the survey and the personal experience of the author as an attempt at proof of concept. This needs to be formally studied and jowls should be added to the target zones for deoxycholate if these studies prove to have a satisfactory safety and efficacy profile.

Conclusions

Deoxycholate may be delivered on-label as described in the submental zone, however, variations to this technique in an off-label extension include adding lignocaine to the agent and tapering to 0.1 mL as one comes to the edge of the fat deposit and injecting the jowls. In the author's opinion, the jowls can be marked out above and below the mandible and injected throughout this zone safely in this study by limited survey. Caution with the marginal mandibular branch of the facial nerve is always still recommended.

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New concepts in botulinum treatment for facial ageing

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OUTLINE: Although there are more similarities between the five botulinum toxins that are currently available, some differences, such as dosage, units, packaging, and methods of dilution, also exist. Understanding these subtle differences enable the different toxins to be harnessed for the cosmetic treatment of common areas, including the glabella, forehead, brow, crow's feet, and eyelids. Understanding the dynamics of these muscles and how they interact enables physicians to successfully treat wrinkles and contour changes in the structure. This article will discuss methods of choice to ensure successful treatment and avoid complications.

KEYWORDS: botulinum toxin, glabella, forehead, brow, crow's feet

ASCD References to come...

Cosmetic treatment with the botulinum toxin molecule has become the most common aesthetic procedure. In the United States, more than 5 million treatments took place in 2019, which reflects a 459% increase from 2000 to 2020, as reported by the American Society of Plastic Surgeons.[1] An estimated 12 million treatments occurred worldwide. Since Dr Carruthers first observed the unintentional smoothing of the glabellar lines when treating blepharospasm,[2] the procedure has evolved over the ensuing 20 years to become the most common cosmetic procedure in the world. The use of botulinum toxin has evolved beyond smoothing wrinkles to altering the facial structure, facial muscle symmetry and balance, as well as improving skin quality. None of the other aesthetic applications, which are currently available to enhance the ageing of facial and neck skin, have surpassed the efficacy, safety, and durability of botulinum toxin treatment.

Following the evolution of the primary botulinum neurotoxin type A complex, over six molecular forms, both complex and naked, are now manufactured. Despite significant manufacturing differences, many similarities exist between the different industrial toxins.

The toxins that are approved in the United States by the Food and Drug Administration (FDA) are outlined in Table 1 and are as follows: OnabotulinumtoxinA (Botox [US], Vistabel [UK]); AbobotulinumtoxinA (Dysport [US], Azzalure [Europe]); IncobotulinumtoxinA (Xeomin [US], Bocouture [UK]); PrabotulinumtoxinA (Jeuveau [US], Nabota [Korea]); DaxibotulinumtoxinA (Daxxify [US]); and Botulinum toxin B (Myobloc). Other type-A botulinum toxins that are used worldwide, include Neuronox, Innotox, and other toxins that are used in Korea and China.

The five FDA-approved type-A botulinum toxins have many attributes in common, including binding sites, mechanism of action, onset, duration, side effects, and immunogenicity. Differences between the toxins include the action, onset, and duration or field of effect, which are related to the dosage and dilution, rather than to the unique properties of each toxin. Subtle differences between the toxins allow the injector to tailor the treatment for each area and patient (Table 2). I will review cosmetic areas treated with injectable botulinum toxin and reflect on the updated treatment protocols in the next section.[3]

Table 1. *Characteristics of approved botulinum toxin A preparations.*

	Onabotulinum toxinA	**Abobotulinum toxinA**	**Incobotulinum toxinA**	**Prabotulinum toxinA**	**Daxibotulinum toxinA**
Preparation	Powder				
Storage conditions	Below 8°C	Below 8°C	Below 25°C	Below 8°C	Room temperature
Shelf-life	24 months	15 months	36 months	24 months	24 months
***Clostridium botulinum* strain**	Hall A	Ipsen strain	Hall A	Hall A	Hall A
SNARE target	SNAP25				
Purification process	Precipitation and chromatography				Chromatography
pH after reconstitution	7.4				
Stabilisation	Vacuum drying	Freeze-drying (lyophilisate)	Vacuum drying	Freeze drying	Lyophilisation
Excipients	Human serum albumin (500 µg/vial) NaCl (900 µg/vial)	Human serum albumin (125µg/vial) Lactose (2500 µg/vial)	Human serum albumin (1 mg/vial) Sucrose (5 mg/vial)	Human serum albumin (1 mg/vial)	Positively charged peptide; albumin-free
Biological activity	100 MU-A/vial	500 MU-I/vial	100 MU-M/vial	100 MU-M/vial	100 MU-M/vial

Table 2. *Comparisons of toxins.*

Comparison of Toxins

Types of botulinum toxin for cosmetic usage	Duration of action
Mechanism of action	Diffusion/field of effect
Compositional differences	Safety
Dosing	Resistance
Efficacy	Storage

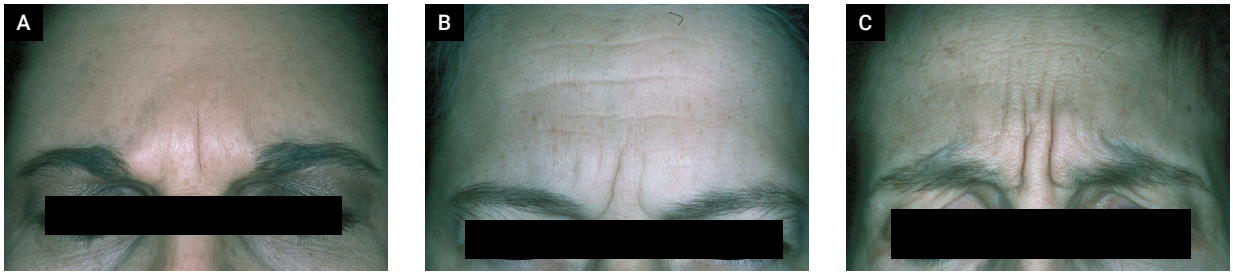

Figure 1. *The number of units depends on the severity of the frown. A: mild; B: moderate; C: severe.*

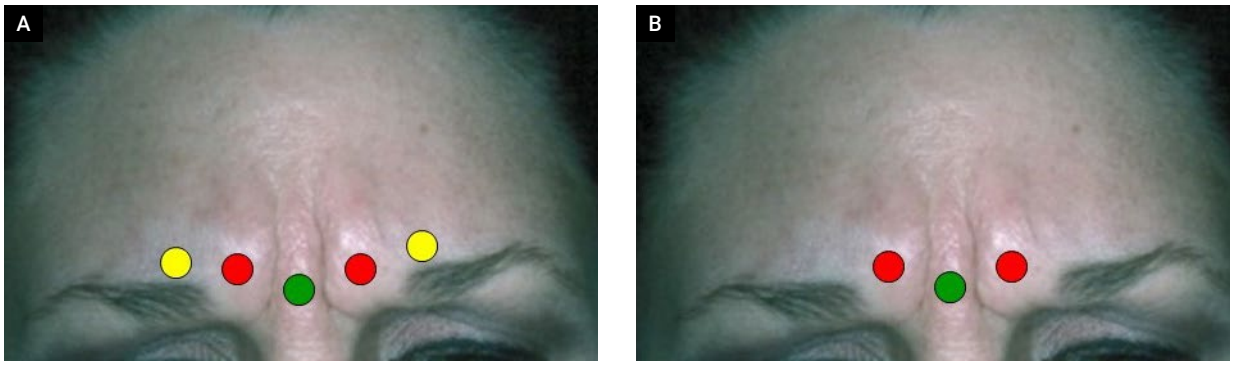

Figure 2. *The Muscle sites for botulinum injection showing the use of (A) five sites versus (B) three sites. Yellow: orbicularis; red: corrugator; green: procerus*

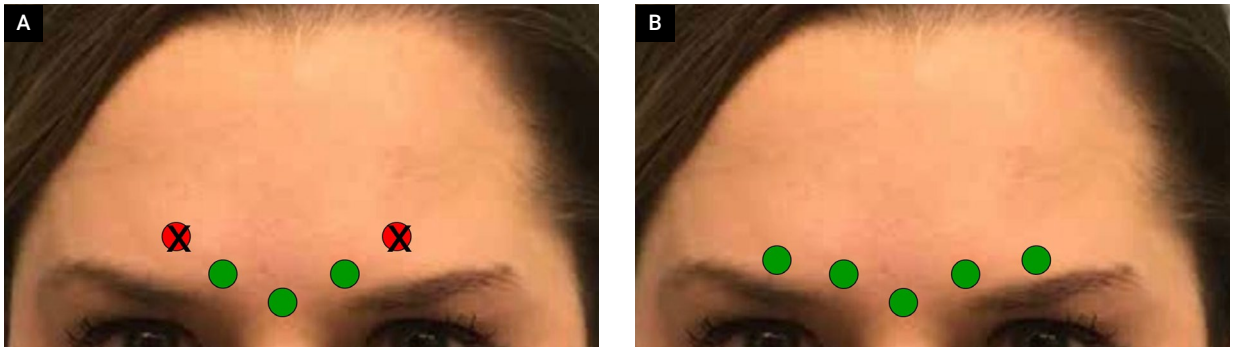

Figure 3. *Figure showing (A) incorrect lateral injection sites for the corrugators and (B) correct injection sites for the corrugators.*

Glabellar

Injection of the glabella is the original and, by far, the most common aesthetic use of type-A botulinum toxins. Although the five standard injection sites are appropriate for most patients, the dosage and injection technique must be individualised to each patient. It is important to assess the size and shape of each of the glabellar muscles, i.e., the corrugator, procerus, and orbicularis oculi. The muscle size and dynamic action determine the dosage. The evaluation places the muscle mass into three categories, i.e., small, medium, and large.[4] Small to medium muscle mass can be treated with 15 botulinum toxin units or 30 Speywood units, while large, more dynamic glabella muscles can take 20–25 botulinum toxin units or 50 to 60 Speywood units (Figure 1A–C). The corrugator should be carefully assessed to determine lateral or horizontal movement making the injection site of the lateral corrugator important.[5] With little lateral movement, only three injection sites may be necessary, especially with small muscle mass (Figure 2). The original injection points position the lateral corrugator site 1 cm above the orbital rim. However, this site is too high and may cause eyebrow/lid ptosis for many patients, particularly those with "lazy elevators" that depend on an auxiliary lift from inferior fibres of the frontalis muscle. It was previously thought the lower injections at the orbital rim could cause ptosis through the dispersion of toxin directly to the levator muscle. It has become apparent that injection of the lower frontalis plays a greater role in eyebrow/lid ptosis in susceptible patients. The lateral injection sites should not be more than 0.5 cm above the orbital rim for safety. Careful pre-treatment evaluation of eyelid position is important to document low-lying upper lids prior to treatment (Figure 3).[6]

Figure 4. *Results shown at rest with (A) no ptosis and (B) ptosis.*

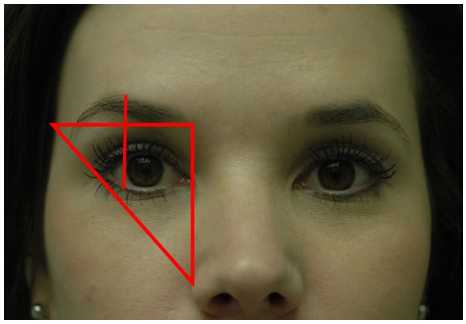

Figure 6. *Dimensions of the ideal brow. Lateral and medial edges of the brow fall into the same horizontal plane. The peak of the brow should be on a vertical line tangential to the lateral limbus.*

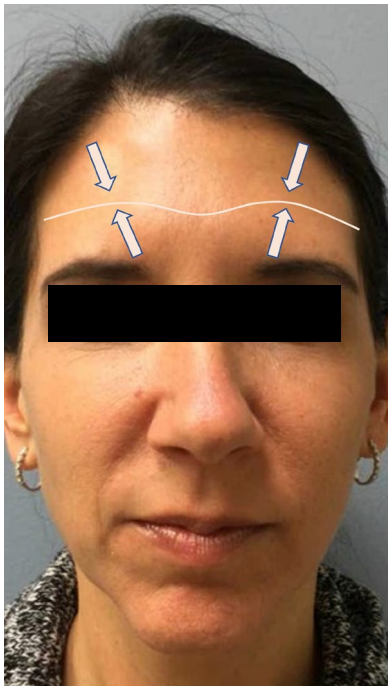

Figure 5. *Image of the forehead showing brow elevators*

The standard eyelid position should be no less than 2 mm above the limbus at the mid-papillary line. Photographic documentation of the upper lid position prior to treatment is important, as well as a levator excursion test. Unrecognised upper lid asymmetry or depressed lid should be pointed out to the patient and used as a guide to the injection point and dosage of the lateral corrugator (Figure 4).[7]

Forehead

The frontalis muscle is the only brow elevator in the upper face. Injections to soften horizontal wrinkle lines on the forehead must consider the brow position. The upper two-thirds of the frontalis can be injected without worry of affecting brow position. The lower third, particularly the lateral areas, may affect the brow and cause unintentional ptosis. It must be noted that deep injection into the frontalis will give a full muscle effect while superficial injections may be safe in superficial wrinkles without causing brow depression (Figure 5).[8]

After the usual five glabellar injection points and six frontalis injections with adequate dosage as per muscle mass, most patients have a full effect of a smooth forehead without brow position change. The fine wrinkle lines above the lateral brows are more difficult to manage. Microdoses of botulinum toxin (0.25–0.5 BUs) injected into skin will only smooth most fine lines without brow changes. The brow position is a result of the balance of the elevator (frontalis) and the depressors (orbicularis, corrugator, and procerus). These are four to six injection points just above the brow injected into the skin only.[9] This is monitored by micro-injections producing a peau d'orange with only a 1–2 mm insertion of the needle.

Brow position can be elevated to correct brow ptosis or asymmetry. The position and shape of the brow is a major factor in determining facial shape. The ideal shape of the female brow has the following characteristics (Figure 6):[10]

- The brow should begin at a point 1 cm above the supraorbital rim along an imaginary line from the ala nasi through the medial canthus.
- The brow ends laterally, where an oblique line begins at the ala nasi through the lateral canthus.
- The beginning and end of the brow should remain in the same horizontal plane.
- The tail of the brow should be 1 to 2 mm higher than the head of the brow.

The shape and position of the brow can thus be changed with appropriate botulinum toxin injections into either elevators or depressors surrounding the brow. The corrugator is a major depressor of the medial brow and the orbicularis muscle laterally. The lateral brow has less support beyond the attachments to the

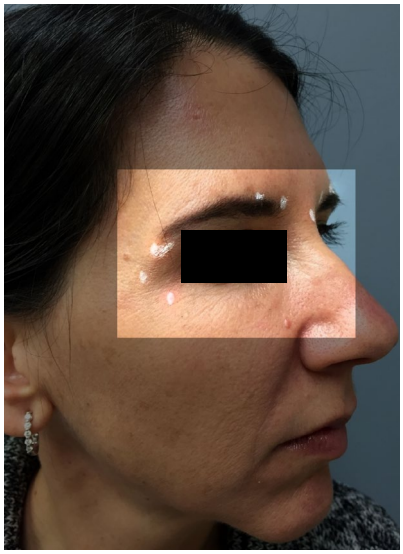

Figure 7. *Injection sites used to raise the brow.*

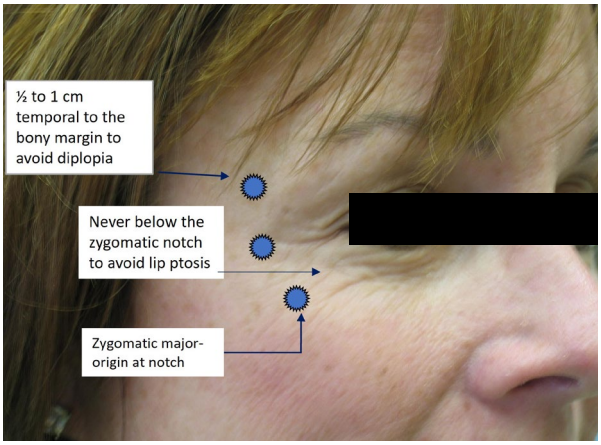

Figure 8. *Correct injection technique for crow's feet.*

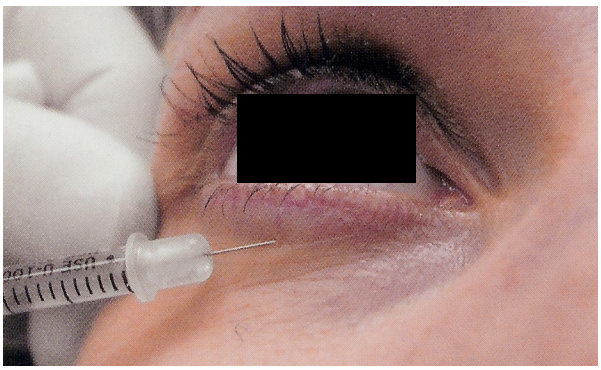

Figure 9. *Correct injection technique for infraorbital area.*

supraorbital ridge. Thus, it has a greater propensity to droop, which can be corrected by injecting small doses of concentrated botulinum toxin (3–4 aliquots lateral to the brow: superior, lateral, and inferior). One to two units should be placed superficially 1–2 cm apart into the orbicularis only, avoiding the superior temporalis muscle.[11] Ensuring this injection remains superficial reduces the risk of the toxin dispersing beyond the intended area and producing eyelid and brow ptosis (Figure 7). Deeper injections in this area may affect the lacrimal gland producing xerophthalmia or dry eye. The deeper injections into orbicularis can also create an abnormal elevation of the lateral brow. This creates a Mephisto or 'Mr. Spock' look on one or both brows. If this does occur, one or two units of botulinum toxin can be injected 1 cm above the tail of the brow, lowering it to a normal position.[12]

Lower eyelid and crow's feet

The third area in the upper face targeted with botulinum toxin treatment is the lower eyelid (including crow's feet). This is a continuation of lateral brow treatments to correct the wrinkle lines spreading to the lateral canthus and lower eyelid. The extent of the problem is demonstrated by smiling and activating the zygomatic muscles. The number of injection sites and positions is determined by the extent of lines beyond the lateral canthus. Care must be taken not to inject inferior to the orbital rim into the zygomaticus muscle as it would weaken the smile. The most common pattern is three injection sites into the orbicularis muscle surrounding the lateral canthus – superior, mid-canthus, and inferior at the lower eyelid. Three botulinum toxin units or six Speywood units are injected into each site (Figure 8).[13]

Lower eyelid wrinkles are also treated with a low dose, superficial intradermal botulinum toxin injection. This not only smooths wrinkles and crepitation of eyelid skin, but also produces a wide-eye youthful appearance. One to three units are placed intradermally to the lower eyelid margin below the mid-pupillary line (Figure 9). Placing this injection too laterally or too deeply can increase the risk of ectropion and the inability to firmly shut the eyes. This can cause photophobia, epiphora, or even corneal abrasion. Over-aggressive treatment can also weaken the pretarsal muscle increasing lower lid bulging.[14] Low volume, high concentrated injections with low doses to pretarsal and septal orbicularis oculi is the recommended treatment program for lower lid smoothing and to prevent complication.[15]

The use of botulinum toxin for facial rejuvenation should be individualised to the patient's facial shape, contour, skin type, and selective problems. Cookbook approaches to using the same injection points and doses in all patients should be avoided as that does not address individual desires or needs. The physician should carefully analyse muscle dynamics, asymmetry, and facial shape to tailor the injection results to fit the patient's overall facial appearance. Remember, a cosmetic procedure is performed to enhance or improve appearance and not detract from it.

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TCA and the treatment of atrophic acne scars

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OUTLINE: Acne scarring is a common sequelae of poorly managed acne vulgaris which has profound implications on the quality of life as well as mental wellbeing of those afflicted. Chemical peels have a long history both in the management of acne as well as subsequent scarring. Trichloroacetic acid (TCA) is a commonly used agent for chemical revision of acne scars. This article aims to explore the current literature on the efficacy and safety of TCA.

KEYWORDS: acne vulgaris, scars, chemical peels, chemoexfoliation, trichloroacetic acid

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Introduction

Acne vulgaris is a common condition for adolescents, with 20-90% being affected by the condition.[1] All body areas with high concentrations of the pilosebaceous unit may be involved and the common sites include the face, back, and chest. Lesions can result in permanent scarring with a marked impact on quality of life and self-confidence.[2] The pathogenesis of acne is complex involving inflammation around the pilosebaceous unit, abnormal keratinisation, and *Cutibacterium acnes*.[3] Trichloroacetic acid (TCA) is a commonly used peeling agent for chemical revision of acne scarring.

Classification of acne scars

Acne scars are polymorphic in nature and the morphology of the scar has profound implications in the modality of treatment that should be used.[4] Acne scars are divided into three main categories – ice pick scars, boxcar scars, and rolling scars, by the widely used classification system by Jacob et al.[5] Ice pick scars are narrow scars (<2 mm) with deep penetration vertically into the deep dermis and even subcutaneous tissue.[5,6] Usually, the surface opening is wider than the infundibulum of the scar but not always. Rolling scars are caused by tethering of the dermis to the subcutis as a result of scar tissues, which causes a shallow, rolling, and undulating appearance of the superficial epidermis.[5,6] Boxcar scars are depressions with sharply formed edges and the opening of the scar and the infundibulum have the same width, unlike ice pick scars.[5,6] Additionally, keloid scars, atrophic scars, and scars with mixed morphology can also occur as sequelae of acne.[4,7]

The treatment of acne scars depends on the morphology of scars that is present in individual patients. The treatment modalities can broadly be divided into energy-based and non-energy based.[4,7] Chemical peels are classified as a non-energy-based treatment modality and have been used in acne scar revision since the beginning of the 1900s.[4] Chemical peels induce controlled destruction of the epidermis and dermis to allow for the regeneration of healthy tissues from the epidermal appendages.

Trichloroacetic acid

The history of chemical peels can be dated back to the ancient Egyptians with the use of sour milk containing lactic acid and alpha-hydroxy acid.[8,9] The advent of TCA peels only began in the late 1950s, the popularity of which was not developed until the 1980s.[8]

TCA is a tricholorinated carbonic acid. The application of TCA concentration induces precipitation of epidermal protein and coagulative necrosis within the epidermis

and collagen bundles in the papillary and upper reticular dermis, allowing the skin to re-epithelialise from the preserved pilosebaceous unit.[9,10]

Chemical peel agents can be categorised depending on the depth of penetration into the skin tissue. Superficial chemical peels induce keratolysis and keratocoagulation in the epidermis and down to the epidermo-dermal junction to a depth of approximately 60 μm.[1,11] Medium-depth peels penetrate the papillary dermis and upper reticular dermis while deep chemical peels penetrate to the mid reticular dermis.[1,11] Extent of frosting, which represents the process of keratocoagulation, is commonly used as a surrogate marker for the depth of peel achieved by TCA (Video 1).

As a peeling agent, TCA harbours several advantages against its counterparts. Firstly, TCA can act as a superficial, medium, or deep peeling agent depending on the concentration. Between a concentration of 10-30%, TCA is a superficial peel, while at a concentration of 35-50% and 50% above, TCA becomes a medium depth and deep peel, respectively. Secondly, TCA is a self-neutralising agent which requires no neutralising agent to stop the peeling process.[12] Lastly, TCA is not absorbed systemically with no systemic side effects compared to other deep peel agents such as phenol.[12]

TCA as a superficial peel

Superficial peels are more commonly used in the treatment of active acne rather than acne scarring. Many of the superficial peeling agents also possess anti-comedonal activity and bacteriocidal properties which make them ideal for managing active acne vulgaris given the pathophysiology of the disease.[13] The efficacy of TCA as a superficial peel for acne scar revision does not have robust evidence within the current literature. A limited trial involving seven patients receiving 20% TCA peel shows moderate improvement to acne scars.[14]

TCA as a medium-depth peel

TCA between the concentration of 35-50% is used as a medium-depth peeling agent. Due to the potential risk of pigmentary alteration and scarring associated with TCA at high concentrations, TCA is often used in conjunction with other weaker peeling agents such as glycolic acid or Jessner's formulation in the setting of a medium-depth peel.[13] The use of a weaker peeling agent prior to TCA peel allows partial removal of the epidermis which facilitates a lower concentration of TCA to minimise complications.[13] Similar to the use of TCA as a superficial peeling agent, the current existing literature regarding medium-depth TCA peel is relatively dearth. In a small trial involving 14 patients, Al-Waiz and colleagues demonstrated moderate efficacy (moderate improvement as per Global Assessment Scale in 53.3% of patients enrolled) after three sessions of Jessner's peel followed by 35% TCA with no significant complications such as dyspigmentation or scar extension.[15]

TCA as a deep peeling agent

With regard to acne scar revision, the role that TCA most commonly serves is as a focal deep peeling agent. Deeper peels with TCA have increased complications of dyspigmentation and scarring; therefore, the application of such agents as a broad surface peel is inherently risky.[10] The current trend with TCA as a deep peeling agent is mainly in the form of TCA CROSS (Chemical Reconstruction of Skin Scars), which involves using high-concentration TCA on focal scars with a fine application to allow accurate targeting of the scar base while minimising spill-over on to healthy skin tissue.

The concept of TCA CROSS was first proposed in 2002 by Lee et al. in a prospective trial involving 65 Korean patients with varying degrees of ice pick acne scarring.[10] This trial involved the novel technique of applying high concentrations of TCA of 65% and 100% with a thin and finely pointed wooden applicator to avoid unnecessary spill-over. Both treatment arms achieved excellent outcomes in terms of scar count reduction; 82% of patients receiving 65% TCA CROSS had good to excellent responses compared to 94% in the 100% TCA group. By precisely targeting the scar tissue, the rate of complications was minimal with no cases of persistent erythema, dyspigmentation, scarring, or keloids.

Currently, the majority of studies involving TCA CROSS have focused on the revision of ice pick scars and the efficacy is well validated. To date, there have been only a few studies involving TCA CROSS monotherapy in the management of other scar types. In a study involving 53 patients, Agarwal et al. treated a mixture of ice pick, boxcar, and rolling scar with 70% TCA CROSS with good or excellent clinical response in 66% of the patients.[16] Garem et al. demonstrated in a cohort of 30 patients managed with 50% TCA CROSS a statistically significant ($P<0.05$) reduction in the Goodman grading score of boxcar scars.[17] In a retrospective study by Lim and Sun, 37 patients with a combination of ice pick, rolling, and polymorphic acne scars had good to marked improvement with 90% TCA CROSS (Figure 1).[18] While all three studies demonstrate promising prospects of TCA CROSS against other scar types, the overall assessment of clinical efficacy in these studies was not discriminated against each scar type. Currently, there are no studies to date that assess the selected efficacy of TCA CROSS against each scar type.

Since the pioneering study by Lee et al., varying concentrations of TCA between 50% and 100% have been trialled with the CROSS technique for acne

Figure 1. *Revision of acne scar with paint brush novel applicator*

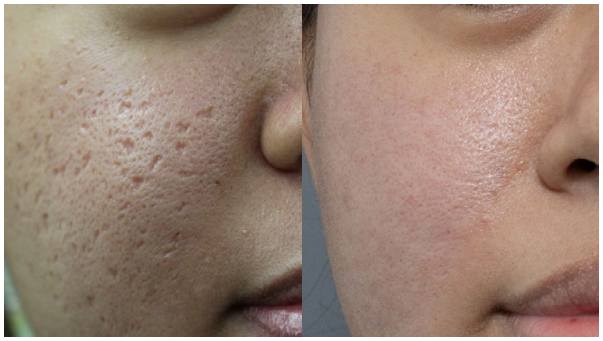

Figure 2. *TCA with the brush technique, followed by light fractional CO2 laser resurfacing*

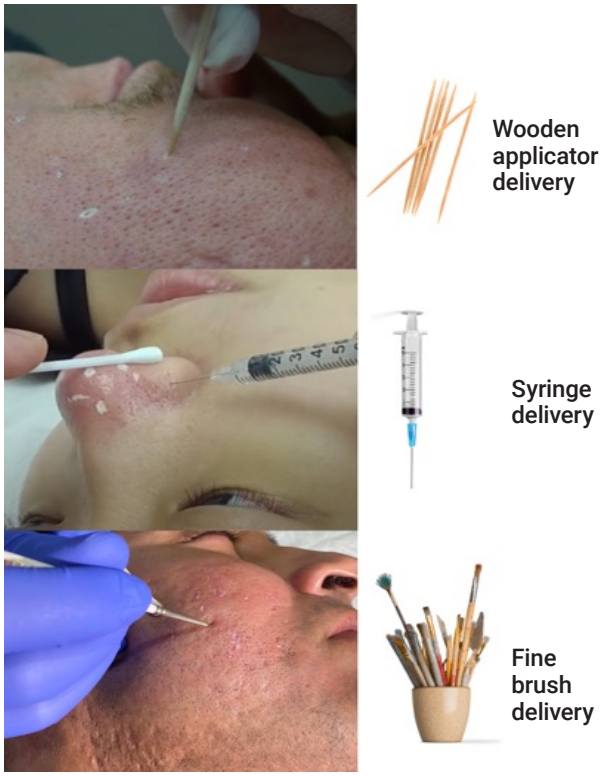

Figure 3. *Common applicators used for TCA CROSS technique*

scar revision.[12] In terms of the efficacy of different concentrations of TCA used in the CROSS technique, most studies demonstrated good improvement in the majority of the patients enrolled.[10,16,18-20] Nevertheless, most of the studies in the existing literature are small scale prospective or retrospective studies with limited power. Besides a few studies to date which compared different concentrations of TCA CROSS in non-blinded trials which are again hindered by the small treatment population, there have been no randomised trials so far to assess the difference in efficacy and rates of complications between different concentration.[10,12] The choice of concentration used by clinicians, therefore, is currently guided more by the personal experience and preference of the operator rather than robust clinical evidence.

Optimal treatment of acne scarring often requires a multimodal approach to achieve the best outcome given the varying morphology of the scars that respond differently depending on the modality of the treatment. Chemical peels have been used in conjunction with subcision, microneedling, dermaroller, as well as non-ablative lasers (Figure 2).[21-23]

The tool that is used to apply TCA has also evolved since the inception of the CROSS technique. The original applicator is a sharp-ended wooden stick. Subsequent studies have employed a range of different applicators which includes toothpick, insulin syringe, and fine-tipped paint brush (Figure 3).[12,18] Interestingly, two studies reported the highest rate of scar extension post-TCA CROSS at respectively 17% and 13%.[16,24] Both used toothpicks as the primary applicator, suggesting possible imprecision with the risk of spill-over on to healthy skin.

General complications of chemical peels

Swelling and pain

All agents used in chemical peels have the potential of causing swelling but this effect is more commonly associated with deeper peels. Dermal oedema appears between 24 to 72 hours post-peel and may take several days to resolve and the extent of oedema may vary from mild to severe.[25,26] Antihistamines are first-line agents for post-peel swelling along with a proper wound care regimen.[27] Systemic steroids are of utility in the case of significant oedema but may result in delayed wound healing.[25] Pain is also a common and expected occurrence in the setting of medium to deep peels but is rarely necessary to warrant the prescription of potent analgesics.[26]

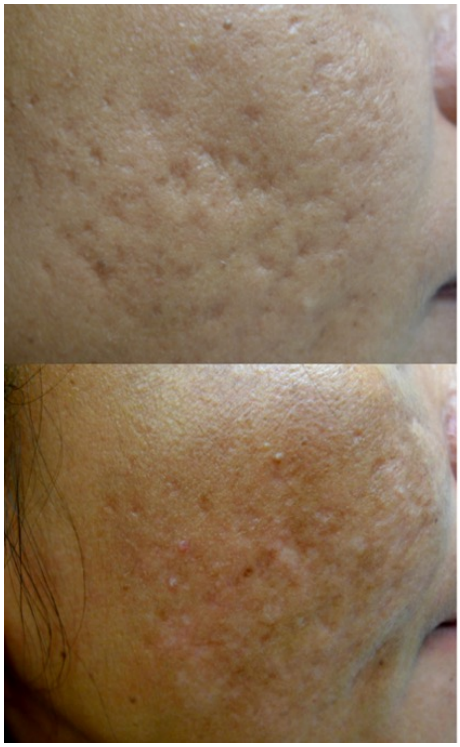

Figure 4. *Post inflammatory hypopigmentation is a rare side effect. Limiting treatment to well defined scars less than 3 mm can reduce the incidence of this adverse reaction*

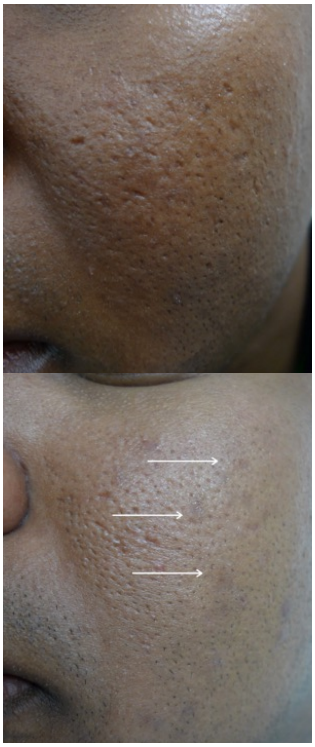

Figure 5. *Post inflammatory hyperpigmentation is expected in skin types 3 and above. In most cases it is self-limiting*

Persistent erythema

Post-peel erythema is common after all depths of peeling, which usually resolves but can persist to become permanent. According to a systemic review on TCA CROSS by Chung et al., persistent erythema is the second most reported complication post-procedure.[12] Persistent erythema indicates a prolonged fibroplasia phase of wound healing with the potential to lead to skin thickening and scarring.[25] Prolonged erythema occurs more often in the instance of medium and deep peels. Association between topical or systemic retinoid use, as well as alcoholic beverage consumption, and pre-existing skin conditions exist in terms of persistent erythema after chemical peels.[25] Persistent erythema should be treated promptly after diagnosis usually with potent topical steroids for 1-2 weeks.[27] Strict sun protection, as well as regular emollients, should also be incorporated into the treatment regimen.[27]

Infections

Post-peel infection is rare with TCA peels given the inherent bacteriostatic nature of the agent.[16] Prolonged occlusive dressing or ointment and poor wound care are risk factors for post-peel infection due to the build-up of necrotic debris leading to secondary impetiginisation.[26,27] Common skin commensals such as *Streptococcus* and *Staphylococcus*, as well as *Pseudomonas*, are the most common pathogens that can lead to infection post chemical peeling.[25] Fungal species, such as Candida, can occur in the setting of immunosuppression. Recurrence of herpetic infection can also occur after chemical peeling which usually occurs 5-12 days after the initial procedure. It is recommended that patients with a history of herpes should be commenced on a prophylactic dose of acyclovir or valacyclovir to prevent secondary herpetic flare.[27] All forms of secondary infections after chemical peeling should be addressed with urgency to prevent potentially delayed wound healing and inflammation leading to an increased risk of scarring.

Dyspigmentation

Mild hypopigmentation after chemical peeling, especially in medium or deep peels, is expected due to the destruction of melanocytes in the epidermo-dermal junction after exfoliation (Figure 4). Hypopigmentation should be temporary as the skin re-epithelialises and melanocytes migrate into the newly formed junction between the dermis and the epidermis.[25] Permanent hypopigmentation, however, can occur in the setting of chemical peels and is a troublesome complication with a higher predilection (more noticeable) for patients with darker skin types.

Conversely, hyperpigmentary changes can also occur after procedures (Figure 5). The risk of this complication is more prominent in those with darker Fitzpatrick skin types. Intense sun exposure, use of

photosensitising agents, oral contraceptive use, and hormone replacement therapy can also increase the risk of this complication.[25] Hydroquinone can be used in patients with darker skin types to reduce the risk of hyperpigmentation; along with strict sun protection and stringent skincare to prevent secondary infections.[27]

Although dyspigmentation is a concern in treating patients with darker skin types, TCA CROSS can still be a viable option given most reported cases of dyspigmentation are temporary with resolution within 6 months.[12,15]

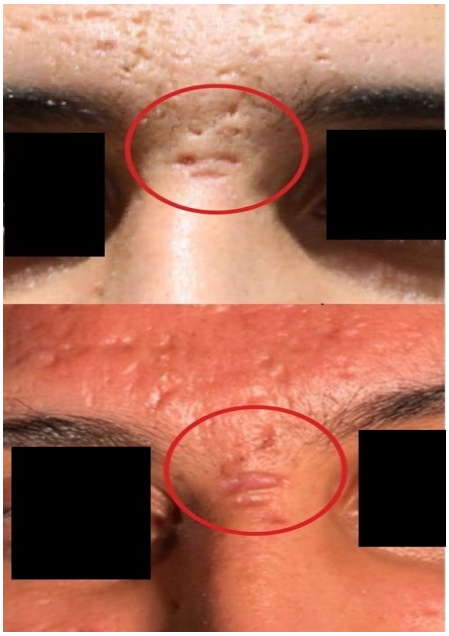

Figure 6. *Scar extension is more common in midline scars. Additionally limiting TCA application to focal areas 2 mm apart will reduce the incidence of scar coalescence*

Scarring

Scarring after chemical peeling is a dreaded complication. Although it is a rare side effect of chemo-exfoliation, significant physical and psychological distress is associated with this complication. Hypertrophic scars are the most common type of scarring after chemical peels; however, keloids, atrophic scars, and contractures may also occur.[25] The specific concern regarding TCA CROSS is the potential for scar extension (Figure 6) due to spill-over of TCA on to normal skin tissue. Delayed healing and prolonged erythema are often the early signs of post-treatment scarring from chemical peels.[25] Both personal and environmental factors can contribute to the risk of scarring. Patients with a previous history of poor wound healing, post-surgical scarring, or darker skin types will be more likely to experience this complication.[25] Certain parts of the body, namely those with a lower density of adnexal structure such as the dorsal hand, chest, and neck, are more prone to scarring as the adnexal germ cells are critical in the re-epithelisation process.[25] Smoking, or recent ablative procedures may lead to poor wound healing and an increased risk of scarring.[18,25] If scarring were to occur after chemical peels, intralesional corticosteroids, mechanical or energy-based ablative therapy, or surgical removal can be considered for revision. Concomitant use of vitamin A analogs are traditionally recognised to contribute to the risk of post-peel scarring due to the delay in epithelialisation. A recent study showed almost equal re-epithelialisation time between those on isotretinoin and the rest of the cohort (6.4 vs 6.2 days) with no higher rate of post-peel complications. However, due the small sample of patients (5 patients on isotretinoin vs 32 patients free of retinol), the true implication of this finding needs to be further elucidated with higher-powered comparative studies.

Conclusion

Since the inception of TCA, this agent has evolved to become one of the main treatments for focal acne scar revision. It shows excellent efficacy as a deep peeling agent for ice pick scars while also demonstrating some promising results for other scar types which need to be further explored. Post-procedural erythema and dyspigmentation are common complications of this modality; however, most prove to be transient. Scar extension is an uncommon but troublesome complication which clinicians need to be aware of to avoid contamination of healthy skin with the peeling agent. Overall, TCA is a useful and accessible treatment for acne scar revision for dermatologists to master.

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Muscle stimulation for the face: a paradigm shift in facial rejuvenation

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Disclosures: Lumenis Be key opinion leader (KOL)

BACKGROUND: Facial ageing is a dynamic and complex process resulting from extrinsic and intrinsic factors. All layers of the facial anatomy are affected due to interrelated changes to skin, fat, muscle and bone. Despite a similar facial ageing sequence, the rate of change of facial characteristics and its extent differs among individuals. In order to tailor the treatment, a special protocol was developed targeting each tissue layer with the appropriate technology to stimulate a complete rejuvenation process.

METHODS: A three step protocol was developed by using: 1) dynamic muscle stimulation (DMSt) that delivers an electrical current to activate skeletal muscles and facilitate contraction; 2) TriPollar® radiofrequency (RF), which produces volumetric heating of dermal and subcutaneous tissues; and 3) triFX™ RF microneedles (RFMN), which uses an array of microneedles that penetrate the skin and heat the deep dermis, creating zones of invisible thermal micro-wounds between areas of unaffected normal healthy skin.

This sequential treatment is repeated at weekly intervals for 4-6 treatment sessions. The triFX step is repeated every two weeks.

RESULTS: The DMSt was previously used in a clinical study and an increase of zygomatic muscle thickness was reported. DMSt increases facial muscle tone which supports the dermal structures. TriPollar® RF has shown effectiveness in improving facial skin tightness and wrinkles. Combining the two technologies (DMSt with TriPollar RF) in a sequential method was beneficial for numerous indications showing an enhanced result when combined than when each modality was evaluated alone. Adding the triFX into the treatment protocol can enhance new collagen deposition and tissue remodeling resulting in an increase of the epidermal layer volume and smoother skin texture, as shown by numerous clinical evaluations. The effects of combining all three technologies in a single treatment lasts for approximately 3-6 months, and may be maintained with additional sessions thereafter.

CONCLUSIONS: The triLift protocol provides rejuvenation treatment for all facial layers in the same treatment session, simulating a facelift without a surgical intervention and with minimal adverse effects and downtime.

KEYWORDS: facial ageing, skeletal muscles, muscle stimulation, facial muscle toning, DMSt

ASCD References to come...

Facial muscles and ageing

Facial ageing is a dynamic and complex process involving all layers of the facial anatomy due to interrelated changes to skin, fat, muscle and bone. It is caused by the intricate interplay of both extrinsic and intrinsic factors. Extrinsic skin ageing is caused by long-term exposure to ultraviolet radiation, wind, pollution, repetitive muscle movements, and various lifestyle habits such as smoking, sleep, diet and poor general health. These lead to upper dermis elastosis, destruction of its fibrillar structure, increased intercellular substance and moderate inflammatory infiltrate.[1,2] As a result, the support and volume of dermal structures are decreased, leading to reduced skin torsion extensibility and causing wrinkling of the

overlying epidermis.[3] Key intrinsic factors include gravitational effects secondary to decreased skin elasticity, volume loss in facial fat compartments, and bony and soft-tissue re-modeling through genetic and hormonal influences. In addition, repetitive mimetic muscle contractions create prolonged cutaneous changes that evolve to static and dynamic wrinkles and folds. Age-related volume loss influences muscle tone and consequently impacts bone re-modeling.[4]

Despite a similar facial ageing sequence, the rate of change of facial characteristics and its extent differs among individuals.[5,6] Rates of bone remodeling, photodamage, wrinkle development, and soft tissue redistribution varies between races and ethnicities, and people with skin of colour may have distinct pigmentation concerns. However, age-related changes in skin texture, pigment, and bone structure affect all populations.[7]

The triLift protocol is a sequenced treatment protocol using the triLift platform (Figure 1) that targets each skin layer with the appropriate technology and energy to stimulate a layered rejuvenation process. Specifically, dynamic in-motion electrical muscle stimulation (DMSt) is used for toning facial muscles, third-generation TriPollar® radiofrequency (RF) is used for thickening and strengthening the dermal layer, and triFX™ RF microneedles (RFMN) are used for increasing the epidermal layer's natural volume and smoothing its texture.

Figure 1. *triLift Platform by Lumenis Be*

Each of the technologies is discussed below, together with evidence for its effectiveness and safety.

Dynamic in-motion electric muscle stimulation (DMSt)

The triLift protocol uses DMSt to tone facial muscles. DMSt, also termed elsewhere as electrical muscle stimulation (EMS), dynamic muscle activation, neuromuscular electrical stimulation, or electromyostimulation, uses an electrical current to activate skeletal muscles and facilitate contraction. The impulses generated by the device are delivered through electrodes on the skin near the muscles being stimulated. To avoid charge accumulation, which may damage the tissue, EMS devices typically deliver a pulsed biphasic current. EMS recruits muscle motor units in a nonselective, spatially fixed, and temporally synchronous pattern, such that both slow and fast fibres are non-selectively activated at low or high force levels. Such nonselective recruitment means that all fibres, regardless of type, have the potential to be activated at relatively low intensities.[8]

EMS is commonly used in clinical settings to mimic voluntary contractions and enhance the rehabilitation of human skeletal muscles. For example, it has been shown to increase muscle strength of the quadriceps femoris and abdominal muscular strength in healthy individuals and as a post-exercise recovery tool for athletes.[9-11] It has also been used as a rehabilitation and preventive tool for partially or totally immobilised patients with muscle atrophy.[12-16]

This technology has also been used to stimulate facial muscles and to restore muscle contour, strengthening the skin's support.[17] It has also been examined for the treatment of facial paralysis[18,19] and Bell's palsy.[20]

The effect of EMS technology on facial muscle tone and signs of facial ageing in healthy women was evaluated in a randomised, controlled, partially blinded study. One-hundred and eight women (mean age 43.7 years, range 32-58 years) were randomised to 12 weeks of treatment with a neuromuscular electrical stimulation facial device (20 min/day, 5 days/week) or to a nonintervention control group. Participants could not alter fitness, diet or facial care routines during the study. Assessments included psychometric evaluations of facial appearance and assessor-blinded ultrasound measurements of the thickness of the zygomatic major muscle. In the neuromuscular electrical stimulation group mean zygomatic muscle thickness increased by 18.6% from baseline but not the control group. Between-group differences were significant at 6 and 12 weeks (P=0.05 and P=0.0001, respectively). In an overall evaluation, ≥80% of neuromuscular electrical stimulation users reported improved firmness, tone

and lift compared to <5% of the control group (P<0.001). As expected with transcutaneous electrical stimulation, transient mild erythema at the site of the electrodes occurred in all users, which resolved shortly after the end of each session. The only other adverse event was persistent fluttering of one participant's eyelid after one session, which did not recur.[21] It was suggested that the exercise-induced increase in the zygomatic major muscle size may be linked to the shortening of the resting length of the muscle, resulting in improvements in facial tone, firmness and lift.[22]

TriPollar® radiofrequency

Third-generation TriPollar® RF is used to thicken and strengthen the dermal layer. TriPollar RF comprises three or more electrodes that deliver radiofrequency energy into the skin at a frequency of 1 MHz and a maximum power of 50 W. This component produces volumetric heating of dermal and subcutaneous tissues through rapidly oscillating electromagnetic fields that cause movement of charged particles within the dermis and generation of heat that is proportional to the tissue's electrical resistance (Figure 2).[23]

TriPollar RF-based systems have shown effectiveness in improving facial skin tightness and wrinkles.[23-26] The biological mechanism involved in skin tightening using TriPollar RF technology included stimulation of dermal fibroblasts with increased collagen synthesis, an increase of 49% in dermal thickness, and focal thickening of collagen fibres.[23,27] In a study that evaluated the improvement in facial wrinkles and rhytids in 37 women (mean age 52.8 years) that received eight weekly treatments with TriPollar RF, improvement was observed in 94 to 97% of study participants.[26] Levenberg reported an improvement of perioral and periorbital wrinkles in nine women treated with RF TriPollar technology.[25]

In all of the studies, treatment was reported to be painless or involved minimal pain. Post-treatment transient mild-moderate erythema and/or slight oedema were reported in some of the studies.[23-26]

In several studies, EMS technology and TriPollar RF were combined. In a study that included 20 women (mean age 43 years) that were treated for facial wrinkles with TriPollar and EMS technology once a week for 8 weeks, significant improvements in skin tonus, skin lift effect and facial skin colour homogeneity were noted as well as reduced sharpness of mimic and static wrinkles. Ultrasound assessment of the skin showed significant dermal-epidermal thickening and increased dermal density.[24]

Levenberg et al. showed that subjects who underwent facial treatments using two TriFractional treatments spaced one month apart, with two TriPollar RF plus EMS technology two weeks after each TriFractional treatment, had improvement in acne scars, skin texture and wrinkles together with facial contouring.[28] In another study, 11 subjects (mean age 43 years; Fitzpatrick skin type II-IV) with Fitzpatrick Elastosis Scale (FES) score 3-6 were treated with TriPollar® RF and EMS technology once a week for 6 weeks.[17] At 1 and 3 months of follow-up, mean physician-rated FES statistically significantly improved from baseline by 2.27 ± 0.45 for both time points (P<0.0001). At 3 months of follow-up, fine lines, wrinkles and skin tightness were improved in 81.8% of patients and were much improved in 18.2%. Pain was minimal and there was no downtime or adverse events.

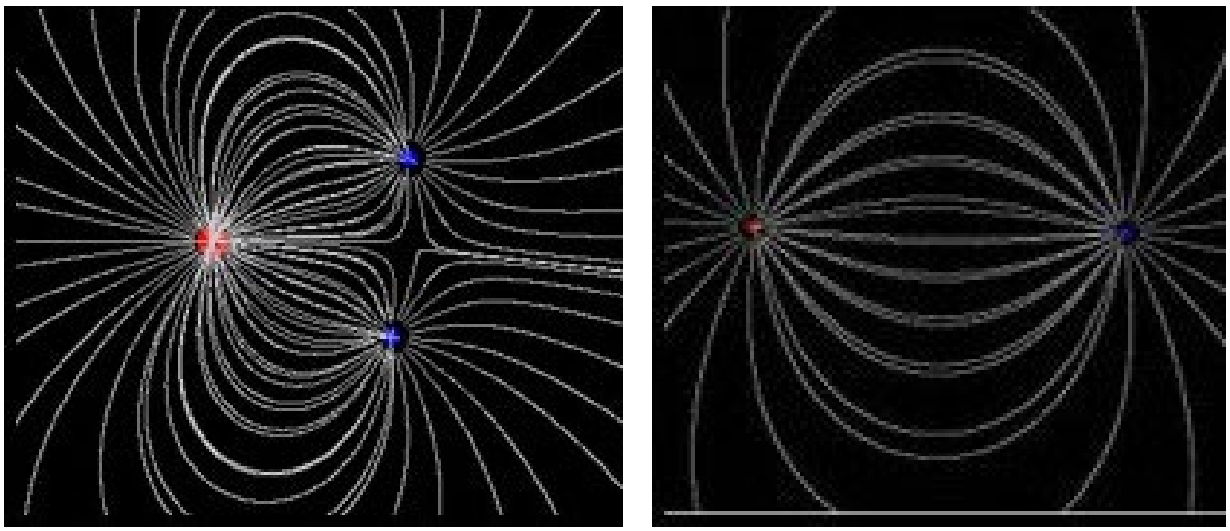

Figure 2. *Electrical field density simulation of TriPollar (left) vs Bipolar (right)*

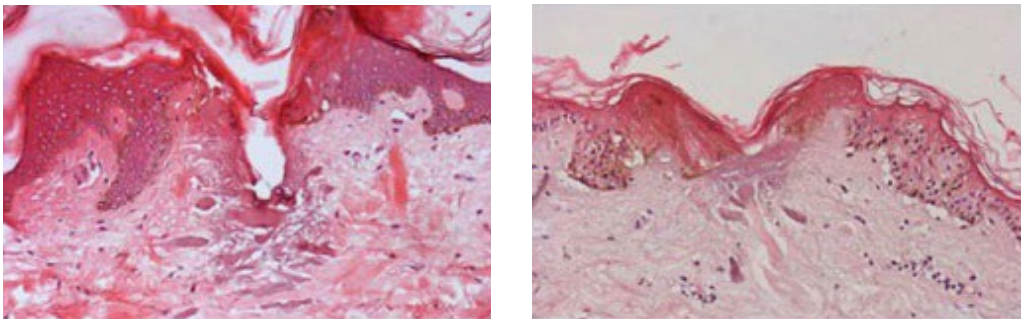

Figure 3. *Ex-vivo UV aged skin histology samples at D0 (left) and D2 in survival medium (right). (H&E stain, magnification: x200 left, x100 right). Left: immediately after treatment with triFX at low level. Micro ablation effect is visible into upper dermal layer. Right: after 2 days in survival medium. Healing process including collagen regeneration and epithelialisation is demonstrated.*

triFX radio frequency microneedling

triFX™ RFMN technology uses an array of microneedles that penetrate the skin and heat the deep dermis, creating zones of invisible thermal micro-wounds between areas of unaffected normal healthy skin. The thermal effects of RF can change the shape, length and diameter of collagen fibres,[29] cause contraction of connective tissue,[30-32] and repair tissue. Repeated thermal wound healing stimulates fibroblasts to enhance new collagen deposition and remodeling, resulting in further collagen tightening and an overall increase in collagen content. As debris is removed and cellular and fibre regeneration takes place, a gradual increase in skin quality is observed. Assessment of changes in tissue, cells, thickness and structure of pigskin after RF application demonstrated an expanded papillary dermis due to oedema and vascular congestion, followed by an accumulation of intercellular substances. Two months after the last application, an increase in the amount of collagen, elastic fibre and mucopolysaccharides was noted.[33,34]

This technology has demonstrated effectiveness when used for skin resurfacing to reduce wrinkles and enhance skin and facial appearance.[35] In a study that evaluated the change in skin texture and wrinkles of 12 subjects (mean age 45.5 years, Fitzpatrick skin types II-III) with FES score 3-6 who were treated with three sessions of triFX RF technology using 100 microneedles at 3-week intervals, all subjects had improved skin texture and pigmentation and in most of them (91.7%) skin brightness, tightness, and wrinkles also improved. Physician-rated FES showed statistically significant improvement of 2.67 ± 1.18 and 2.33 ± 1.03, respectively ($P<0.0001$) from baseline to 1 and 3 months of follow-up. The treatments were well tolerated, with no downtime or adverse events.[36]

triLift protocol

Step 1: Apply a thin layer of 87% pure medical grade glycerin. Use applicator #2 (medium applicator) in lift mode to pass over the zygomaticus major and minor and the masseter for 8 minutes on each side, adjusting parameters to reach 40°C, and to visually see a contraction of these muscles.

Step 2: Use applicator #3 (small applicator) in TriPollar® RF mode at 39°C to pass over the perioral and periorbital regions for up to 10 minutes each side of the face.

Step 3: Clean the area well with warm water to remove all glycerin. Thoroughly dry the skin, stretching it so as not to miss skin folds. Attach a new triFX tip (depending on the subject's condition use one of the five available triFX disposable tips). Start with low presets and increase the exposure and power each treatment session.

Perform 4-6 treatment sessions (triLIFT steps 1, 2) at weekly intervals. The triFX step (triLIFT step 3) should be done every two weeks (i.e., at weeks 1, 3 and 6). Maintenance sessions and follow-up should be performed 3-6 months after the last treatment session, depending on the individual's baseline condition.

Conclusion

The clinical data summarised above shows that each of the triLift protocol components targets a specific facial layer: DMSt increases facial muscle tone which supports the dermal structures; TriPollar® RF thickens and strengthens the dermal layer; and triFX increases the epidermal layer's natural volume and smooths its texture. Therefore, altogether the triLift protocol provides rejuvenation treatment for all facial layers in the same treatment session, simulating a facelift without a surgical intervention and with minimal adverse effects and downtime. The effects last for approximately 3-6 months, and may be maintained with additional sessions thereafter.

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Dynamic muscle stimulation (DMSt™) for muscle toning and improving facial ageing features

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BACKGROUND: With the progression of ageing, human features undergo undesirable changes to facial contour, volume, and overall expression. Changes in skeletal muscles and repetitive muscle contraction result in the appearance of superficial and deep wrinkles. The superficial musculo-aponeurotic system (SMAS) plays an important role in maintaining facial elasticity and facilitating the coordination of the facial muscles, which decreases in activity with ageing. Therefore, treatment of ageing skin should involve recontouring of the SMAS.

METHODS: Dynamic muscle stimulation (DMSt) uses the application of transcutaneous electrical muscle stimulation (EMS) through skin contact to activate healthy skeletal muscle and facilitate contraction. Muscle toning is achieved by targeting different layers of muscles using directly applied electrical current, which allows intervals of stimulation and relaxation to effectively augment muscle workload. DMSt is controlled by three parameters: pulse duration (ms); pulse power (mA) and frequency (Hz). These parameters can be adjusted by the operator on the triLift system according to the patient's reaction and skin tolerance. No active cooling of the electrodes or the skin is required.

SUMMARY: The triLift system (by Lumenis Be), is the only device using DMSt to date, delivering electrical current during non-stationary application to activate both low- and high-threshold muscle endplates, helping to tone muscles and restore the contour of the SMAS, thereby strengthening facial skin support and improving the facial appearance.

KEYWORDS: facial ageing, muscle stimulation, collagen and elastin, muscle toning, EMS

ASCD References to come...

Facial muscles and ageing

Muscle tissue comprises a large portion of human body composition (38% in males and 31% in females).[1] Nearly all movement in the body is the result of muscle contraction. The integrated action of joints, bones, and skeletal muscles produces obvious movements such as walking and running. Skeletal muscles also produce more subtle movements that result in various facial expressions, eye movements, and respiration.

The distribution of skeletal muscles in the body changes with age. Relative skeletal muscle mass starts to decrease in the third decade of life and a noticeable decrease in absolute skeletal muscle mass is observed at the end of the fifth decade.[1,2] The change in skeletal muscle mass results in loss of strength and physical function.

The skin, soft tissues (muscle, subcutaneous fat, fascia), and structural support (bone and teeth) act in dynamic unison to determine facial appearance throughout life. Humans have well-developed muscles in the face that permit a large variety of facial expressions. Muscles of facial expression include frontalis, orbicularis oris, laris oculi, buccinator, and zygomaticus (Figure 1).[3] Facial mimetic muscles insert into the dermis and therefore play a role in the suspension and structural integrity of the soft tissue surrounding them.[4] Each facial feature is affected by the ageing process, resulting in changes to facial contour and volume.[4,5] Changes in muscle tone and repetitive muscle contraction result in the appearance of superficial and deep dynamic wrinkles during animation.[5]

The superficial musculo-aponeurotic system (SMAS) is a continuous, organised fibrous network composed of the platysma muscle, parotid fascia, and fibromuscular layer covering the cheek that connects the facial muscles to the dermis.[6,7] The SMAS surrounds facial tissues and connects the facial muscles to the dermis, playing an essential role in providing facial elasticity and support while also facilitating coordination of the facial muscles.[8-10] It has a close relationship with the most superficial fascial planes of the face and neck area. Ageing renders the SMAS less elastic, weakening this skin support frame. Therefore, treatment of ageing skin should involve recontouring of the SMAS.[11]

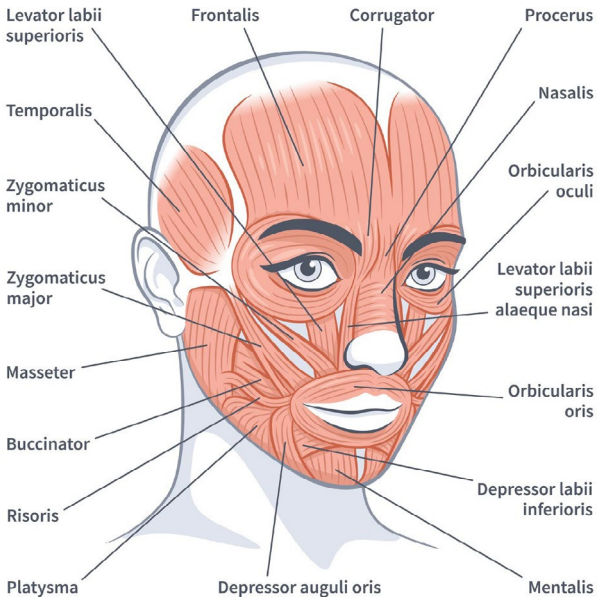

Figure 1: *Facial muscles*[3]

Skeletal muscles anatomy and mechanism of action

Skeletal muscle contraction is normally activated by electrical impulses sent from the brain through the central nervous system.[3]

Each skeletal muscle fibre is a single cylindrical muscle cell. An individual skeletal muscle may be made up of hundreds, or even thousands, of muscle fibres bundled together and wrapped in a connective tissue covering. Each muscle is surrounded by a connective tissue sheath called the epimysium. Fascia, connective tissue outside the epimysium, surrounds and separates the muscles. Portions of the epimysium project inward to divide the muscle into compartments. Each compartment contains bundles of muscle fibres. Each bundle of muscle fibres is called a fasciculus and is surrounded by a layer of connective tissue called the perimysium. Within the fasciculus, each individual muscle cell, called a muscle fibre, is surrounded by connective tissue called the endomysium.[3]

The connective tissue covering the muscle fibres provide support and protection and allow them to withstand the forces of contraction. The coverings also provide pathways for the passage of blood vessels and nerves.

Commonly, the epimysium, perimysium, and endomysium extend beyond the gaster (the fleshy part of the muscle) to form a thick ropelike tendon or a broad, flat sheet-like aponeurosis. The tendon and aponeurosis form indirect attachments from muscles to the periosteum of bones or to the connective tissue of other muscles. Typically, a muscle spans a joint and is attached to bones by tendons at both ends. One of the bones remains relatively fixed or stable while the other end moves as a result of muscle contraction.[3]

Skeletal muscles have an abundant supply of blood vessels and nerves. This is directly related to the primary function of skeletal muscle contraction. Before a skeletal muscle fibre can contract, it has to receive an impulse from a nerve cell. Usually, an artery and at least one vein accompany each nerve that penetrates the epimysium of a skeletal muscle. Branches of the nerve and blood vessels follow the connective tissue components of the muscle of a nerve cell and with one or more capillaries.[3]

Muscle fibres are classified using three different methods: histochemical staining for myosin ATPase, myosin heavy chain isoform identification, and biochemical identification of metabolic enzymes. In humans ATPase histochemical staining intensity at different pH levels identified seven muscle fibre types (from slowest to fastest): types I, IC, IIC, IIAC, IIA, IIAB, and IIB.[12] The functional unit of the neuromuscular system is the motor unit. It comprises an alpha motoneuron (originating in the spinal cord) and all of the muscle fibres that it innervates. All muscle fibres of a motor unit have similar characteristics. Motor units can be classified according to the contractile and fatigue characteristics of the muscle fibres. Based on contractile speed, motor units are classified as either slow-twitch (S) or fast-twitch (F). The F motor units are further subdivided into fast-twitch fatigue-resistant (FR), fast-twitch fatigue-intermediate (Fint), and fast-twitch fatigable (FF).[12] Muscle fibres have contractile and metabolic properties that allow them (and their motor units) to change in size, and convert from one type to another in response to stimuli such as training and rehabilitation; therefore, that can adapt to different functional demands.[12]

Dynamic muscle stimulation (DMSt™)

Dynamic muscle stimulation (DMSt) uses application of transcutaneous electrical muscle stimulation (EMS) through skin contact to activate healthy skeletal muscle and facilitate contraction. EMS technology is commonly used in clinical settings to mimic voluntary contractions and enhance the rehabilitation of human skeletal muscles. Muscle toning is achieved by targeting different layers of muscles using directly applied electrical current, which allows intervals of stimulation and relaxation for effectively augmenting muscle workload. Delivery of electrical current during non-static application activates both low- and high-threshold muscle endplates. In the triLift system (by Lumenis Be), the only device using DMSt to date, the impulses are delivered through metal (stainless steel) electrodes that are placed on the skin close to the muscles being stimulated, while the system applicator is moved across the treatment area (Figure 2). To avoid charge accumulation, which could damage the tissue, a biphasic current is used. It may or may not produce a muscle contraction depending on the intensity of the current. Electrical stimulation that elicits a muscle contraction can be applied to single or multiple muscle groups, during functional activities and in combination with voluntary effort.

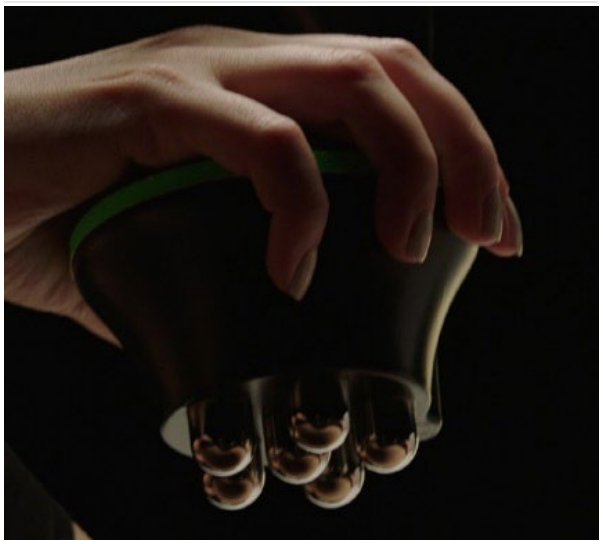

Figure 2. *triLift large applicator stainless steel electrodes*

In contrast to Henneman's size principle of voluntary motor unit recruitment, in which recruitment of small, typically slow, motor units is followed in order of increasing size to the larger, typically fast, motor units,[13] EMS technology recruits motor units in a nonselective, spatially fixed, and temporally synchronous pattern, such that both slow and fast fibres are non-selectively activated at low or high force levels. Such nonselective recruitment means that all fibres, regardless of type, have the potential to be activated at relatively low intensities. This activation may be beneficial in the clinical setting and potentially thought of as preferential activation.[14]

DMSt is controlled by three parameters: pulse width, pulse amplitude, and frequency. Pulse width refers to the duration of time each group of pulses of stimulation is set to, measured in microseconds. A short pulse width can be more comfortable for some patients, but a longer pulse width may recruit more motor neurons and thus improve the muscle contraction produced. Pulse amplitude (sometimes referred to as power) refers to the strength of the stimulation delivered, measured in milliamps (mA). It relates to how much energy is being induced onto the patient's tissue. It needs to be high enough to evoke the desired effect and low enough in order to induce as little inconvenience to the patient as possible. Frequency is the number of electrical impulses delivered in a given period of time and is measured in hertz (Hz). Default parameters are pre-programmed to facilitate treatment initiation. The parameters should be further adjusted by the operator from the user interface on the triLift system according to the patient's reaction and skin tolerance. No active cooling of the electrodes or the skin is required.

The ability of protocols using EMS technology to improve skeletal muscle performance in healthy and dysfunctional muscle was demonstrated in several studies and it is also used in clinical practice. For example, this modality has been used in clinical settings to mimic voluntary contractions and enhance the rehabilitation of human skeletal muscles. It has been shown to increase muscle strength of the quadriceps femoris and abdominal muscular strength in healthy individuals and as a post-exercise recovery tool for athletes.[15-17] It can be used as a rehabilitation and preventive tool for partially or totally immobilised patients with muscle atrophy.[18-22]

EMS technology has also been applied to facial muscles for the treatment of facial paralysis[23,24] and Bell's palsy.[25] The effect of this technology on facial muscle tone and signs of facial ageing in healthy women was evaluated in a randomised, controlled, partially blinded study. Onehundred and eight women (mean age 43.7 years, range 32-58 years) were randomised to 12 weeks of treatment with a neuromuscular electrical stimulation facial device (20 min/day, 5 days/week) or to a nonintervention control group. Participants could not alter fitness, diet or facial care routines during the study. Assessments included psychometric evaluations of facial appearance and assessor-blinded ultrasound measurements of the thickness of the zygomatic major muscle. Mean zygomatic muscle thickness increased by 18.6% from baseline in the neuromuscular electrical stimulation group but not in the control group. Between-group differences were significant at 6 and

12 weeks (P=0.05 and P=0.0001, respectively). In an overall evaluation, ≥80% of neuromuscular electrical stimulation users reported improved firmness, tone and lift compared to <5% of the control group (P<0.001). As expected with transcutaneous electrical stimulation, transient mild erythema at the site of the electrodes occurred in all users, which resolved shortly after the end of each session.

The only other adverse event was persistent fluttering of one participant's eyelid after one session, which did not recur.[26] It was suggested that the exercise-induced increase in the zygomatic major muscle size may be linked to the shortening of the resting length of the muscle, resulting in improvements in facial tone, firmness and lift.[27]

Summary

DMSt helps to tone muscles and restore the contour of the SMAS, thereby strengthening facial skin support and improving facial appearance. It provides a non-invasive non-surgical alternative for toning simply by applying energy through a single applicator. It may be used sequentially with other types of energies, such as radiofrequency (e.g., TriPollar or triFX microneedling) to achieve complete facial rejuvenation of all skin layers.

Figure 3. *triLift system by Lumenis Be*

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