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Annals of Psychiatry and Mental Health

Review Article

Smiling and Crying

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Submitted: 21 June 2016

Accepted: 03 August 2016

Published: 05 August 2016

ISSN: 2374-0124

OPEN ACCESS

Keywords
• Smiling
• Crying
• Behaviors
• Pain or tension

Abstract

Smiling and crying has always caught the interest of the scientists. These basic human expressions are known to provide an insight into the mind of humans. Thus a need to regulate these behaviors is seen across all cultures. Both smiling and crying can be either emotional congruent or incongruent depending on the learned behavior or it can entirely be a brainstem mediated response having no connection to emotions or social behavior. Since smile and crying generally depict an emotional state of euphoria and sadness, respectively, these behaviors are seen across many psychiatric disorders. Malfunctioning of brain parts and gender differences involved in smiling and crying are discussed in this review article.

INTRODUCTION

Smile

Ekman and Friesen (1982) classified Smile into -felt smile, in response to a positive modification in circumstances or encounter of a pleasant stimuli 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 dampened 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 brainstem 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

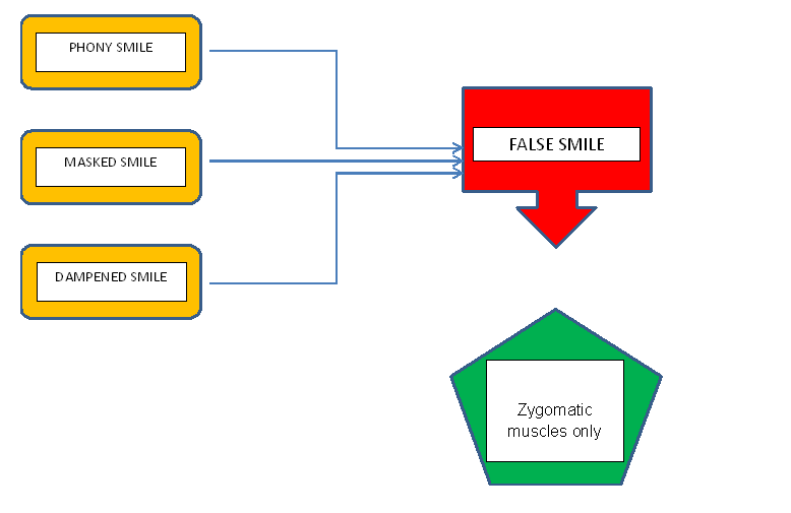

Figure 1 Genuine smile in response to pleasant stimulus. Genuine smiles results in Zygomatic and Orbicularis oculi both muscles contraction.

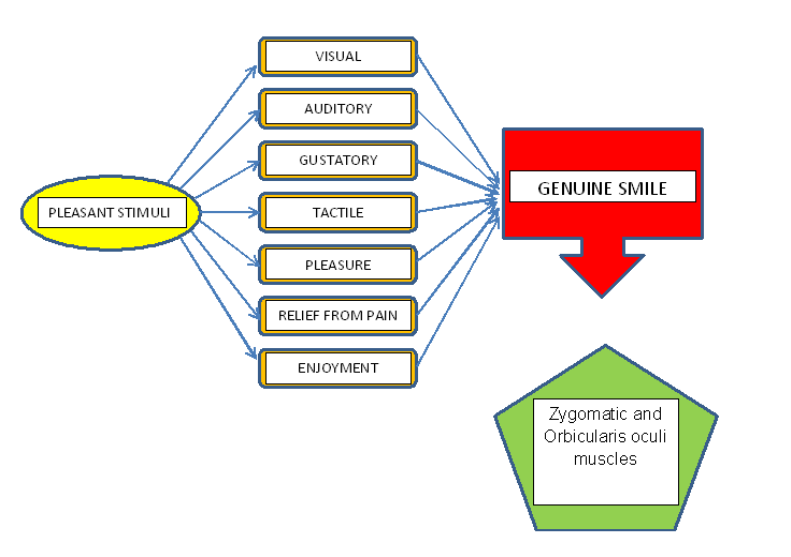

Figure 2 Classification of False smile. False smiles results in only Zygomatic muscles contraction, no contraction of Orbicularis oculi muscle.

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 andfronto-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

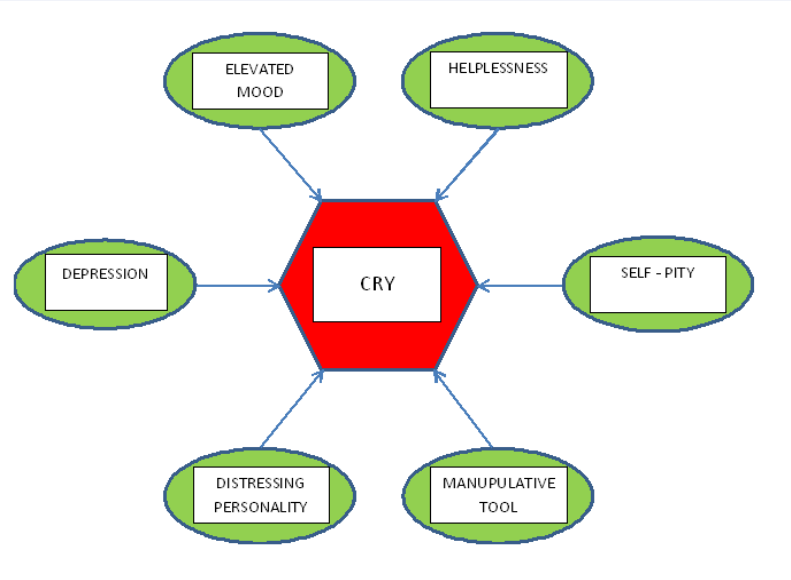

Figure 3 Significance of CRY.

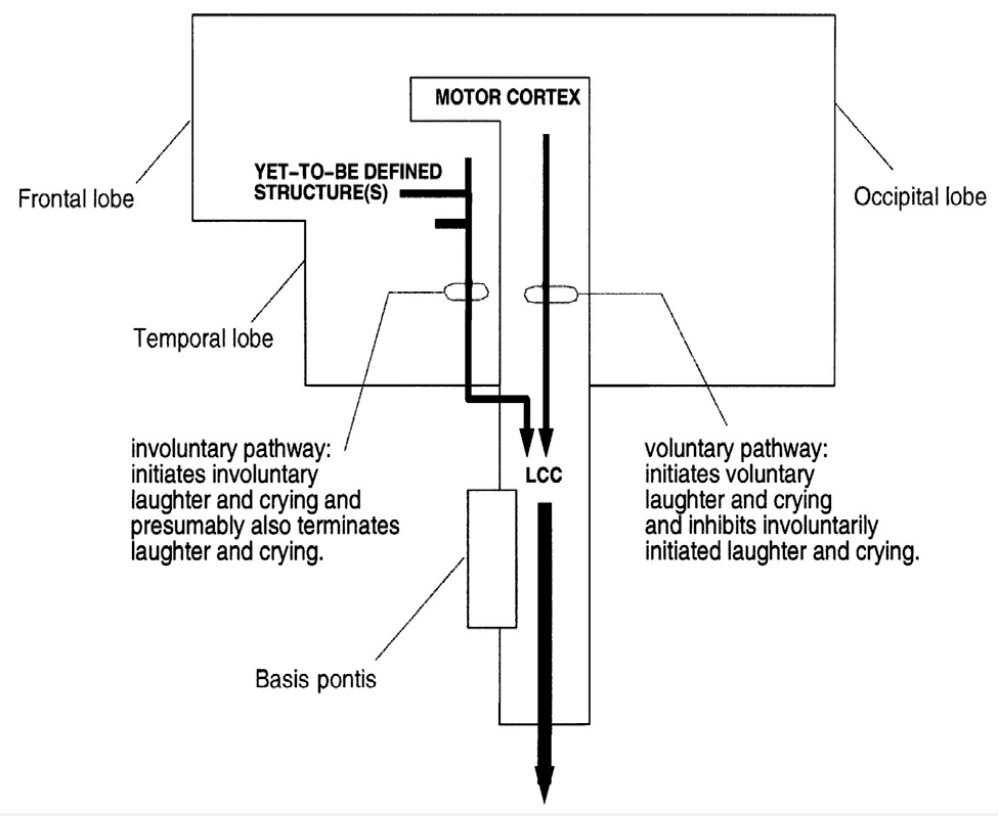

Figure 4 Neuro anatomical pathways for laughter and crying center (LCC). [21] (SOURCE: This figure is taken as it is) from Reference [21].

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, metalizing 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 gyruswhile 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 excitation [67]. Negative valenced stimuli have greater emotional salience than positive emotional stimuli [68]. Hence, a greater is 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 tobe 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 valanced 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 a 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 concludedleft 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 affectglobal 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 neuroimagingand 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 sconduct 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].

Posner 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].

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).

Table 1: Conditions Leading To Smiling 0r Crying Behavior and Their Neural Correlates.

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

Table 2: Gender Differences.

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

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.

REFERENCES

1. Ekman P, Friesen W. Felt, false and miserable smiles. Jrn Nonverbal Behav. 1982; 6: 238–252.
2. Ekman P. The Argument and Evidence about Universals in Facial Expression of Emotion. In: Wagner H & Manstead A, editors. Handbook of Social Psychophysiology. Chichester: John Wiley & Sons Ltd. 1989: 143-164.
3. Guillaume Duchenne. The Mechanism of Human Facial Expression. New York: Cambridge University Press. Originally published as Mecanisme de la Physionomie Humaine in 1862. 1990.
4. Ambadar Z, Cohn J, Reed L. All Smiles are Not Created Equal: Morphology and Timing of Smiles Perceived as Amused, Polite, and Embarrassed/Nervous. J Nonverbal Behav. 2009; 33: 17-34.
5. Ekman P, Davidson R, Friesen W. The Duchenne smile: Emotional expression and brain physiology: II. J Pers Soc Psychol. 1990; 58: 342–353.
6. Patel V. Crying behavior and psychiatric disorder in adults: a review. Compr Psychiatry 1993; 34: 206-211.
7. Green R, McAllister T, Bernat J. A study of crying in medically and surgically hospitalized patients. Am J Psychiatry. 1987; 144: 442-447.
8. Bindra D. Weeping, a problem of many facets. Bulletin of the British Psychological Society. 1972; 25: 281-284.
9. Frijda N. The laws of emotion. Am Psychol. 1988; 43: 349-358.
10. Koestler A. The act of creation. London: Hutchinson; 1964.
11. Kottler J. The language of tears. San Francisco, CA: Jossey-Bass; 1996.
12. Frey W, Hofman-Ahern C, Johnson R, Lykken D, Tuason V. Crying behavior in the human adult. Integrative Psychiatry. 1983; 1: 94-98.
13. Hastrup J, Baker J, Kraemer D, Bornstein R. Crying and depression among older adults. Gerontologist. 1986; 26: 91-96.
14. Lemire R, Beckwith J, Warkany J. Incidences, etiology, and epidemiology. Anencephaly. Raven, New York. 1978: 11-47.
15. Woods Michael. New Dimensions in Prenatal Ultrasound: 3D and 4D. 2014.
16. Yakovlev P, Lecours A. The myelogenetic cycles of regional maturation of the brain. In: Minkowski A, editor. Regional development of the brain in early life. Blackwell Scientific; Oxford, UK; 1967: 3-70.
17. Jürgens U. Neuronal control of mammalian vocalization, with special reference to the squirrel monkey. Naturwissenschaften. 1998; 85: 376-388.
18. Luyendijk W, Treffers P. The smile in anencephalic infants. Clin Neurol Neurosurg 1992; 94: 113-117.
19. Parvizi J, Anderson S, Martin C, Damasio H, Damasio A. Pathological laughter and crying: a link to the cerebellum. Brain. 2001; 124: 1708-1719.
20. Lopez O, Gonzalez M, Becker J, Reynolds C, Sudilosky A, DeKosky S. Symptoms of depression and psychosis in Alzheimer's disease and frontotemporal dementia: exploration of underlying mechanisms. Neuropsychiatry, Neuropsychology, and Behavioral Neurology. 1996; 9: 154–161.
21. Parvizi J, Anderson SW, Martin CO, Damasio H, Damasio AR. Pathological laughter and cryingA link to the cerebellum. Brain. 2001; 124: 1708-1719.
22. Jurgens U, Ploog D. On the neural control of mammalian vocalization. Trends in neurosciences. 1981; 4: 135-137.
23. López-Laso E, Mateos G, Camino L, Jimenez G, Esparza R. Giant hypothalamic hamartoma and dacrystic seizures. Epileptic Disord. 2007; 9: 90-93.
24. Spitz R, Wolf K. The smiling response: a contribution to the ontogenesis of social relations. Genet Psychol Monographs. 1946; 34: 57-125.
25. Joseph R. Neuropsychiatry, Neuropsychology, AND Clinical Neuroscience, second edition; 1996: 168.

26. Wolff P. Observations on the early development of smiling. In B.M. Foss, editors. Determinants of infant behavior. New York: John Wiley. 1963; 2: 113-138.

27. Herschkowitz N, Kagan J, Zilles K. Neurobiological bases of behavioral development in the first year. Neuropediatrics. 1997; 28: 296-306.

28. Grossmann T, Johnson M, Lloyd-Fox S, Biasi A, Deligianni F, Elwell C, et al. Early cortical specialization for face-to-face communication in human infants. Proc Biol Sci. 2008; 275: 2803-2811.

29. Hennenlotter A, Schroeder U, Erhard P, Castrop F, Haslinger B, Stoecker D, et al. A common neural basis for receptive and expressive communication of pleasant facial affect. Neuroimage. 2005; 26: 581-591.

30. Moscovitch M, Winocur G, Behrmann M. What is special about face recognition? Nineteen experiments on a person with visual object agnosia and dyslexia but normal face recognition. J Cogn Neurosci. 1997; 9: 555-604.

31. Haxby J, Hoffman E, Gobbini M. The distributed human neural system for face perception. Trends Cogn Sci. 2000; 4: 223–233.

32. Frith C. The social brain? Philos Trans R Soc Lond B Biol Sci. 2007; 362: 671–678.

33. Urakawa S, Takamoto K, Ishikawa A, Ono T, Nishijo H. Selective Medial Prefrontal Cortex Responses During Live Mutual Gaze Interactions in Human Infants: An fNIRS Study. Brain Topogr. 2015; 28: 691-701.

34. Carmody DP, Lewis M. Brain activation when hearing one's own and others' names. Brain Res. 2006; 1116:153-158.

35. Kawashima R, Sugiura M, Kato T, Nakanura A, Ito K, Fukuda H, et al. The human amygdala plays an important role in gaze monitoring. A PET study. Brain. 1999; 122: 779-783.

36. Nichols C, Ibañez L, Foss-Feig J, Stone W. Social smiling and its components in high-risk infant siblings without later ASD symptomatology. J Autism Dev Disord. 2014; 44: 894-902.

37. Schultz R, Grelotti D, Klin A, Kleinman J, Van der G, Marios R, et al. The role of the fusiform face area in social cognition: implications for the pathobiology of autism. Philos Trans R Soc Lond B Biol Sci. 2003; 358: 415-427.

38. Zilbovicius M, Meresse I, Boddaert N. [Autism: neuroimaging]. Rev Bras Psiquiatr. 2006; 28: 21-28.

39. Courchesne E, Mouton P, Calhoun M, Semendeferi K, Ahrens-Barbeau C, Gallet M, et al. Neuron number and size in prefrontal cortex of children with autism. JAMA. 2011; 306: 2001-2010.

40. Bell S, Ainsworth M. Infant crying and maternal responsiveness. Child Dev. 1972; 43: 1171-1190.

41. Nelson J. An ego psychological and object relations study of crying. Unpublished doctoral dissertation, California Institute for Clinical Social Work; 1979.

42. Byrne G, Raphael B, Arnold E. Alcohol consumption and psychological distress in recently widowed older men. Aust N Z J Psychiatry. 1999; 33: 740-747.

43. Maciejewski P, Zhang B, Block S, Prigerson H. An empirical examination of the stage theory of grief. JAMA. 2007; 297:716–723.

44. Bartels A, Zeki S. The neural correlates of maternal and romantic love. Neuroimage. 2004; 21:1155-1166.

45. Freed P, Yanagihara T, Hirsch J, Mann J. Neural mechanisms of grief regulation. Biol Psychiatry. 2009; 66: 33-40.

46. Dimberg U. Facial reactions to facial expressions. Psychophysiology. 1982; 19: 643-647.

47. Bandura A, Rosenthal T. Vicarious classical conditioning as a function of arousal level. J Pers Soc Psychol. 1966; 3: 54–62.

48. Hatfield E, Cacioppo J, Rapson R. Emotional Contagion. Paris: Cambridge University Press; 1994.

49. Decety J, Jackson P. The functional architecture of human empathy. Behav Cogn Neurosci Rev. 2004; 3: 71-100.

50. van Baaren R, Holland R, Kawakami K, van Knippenberg A. Mimicry and prosocial behavior. Psychol Sci. 2004; 15: 71-74.

51. Leighton J, Bird G, Orsini C, Heyes C. Social attitudes modulate automatic imitation. J Exp Soc Psych. 2010; 46: 905-910.

52. Rychlowska M, Cañadas E, Wood A, Krumhuber E, Fischer A, Niedenthal P. Blocking mimicry makes true and false smiles look the same. PLoS One. 2014. 9: e90876.

53. Iacoboni M, Woods RP, Brass M, Bekkering H, Mazziotta JC, Rizzolatti G. Cortical mechanisms of human imitation. Science. 1999; 286: 2526-2528.

54. Carr L, Iacoboni M, Dubeau , Mazziotta JC, Lenzi GL. Neural mechanisms of empathy in humans: a relay from neural systems for imitation to limbic areas. Proc Natl Acad Sci USA 2003; 100: 5497-5502.

55. Leslie K, Johnson-Frey S, Grafton S. Functional imaging of face and hand imitation: towards a motor theory of empathy. Neuroimage. 2004; 21: 601-607.

56. Iacoboni M. Neural mechanisms of imitation. Curr Opin Neurobiol. 2005; 15: 632-637.

57. Hoffman E, Haxby J. Distinct representations of eye gaze and identity in the distributed human neural system for face perception. Nat Neurosci. 2000; 3: 80–84.

58. Wang Y, Ramsey R, Hamilton A. The control of mimicry by eye contact is mediated by medial prefrontal cortex. J Neurosci. 2011; 31:12001-12010.

59. Luria A. Higher cortical functions in man. 2nd edition. New York: Basic Books; 1980.

60. Dapretto M, Davies M, Pfeifer J, Scott AA, Sigman M, Bookheimer SY, et al. Understanding emotions in others: mirror neuron dysfunction in children with autism spectrum disorders. Nat Neurosci. 2006; 9:28-30.

61. Yuan T, Hoff R. Mirror neuron system based therapy for emotional disorders. Med Hypotheses. 2008; 71:722-726.

62. Camras L, Allison K. Children's understanding of emotional facial expressions and verbal labels. Journal of Nonverbal Behavior. 1985; 9: 84-94.

63. Davidson R. Anterior cerebral asymmetry and the nature of emotion. Brain Cogn. 1992; 20: 125-151.

64. Ahern G, Schwartz G. Differential lateralization for positive vs negative emotion. Neuropsychologia. 1979; 17: 693–698.

65. Schlosberg H. Three dimensions of emotion. Psychol Rev. 1954; 61:81-88.

66. Frijda N. The Emotions. New York: Cambridge University Press; 1987.

67. Lang P, Greenwald M, Bradley M, Hamm A. Looking at pictures: affective, facial, visceral, and behavioral reactions. Psychophysiology. 1993; 30: 261-273.

68. Posner J, Russell J, Gerber A, Gorman D, Colibazzi Y, Yu S, et al. The neurophysiological bases of emotion: An fMRI study of the affective circumflex using emotion-denoting words. Hum Brain Mapp. 2009; 30: 883-895.

69. Murphy F, Nimmo-Smith I, Lawrence A. Functional neuroanatomy of emotion: A meta-analysis. Cogn Affect Behav Neurosci. 2003; 3: 207-233.

70. MacNamara A, Rabinak CA, Fitzgerald DA, Zhou XJ, Shankman SA, Milad MR, et al. Neural correlates of individual differences in fear learning. Behav Brain Res. 2015; 287: 34-41.

71. Donofry SD, Roecklein KA, Wildes JE, Miller MA, Erickson KI. Alterations in emotion generation and regulation neurocircuitry in depression and eating disorders: A comparative review of structural and functional neuroimagingstudies. Neurosci Biobehav Rev. 2016; 68: 911-927.

72. McCloskey MS, Phan KL, Angstadt M, Fettich KC, Keedy S, Coccaro EF,et al . Amygdala hyperactivation to angry faces in intermittent explosive disorder. J Psychiatr Res. 2016; 79: 34-41.

73. Davis M, Whalen PJ. The amygdala: vigilance and emotion. Mol Psychiatry. 2001; 6: 13-34.

74. Murray EA. The amygdala, reward and emotion. Trends Cogn Sci. 2007; 11: 489-497.

75. Phan KL, Wager T, Taylor SF, Liberzon I. Functional neuroanatomy of emotion: a meta-analysis of emotion activation studies in PET and fMRI. Neuroimage. 2002; 16: 331-348.

76. Kim H, Somerville LH, Johnstone T, Alexander AL, Whalen PJ. Inverse amygdala and medial prefrontal cortex responses to surprised faces. Neuroreport. 2003; 14: 2317-2322.

77. Ghashghaei HT, Hilgetag CC, Barbas H. Sequence of information processing for emotions based on the anatomic dialogue between prefrontal cortex and amygdala. Neuroimage. 2007; 34: 905-923.

78. Damasio AR, Grabowski TJ, Bechara A, Damasio H, Ponto LL, Parvizi J, et al. Subcortical and cortical brain activity during the feeling of self-generated emotions. Nat Neurosci. 2000; 3: 1049-1056.

79. Bush G, Luu P, Posner MI. Cognitive and emotional influences in anterior cingulate cortex. Trends Cogn Sci. 2000; 4: 215-222.

80. Coppens E, Spruyt A, Vandenbulcke M, Van Paesschen W, Vansteenwegen D. Classically conditioned fear responses are preserved following unilateral temporal lobectomy in humans when concurrent US-expectancy ratings are used. Neuropsychologia. 2009; 47: 2496-2503.

81. Beauregard M, Leroux JM, Bergman S, Arzoumanian Y, Beaudoin G, Bourgouin P, et al. The functional neuroanatomy of major depression: an fMRI study using an emotional activation paradigm. Neuroreport. 1998; 9: 3253-3258.

82. Veissier I, Boissy A. Stress and welfare: two complementary concepts that are intrinsically related to the animal's point of view. Physiol Behav. 2007; 92: 429-433.

83. LeDoux JE. Emotion circuits in the brain. Annu Rev Neurosci. 2000; 23: 155-184.

84. Olsson A, Nearing KI, Phelps EA. Learning fears by observing others: the neural systems of social fear transmission. Soc Cogn Affect Neurosci. 2007; 2: 3-11.

85. Pessoa L, Kastner S, Ungerleider LG. Attentional control of the processing of neural and emotional stimuli. Brain Res Cogn Brain Res. 2002; 15: 31-45.

86. Britton JC, Taylor SF, Sudheimer KD, Liberzon I. Facial expressions and complex IAPS pictures: common and differential networks. Neuroimage. 2006; 31: 906-919.

87. Ochsner KN, Ray RR, Hughes B, McRae K, Cooper JC, Weber J, et al. Bottom-up and top-down processes in emotion generation: common and distinct neural mechanisms. Psychological Science. 2009; 20(11): 1322–1331.

88. Anderson A, Phelps E. Is the human amygdala critical for the subjective experience of emotion? Evidence of intact dispositional affect in patients with amygdala lesions. Journal of Cognitive Neuroscience. 2002; 14: 709-720.

89. Comte M, Schön D, Coull J, Reynaud E, Khalfa S, Belzeaux R et al. Dissociating Bottom-Up and Top-Down Mechanisms in the Cortico-Limbic System during Emotion Processing. Cereb Cortex 2016; 26: 144-155.

90. Gu X1, Hof PR, Friston KJ, Fan J. Anterior insular cortex and emotional awareness. J Comp Neurol. 2013; 521: 3371-3388.

91. Phillips ML, Drevets WC, Rauch SL, Lane R. Neurobiology of emotion perception I: The neural basis of normal emotion perception. Biol Psychiatry. 2003; 54: 504-514.

92. Hermans EJ, van Wingen G, Bos PA, Putman P, van Honk J. Reduced spontaneous facial mimicry in women with autistic traits. Biol Psychol. 2009; 80: 348-353.

93. de Wied M, van Boxtel A, Zaalberg R, Goudena PP, Matthys W. Facial EMG responses to dynamic emotional facial expressions in boys with disruptive behavior disorders. J Psychiatr Res. 2006; 40: 112-121.

94. Schwartz GE, Fair PL, Mandel MR, Salt P, Mieske M, Klerman GL, et al. Facial electromyography in the assessment of improvement in depression. Psychosom Med. 1978; 40: 355-360.

95. Medford N, Critchley HD. Conjoint activity of anterior insular and anterior cingulate cortex: awareness and response. Brain Struct Funct. 2010; 214: 535-549.

96. Posner J, Russell JA, Peterson BS. The circumplex model of affect: an integrative approach to affective neuroscience, cognitive development, and psychopathology. Dev Psychopathol. 2005; 17: 715-734.

97. O'Doherty J, Kringelbach ML, Rolls ET, Hornak J, Andrews C. Abstract reward and punishment representations in the human orbitofrontal cortex. Nat Neurosci. 2001; 4: 95-102.

98. Peciña S, Schulkin J, Berridge KC. Nucleus accumbens corticotropin-releasing factor increases cue-triggered motivation for sucrose reward: paradoxical positive incentive effects in stress. BMC Biol. 2006; 4: 8.

99. Kühn S, Gallinat J. The neural correlates of subjective pleasantness. Neuroimage. 2012; 61: 289-294.

100. Schoenbaum G, Takahashi Y, Liu TL, McDannald MA. Does the orbitofrontal cortex signal value. Ann N Y Acad Sci. 2011; 1239: 87-99.

101. Shore DM, Heerey EA. The value of genuine and polite smiles. Emotion. 2011; 11: 169-174.

102. O'Doherty J, Critchley H, Deichmann R, Dolan, R. Dissociating valence of outcome from behavioral control in human orbital and ventral prefrontal cortices. J Neurosci. 2003; 23: 7931-7939.

103. Okun M, Bowers D, Springer U, Shapira NA, Malone D, Rezai AR, et al. What's in a"smile" Intra-operative observations of contralateral smiles induced by deep brain stimulation. Neurocase. 2004; 10: 271-279.

104. Kringelbach ML, O'Doherty J, Rolls ET, Andrews C. Activation of the human orbitofrontal cortex to a liquid food stimulus is correlated with its subjective pleasantness. Cereb Cortex. 2003; 13: 1064-1071.

105. Kringelbach ML, Rolls ET. The functional neuroanatomy of the human orbitofrontal cortex: evidence from neuroimaging and neuropsychology. Prog Neurobiol. 2004; 72: 341-372.

106. Goel V, Dolan R. Reciprocal neural response within lateral and ventral medial prefrontal cortex during hot and cold reasoning. Neuroimage. 2003; 20: 2314-2321.

107. Kühn S, Müller BC, van der Leij A, Dijksterhuis A, Brass M, van Baaren RB, et al. Neural correlates of emotional synchrony. Soc Cogn Affect Neurosci. 2011; 6: 368-374.

108. Etkin A, Egner T, Kalisch R. Emotional processing in anterior cingulate and medial prefrontal cortex. Trends Cogn Sci. 2011; 15: 85-93.

109. Barrash J, Tranel D, Anderson S. Acquired personality disturbances associated with bilateral damage to the ventromedial prefrontal region. Dev Neuropsychol 2000; 18: 355-381.

110. Blair KS, Smith BW, Mitchell DG, Morton J, Vythilingam M, Pessoa L, et al. Modulation of emotion by cognition and cognition by emotion. Neuroimage. 2007; 35: 430-440.

111. Wager TD, Davidson ML, Hughes BL, Lindquist MA, Ochsner KN. Prefrontal-subcortical pathways mediating successful emotion regulation. Neuron. 2008; 59: 1037-1050.

112. Beck A, Rush A, Shaw B, Emery G. Cognitive therapy of depression. New York: Guilford; 1979.

113. Surguladze SA, Young AW, Senior C, Brébion G, Travis MJ, Phillips ML, et al. Recognition accuracy and response bias to happy and sad facial expressions in patients with major depression. Neuropsychology. 2004; 18: 212-218.

114. Persad SM, Polivy J. Differences between depressed and nondepressed individuals in the recognition of and response to facial emotional cues. J Abnorm Psychol. 1993; 102: 358-368.

115. Fu C, Williams S, Cleare A, Brammer MJ, Walsh ND, Kim J, et al. Attenuation of the neural response to sad faces in major depression by antidepressant treatment: a prospective, event-related functional magnetic resonance imaging study. Arch Gen Psychiat 2004; 61: 877-889.

116. Fu C, Williams S, Brammer M, Suckling, Kim J, Cleare AJ et al. Neural responses to happy facial expressions in major depression following antidepressant treatment. Am J Psychiatry. 2007; 164: 599-607.

117. Critchley H, Daly E, Phillips M, Brammer M, Bullmore E, Williams S, et al. Explicit and implicit neural mechanisms for processing of social information from facial expressions: a functional magnetic resonance imaging study. Hum Brain Mapp. 2000; 9: 93-105.

118. Mayberg HS, Brannan SK, Tekell JL, Silva JA, Mahurin RK, McGinnis S, et al. Regional metabolic effects of fluoxetine in major depression: serial changes and relationship to clinical response. Biol Psychiatry. 2000; 48: 830-843.

119. Kempton MJ, Salvador Z, Munafò MR, Geddes JR, Simmons A, Frangou S, et al. Structural neuroimaging studies in major depressive disorder. Meta-analysis and comparison with bipolar disorder. Arch Gen Psychiatry. 2011; 68: 675-690.

120. Wagner G, Koch K, Schachtzabel C, Reichenback, Sauer H, Schlosser Md RG, et al. Enhanced rostral anterior cingulate cortex activation during cognitive control is related to orbitofrontal volume reduction in unipolar depression. J Psychiatry Neurosci 2008; 33: 199-208.

121. Costafreda S, McCann P, Saker P, Cole JH, Cohen-Woods S, Farmer AE, et al. Modulation of amygdala response and connectivity in depression by serotonin transporter polymorphism and diagnosis. J Affect Disord. 2013; 150: 96-103.

122. Rubinsztein JS, Fletcher PC, Rogers RD, Ho LW, Aigbirhio FI, Paykel ES, et al. Decision-making in mania: a PET study. Brain. 2001; 124: 2550-2563.

123. Bermpohl F, Kahnt T, Dalanay U, Hägele C, Sajonz B, Wegner T, et al. Altered representation of expected value in the orbitofrontal cortex in mania. Hum Brain Mapp. 2010; 31: 958-969.

124. Risinger RC, Salmeron BJ, Ross TJ, Amen SL, Sanfilipo M, Hoffmann RG, et al. Neural correlates of high and craving during cocaine self-administration using BOLD fMRI. Neuroimage. 2005; 26: 1097-1108.

125. Barr J, Forster G, Unterwald E. Repeated cocaine enhances ventral hippocampal-stimulated dopamine efflux in the nucleus accumbens and alters ventral hippocampal NMDA receptor subunit expression. J Neurochem. 2014; 130: 583-590.

126. Sellings LH, Clarke PB. Segregation of amphetamine reward and locomotor stimulation between nucleus accumbens medial shell and core. J Neurosci. 2003; 23: 6295-6303.

127. Payer DE, Lieberman MD, London ED. Neural correlates of affect processing and aggression in methamphetamine dependence. Arch Gen Psychiatry. 2011; 68: 271-282.

128. Paulus MP, Hozack NE, Zauscher BE, Frank L, Brown GG, Braff DL, et al. Behavioral and functional neuroimaging evidence for prefrontal dysfunction in methamphetamine-dependent subjects. Neuropsychopharmacology. 2002; 26: 53-63.

129. French ED, Dillon K, Wu X. Cannabinoids excite dopamine neurons in the ventral tegmentum and substantia nigra. Neuroreport. 1997; 8: 649-652.

130. Frenzilli G, Ferrucci M, Giorgi F, Blandini F, Nigro M, Ruggieri S, et al. DNA fragmentation and oxidative stress in the hippocampal formation: a bridge between 3, 4-methylenedioxymethamphetamine (ecstasy) intake and long-lasting behavioral alterations. Behav Pharmacol. 2007; 18: 471-481.

131. Carhart-Harris R, Murphy K, Leech R, Erritzoe, Wall MD, Ferguson B, et al. The effects of acutely administered 3,4-methylenedioxymethamphetamine on spontaneous brain function in healthy volunteers measured with arterial spin labeling and blood oxygen level-dependent resting state functional connectivity. Biol Psychiatry. 2015; 78: 554-562.

132. LaFrance M, Hecht M. Option or obligation to smile: The effect of power and gender on facial expression. In P. Philippot, R. S. Feldman,& E. J. Coats (Eds.), The social context of nonverbal behavior Cambridge, England: Cambridge University Press. 1999; 45-70.

133. Montagne B, Kessels RP, Frigerio E, de Haan EH, Perrett DI. Sex differences in the perception of affective facial expressions: do men really lack emotional sensitivity. Cogn Process. 2005; 6: 136-141.

134. Gohier B, Senior C, Brittain PJ, Lounes N, El-Hage W, Law V, et al. Gender differences in the sensitivity to negative stimuli: cross-modal affective priming study. Eur Psychiatry. 2013; 28: 74-80.

135. Weisenbach SL, Rapport LJ, Briceno EM, Haase BD, Vederman AC, Bieliauskas LA, et al. Reduced emotion processing efficiency in healthy males relative to females. Soc Cogn Affect Neurosci. 2014; 9: 316-325.

136. Vingerhoets AJ, Van Heck GL. Gender, coping and psychosomatic symptoms. Psychol Med. 1990; 20: 125-135.

137. Mak AK, Hu ZG, Zhang JX, Xiao Z, Lee TM. Sex-related differences in neural activity during emotion regulation. Neuropsychologia. 2009; 47: 2900-2908.

138. McRae K, Ochsner K, Mauss I, Gabrieli J, Gross J. Gender differences in emotion regulation: An fMRI study of cognitive reappraisal. Group Processes and Intergroup Relations 2008; 11: 143-162.

139. Williams LM, Barton MJ, Kemp AH, Liddell BJ, Peduto A, Gordon E,

et al. Distinct amygdala-autonomic arousal profiles in response to fear signals in healthy males and females. Neuroimage. 2005; 28: 618-626.

140. Doherty R, Orimoto L, Singelis T, Hatfield E, Hebb J. Emotional contagion. Gender and occupational differences. Psychol Women Q. 1995; 19: 355-371.

141. Cheng Y, Chou KH, Decety J, Chen IY, Hung D, Tzeng OJ, et al . Sex differences in the neuroanatomy of human mirror-neuron system: a voxel-based morphometric investigation. Neuroscience. 2009; 158: 713-720.

Cite this article

Ali S, Jab S, Kaur A, Nathani M, Shahid M (2016) Smiling and Crying. Ann Psychiatry Ment Health 4(5): 1076.