Differentiation of Primary Emotions through Neuromodulators: Review of Literature

Emotional mental disorders are a leading cause of disabilities worldwide. Despite the obvious importance of emotion for human existence, controversy still abounds over the definition of emotion, the number of emotions that exit, whether different emotions have different physiological signatures. In this paper, we review papers about the emotional functions of neuromodulators: dopamine (DA), norepinephrine (NE), serotonin (5-HT) and acetyl choline (ACh), and propose that they act as the primary neural basis for 5 primary emotions (joy, anger/fear, sadness and missing). DA is a rewarding signal for salient stimuli such as food, sex and other needs. 5-HT has been related to depression for decades, it might be a marker for dislike/disgusting things. NE is the substrate for surprise, whose release induces “fight or flight” behaviors, or fear and anger emotions. Even though anger and fear are common emotions, there are no previous papers differentiating them or probing into their relationships. Here we found that fear and anger usually come hand in hand with fear followed by anger. And we give an equation to describe their relationships: The total amount of Surprise/stress = fear + anger. This equation fits in both duration and tension. Finally we introduced ACh emotions, which are opposite to NE induced stressful emotions. These emotions include “missing and wanting”, “calm/tame”, affectionate, submissive, et al. The significance of ACh emotions lies in the treatment for aggressive, impulsive behaviors, or phobia and manic disorders. In all, there might be 5 basic emotions (joy, anger/fear, sadness and missing).

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INTRODUCTION

Emotional mental disorders are a leading cause of disabilities worldwide. Despite the obvious importance of emotion for human existence, scientists concerned with human nature have not been able to reach a consensus about what emotion is[1]. Controversy still abounds over the definition of emotion, the number of emotions that exit, whether different emotions have different physiological signatures[1-4]. Although there has been no shortage of psychological research on these topics, the findings have not resolved many of the issues. Information about the representation of emotions in the brain may shed light on the nature of emotional processes[5-11]. Like color, people will never imagine that there are only three primary colors, if not for the physiological proof of three different kinds of conic cells in the eye, but emotion seems much more complicated.

Studies of the neural basis of emotion have a long history, and are
still an active field of experimental and theoretical research. Most of the work focuses on identifying neural structures responsible for the experience of particular emotions. This kind of research culminated around mid-century in the limbic system theory of emotion. The neural anatomical basis of emotion helped us find important structures involved in primary emotions, but the structure for emotions is usually mixed and widespread in the brain, which suggests an alternative approach: neuromodulators. With the development of drugs for the affective disorders, catecholamine was shown to be the substrate for emotion ever since the 50s-60s in last century\[9]. Even though many years have passed, the effects of catecholamine in treating affective diseases are still quite mixed. For example, antidepressant drugs affect almost all the catecholamine neuromodulators and are used for almost all affective diseases such as anxiety, phobia, depression et al. There is a need to improve the conceptualization and classification of the emotional states and the involvement of the neuromodulators. Here we review papers about their differences in modulating emotions, and propose that these three monoamine neuromodulators together with ACh underlie the

5 primary emotions of human (DA-pleasant, 5-HT-unpleasant, NE-sadness, AMPH-anxiety, ACh-anger).

**DOPAMINE (DA) – PLEASANT MARKER**

DA has been linked to rewarding processes in the brain since 1980, when Wise first proposed the hedonic hypothesis of DA\[8\], who proposed that DA is a signal of stimulus salience, providing feeling of enjoyment. DA is a rewarding signal for salient stimuli such as food, sex and other needs, which are excited by reward seeking behaviors. The reward depends on the needs of subjects. Human needs were classified by Maslow (1943) as the hierarchy of needs, from physiological needs to social needs. The physiological needs are required for human survival; they are so primitive in evolution that they responded just automatically, and they were used as unconditioned rewarding stimuli in Pavlovian experiments. Social needs include love, trust, esteem, pride and self-actualization etc, which depends upon human appraisal.

Many pharmacological and behavioral studies on intracranial self-stimulation established the important role of the medial prefrontal DA system in positively motivated behavior\[16\]. We don’t need to review them here, for the rewarding role of DA has been well documented in many aspects. For example, drug addictions are involved in the reward system by affecting DA release and reuptake. Imaging studies revealed that drug-naive individuals show enhanced DA levels in the striatum upon exposure to psycho-stimulants. Imaging studies revealed that drug-naive individuals show enhanced DA levels in the striatum upon exposure to psycho-stimulants. Social incentives include love, trust, esteem, pride and self-actualization etc, which depends upon human appraisal.

Over the past several decades, extensive studies have refined the function of DA, and theories concerning the role of midbrain DA on behavior have also changed dramatically\[12,13\]. For example, empirical evidence revealed that global DA depletion does not impair hedonic (liking) response to primary reward such as the preference for sucrose over water. These observations have led to a formulation that the contribution of DA includes an effect on motivated behavior toward desired goals. Mesolimbic DA is also involved in aversively motivated behaviors. Medial hypothalamic stimulation, which has been considered to cause a primary aversive state, cause significant decrease in extracellular DA\[10]. However, when rats can lever-press to escape the stimulation, NAc DA increased instead. This is similar to findings from Cabib\[11\], that when animals were allowed to control the painful shock experience, NAc DA metabolites increased.

The most interesting study about DA is prediction error. Prediction error was proposed by Schultz et al\[16\] who discriminate role of DA in reward and prediction. Their connection derives from a wide variety of conditioning experiments. They proposed that the function of DA is a signal of salience of stimulus, for example, DA neurons are activated when animals touch a small morsel of apple or receive a small quantity of fruit juice to the mouth. These phased activations do not, however, discriminate between these different types of rewarding stimuli. Aversive stimuli like air puffs to the hand or drops of saline to the mouth do not cause these same transient activations. This unconditioned reaction can be connected to some neutral stimuli. For example, if presentation of a light is consistently followed by food, a rat will learn that the light predicts the future arrival of food. Only the light with food will induce DA release. The prediction-based explanation is that the light fully predicts the food that arrives. Surprisingly, once the stimulus-reward association is learned, reward delivery no longer elicits an increase in the activity of DA neurons as it is fully expected\[16\]. It appears therefore that learning is driven by deviations or “errors” between the predicted time and amount of rewards. So Schultz et al proposed that DA encodes expectations about external stimuli or reward, especially when it is uncertain or deviation or error (prediction error)\[14\].

**SEROTONIN (5-HT) – UNPLEASANT MARKER**

5-HT has been related to depression for decades mostly because of the antidepressant drugs, and till now, the mostly prescribed drugs for depression are drugs that alter 5-HT levels. Even though 5-HT functions as a neurotransmitter in the nervous system, it is mostly released in the gut (about 90%), where enterochromaffin cells release 5-HT in response to noxious substance in the food. 5-HT makes the gut move faster, causing vomiting or diarrhea. Plant seeds with 5-HT exploit this reaction to speed the passage of seeds through the digestive tract. Animals such as wasps and scorpion sting can induce pain, for 5-HT presents in their venom. So 5-HT give objects a negative/disliked marker. Even in phylogenetically distant animals such as the mollusk, 5-HT release is involved in the regulation of avoidance behavior\[17\]. Drugs which block 5-HT (5-HT2c receptors) make the body unable to shut off appetite, and are associated with increased weight gain\[18\]. It is really the case that people with low 5-HT showed more aggressive behavior, don’t care about the negative (punishment) results, they even commit suicide. In 1976, Asberg and Traskman-Bendz identified a subgroup of depressed subjects with low cerebrospinal fluid (CSF) concentrations of 5-HIAA who had a history of serious suicide attempts\[19,20\]. And thereafter, the correlation between low CSF 5-HIAA concentrations and increased risk for suicide has become one of the most reproducible findings in biological psychiatry, with over 20 studies finding low CSF 5-HIAA concentrations in populations that attempt suicide\[21,22\]. Some other studies have suggested that the deficits in 5-HT functioning within the frontal cortex may underlie the other behavior disturbance, such as impaired impulse control, increased incidence of violent episodes\[23,24\]. Some studies even suggested that low 5-HIAA (5-HT metabolite) in CSF is a marker for the predisposition to a wide array of psychopathological problems, such as impulse control, suicide, impulsive fire setting, or frequent violent criminal behavior, even
alcohol intake and dependence\textsuperscript{[29]}. 5-HT in the central nervous system (CNS) mostly produced by neurons in raphe nuclei, and released into extracellular space between neurons in almost every part of the CNS, such as medial prefrontal cortex, amygdala, hippocampus, and is involved in appetite, sleep, mood, emotion\textsuperscript{[30]}. These functions have been revised in the past years. For example, it was thought that 5-HT induced sleep, one of the main arguments related to the insomnia following the destruction of midbrain raphe nuclei in the cat\textsuperscript{[27,28]}. But it is currently accepted that 5-HT functions predominantly to promote wakefulness and to inhibit REM sleep\textsuperscript{[29]}, for the activity of serotonergic neurons of the dorsal raphe nuclei decreases from waking through slow wave sleep to REM sleep. Only under some circumstance it contribute to the increase in sleep propensity\textsuperscript{[30]}. So the function of 5-HT might act as a tranquilizer, and many studies support this. For example, tryptophan, which is easily converted to 5-HT in the body, is used as a tranquilizer, and it has been proved that 5-HT is involved in the biology of torpor and hibernation and it inhibits mitochondrial respiration.

Abundant evidence points to a decrease of the seotoninergic system in anxiety, phobias, panic attacks and post-traumatic stress and depression disorders based on effects of treatments that enhance 5-HT, and the most popular kind of antidepressant is said to increase the action of 5-HT in the brain, however, it has been difficult to establish a primary role for 5-HT deficiency in these diseases\textsuperscript{[30]}. Even though the company called these drugs antidepressant, actually they are good for aggressive or anxiety behaviors instead of depression. Actually these drugs affect all the catecholamine (DA, NE and 5-HT). For example, the first generation of antidepressants: the monoamine oxidase inhibitor has similar effect on all catecholamine neuromodulators. The drug companies are finding more specific drugs, the third generation of antidepressants: the 5-HT specific reuptake inhibitors. While it is true that these newer drugs increase the actions of 5-HT, it is hard to find their effects on depressant. On the contrary, selective 5-HT reuptake inhibitors have many depression-like side effects, such as apathy, nausea/vomiting, drowsiness, weight loss or diminished libido, even a higher risk of suicidal behavior in children. These drugs’ improvement of depressive symptoms, sometimes better than placebo, might be the result of increased social interaction subsequent to a reduction in fear and avoidance\textsuperscript{[31]}. Injecting 5-HT never reduced depression, sometimes NE or DA does. Injecting 5-HT, or increasing its activity, can cause sedation, helplessness. Helpless learning, a behavioral depression caused by exposure to inescapable stress, is considered to be an animal model of human depressive disorder. Cortical 5-HTergic excess is causally related to the development of learned helplessness\textsuperscript{[32]}, and learned helplessness patients have high level of 5-HT. If increasing 5-HT is not the cure, then maybe decreasing 5-HT will be the cure. Indeed, chemicals that antagonize 5-HT do seem to function as antidepressants\textsuperscript{[33]}. It has been found that 1-tryptophan depresses while levodopa intensifies emotional reactivity, the former lowering the level of endogenous 5-HT. And it was also found that lack of 5-HT enhances the emotional reactivity to learned fear memories\textsuperscript{[34]}. So the major benefit of the 5-HT drug is alleviated anxiety and improved sleep. So the term antidepressants is not correct, actually it acts as a tranquilizer. It never relieved depression, actually it induces more depression.

Genetic proofs also support this point. Pet1 is specifically expressed in the 5-HT neurons and directly activates the transcription of genes implicated in the serotonergic machinery. Compared to WT and Pet +/- littermates, Pet +/- mice has an 80% reduction in 5-HT tissue levels, with no significant changes in the levels of DA\textsuperscript{[35]}. These mice showed normal ambulatory activity in a novel arena; however, they explored more in the aversive areas, which suggests lacking of dislike marker due to low 5-HT. Additionally, these mice showed increased levels of aggression as measured by the resident-intruder assay\textsuperscript{[36]}. The hydroxylation of tryptophan into 5-hydroxy-tryptophan is the rate-limiting step in the synthesis of 5-HT. Two isoforms of the enzyme tryptophan hydroxylase, Tph1 and Tph2 are responsible for catalyzing this reaction. Tph2 is exclusively expressed in the 5-HT neurons of the raphe nuclei. Genetic deletion of Tph2 was obtained in several groups. The depressive-like behavior of the Tph2 +/- mice was evaluated. Savelieva and colleagues\textsuperscript{[37]} found that these mice spent less time immobile in the test suggestive of an antidepressant phenotype.

Another example is the vesicular monoamine transporter (Vmat) 2, which transports monoamines into the synaptic vesicle. The homozygote Vmat2 +/- induced a major depletion of all monoamines and die within a few days after birth\textsuperscript{[37]}. In contrast, heterozygote Vmat2 +/- mice, have 34% decrease in brain 5-HT, are viable and showed normal growth rate and behavior. These animals also showed significant reduction in level of DA 42% and NE, 23%. Interestingly, they showed pronounced depressive-like phenotype characterized by increased immobility\textsuperscript{[38]}. In contrast, no anxiety phenotype was detected in a large battery of tests. To overcome the lack of specificity, a conditional knockout mouse was created by crossing Sert mice with Cmat2, allowing for the deletion of the Vmat2 gene specifically in 5-HT neurons\textsuperscript{[39]}. No depression like phenotype was observed; instead they showed less latency to reach out the food pellet in a NSF test, suggesting an anxiolytic-like phenotype. Overall, both pharmacological and genetic studies reviewed above prove that 5-HT is an aversive marker, which induces emotional depression.

**NOREPINEPHRINE (NE)– SURPRISE MARKER**

DA and 5-HT give the value of the objects around us according to whether they fit our needs or if they are dangerous to us. But everything in the world, whether it is positive or negative, occurs in anticipated ways (expected) or unanticipated ways (surprisingly). If it happens as anticipated, people feel calm; instead if it happened surprisingly, the first reaction of people would be scared and angry. For example, the door was knocked very loudly, while you were focusing on your reading. The first reaction is scared and then angry about why it was knocking so loudly. After opening the door, you can decide if it is happy (if it is what you wanted) or sad (if it is not what you wanted) (refer to figure 1). So fear and anger are not due to likeness or dislikeness of things themselves, instead they are due to unexpected ways these things happen. Let us take one example from Izzard’s paper\textsuperscript{[40]}, when Rafe was hit from the back by a wheel chair, the first reaction of him was scared and angry, and showed angry expression and clenched fist. But after he turned back to see Rebecca, a person with hemiplegia whose wheelchair had gone out of control and cause her to crash into Rafe. Rafe’s understanding changed his anger to sadness and sympathy.

The substrate for both fear and anger is NE. In the periphery autonomous nervous system, NE acts as a sympathetic neurotransmitter, together with hypothalamic-pituitary-adrenal (HPA), they act as stress hormones, which underlie “fight-or-flight” responses, directly increasing heart rate, triggering the release of glucose, and increasing blood flow to skeletal muscles. In the CNS, the NE system is considered to play an important role in attention, sleep/wakefulness, emotion and central responses to...
Emotions are due to catecholamine (DA, NE and 5-HT) and Ach. DA is a pleasant marker, 5-HT is an unpleasant marker. NE is a surprise marker, while Ach is a marker for anticipation. NE is well known for fight (anger) or flight (fear) behaviors. Fear and anger are twin emotions, which usually come together hand-in-hand with fear followed by anger, but they never happen at the same time in a same person. The total surprise = fear + anger. Therefore, it is usually the more scared, the less angry; and vice versa. Ach is the cognitive part of emotion, which gives the subject a quiet anticipated emotion, such as calm, tame, affectionate, missing and wanting, nostalgia, et al., which can be called “Ach related emotions”. The figure is Modified from our previous paper in Mens Sana Monographs (2015 in press).

The total amount of Stress = fear (duration, tension) + anger (duration, tension).

If fear is stronger, anger would be relatively less for certain amount of stress; for example, when people feel highly scared at strong dangerous things, such as tornado, there is no time left for anger. On the contrary, when the fear is less, anger should dominate, such as betray by a lover; or when your kids did not listen to you and result in failure of a test. So it seems that fear is facing dangerous/disliked things, while anger is for liked things; this is correct at the poles of liked or disliked things. If fear duration is similar to anger duration, they are mixed together. For example, after class begins, the teacher tells you unexpectedly there will be test, you will be very nervous: your first reaction will be scared, next you will be angry. Not only the total amount of surprise will be less, the distribution of mixed feelings will change too, and result in fear or anger dominating, if things happened the second time or many times again. The same example: if the teacher always gives you this kind of unexpected tests, your fear will dwindle, and your anger will dominate. The typical example is that when two cocks first meet, mixed feelings of fear and anger arise in both of them. Then they will start to fight, and the results will be that one wins with the other one fails and runs away. They don’t need to fight anymore the next time they meet, the failed one would run away with fear, and succeeded one would threaten with anger.

It is well documented that NE and DA are usually released together to induce aggressive behaviors (anger); and they have come to be recognized as playing an important role in attention and focus, which are the most activated state of emotion. On the contrary, the interaction between NE and 5-HT to make the fear emotions is much less documented. A considerable amount of research has focused on the finding of low 5-HT metabolite levels in abnormal aggression, but the mechanism is not clear. It is easy to explain using our equation shown above. At low levels of 5-HT, people cannot feel aggression, but the mechanism is not clear. It is easy to explain using our equation shown above. At low levels of 5-HT, people cannot feel aggression, but the mechanism is not clear.
ACETYLCOLINE (ACH) – ANTICIPATION MARKER

There are considerable evidences suggesting that monoamine (including DA, NA and 5-HT) in emotion. So far, it might be enough to propose 3 basic emotional neuromodulators in the brain to form 4 primary emotions (joy, sadness, fear and anger), like 3 basic colors. So it might be redundant to involve one additional neuromodulator ACh in emotion. Indeed, there are few reports about cholinergic involvement in emotion. In the periphery nervous system, ACh acts as a parasympathetic neurotransmitter, and it is responsible for stimulation of “rest and digest” activities that occur when the body is at rest: including sexual arousal, salivation, tears, urination, digestion and defecation, which is compensatory to the NE. In the CNS, ACh might act as “rest and thinking”, it is the neuromodulator in the brain that can boost cognitive function and its uptake inhibitors have been used for Alzheimer’s disease. So its function is well known in learning/reinforcement, or to form the association between unconditioned stimulus with neutral stimulus in Pavlovian conditioning. ACh might be the cognitive part of emotion, and the only cognitive part in Izard’s emotional theory. Even though using cognitive strategies is a good way to regulate the expectation of reward[59], cognition is not an emotion in itself. Plutchik was the first to include anticipation in primary emotion list. Here we still want to propose another batch of emotions, which were omitted by many psychologists before: “missing and wanting”, which was found when we tried to look for the opposite of anger and fear. Actually the same question was already mentioned at the start of the last section: “Everything in the world, whether it is positive or negative, occurs in ways anticipated (expected) or not anticipated (surprisingly). If it happens anticipated, people feel calm; instead if it happened surprisingly, the first reaction of people would be scared and angry.”

What is the opposite of anger? It is hard to find a proper English word for it. Darwin used the related words, “aggressive-submissive” in his famous book about emotions more than 100 years ago[60]. While probing into the angry emotion, he found that even bees get angry. He described dogs at anger as retracting upper lips, exposing teeth for biting. And in a similar fashion, dogs stand erect, hairs on its back upright to appear large, thus threatening. On the contrary, the dog slinks, with back down and close to the ground to show affectionate or submissive. Therefore Darwin invokes the principles of antithesis. He even extended it to human by showing a stance of an aggressive man as compared to a helpless man shrugging. So the opposite emotion for anger should be affectionate, and opposite emotion for fear might be “missing/wanting”. These related emotions include/ not limited to: calm, affectionate, tame, submissive, pensiveness, and nostalgia, and ACh underlies these emotions by inhibiting the excitation of cortex[61,62]. The significance of ACh related emotion lies in that they can help people relax from the stressful events. For example, nostalgia music (not sad music) can make people calm down. Furthermore, long term stressful events usually lead to back pain, because of long time back stance, while slink-like activity help relax. Although ACh related emotions are not all positive, such as apprehension and worrying which also belong to this group, the benefit of ACh related emotions is that they are not so motivated to affect the body and spirits, especially for the highly motivated people, who are very easily to burst into angry even for very small unexpected things, such as the baby not listening to him. So these emotions are the long time pursuit of many religions.

Actually, it has long been suggested for cholinergic involvement in the depression since 1972[63]. At the time when the hypothesis was first published, the primary evidence was that a number of cholinesterase inhibitors have been shown to induce depression and to exhibit anergic effects[64], presumably by increasing central acetylcholine levels[65]. For example, physostigmine, a centrally acting cholinesterase inhibitor, was shown to decrease manic symptoms and increase depressed reaction[66]. Later on, many studies provided information that largely supported the hypothesis[64,65,67].

The theory was further supported by animal studies showing that mice bred specifically for sensitivity to cholinergic agents demonstrated depression-like behaviors[68]. Other studies found that learned helplessness and swim stress, widely used preclinical models of depression, could induced sensitivity to cholinergic agents[69]. In addition, the effects of the monoamine depletory, reserpine, the mood depressing effects of which have been used to support the monoamine hypothesis of depression, are remarkably similar to those of the cholinesterase inhibitors. Overlapping symptoms include apathy, lassitude, slowed down thinking, psychomotor retardation, lack of interest, fatigue, lethargy, nightmares, and depression[70]. In addition, reserpine has been reported to have central cholinergic properties[71]. Thus the reserpine induced effect might be due to a combination of monoamine depletion and cholinergic activation, shifting NE-ACh balance to a cholinergic dominance[72].

There are a lot of reports about the interaction between ACh with catecholamine. For example, the contradiction of NE and ACh in the affective disorder in the CNS was reflected in their synaptic wire in amygdala[73]: ACh carries the influence of the amygdala to other brain structures, NE inhibits the activity of ACh[74]. Through these synaptic wires, the amygdala filters its incoming sensory streams of information, looking for those ‘dangerous’ stimulus features which would require the organism to engage in certain species-specific instincts, such as freezing or starting[75]. Similarly, the inhibition of ACh on DA is also involved in the predication error experiments. As we know, ACh is involved in the conditioned learning, after learning, light will not induce DA release, because ACh dominates and inhibits DA release. Similarly, DA release during feeding signals food reward, the increase in ACh in NAc has a role in the onset of satiation[76]. This increase in ACh is attenuated with sham feeding where food drained from a gastric fistula after ingestion. Thus the food-induced DA release goes unopposed by ACh. Hoebel and colleagues[77] suggested that the DA and ACh balance in the NAc may affect motivation, while DA enables a person to start moving, which ACh acts as a control to prevent over-responding and facilitate stopping, this interaction between DA and ACh is well documented in Parkinson’s diseases.

The interaction between ACh with 5-HT has also been reported. Selective 5-HT reuptake inhibitor fluoxetine has demonstrated the ability to alleviate behavioral depression in the forced swim test, one of the potential mechanisms is to suppress cholinergic activities in the nucleus accumbens[78]. Tobacco smoking also supports the relationship between ACh and depression. The depression rates are much higher in smokers, smokers with a history of major depression have a harder time quitting smoking, and patients with depression are at a risk for developing a major depression episode. Smoker also have lower levels of monoamine oxidase A[79]. Many patients with depression fail to derive sufficient benefits from available treatment options, and with up to a third never reaching remission despite
CONCLUSIONS

Ekman devised a list of 5 basic emotions: anger, disgust, fear, happiness, sadness and surprise[79], which are similar to our model. Robert Plutchik proposed 8 primary emotions: anger, fear, sadness, disgust, surprise, anticipation, trust and joy, and arranged them in a color wheel. He was the first to put anticipation as emotion. The earliest dimensional study of emotion came from Wundt (1897), who identified three dimensions of emotion as pleasant-unpleasant, tension-relaxation, and excitation-calm. Actually his dimension is very similar to our model, except that we put the last two dimensions which are actually overlapping into one dimension.

James Russell’s proposed two dimensions for emotions: hedonic (pleasure-displeasure) and arousal (rest-activated), which is similar to our model here[80]. Combined, our two-dimensions model (Figure1) derived from neuromodulators are very similar to the previous emotional theories. In conclusion, we in this paper propose 4 different kinds of neuromodulators that are the substrates of emotion, which can made up 5 kinds of primary emotions: pleasant, unpleasant, (fear and anger), and wanting. Even though there are many standards for primary emotions, the most important is that they should exclude each other, which means that they should not occur at the same time. Like we talked before, anger and fear never happen in at the same time in a same person, so it is with happiness and sadness. It might also be right with calm and wanting. The second rule is that they should be able to make all of the emotions, like the 7 colors in rainbows. Let us end our paper with an example of the emotional changes in everyday life (Figure 2): life is normally calm, everything is as expected; then something unexpected happens, people first feel scared (fear) and then blame (anger) the reasons for the unexpectancy after fear is gone; then people feel happy or sad depending on whether the thing happened fits for their needs or not. Finally, the thing passes away, and people calm down from the happiness, or feel missing and wanting for the lost thing. The peaceful mind will be broken again by apprehension/worrying about the uncertain upcoming events, which will induced new emotional waves. Many similar emotional waves, big or small, long or short, constitute our everyday emotions. So fear-anger-happiness-sadness-calm-wanting constitute the rainbow of emotions, they exclude each other and constitute almost all the emotions in everyday life.

Figure 2 life is normally calm, everything is as expected; then something unexpected happens, people first feel scared (fear) and then blame (anger) the reasons for the unexpectancy after fear is gone; then people feel happy or sad depending on whether the thing happened fits for their needs or not. Finally, the thing passes away, and people calm down from the happiness, or feel missing and wanting for the lost thing.

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