INTRODUCTION

Smile

Ekman and Friesen (1982) classified smile into -felt smile, in response to a positive modification in circumstances or a counter of a pleasant stimulus which may be in the form of visual, auditory, gustatory, tactile stimulation, pleasure, relief from pain or tension and enjoyment of other person [1]. False smiles was further classified into phony smile when nothing is felt but person smiles to show a genuine emotion felt, masked smile in which individual acts opposite to what he is actually feeling, and damped smile where the person performs as if the positive emotion felt is less than what is actually felt [1]. There is also another type of smile, known as miserable smile in which negative features of emotions are evident [1] (Figures 1, 2).

Genuine smile also known as Duchene smile involves the contraction of both Zygomatic and Orbicularis oculi muscles [2]. Non-Duchene smile involves only zygomatic muscles [3]. A recent study done by using FACS (Facial action coding system) and Automated Facial Image Analysis showed humans to be able to differentiate various types of smiles [4]. Duchene smile i.e. felt smile was associated with positive affect and non-Duchene smile with a negative affect [5]. Genuine and False smiles are described in Figure 1 and 2 above.

Cry

Patel (1993) defined crying as complex secreto motor phenomenon leading to production of tears from lacrimal apparatus without irritation of any ocular structures. It is often accompanied with vocalizations and sobbing with changes in muscles of facial expression, respiration and trunk [6]. Although major depressive disorder is most commonly associated with crying [7], it may also signify elevated mood [8] or helplessness [9] and self-pity [10]. It can also be a manipulative tool [11] or a distressing personality trait in adults with no relation to mood disorders [7]. Crying, a unique behavior [12] in humans has a survival role to play [13]. Most crying episodes are due to interpersonal issues. It is also a byproduct of emotional responses evoked by media such as watching a movie. Occurrence of sad thought also leads to crying [12]. Figure (3) demonstrates different factors associated with cry as mentioned above.

DISCUSSION

Smiling and crying always an emotion?

Smiling and crying behavior has been observed in anencephalic [14]. 4-D fetal Ultrasound studies have identified crying and smiling in fetus [15]. The brain is not myelinated beyond brainstem in infants [16]. Hence, the facial and vocal expression center is believed to be situated in brainstem. The brainstem structure involved fundamentally in vocalizations in mammals is identified as periaqueductal grey (PAG) [17]. Another study done on mero-anencephalics lays hypothesis that motor center for smiling is localized in pontine tegmentum [18]. Failure of higher cortical control on these brainstem centers as found in neurological injuries [19] leads to aberrant episodes of crying and laughing. According to the traditional view, laughter and crying would be triggered normally via two separate neuroanatomical pathways to the brainstorm laughter and crying center (LCC), and pathological laughter and crying is caused by lesions of the voluntary paths to the LCC [20]. These pathways are illustrated in Figure (4) below [21].

Crying in mammals is genetically pre-programmed with limbic cortex including hypothalamus and amygdala having some control in the production of this vocalization [22]. This is supported by the fact that hypothalamic hamartomas are associated with both dacrystic and gelastic seizures [23]. It is debatable whether the initial crying and smiling often observed in infants is mediated by hypothalamus in response to state of...
Figure 1 Genuine smile in response to pleasant stimulus. Genuine smiles result in Zygomatic and Orbicularis oculi both muscles contraction.

Figure 2 Classification of False smile. False smiles result in only Zygomatic muscles contraction, no contraction of Orbicularis oculi muscle.

discomfort or quiescence [24] constitutes true emotions or not [25].

Social Smile

Starting at about 2 months, infants begin to smile when fully alert [26]. As the prefrontal cortex begins to mature, the endogenous crying and spontaneous smile diminishes and true smiling emerges [27]. Neuroimaging and electroencephalographic (EEG) study (which includes event-related potentials (ERPs) and gamma-band oscillatory activity) done in four month old infants to study early social cognitive found that cortical networks responsible for facial communication cues including integration of eye and head orientation has early specialization. They found mutual gaze, eyebrow raise with accompanying smile in context of mutual gaze and perceiving facial communication signals activate areas in infant superior posterior temporal cortex and fronto-polar cortex. These are the same areas which are also activated in adult humans [28]. Another study concludes that more observation of pleasant facial affect such as smile activated posterior superior temporal sulcus, fusiform gyrus and ventral amygdala [29]. Right premotor cortex and pars opercularis of the inferior frontal gyrus, right parietal operculum and left anterior insula were activated by both observation and production of such pleasant affect [29].

Fusiform face area (FFA) is responsible for processing face in normal orientation [30]. Superior temporal sulcus (STS) and areas around it including superior and middle temporal gyri process changeable features of face such as emotional facial expressions [31]. Medial Prefrontal cortex (MPFC) is activated in reading communicative intentions [32], mutual gaze [33] and calling person’s name [34]. Amygdala has the ability to distinguish male from female faces and different emotions [35]. The right
Amygdala is stimulated in eye to eye contact while left amygdala can detect direction of other person’s gaze [35]. Social smile including perception of eye gaze and eye contact is impaired in Autistic Spectrum Disorder (ASD) [36]. During processing of face, there is also a failure to activate FFA in ASD [37]. Several studies have shown a link between autistic behavior and temporal lobe [38] and prefrontal cortex (PFC) dysfunction [39].

**Crying: An Attachment Behavior**

Separation from loved one in infancy [40] or loss of loved one at any stage of life results in crying [41]. Bereaved individual have high risk of increase in alcohol consumption, major/minor...
depression including suicide risk [42]. In such individuals presence of intrusive thoughts might lead to complicated grief [43]. Neuroimaging study done by Bartels A and Zeki S (2004) to find neural correlate of maternal and romantic love showed activation of reward system that are rich in oxytocin and vasopressin receptors in both types of love [44]. Ventral tegmental area (VTA) is activated by both maternal and romantic love [44]. PAG and orbitofrontal cortex (OFC) are specifically associated with maternal love whereas a dorsally located region in anterior cingulate is specifically associated with romantic love [44]. These attachment behaviors also deactivated brain region associated with negative emotions, metaizing and social judgments i.e. middle prefrontal, inferior parietal, middle temporal cortices, amygdala, temporal poles, parietotemporal junction and mesial prefrontal cortex regions [44]. A Functional Magnetic Resonance imaging (fMRI) study done on recently bereaved individuals found a direct link between intrusive thoughts, ventral amygdala and rostral anterior cingulate cortex (ACC). The activity in amygdala was shown to be modulated by dorsolateral prefrontal cortex (DLPFC) and rostral anterior cingulate cortex (rACC) [45].

Smiling and Crying in Social Setting

Humans mimic facial expressions of people surrounding them [46]. Observing facial expressions in others leads to development of corresponding emotions in self [47,48]. Hatfield Cacioppo and Rapson (1994) defined emotional contagion as the predisposition to mimic and synchronize human emotions including facial expressions, thereby converging emotionally with other person. They emphasized the importance of mimicry including facial mimicry in emotional contagion [48]. This imitation of human behavior which is essential for emotional contagion is also thought to be important for the development of empathy [49] and is widely regarded a prosocial behavior [50]. Prosocial words enhance imitation tendencies [51]. Such is the importance of mimicry in decoding the emotional meaning underlying facial expression that blocking mimicry has been shown to compromise the decoding of genuine vs. false smile [52].

Functional imaging studies in humans have identified mirror neuron system (MNS) constituted by inferior frontal cortex and rostral part of inferior parietal lobe [53]. Limbic system and the insula are also activated upon observation and imitation of facial expression, hence emphasizing the collaboration of MNS with limbic system in social interaction [54] and empathy [55]. Additionally, superior temporal sulcus, an area involved in visual processing of biological motion i.e. eye gaze, lip movement and gestures to play an important part of the imitation circuit [56,57]. Eye contact is thought to regulate the interaction between PFC and STS [58]. Damage to PFC has been shown to result in over imitation [59].

A flow MRI study done on highly functioning children with autism showed no mirror activation in inferior frontal gyrus while observing and imitating emotional expression [60]. Given the dysfunction of MNS in autism [60], Yuan T (2008) proposed MNS-based therapies might be useful for the treatment of emotional dysfunction observed in autism, Post Stroke Depression And Other Mood Disorders [61].

Smiling and crying as emotions

Happiness and sadness are most easily recognized human emotions [62]. Various studies have put forward the role of left vs. right hemisphere in emotions. The right hemisphere hypothesis posits that the right half of brain is involved in all emotional processing irrespective of the valence [63]. The valence specific hypothesis maintains that left half is dominant in processing positive emotions and right half negative emotions [64].

The two cardinal dimensions used to study emotions are – valence and arousal [65]. Valence measures whether an emotion is positive or negative affect [66]. Arousal measures calmness or excitement [67]. Negative valenced stimuli have greater emotional salience than positive emotional stimuli [68]. Hence, a greater area is involved in processing of negative valenced stimuli as compared to positive valenced ones [68]. Role of amygdala in processing of negative emotion is well known [69]. Amygdala has been shown to be activated in about 40% of all studies that examined neural correlates of fear [69]. It is the activation of amygdala which differs from person to person and therefore, is responsible for inter-individual differences seen in fear conditioning observed in humans [70]. Besides fear, amygdala has been shown to be involved in other negative valenced emotions such as depression [71] and is activated when threat or aggression is perceived [72]. However, many recent studies have shown amygdala to be stimulated in positive valenced emotion as well [73]. Moreover, activity in amygdala increases when valence of stimuli shifts from a negative or positive stimulus to a more neutral value [74]. This shows that amygdala has a critical role in relevance detection or salience [75]. MPFC also seem to be involved in processing of both negative and positive valenced stimuli [68,75]. The activity in PFC is inversely proportional to that in amygdala [76], thereby suggesting a regulatory system to keep excess emotions in check [77]. Dorsal ACC processes negatively valenced stimuli [78]. It is also involved in executive function [79], suggesting a highly emotional stimulus is capable of arousal as well [80]. Subcallosal cingulate is known to have a role in depression [81]. Other brain regions shown to be activated by valence are left DLPFC, amygdala, cingulate gyrus, bilateral medial PFC, insular cortex, and precuneus [68]. Dorsal ACC, left DLPFC, left parahippocampus and dorsal cerebellum are shown to be activated in arousal. Hippocampus a structure closely placed near parahippocampal gyrus is known to be involved in cortical arousal [68]. Left DLPFC and dorsal cerebellum seem to have a regulatory control on arousal as activity in these areas was observed to be inversely related to arousal [68].

Emotions are thought to be generated either by bottom up model or top down model [82]. Stimuli that inherently elicit emotional response such as encountering a snake are thought to be processed by bottom up mechanism [83]. Emotions linked with conscious awareness [84] and cognition is thought to be modulated by top-down mechanism [85]. Pessoa et al., (2002) hypothesize that processing of facial emotional expressions is under top-down control. These are also more self-reported as compared to bottom up [86]. A fMRI study done to find out the generation of emotions concluded that PFC, cingulate, temporal regions, left amygdala and dorsal MPFC were activated in top-down emotional processing whereas right PFC and parietal...
regions, both amygdala were activated in bottom-up [87]. Left amygdala showed overlapping activity in processing top-down and bottom-up emotions suggesting the more important role of left amygdala in influencing top-down emotions and anxiety as compared to right amygdala. Since lesions in amygdala have not been shown to affect global mood at the end of the day [88], it is argued that global mood depends on top-down and medial PFC mechanism [87]. Amygdala and MPFC activation was noted when the affect was self-reported in bottom up and top down responses respectively [87]. Greater amygdala activation along with more strong functional connection between amygdala, dorsal ACC and DLPFC was seen in bottom up model in response to negative stimuli as compared to positive in bottom up model [87]. Activation of ACC, its connection with amygdala and DLPFC was enhanced in top-down resolution of emotional conflict [89]. Gu X et al., (2013) proposed that anterior insular cortex creates awareness of emotions by bridging the top-down and bottom-up mechanism thereby giving rise to physiological component of emotional experience [90].

Phillips ML et al., (2003) took into account studies done on animals, human lesions and functional neuroimaging and proposed two neural systems - ventral system and dorsal system [91]. The ventral system includes amygdala, insula, ventral striatum, and ventral regions of PFC and ACC in identification, perception and automatic regulation of emotions [91]. The dorsal system consisting of hippocampus, dorsal regions of ACC and PFC is involved in regulation of emotions [91].

Perception of emotions activates basal and lateral nuclei of amygdala [83]. Basal nucleus has direct connections with cingulate motor cortex which represents upper face unlike lateral nuclei which is connected to regions involving subjective emotional response. Thus, upper part of face is a more direct function of efferent amygdala in response to emotional stimuli. Individuals with social communication disorder such as autism, Asperger’s syndrome conduct disorder, oppositional defiant disorder and depression show upper face to show more impairment as compared to lower part [92-94].

Medford and Critchley (2010) fortify that it is the anterior insular cortex (AIC) along with ACC that produces subjective feeling and coordinates appropriate responses to stimuli both external and internal with AIC acting as input and ACC as output [95].

Table 1: Conditions Leading To Smiling Or Crying Behavior and Their Neural Correlates.

<table>
<thead>
<tr>
<th>Depression</th>
<th>Decrease in hippocampal [119] DLPFC volume [120], decrease functional connectivity between amygdala, PFC and ACC [121], Greater and abnormal activation of amygdala [115].</th>
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<tbody>
<tr>
<td>Mania</td>
<td>Increase activation of dorsal ACC, decrease activation of ventral PFC [122], dysfunction OFC [123].</td>
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<tr>
<td>Cocaine Use</td>
<td>Craving is directly related to activity in limbic, paralimbic, and mesocortical regions including the NAc, inferior frontal/orbitofrontal gyrus [OF C], and anterior cingulate (AC) [124] repeated exposure to cocaine has been found to be associated with increased ventral hippocampus-nucleus accumbens communication [125].</td>
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<tr>
<td>Amphetamine Use</td>
<td>Increased dopaminergic transmission in the NAc [126], low ventral inferior frontal gyrus activity [127], reduce activation of DLPFC and OFC [128].</td>
</tr>
<tr>
<td>Marijuana Use</td>
<td>Increase DA efflux in the NAc and PFC and increase DA cell firing in the VTA [129].</td>
</tr>
<tr>
<td>MDMA Use</td>
<td>Hippocampal hyperexcitability [130], decrease in Communication between the medial temporal lobe and medial prefrontal cortex, increase communication between amygdala and hippocampus [131].</td>
</tr>
</tbody>
</table>

Depressed individuals have negative bias in perception of facial expressions, emotions and memory [112]. Acute episode of depression diminishes the capability to distinguish happy and sad faces [113]. Even happy faces are perceived as sad [114]. Sad facial expressions activate amygdala, ventral striatum and fronto-parietal cortices in depressed patients. Fluoxetine decrease in activity of these areas [115].

AnfMRI study done on depressed patients to see the neural responses to happy facial expression showed reduce activation of hippocampus, putamen and extra-striate cortex in depressed patients [116]. Hippocampus is involved in implicit processing happy faces [117]. In general, anti-depressants decreased the increased activation of limbic-sub cortical activity seen in depressed patients though [118] the slight impairments were shown to persist in remission phase; thus indicating impairment in limbic, sub cortical and extra-striate regions to be trait feature of depression [116] (Tables 1, 2).

Pössner J (2005) argues that it is the PFC which takes into account valence and arousal along with past, present and future events, goals and expectations leading to subjective emotional experience [96]. More complex and higher pleasant stimulus are represented more anteriorly in the PFC while fundamental pleasures are located posteriorly [97]. Cortical subcortical area encoding subjective pleasure is situated in mid-anterior sub region of OFC [98] and ventral striatum [99]. These regions also constitute reward pathway [100]. Smiling, a facial expression having intrinsic reward value [101] activates medial OFC [102] and is also produced by deep brain stimulation of nucleus accumbens (NAc) resulting in feeling of euphoria [103]. OFC is capable of changing the subjective feeling of pleasure [104]. Medial edge of OFC deals with both positive and negative valenced events [105] while the lateral part encodes unpleasant events [97]. Ventromedial prefrontal cortex (VMPFC) activated by emotional salient reasoning [106] also seem to be involved in processing of reward [107] and is speculated to have regulatory control over limbic system [108]. Lesion in VMPFC leads to apathy, blunted emotional experience and expression with poor decision making [109]. DLPFC is involved in emotional self-regulation. Hence, it can suppress sadness [110]. Wager TD et al., (2008) posits that VLPFC regulates emotion through its effect either on amygdala or on NAc [111].

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Table 2: Gender Differences.

<table>
<thead>
<tr>
<th></th>
<th>FEMALE</th>
<th>MALE</th>
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<tbody>
<tr>
<td>Onset and Frequency of smiling</td>
<td>More frequent than males [132]</td>
<td>Delayed and less frequent as compared to females [132]</td>
</tr>
<tr>
<td>Decoding and recognition of facial emotional expression</td>
<td>More efficient [133]</td>
<td>Less efficient than females [133]</td>
</tr>
<tr>
<td>Negative valenced emotional stimuli</td>
<td>female more reactive [134]</td>
<td></td>
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<tr>
<td>Parts of brain activated upon identification of happy emotional expression</td>
<td>significantly activated right precuneus/cuneus and left caudate body [135]</td>
<td>more diffuse activation of cortical and limbic regions in males [135]</td>
</tr>
<tr>
<td>Parts of brain activated upon identification of sad facial expression</td>
<td>Only activated posterior cingulate/precuneus [75,135,]</td>
<td>Posterior cingulate precuneus more activated than female, also activated mid-cingulate, insula, caudate, precuneus, middle frontal gyrus [75,135].</td>
</tr>
<tr>
<td>Emotional regulation</td>
<td>More use of emotion based strategy [136].</td>
<td>more activation of prefrontal regions and anterior cingulate [137], more use of cognitive based strategy [138]; early engagement in males [139]</td>
</tr>
<tr>
<td>Emotional contagion and imitation</td>
<td>More readily deployment of mimicry and emotional contagion [140]</td>
<td></td>
</tr>
<tr>
<td>Sex differences in the human mirror-neuron system</td>
<td>larger gray matter volume in the pars opercularis and inferior parietal lobule as compared to males; participants [141]</td>
<td>emotional empathic disposition was tightly coupled with larger gray matter volume of the pars opercularis across all female and male [141]</td>
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</table>

CONCLUSION

Research done on psychiatric conditions with their relations on smiling and crying behavior opens new door in understanding, managing and treating these patients. Gender differences in the neural mechanism processing smiling and crying answers long puzzled questions as to why women and men act differently when it comes to emotions. More research needs to be done in understanding how these facial expressions are regulated.

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