A NEUROCOGNITIVE MODEL OF HIGH ANXIETY TRAIT IN VICTIMS WITH POST DISASTERS EXPERIENCE

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Abstrak

Mereka yang mempunyai pengalaman berkaitan bencana amat mudah sekali menjadi mangsa yang terdedah kepada sifat kebimbangan yang tinggi. Tingkah laku ini boleh berkembang dari semasa ke semasa menjadi kebimbangan yang tulen sekiranya individu tersebut tidak mempunyai sebarang bentuk sokongan. Oleh sebab itu, pemahaman terhadap tingkah laku individu tersebut merupakan suatu cara yang penting untuk merungkai kewujudan kebimbangan itu. Beberapa tahun kebelakangan ini, focus terhadap kebimbangan ini telah menjadi fenomena. Manifestasinya telah dikaji secara meluas di peringkat bawah tentang sistem fungsi manusia (tubuh badan). Sebahagian penyelidik juga telah meneruskan kajian tersebut di peringkat yang lebih tinggi tentang fungsi kognitif. Akan tetapi, masih lagi terdapat bukti-bukti yang menunjukkan bahawa pendekatan yang tepat tidak disediakan untuk mendapatkan jawapan tentang kewujudannya dalam tingkah laku manusia. Sementara itu, maklumat-maklumat yang masih ada menunjukkan gangguan kebimbangan ini merupakan masalah psikologi yang paling lazim yang dihadapi oleh dunia sekarang ini. Tambahan pula, mereka yang mengalami gangguan ini mencatatkan angka yang sangat tinggi dalam kalangan penduduk di seluruh dunia. Oleh sebab itu, kajian ini lebih tertumpu kepada bagaimana individu yang telah mengalami bencana ini boleh memanfaatkan kebimbangan melalui pendedahan yang baik terhadap peristiwa-peristiwa yang mendatang dalam persekitaran mereka. Ini adalah langkah yang proaktif untuk menampung kewujudan gangguan kebimbangan yang lebih luas yang mungkin timbul melalui bencana yang berlaku yang mana ianya kini merupakan hal ehwal seluruh dunia. Aspek ini dicapai melalui pertimbangan terhadap Peranan mekanisma neurokognitif dalam kewujudan kebimbangan. Hasil penyiasatan menunujukkan mekanisma neurokognitif memainkan Peranan dalam kewujudan kebimbangan. Hal ini telah didemonstrasikan melalui konsep pemodelan pengkomputeran untuk mensimulasikan mekanisma yang dikenalpasti melalui dapatan kajian dan pendapat-pendapat pakar. Peningkatan dalam pengaktifan amygdala diperhatikan bagi membantu pembangunan kebimbangan sementara perkara yang sama dilakukan kepada korteks prefrontal untuk membantu menghalang kebimbangan dan sebaliknya. Tambahan pula, transformasi yang sesuai terhadap kondisi individu telah ditaksirkan menggunakan persamaan matematik untuk menunjukkan perubahan yang munasabah dari semasa ke semasa.

Kata kunci: Mekanisma neurokognitif, Sifat kebimbangan yang tinggi, Pemodelan pengkomputeran, Pengalaman pascabencana

Abstract

People with disasters experience are the most vulnerable victims of high anxiety trait. This behavior could develop overtime to pure anxiety if the individuals do not have any means of support. Hence, understanding this behaviour in the individuals is an essential means of unveiling anxiety emergence. Anxiety has been a phenomenon of focus over the years. Its manifestations have been extensively studied at the lower level of human functioning system (the body). Also, some researches have extended to the higher level of cognitive functions. Still, evidences showed that a precise approach have not been provided to elicit its emergence in human behavior. Meanwhile, extant literatures showed that anxiety disorders are the most prevalent psychological problems the world is facing today. More so, numerous numbers of people around the globe were suffering from these disorders. Therefore, this study examines how individuals with post disasters experience could develop anxiety by virtue of exposure to further events in the environment. This is a proactive measure to cater for wider emergence of anxiety disorders that might arise through disasters occurrence which is now a worldwide affair. This aspect was achieved through consideration for the role of neurocognitive mechanisms in the emergence of anxiety. The outcome of the investigation shows that, neurocognitive mechanisms play role in the emergence of anxiety. This was demonstrated through computational modeling concept to simulate those mechanisms identified through literatures and expert opinions. Increased activation of amygdala is observed to favor the development of anxiety while that of the prefrontal cortex favor the prevention of anxiety and vice versa. In addition, possible transformation of the individuals' conditions was assessed using mathematical equations to show the possible changes overtime.

Keywords: Neurocognitive mechanisms, High anxiety trait, Computational modeling, Post disasters experience.

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CHAPTER ONE
BACKGROUND OF STUDY

This chapter presents the introduction to this research by explaining the background information underlying the concepts in the study, the problem statement, research objectives as well as scope and significance of the study. It explicitly defined the focused of the study and provides brief insight into the target model.

1.1 Introduction

Anxiety is a feeling and emotion exhibited in response to a particular threat. It is characterized by set of physiological and behavioral patterns such as arousal, vigilance, and avoidance that protect individuals from the possible danger associated with that threat (Gross & Hen, 2004).

These patterns of behaviour form part of the psychological and universal mechanisms employed to excite the states of the mind towards a threat (Choi et al., 2011). Physiologically, these are normal reactions, but, if the condition associates with the cognitive functioning process, it becomes problem and if not given the necessary attention could lead to chronic condition that could affect the normal psychological state of individuals (Eysenck, 2013).

The symptoms of anxiety share similar features with fear, but, a clear distinction could be made between these and fear in term of response to a specific threat that is short lived (Rachman & Maser, 2013). In the pathological form, anxiety exist in six forms as provided in the Diagnostic and Statistical Manual of the American Psychological association (Gross & Hen, 2004). These classifications include Panic

disorder, Posttraumatic stress disorder, social phobia, generalized anxiety disorder, obsessive compulsive disorder and simple phobia. All these form most of the psychological problems the world is facing today. Also, in the non-pathological form, it is of two types: state anxiety and trait anxiety (Gross & Hen, 2004).

State anxiety represents the immediate individual disposition to perceiving environmental stimuli as threatening, thereby resulting to responses that predicate anxious behaviour/mood (Gross & Hen, 2004; Eysenck, 2013). This is common among people of varying ages and sex (Zavos et al., 2012). Individuals with state anxiety are much more vulnerable to developing trait anxiety and other forms of anxiety disorders as well (Eysenck, 2014). They possess the fundamental mechanisms needed to initiate behavioral changes towards anticipated threats.

Trait anxiety on the other hand describes the condition that influences the severity or acuteness of anxiety (Kindt & Soeter, 2014). It is a stage where the condition of anxiety may evoke maladaptive expressions such as worry associated with one's experience of calamitous events such as disaster (Eysenck, 2013, 2014). Kindt and Soeter (2014) mentioned that Trait anxiety is a recognized individual risk factor for the development of anxiety, yet the neurobiological mechanisms predicating it still remain ambiguous.

Individuals with previous experience of distressing events such as disaster, usually develop high anxiety trait over time due to the level of impact and exposure to such events (Goldmann & Galea, 2014). The unwanted behavior may accumulate over time and become collective dysfunctional behavior called anxiety (Westen, 1996;

Zettle, 2012). This constitutes greater risks to health and psychological behavior of individuals and if not given the necessary support, could lead to complex problems. Therefore, this study developed a model that could serve as basis for the design of intelligent systems that can be use to provide such support.

1.2 Problem Statement

In recent years, several studies have focused on the development of intelligent systems that can support victims with stressful events experience through the implementation of models and software systems (Rakes, Deane, Rees, & Fetter, 2014; vom Brocke, Riedl, & Léger, 2013; Zlatanova, Peters, Dilo, & Scholten, 2012). Meanwhile, those that look into the neurocognitive and the actual biological correlates for the functionality of such artifact are very few.

Hence, there is need to consider these in intelligent system design so that information technology designs or specifically human-computer interaction in general may significantly benefit from theories, concepts, methods and data related to the field of psychology, cognitive as well as neuroscience (Dimoka, Pavlou, & Davis, 2011; Velik, 2013). In particular, state of the art neurological knowledge may improve IT artifacts which may clearly enhance the functionality of such artifact towards understanding variability in human behaviours (Dimoka, et al., 2011).

High anxiety trait has been referred to as a fundamental risk factor to anxiety development (Bishop, 2007; Ding, 2007; Sandi & Richter, 2009). In particularly, it

said that people with adverse effects of disasters when received little or no supports could develop such behavioral patterns overtime leading to major anxiety that agitates their body state to other environmental events (Ding, 2007).

These may lead to multiple complications ranging from difficulty in sleeping, bowel problems, headaches, teeth grinding, depression (Comer, Pincus, & Hofmann, 2012). These could as well enhance complex behavioral/psychological patterns leading to substance abuse or the thought of committing suicide (Cougle, Keough, Riccardi, & Sachs-Ericsson, 2009).

Hence, as way to assist people in coping with the aftermath of disasters occurrence, this study developed a neurocognitive model of high anxiety trait that can serve as basis for the realization of several behavior-based systems that can help and support these individuals. Through the systems, the needed assistance could be provided to avoid future complications and possible emergence of psychological problems.

Therefore, to achieve this kind of behavior based model, this study maintain consistency between theories and abstractions related to the conditions suggested to enhance development of anxiety in these individuals. These were mathematically formalized into a form that was computationally simulated to explain the development of anxiety in the individuals.

1.3 Research Questions

In the study, in order to maintain consistency between theories and several assumptions related to the conditions suggested to enhance the development of

anxiety in the field of affective science, the following research questions were specified for the study:

1. What are the neurocognitive mechanisms associated with the development of high anxiety trait?
2. How to develop a model (neurocognitive) that formalizes these mechanisms?
3. How to evaluate the model?

1.4 Research Objectives

Similarly, to provide the necessary answers to the above questions, the following objectives were made as target for the study.

1. To identify the neurocognitive mechanisms associated with the development of high anxiety trait.
2. To develop a model (neurocognitive) that formalizes those mechanisms.
3. To evaluate the model using mathematical verification analysis.

1.5 Research Scope and Limit

The study developed a neurocognitive model specifically within these limits:

i) Identification of the neurocognitive mechanisms predicting the psychological state of victims with post disasters experience when responding to further environmental events.

ii) Development of a formal (neurocognitive) model that is based on the following criteria:

a) Abstractions from the field of Psychology, cognitive and neuroscience specifying the role of the neurocognitive mechanisms in the development of anxiety.

b) Computational simulation of the relationship between the mechanisms to provide justification for the emergence of anxiety.

c) Logical description of the relationships among the mechanisms as specified in the model

iii) Mathematical verification of the model and the problem described by the model with differential equations.

1.6 Significance of Study

The neurocognitive model developed in this study is suitable for investigating the development of high anxiety in victims with post disasters experience. The model revealed some observable features of anxiety that assists in quick assessment of the psychological impact of disasters. It exemplifies the implementation of theories and concepts from psychology, cognitive and neurological science in providing better opportunity for designing support systems that can aid victims with post disasters experience. Mathematical quantification of the domain issues provided means for investigating the trends and possible transformation of the individuals' condition that can enhance treatment or intervention.

1.6.1 Theoretical Contribution

The model developed in this study is a clear representation of the interactions of neurocognitive mechanisms involved in the development of anxiety and how the condition emerges from a hidden behaviour (high anxiety trait). It also shows the implementation of assumptions (theories) from the field of psychology, cognitive and neuroscience that suggests possible link between the mechanisms of the brain and human behaviour.

1.6.2 Practical Contribution

This model could be use in developing simulators for reading the anxiety level of victims with post disasters experience and provide the corresponding guide needed to support such individuals. It could also be used as a domain model for the design of intelligent systems for response preparation such as targeting interventions and resources allocation.

CHAPTER TWO
LITERATURE REVIEW

2.1 Introduction

Experts in the public health service have studied the possible long-term psychological consequences of a disaster. It was discovered that in addition to physical property damages, the consequence of disastrous events, whether natural or man-made, affects people's mindsets and results in different levels of psychological damages (Briere & Elliott, 2000; Kates, Colten, Laska, & Leatherman, 2006; Norris, Friedman, & Watson, 2002). For instance, Acierno and colleagues (2007) in a post-disaster analysis of the 2004 Florida hurricanes uncovers the profound psychological impacts of the disaster both on victims involved and those that lost relatives to such a catastrophic event.

Also, the survey conducted immediately after the occurrence of September 11 terrorist attacks revealed that significant sample of the entire country population experienced substantial psychological distress, both in the cities where the attacks occurred and across the country (Neria et al., 2007; Schlenger et al., 2002).

Similarly, a post war events analysis in Palestine (Gaza and Southern Lebanon) Bosnia, Iraq, Afghanistan and Syria, showed the impacts of disasters on both parents and the children (Jabbar & Zaza, 2014; Khamis, 2012; Petrie et al., 2014; Thabet, Tawahina, Sarraj, & Vostanis, 2013).

Disaster-induced negative psychological outcomes that may take time to develop (Ding, 2007).

A typical phenomenon within this context is fear and collective anxiety, which represents common responses to imminent threats and actual disaster consequences (Kelly, Iannone, & McCarty, 2014). Such collective anxiety can evolve into a major reaction to produce a wave of massive sickness that may not only specify individual anxiety after disasters but other psychopathological scenarios as well.

2.2 Disasters and its Prevalence

Disasters could both be a natural or manmade event and when it occurs, may have profound effect on the society or the individuals directly involved. Several reports of post-disaster analyses showed that disaster-induced negative outcomes and it influences the effectiveness of response efforts, such as disaster control procedures, rescue processes, and participation by health care workers and others (Jabbar & Zaza, 2014; Khamis, 2012; Neria, et al., 2007; Petrie, et al., 2014; Schlenger, et al., 2002; Thabet, et al., 2013).

In the case of sudden emergence of a disaster such as a disease outbreak, many measures usually get applied, some of which may appeared too extreme and dictated by panic and fear (Ding, 2007).

The recent outbreak of Ebola virus, for example, led to psychological distress for health workers and the general public because of the gruesome death that followed

the hemorrhagic fever and the stigmatization of groups perceived to be at high risk coupled with the general fears about safety and health implication contributed to massive anxiety (Kinsman, 2012; Tattevin, Durante-Mangoni, & Massaquoi, 2014). In this sense, a disaster event's physical consequence and associated characteristics results to mass anxiety which is a terrible reality to the potential devastation of public health and greater economic loss.

Disasters experience contributes to the emergence of some other psychopathological feature as well aside from anxiety. Typical example is the posttraumatic stress disorder and depressive symptoms. PTSD is one of the acute symptoms of the consequence of post disasters experience. It is usually caused by prolong anxiety and fear arising from the occurrence of disastrous events such as war or terrorist attack. It could even develop as a result of the excessive fear of unemployment (Arnberg, Bergh Johannesson, & Michel, 2013).

The prevalence of posttraumatic stress disorder after the occurrence of disasters has been recently estimated to range from 4-60% with majority of studies recording below 30% (Arnberg, et al., 2013; Neria, et al., 2007). Similarly, depressive symptoms have been categorized as one of those symptoms following exposure to stressful events. It also has a direct relation with post disasters experience with very high comorbidity with anxiety.

Hence, due to the consequences associated with disasters occurrences, there is significance tendency for victims with this experience to possess high anxiety trait (Ding, 2007).

2.3 Potential Impact of a Disaster

Studies examining the potential impact of disasters have focused on two dimensions. One dimension was on the economic impact and the potential loss associated with the occurrence of disasters (Li, Crawford-Brown, Syddall, & Guan, 2013; Xie, Li, Wu, & Hao, 2013, 2014). The other dimension was on examining the long-term psychological effects of disasters occurrence (Ding, 2007; Fergusson & Boden, 2014; Fergusson, Horwood, Boden, & Mulder, 2014; Shultz, Yuval Neria, Allen, & Zelde Espinel, 2013).

Researchers in the second dimension have mentioned that, a disaster can impair people's physical bodies and minds and that those who are exposed to this situation often exhibits different reactions and symptoms during and after the occurrence of the disaster. These reactions and symptoms were said to be more significant in some individuals than others depending on the nature of the events and the personal characteristics of the individuals involved (Ding, 2007).

Meanwhile, the significant implication on the individuals deeply concerned may enhance subjective discomfort or avoidance behaviour (such as anxiety) that can cause impairment in the day to day activities or work productivity.

2.4 Anxiety

Avoidance behaviour or as well known as anxiety is a negative affect that lend itself to both neural and cognitive process. It is an unpleasant state that is non-reflexive of

any biological tendencies (Horikawa, 2013; Kausar, Khan, Rasool, Yusuf, & Spielberger, 2012).

Extant review in the domain of affective science identified that one of the most frequently occurring category of mental illness in the world today is related to disorders of anxiety (Arboleda-Flórez & Stuart, 2012; D. Goldberg & Huxley, 2012; Leach, Butterworth, Olesen, & Mackinnon, 2013; Rasic, Hajek, Alda, & Uher, 2013; Wigman et al., 2012). About 18 percent of the American populations have been diagnosed of these disorders (Walker & Leach, 2014). Also, recently, a link was established between these disorders and stroke in term of factors such as depression.

Anxiety disorders cover several aspects of mental illness and pathological conditions (Rachman & Maser 2013). It enhances significant negative affect that is associated with cognitive functioning processes. In the domain of neuro- psychology, anxiety disorders have been accounted for at the level of high mental functioning, working memory, mental consciousness and new learning (Castaneda, Tuulio-Henriksson, Marttunen, Suvisaari, & Lönnqvist, 2008).

Meanwhile, recently, because of the psychological implication of this on the well being of individuals, many studies examined its cause through the emergence of anxiety (Perkins, Inchley-Mort, Pickering, Corr, & Burgess, 2012; Weymar, Löw, Öhman, & Hamm, 2011; Whalen et al., 2013).

Also, justification for the vulnerability of individuals to high anxious behaviours was attributed to life time experience of stressful events (Sandi & Richter-Levin, 2009).

The role of neural components in the elicitation of anxiety was also investigated (Davis, 2002; Liang, 2009; Lissek, 2012; Shin & Liberzon, 2010). Electrical stimulation of amygdala through conditioned and unconditioned stimulus was said to influence such a behavioral pattern that are indicative of anxiety.

In some other studies, childhood sexual abuse was reported to have severe implication in the etiology of some specific anxiety disorders such as SAD and PTSD. Similarly, physical abuse was also mentioned to be particular to posttraumatic stress disorder and social phobia. Maniglio, (2013) claimed that childhood sex abuse need be considered one of the numerous risk factors for anxiety development as it sometimes indicate additional risk factors for the manifestation of anxiety disorders either as a major or minor contributors.

In another study, Neumann and Landgraf, (2012) and some other authors such as (Binder & Nemeroff, 2010; Uher & McGuffin, 2010) considered the role of certain genes or generally genetic implication for the development of anxiety. Both oxytocin and vasopressin were said to be released from the hypothalamic and limbic region from axion, dendrite and perikarya which is independent or being coordinated by the secretion produced from the neurohypophysial terminals.

The central oxytocin was reported to always exerts anxiolytic and antidepreesive effects, whereas, vasopressin always shows anxiogenic and depressive actions. Thus, the study draw inference based on the dichotomy existing between these two genes and justifies that the balance between the activities of the brain neuropeptide systems is essential in order to maintain proper emotional behaviours.

Also, it was confirmed that evidence for this justification exist in the pharmacological and genetics association studies that identified the role of these genes in the variation of individual emotional traits that can lend itself to psychopathological problems.

Similarly, Binder and Nemeroff, (2010) identified the role of corticotrophin-releasing factor (CRF) system in enhancing physiological reactions to external stressors and in the pathophysiological emergence of anxiety and depression. The authors analyzed that variations in the activities of the gene encrypting the CRF receptor collaborate with the detrimental environmental factors to ensue risk for stress-related psychiatric disorders.

These authors also claimed that this was also evident in many other studies that identified the importance of CRF in mediating stress-related psychopathology while contributing to long term implication for detrimental conditions. Meanwhile, Uher and McGuffin, (2010) presented a review of 34 human observational studies that polymorphism of the serotonin transporter gene regulates the adverse effects of the environmental factors (such as stress) in the manifestation of depression.

The impact of environmental factors such as stress and trauma were explained by (Eiland & McEwen, 2012; Martinowich et al., 2012). In the study of Martinowich, et al., (2012), it was mentioned that exposure to stressful life situations is a risk factor that can predicate the development of anxiety disorder and that stress exposure enhance the activation of the hypothalamic-pituitary adrenal (HPA) and this cause rapid advancement in the glucocorticoid level. It is further said that improper attempt

to regulate the severity of stress response may lead to maladaptive neurobiological changes that can lend it to stress-related anxiety disorders.

Meanwhile, the authors argued that the mechanisms given an integrative account of this evidence still remain unclear but emphasized that one of the clues to studying the pathogenesis of anxiety is to investigate the behavioral and physiological responses to severe stress. Also, Eiland and McEwen, (2012) added that early life exposure to stress-related events is capable of causing long term impairment in emotion, cognition as well as stress sensitivity.

Furthermore, Laugharne, Lillee, and Janca, (2010) claimed that the impact of traumatic events on the development of Posttraumatic stress disorder has been well established but argued that their significant role in the emergence of other anxiety disorders still remain vague. Hence, the authors investigated the causative implication of traumatic events in the development of anxiety and depressive disorder. It was then observed that there are enormous evidences for the potential relevance of trauma in the development of anxiety and depressive disorders aside from the PTSD.

Kotov, et al., (2010) examined how personality trait factors (such as Neuroticism, disinhibition, extraversion, conscientiousness, agreeableness, and openness) contribute to influence the development of anxiety, specific depressive, and substance use disorders in adults. The study exploits information from the review of 175 related articles published between 1980 and 2007. The result indicated that mean deviation (*d)* for the whole diagnostic groups across the studies was high on

neuroticism but low on conscientiousness. Also, many other disorders except dysthmic disorder and social phobia with largest effects size were low on extraversion. Only few conditions were linked to disinhibition while agreeableness and openness were reported not related to the analysis diagnosed.

Thus, kotov and colleagues concluded that, strong link was justified between mental disorders and personality trait while neuroticism possessed the strongest affiliation with several disorders across the line of focus.

In sum, some studies provided evidence for the role of neural mechanisms in the emergence of anxiety. The investigations predict the activation of amygdala-prefrontal neural circuits in enhancing anxious behaviour. The electrical stimulation of the amygdala through natural stimuli such as conditioned (neutral) and unconditioned (aversive) fear stimulus enhance the transmission of impulses that radiates to influence other various body mechanisms that are themselves possess the tendency for specific symptoms of anxiety. Also, emotional response produced due to the excitation of the amygdala is an indication of worry, anxiety or abnormal apprehension.

Amygdala was implicated due to its increased response to aversive stimuli. Davis, (2002), claimed that for a neutral (conditioned) stimulus to produce a significant behavioral changes that can influence a state of fear or anxiety, such stimulus must be capable of activating the amygdala that will in turn enhance emotional responses due to its links between other component of the brain structure. Hence, this is a

preliminary evidence for the involvement of neural mechanisms in the propagation of anxiety.

Also, the hippocampus has been implicated in some studies to be involved in the extinction of aversive stimulus (McDonald & White, 2013; Orsini, Kim, Knapska, & Maren, 2011) while some express contrast opinion (Davis, 2002; Lonsdorf, Haaker, & Kalisch, 2014). Those studies that implicated the hippocampus regarded it as a non reinforcer (i.e. a detector) that provides the organisms the feeling of deviating attention from aversive signals, whereas, those that express diverse opinion were of the view that the actual role of hippocampus in extinction still remains ambiguous.

Unlike, the other neural components, the sensory cortices consists of all the projections from the visual, auditory, as well as the somato-sensory regions associated with focusing attention towards relevant stimuli and the corresponding responses from a specific task. The sensory cortices process distinctly information received through the environment alongside other components prefrontal cortex such as the lateral prefrontal and the posterior orbitofrontal cortex to enhance representation of sensory modalities in the structures associated with emotional or internal and external or environmental responses.

Hence, because the behaviours of humans are not only determined by the occurrence of stressful events in itself alone, but how the various body mechanisms assist individuals to processed the risks associated with the events. These mechanisms predicate the development of a fundamental condition that associates with

individuals' anxious behaviour and became active when they respond to further events in the environment.

2.5 High Anxiety Trait: An antecedent of Anxiety

Sandi and Richter-Levin (2009) claimed that several studies have analyzed that the vulnerability to high anxiety is indicated in the individual life time experience of stressful events. Meanwhile, adequate facts underlying the connectivity of anxiety and depression suggests that equal influence of etiological factors on high anxiety trait facilitates increase vulnerability to major depression and anxiety disorders (Hettema, 2008).

Although, the detection of risk factors (i.e. genetic, personality trait) is fundament to development of the understanding of depression but does not provide basic evidence of the actual mechanism mediating susceptibility to develop the disorder and its sequence (Sandi & Richter-Levin, 2009).

Extant literature on cognitive science have identified that highly anxious individuals demonstrate exceptional feature of cognition in processing threat related stimuli. They show biases in attention to perceived threat and interpret ambiguous events as threatening so as to enhance emotional responses to such conditions (Bishop, 2007).

Furthermore, the response of the amygdala relative to emotional information processing and the rest of the brain structures is another determinant of high anxiety trait in high anxious individuals.

Amygdala has been described in the neuro-imaging studies as a region within the brain which enhances emotional response to unfamiliar or unexpected situation. Amygdala activation is a reaction usually exhibited when individual potentially encounter a strange situation.

However, low anxious individual demonstrate minimal activation of the amygdala while responding to unfamiliar or threatening situations, this indeed signifies individual differences in trait anxiety as predicted by the amygdala responses to unintended stimuli (Sandi & Richter-Levin, 2009).

Also, some studies emphasized that high anxious often demonstrate increased activation of amygdala to non-threatening events irrespective of their attention focus whereas low anxious individual show no such reaction, this however, give a further evidence of anxiety in a high anxious individual due to the connection between the trait and amygdala activation within the brain region (Mattavelli et al., 2013; Somerville, Kim, Johnstone, Alexander, & Whalen, 2004; Whalen et al., 2013).

In genetic etiology, it was identified that high anxiety trait is inherent in individual having the short (*s*) allele of the gene enhancing greater amygdala activation than individuals with long (*l*) allele homozygous (Hariri et al., 2002). These genetic phenomenon strongly support the distinctive responses of the amygdala to emotional stimuli that might signify individuals variability in emotional reactions related to fear and anxiety(Sandi & Richter-Levin, 2009).

There are diverse opinions as regards the connection between stress-induced stimuli and anxiety trait, though; Sandi and Richter-Levin (2009) argued that evidence for its existence has been provided in the Wistar Rats experiment by (Landgraf & Wigger, 2003) to distinguish between high and low anxiety related behaviours. It was reported that Rats with high anxiety behaviour revealed stress-induced hyper-reactivity of the HPA axis, including elevated glucocorticoid levels (Stress-induced hormone).

Moreover, neuroticism has been described as a dimensional model of an individual vulnerability to experience negative emotions that manifest at a critical stage of ill-health as anxiety, depression and other related disorders (Portella, Harmer, Flint, Cowen, & Goodwin, 2005). Hence, individual with high neuroticism show notably high levels of salivary cortisol after waking up in the morning compare to those with lower cortisol. Stronger influence of amygdala response is significant in individual with greater endogenous cortisol levels than those with low cortisol levels.

The potential functioning of human memory to emotional feelings is also correlated to amygdala responses and its various interactions with other brain structures especially the hippocampal and the prefrontal cortex (Sandi & Richter-Levin, 2009). Similarly, Stress and the glucocorticoid also influence the memory particularly during emotionally arousing conditions (Barsegyan, Mackenzie, Kurose, McGaugh, & Roozendaal, 2010). Therefore, variations in several body mechanisms may play active role in intensifying the emergence of anxiety characterized with high anxious individuals.

2.6 Neural Mechanisms in the Development of High Anxiety Trait

The activations of some neural components have been mentioned to be responsible for the elicitation of anxious behaviour. Amygdala, prefrontal cortex, hippocampus, thalamus, sensory cortex and hypothalamus are such major components identified through various studies to be responsible for the development of anxiety.

Though, several other components may be attributed, and there has not being consensus on the precise numbers of neural components predicating the development of anxiety.

Electrical stimulation of amygdala through both conditioned and unconditioned stimulus was said to cause excitation of several body mechanisms which in turn transforms to responses evolving changes within individuals (McKay, Storch, & Haight, 2011). The degree of these responses and the variability among people is as a result of the interplay of several other conditions distinguishing individual traits when encountering challenges related to threat. The interaction of these factors play important role in explaining the manifestation of anxiety especially in the excitation and inhibition process that can explain the top down and bottom up control of such condition thereby, providing clue for treatment approach.

Various studies have implicated the amygdala in the excitation process while the prefrontal cortex was mentioned as being active in the suppression or inhibition process of emotional responses (Grossberg, 2014; Perry et al., 2011).

The hippocampus was mentioned to maintain long time memory, and interacts with both the prefrontal cortex and the amygdala though, with specific role of memory formation in the prefrontal cortex and the amygdala (Zeithamova, Schlichting, & Preston, 2012).

Thalamus was said to receive stimulus from the external world through the sensory mechanisms (visual, auditory e.t.c.) inform of signals, relay these signals to the sensory cortex which maintains sensory representation (see for review(Cardinali, 2011)).

The prefrontal cortex and the amygdala receive the inputs and process the signal. The increased activation of the amygdala is considered in term of low commitment of attentional control strategy (Kross & Mischel, 2010). But, if the stimulus is characterized by features not exemplified by recurrent cognitive reappraisal of negative event, the outcome of the process might not be active enough to be suppressed by the positive emotional disposition technique of individual that will enhance the inhibitory power of the prefrontal cortex (Kross & Mischel, 2010; Perry, et al., 2011).

Alternatively, recurrent cognitive process increases the activity of the amygdala resulting from negative emotional disposition, thereby, reducing the inhibitory power of the prefrontal cortex, thus making a way for the resultant process to the emergence or manifestation of abnormal behaviour that lead to significant body changes over time. Therefore, Table 2.1 below summarizes the neural components as identified in above discussion:

Table 2.1: *Neural Mechanisms*

Neural components	Descriptions
Thalamus	Receives stimulus from the environment due to its direct relationship with the sensory mechanisms and gives input to the sensory cortex as well as the amygdala and hippocampus.
Sensory cortex	Receives input stimulus from the thalamus and send it directly to both amygdala and prefrontal cortex and receives feedback only from amygdala.
Prefrontal cortex	Receives inputs from sensory cortex, amygdala and hippocampus and serving the role of inhibition or suppression of the possible consequences of events receives through the environment.
Amygdala	Receives input from thalamus, sensory cortex, hippocampus and the prefrontal cortex. It is an important component identified as being responsible for the activation of emotion to aversive stimulus.
Hippocampus	It receives inputs from both thalamus and amygdala and suppresses the activation of amygdala by enhancing that of the prefrontal cortex.
Hypothalamus	Receives input majorly from amygdala while also serve as a gate way for the recursive loop after bodily representation of the reactions manifested by the amygdala.It relates directly to body effectors manifested in term of actual mood generated through the hyper-activation of the amygdala.

Cognitive state on the other hand is the individual tendency to maintaining cognitive evaluation of events and how that contributes to a large extent in determining the resulting emotional disposition. Critchley and Nagai, (2012) suggested that one's emotional state may be considered an actual state of his/her physiological arousal and cognition relative to such a state; whether such a state embedded positive or

negative proposition, the nature or kind of emotion experienced, depends solely on how the individual interpret the arousal state. Meanwhile, evidence from another study showed that emotion is not only associated with bodily arousal and that there is varying physiological features for different kinds of emotions (Collier, 2014).

However, in order to explain this process for anxiety manifestation during post disaster experience, the chain of interactions between the neural properties, cognitive and the somatosensory mechanisms should be formally represented. By this, this study develops a formal model that serves a global representation of the neurocognitive mechanisms.

The model is undoubtedly suitable model for exploring the interactions and the consistency between the appropriate assumption for the relationship between such a human behaviour and the brain functionality.

2.7 Related Theories

In the last few decades several theories have been proposed in the domain of affective and cognitive sciences to explain how emotion-like behaviours developed. Specifically, those that focused on how particular behaviour associates with the brain functionality. These show how certain dysfunctions transform to behavioral changes. Thus, theories relating to High anxiety trait during or after the occurrence of disasters could be seen as those that explain the mechanisms or some underlying assumptions behind the development of the variable behaviour across the individuals with certain experience of these events.

According to Sylvester et al., (2012), anxiety is associate with a particular pattern of network-level dysfunction which include increase and decrease functioning of some brain mechanisms and default mode networks. Individuals with anxiety demonstrate alterations in behaviour, task-dependent activity and general functional connectivity between the brain components and this gives an end result of dysfunctions in the components of cognition.

Similarly, in another theory proposed by Basten, Stelzel and Fiebach (2011), individuals with high anxiety trait are easily distracted by threat related events and are impaired in their ability to regulate attention to threatening stimuli. This is as a result of the imbalance in the activities of some neural mechanisms, specifically, the amygdala and the prefrontal cortex which are respectively involved in the hyper-responsivity and attention control deficiency common to individuals with high anxiety trait.

The tendency for the high anxious individuals to maintain high activation of the amygdala and low activation of the prefrontal cortex during threat response after a prolong exposure to stressful experience inherited during disasters events contribute to their future anxiety being exhibited towards that threat. Thus, these evidences generally suggest how impairment in brain functionality, specifically its mechanisms aided by cognitive functioning process of certain events such as disasters could enhance high anxious behavior.

2.8 Discussion of Related Works

This section reviews models and conceptual frameworks describing the concept of anxiety development.

2.8.1 Cognitive Model of Anxiety

Appraisal is a fundamental cognitive feature of anxiety, and has been reported to be characterized by experience of thoughts about negative events (C. Hirsch & Mathews, 1997; C. R. Hirsch & Mathews, 2012). Its phenomenonical content is usually characterized by occurrences whose results are uncertain, but contain the likelihood of one or more potential negative outcomes (C. R. Hirsch & Mathews, 2012; Sibrava & Borkovec, 2006).

Cognitive approach to anxiety is an attempt to explain how high anxious trait individuals cognitively appraised and interpret cues received through the environments based on previous experience similar or non-similar events. The premise of this model is to analyze the underlying mechanisms that describe the various factors associated with individual behaviours with respect to what is being exposed to in the environmental.

Westbrook, Kennerly and Kirk (2011), analyzed a framework shown by Figure 2.1 to explain how persistence interaction between the negative thoughts, emotions, physiological and behavioral changes from environment experiences can result in dysfunctional beliefs. This reflection is explained below:

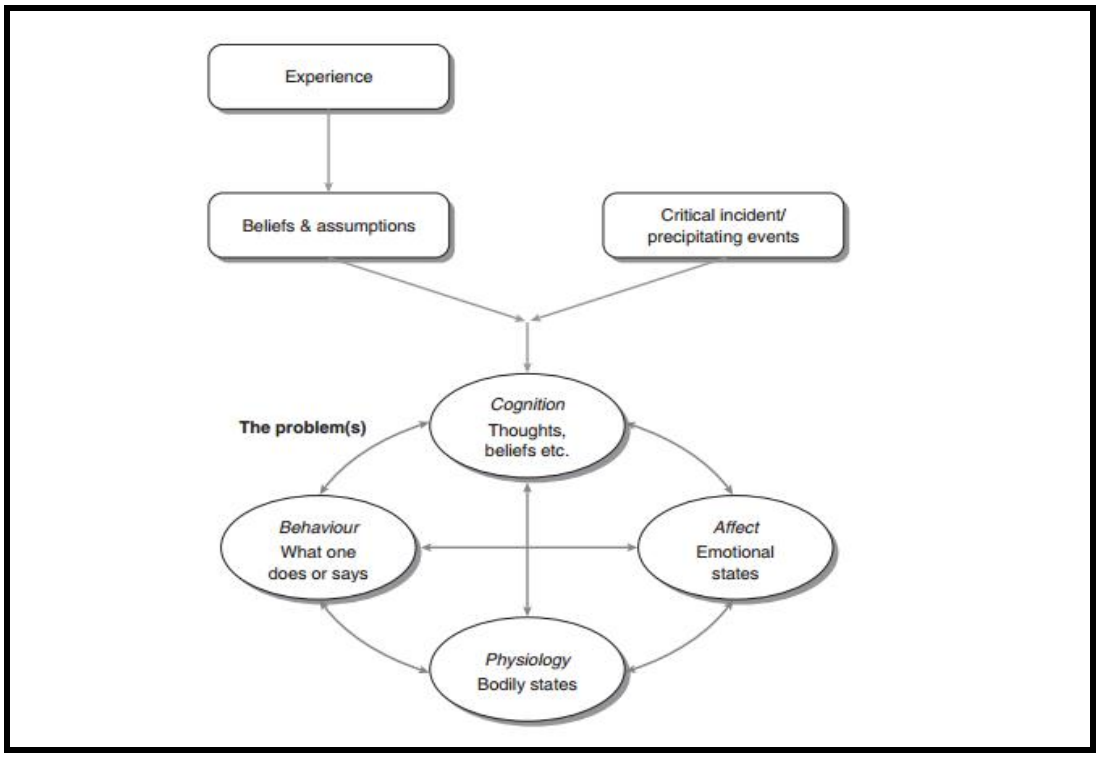

Figure 2.1. Cognitive model of event appraisal in anxiety (Westbrook et. al., 2011).

At a point in life, some people have been exposed to different calamities and events that have a residual reflection on the actual pattern they lead their subsequent life. Several studies have confirmed that individuals with certain forms of anxiety symptoms were diagnosed to have been exposed to some forms of stressful, traumatic and fearful events and these have contributed to the possible vulnerability of such individuals to various dysfunctional attributes. Thus, differences in commitment to beliefs and assumptions of this early life experiences differentiate high anxious individuals from low anxious individual.

For the high anxious individuals, they attribute much beliefs and assumptions to this early life experiences trying to appraise such events in a way that it interferes with

their cognitive thoughts and beliefs. This thoughts and beliefs enhance emotional states which in turn brought about physiological reactions; these reactions determine the perception of such individuals to their experiences. If the events are perceived and interpreted as positive, they exhibit positive emotion and that will determine the level of their commitment to such events but if otherwise, the situation will tend to result in behavioral changes that can result to possible future problems.

2.8.2 Personality Trait Model of Anxiety

Personality trait explains the underlying mechanisms predicting human behaviours when faced with circumstances of life. It determines individual differences and variability in controlling psychological processes involved in experience and action coordination (Petrides, 2011). Its basic concepts were discussed by Costa and McCrae, (2011) to explain the dynamics of individual attitude when exposed to worldly circumstances. The authors described a model termed Five-factors dimension (model) (FFM) of personality attributes to analyze the factors distinguishing individuals' characteristics.

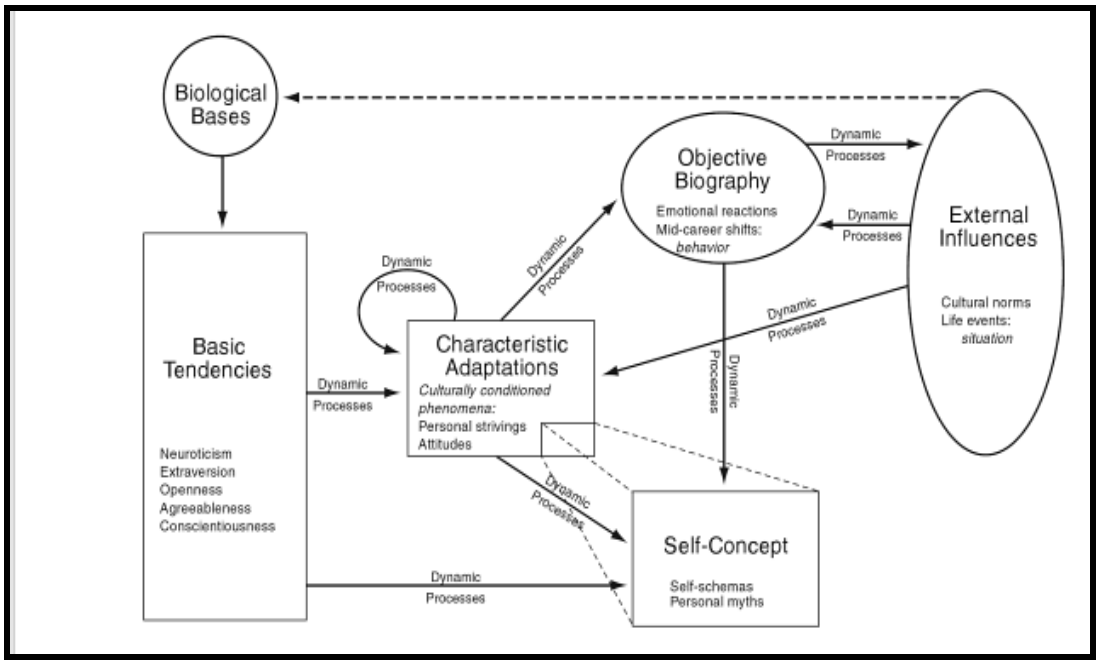

Figure 2.2. Five factor Model of Personality Trait (Costa & McCrae, 2011).

The five-factors model as shown by Figure 2.2 presented above comprises of: External influences: The external influences include cultural norms: life events/situations experienced by an individual. It is capable of influencing emotional changes and characteristics adaptations. Objective biography includes emotional reactions to situations. These reactions influence individual behaviour towards the development of self-schemas/personal myth. Consequently, Neuroticism, Extraversion, Openness, Agreeableness and Conscientiousness are the basic tendencies that can influence individual characteristic adaptations and self exhibited behaviours to various situations.

Most of these factors have been investigated in various studies, only neuroticism was seen to possess highest influential impact on anxiety and some of its several disorders. Neuroticism represents one's emotional tendencies to instability (such

as hyper anxiousness, excessive worry, pessimism e.t.c.). It is an important vulnerability factor to high anxiety trait as it determines the biological origin of anxious behavioral changes.

2.8.3 Information Processing Bias Model

Individual with high anxiety trait exhibit exceptional cognitive features characterized by changes in processing environmental stimuli that include selective attention (attention or interpretation) bias to threat related conditions (Sandi & Richter-Levin, 2009). Studies suggested that highly anxious individuals gradually respond to perceived threat cues in the environment and this enhances their emotional response (Holmes, Mogg, de Fockert, Nielsen, & Bradley, 2013; Oehlberg & Mineka, 2011; Charles Donald Spielberger, 1966; Charles D Spielberger, 1972; Weymar, et al., 2011).

Also, these individuals may interpret ambiguous events as threatening, thereby given rise to anxious behaviour due to their tendency for disastrous predictions of the consequences of events (i.e. mental inclination to imaginary events) (Hayes, Hirsch, Krebs, & Mathews, 2010).

Hence, to show how this process might lead to worry use as a coping strategy to stay on in life. Figure 2.3 presents an illustration signifying how the worry develops which may develop into a complex behavioral pattern with time (Marker & Aylward, 2012).

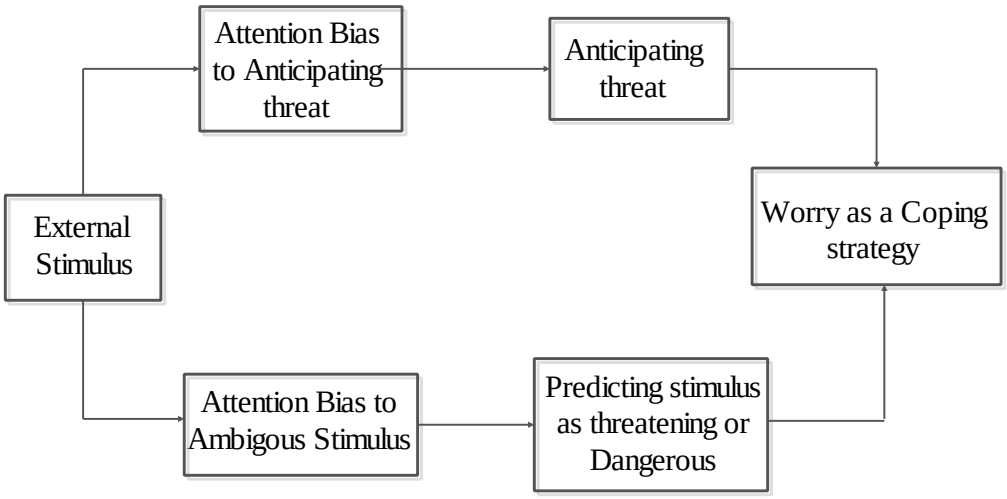

Figure 2.3. Information processing bias model.

2.8.4 Model of Intolerance of Uncertainty

Generally, intolerance of uncertainty is viewed as individual cognitive, behavioral and emotional response to perceived stimuli in ambiguous situations and the corresponding reactions to such information. It is considered as a higher-order process that has direct resultant effects on anxiety through other processes that include; cognitive avoidance, negative problem orientation and positive beliefs about worry (Davey & Wells, 2006). It is specifically seen as an excessive vulnerability of an individual to regard the occurrence of certain negative events as unacceptable no matter the degree of its certainty (Sexton, 2011).

Previous studies claimed that intolerance of uncertainty has a higher correlate with worry in both clinical and non-clinical populations (Dugas, Gosselin, & Ladouceur, 2001; Yook, Kim, Suh, & Lee, 2010). It was reported that the relationship existing between worry and intolerance of uncertainty is not justified by the shared variation

of negative affect but the intolerable habit of high anxious individuals towards ambiguous events distinguish them from the low anxious ones.

Also, it has been claimed that individuals with high anxiety trait, find it more difficult to tolerate and bear uncertainty compared with individuals with low traits, because of the belief that any uncertain events may be catastrophic in their outcomes (Koutstaal, 2011; Nicholas Carleton, Sharpe, & Asmundson, 2007). Consequently, (Berman, Wheaton, Fabricant, Jacobson, & Abramowitz, 2011; Dugas, Freeston, & Ladouceur, 1997) argued that the cognitive assessment of the ambiguity of task (i.e. without adequate guide to the nature of perception) set distinction between worrying individuals and non- worrying individuals, rather than considering the level of difficulties involved in tolerating uncertainty.

Some worry prone individuals are so attentive to what goes on within their environment and extremely seek for disengagement between the anticipated threats, whereas, others without worry do not possess such kind of notion.

Therefore, the understanding of the worry reaction to perceived threat is characterized by the feeble representation of the inherent danger associated with such an event and this determine the behaviour of individual with post disasters experience.

2.8.5 Neurocognitive Model of High Anxiety Trait to Depression

According to this model shown in Figure 2.4 below, high anxious individuals usually demonstrate attention bias and increased activation of amygdala while responding to

aversive stimuli. The hyper-responsiveness is characterized by both amygdala activation and an impaired attention control that makes a high anxious individual a subjective case to experiencing fear and anxiety while confronting threat related events. The overall consequence of these activations promotes anxious behaviour.

Enhanced activation of the amygdala and other mental mechanisms contribute to enhance negative emotional episodic memory and this determines the pattern of behaviour such a high anxious individual will attached to a similar event in the future.

As a result of their confrontation with major life disrupting events, highly anxious individuals tend to develop such a vulnerable neurocognitive phenotype and these serve as sensitized mechanisms that exaggerate the negative consequence of an event. These mechanisms in turn stores the event informs of a strong aversive memory and when such individuals encounter similar or related events, they demonstrate rapidly enhanced emotion that influences congruent behaviour.

In contrast, low anxious subjects show reduced amygdala activation to an impending stimulus with basic content of fear and this is due to the strong personality influences and good cognitive ability to appraise such events as non-threatening.

Therefore, the interaction/activation of some neurocognitive mechanisms such as amygdala, prefrontal cortex, and hippocampus coupled with cognitive ability and individual personality trait developed through prior exposure to stressful events were

identified as an important measure of the behaviour of people with high anxiety trait as these contribute to long term episodic memory.

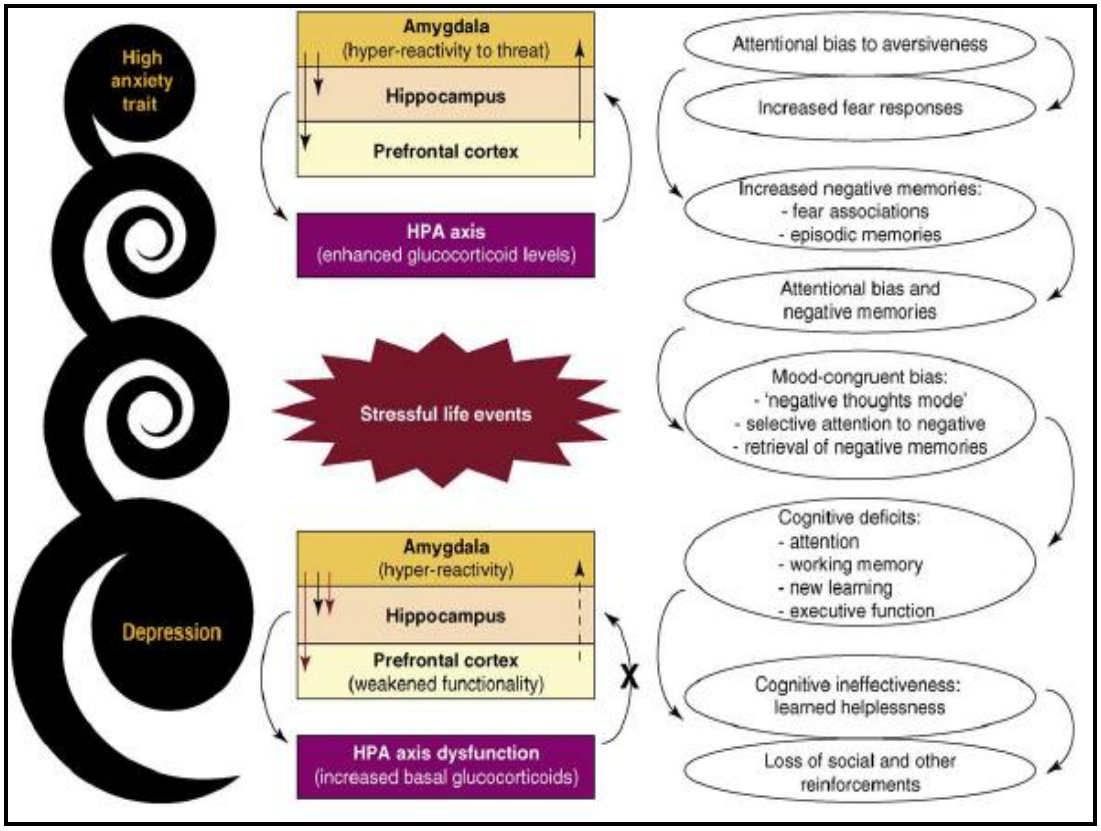

Figure 2.4. Neurocognitive model of high anxiety to depression (Sandi & Richter, 2009).

2.8.6 Model of Psychological Impact of Disaster

Ding (2007) analyzed that when a disaster occurs, the situation generates impulse in form of an impending stimulus (signals) and this will be felt by human being and other external agents. Hence, the consequence of this will bring about mental reaction by human which often affects other body mechanisms. The amygdala, precisely, its lateral nucleus acquires the inputs.

The sensory systems, as well as the conscious regions of the prefrontal cortex and the hippocampus, also receives this and set off impulses effecting the rapid release of several hormones (such as oxytocin, vasopressin e.t.c.) and increased activity of the corticotrophin-releasing factor (CRF) in the limbic system which in turn stimulate the sympathetic nervous system (Allen, Stoney, Owens, & Matthews, 1993; Cannon, 1932; Ding, 2007). The adrenal glands in response, triggers the release of adrenaline, and cortisol through a different route, both enhancing the body for either acceptance or withdrawal from the threat related stimuli.

Also, the hippocampus creates an integrated memory of the received impulse with the distinctive features of the past experiences to assist the individual in making logical decisions. Hence, these individuals demonstrates responses (in term of feeling of shocks and helplessness), cognitive (such as attention bias), physical (such as exhaustion) and interpersonal (such as social withdrawal). The response is in turn send back to the brain majorly the hypothalamic region for mental representation which generates mental pressure or psychological reactance that enhances anxious behaviour.

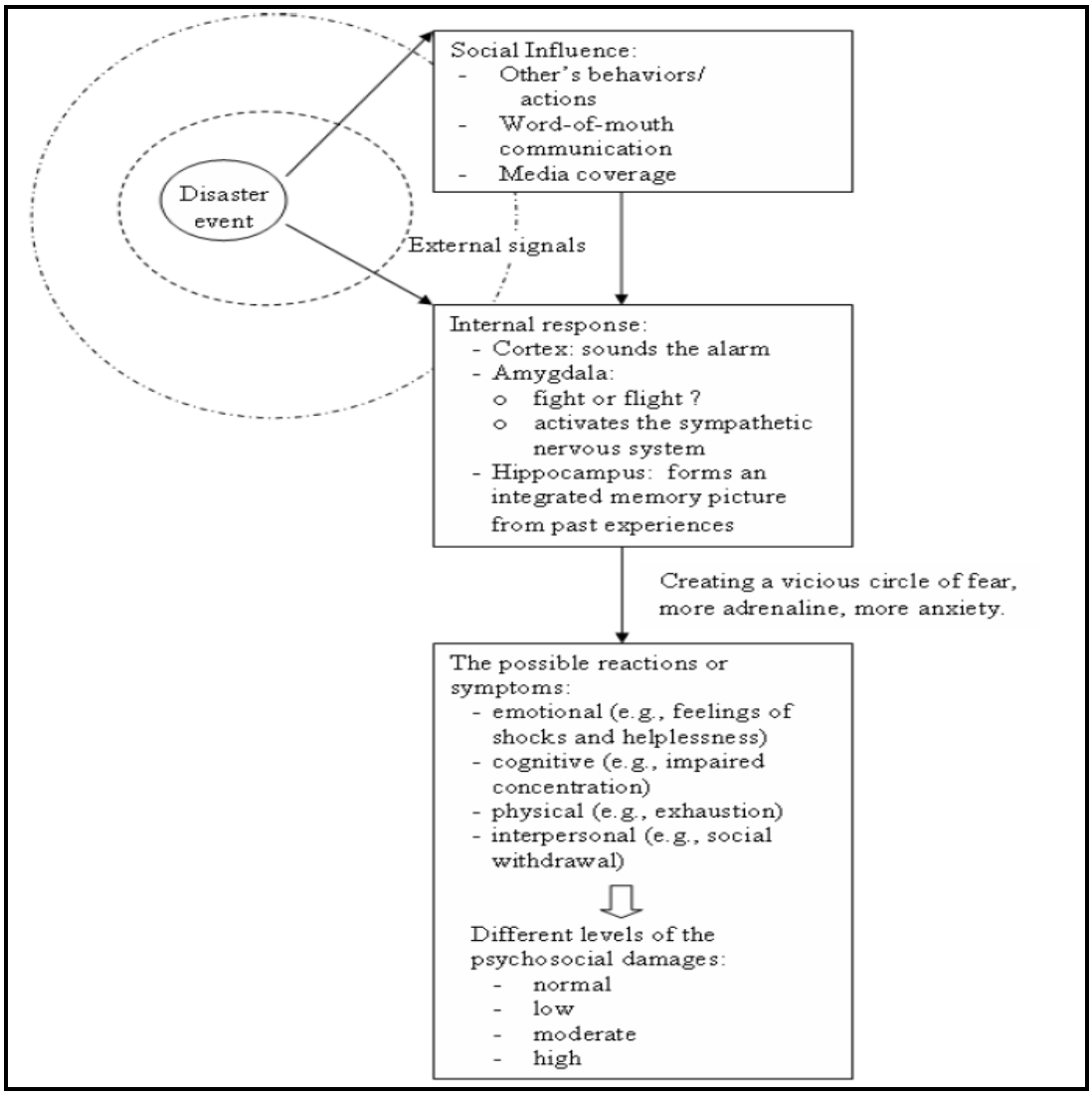

Figure 2.5. Model of psychological impact of disaster (Ding, 2007)

Hence, this process is represented in Figure 2.5 above. However, the combined effects of long time experience of this kind of this event is a predicate for several categories of anxious behaviors (Ding, 2007).

2.9 Discussion of Related Works

Neuro-cognitive models are models showing interpretative mapping of neurocognitive mechanisms with cognitive properties to predict certain human behaviour. The models are usually designed based on abstractions related to theories and expert opinions in the field of psychology, cognitive and neuroscience. Such abstractions allows modeler to exploit neurological knowledge to establish interpretative mapping between neural components and the cognitive state (Bosse, Memon, & Treur, 2012). The result of the mapping gives a concept describing the certain phenomenon at the neural as well as cognitive level to describe a certain phenomenon relating to human behaviour variability.

The final model developed through this approach, represents formally defined objects that are assumed to describe the same process in reality. Figure 2.6 below shows how these are formally related to each other.

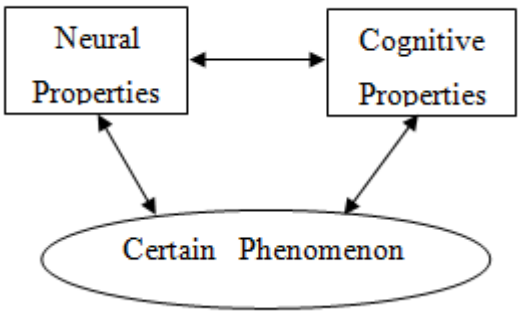

Figure 2.6. Representation of neurocognitive description

2.10 Computational Modeling Technique

Inspired by the principle of artificial neural networks, neuro-cognitive model offers a range of application for designing systems capable of understanding and relating with human been by offering support through the provision of series of instructions and guide encode from the individual condition at a certain point in time.

In a different focus, due to its realistic properties, neuro-cognitive model also serve as an ideal approach in representing and analyzing the biological correlates of the neural circuits (Stewart & Eliasmith, 2011), connections among the neural properties as well as possible link of the brain to the somatosensory organs that determine the corresponding response of individuals to environmental events.

The central strategy of such neurally inspired model is the use of the artificial neural networks principle which exemplifies the real neural system exhibiting a certain properties relating to information processing. Several studies have also implemented this principle to simulate particular interesting area of cognitive behaviours as well as impairment related to human problems. For example, Bin Ab Aziz (2011) proposed a model of cognitive analysis of human depression. Also, recently, are models describing the interpretative mapping of the neural and cognitive properties in determining the state of emotion to certain environmental stimulus (Bosse, et al., 2012).

However, in designing a model describing certain pattern behaviours to represent high anxiety trait post events relating human experience of disaster, there must be a consistency between the neurocognitive process underlying the phenomenon and

the corresponding behaviour specifying the individual state. This will formalize the assumption behind the design of the model.

Table 2.2 below show some of the studies that have implemented the proposed concept in this study and describe their area of contribution in modeling certain human behaviour and the limitations.

Table 2.2: *Related studies on human behaviour computational modelling*

Models/Frameworks	**Descriptions**	**Limitations**
Cognitive and neural model of dynamics of trust in competitive trustees(hoogendoorn et al., (2012)	The model implemented abstractions of neural and cognitive properties to describe the dynamics of trust among individuals.	This model does not identify the actual mechanisms involved in both cognitive and the neural processes.
A generic adaptive agent architecture integrating cognitive and affective states and their interaction (Memon & Treur, 2010).	The model implemented abstractions of neural and cognitive properties to describe how cognitive and affective state could be integrated. Concept from the theory of mind (ToM) was used.	The limitation of the model is that it does not identify any real attribute of both neural and cognitive properties. but this concept is also useful in our model to established coordinate interaction of the respective properties.

A cognitive and neural model for adaptive emotion reading by mirroring preparation states and hebbian learning (Bosse, Memon & Treur, 2010).	The model implemented abstractions of neural and cognitive properties to explain an individual emotional state and how such could be used to understand other people's emotion. Concept from the theory of mind (ToM) and theory theory was used . The limitation of the model is that it does not identify any real attribute of both neural and cognitive properties, but its concept is as well useful in our study.

2.11 Summary

This chapter presents review of literature related to the proposed study. It gave vivid account of how high anxiety trait could predicate in the behaviour of individuals with post disasters experience and how this contribute to major anxiety exhibited in response to other circumstances in life with an inherent feature of fear. Evidences from the various fields of consideration provide combined facts that can be implemented in the design of the proposed model.

CHAPTER THREE
RESEARCH METHODOLOGY

3.1 Introduction

The selection of appropriate design and methodology is one of the fundamental issues bothering the setting up of this research in order to develop the neurocognitive model and how this could represent a global entity specifying the possible trajectories between the neural and cognitive properties and predict possible indications underlying the development of high anxiety trait.

Hence, when considering such a suitable approach to be adopted, adequate selection of procedures and techniques were put into consideration in order to come out with a particular methodology that can guide towards achieving the aim and objectives of this study. The methodological approaches adopted are presented in five (5) phases of design plan to explore the state of the art neurological knowledge in the study as proposed by the title and this is presented in Figure 3.1 and explained below:

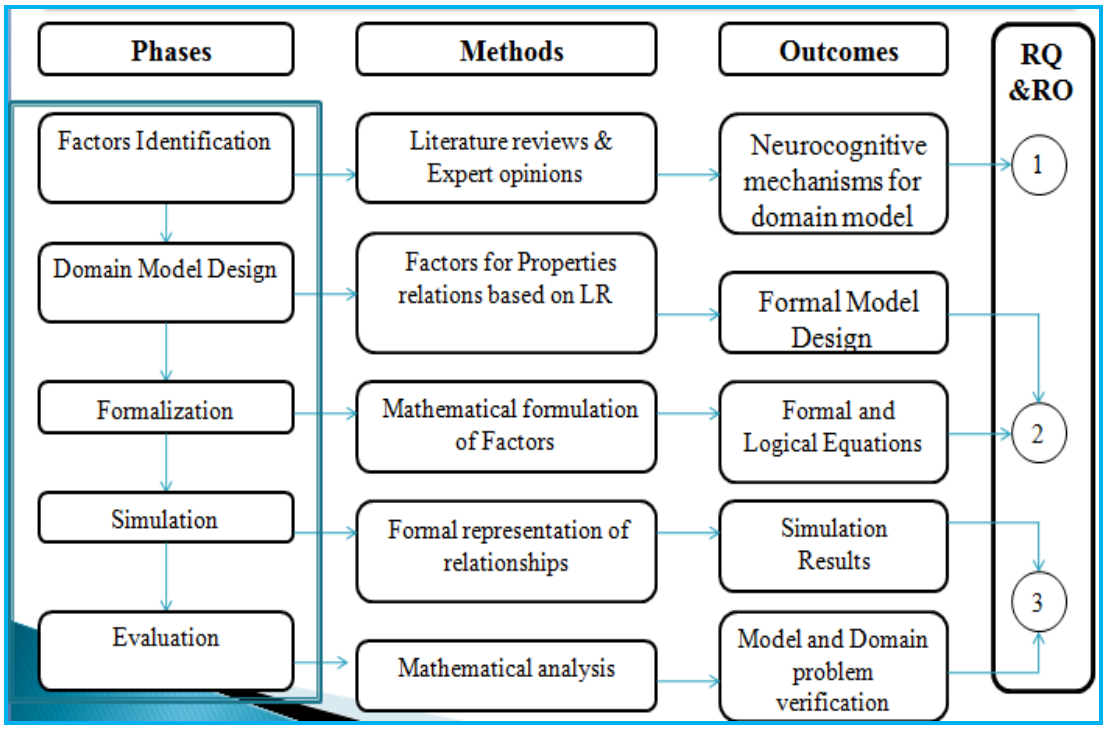

Figure 3.1. Methodology phase

3.2 Factors Identification

Neurocognitive factors were identified through literatures and opinions of expert in the field of Cognitive and Neuroscience specifying direct (known) or indirect (unknown) conditions predicting the development of anxiety (this is shown in Table 3.1). The identification process gave more insight to the fundamental mechanisms underlying the development of high anxiety trait. The result of this stage represents an important outcome leading to the several assumptions behind the setting up of the study. The factors represent nodes and each of the nodes with its own corresponding local properties as well as the degree of activation/ contribution towards the processing of sensed stimulus received through the environment.

Table 3.1: *Factors of neurocognitive process in the development of anxiety*

Factors of neurocognitive process in the development of anxiety	**Descriptions**
Environmental Event	This include disastrous or catastrophic event that occurs within an area.
Sensed Stimulus	This is the stimulus generated by the environmental event.
Sensor_ State	This is the actual state of the various sensory organs after receiving the stimulus.
Thalamus	This is a component of the brain that receives the signal produced from the environment.
Sensory Cortex	It receives input signals from the thalamus and sends it directly to both amygdala and prefrontal cortex.
Prefrontal Cortex	This receives inputs from sensory cortex, amygdala and hippocampus.
Amygdala	It receives input from thalamus, sensory cortex, hippocampus and the prefrontal cortex.
Hippocampus	It receives inputs from both thalamus and amygdala. It suppresses the activation of amygdala and enhances that of the prefrontal cortex.
Hypothalamus	It receives input majorly from amygdala while also serve as a gate way for the recursive loop after bodily representation of the reactions manifested by the amygdala.

Emotional Response	This is the preliminary behavior exhibited by the individual as a result of the activation of the amygdala.
Cognitive State	The cognitive state is the ability of the individual to provide adequate mental assessment of the stimulus as harm, threat (negative interpretation) or benign (positive interpretation).
Anxious Mood	This is the output from the neural process that enhances the release of several hormones influencing the development of anxiety at the body stage.
Body_State	The body state is the interface between the signal from the brain and the input of such to the human body.
Sensor_State	This is another sensory state of the signal but this time by the internal mechanisms such as the kidney which controls the release of the adrenalin.
Sensory rep. of Body State	It represents the actual state of the human body after sensing the stimulus/signal.
Feeling	This is the actual result of the representation.
Anxiety	This is the final response of the individual to the environmental stimulus that may lead to maladaptive behavior.

3.3 Domain Model Design

Figure 3.2 is the domain model designed to establish coordinate relationships among the corresponding factors identified above through the literatures, theories and expert opinions to explain the development of anxiety in victims with post disasters experience. The design yields an important outcome that represents the global dynamic properties and relationships of the identified factors. It also unveils the strength of impact of each factor on one another which clearly specifies the contribution of each on the output condition under investigation.

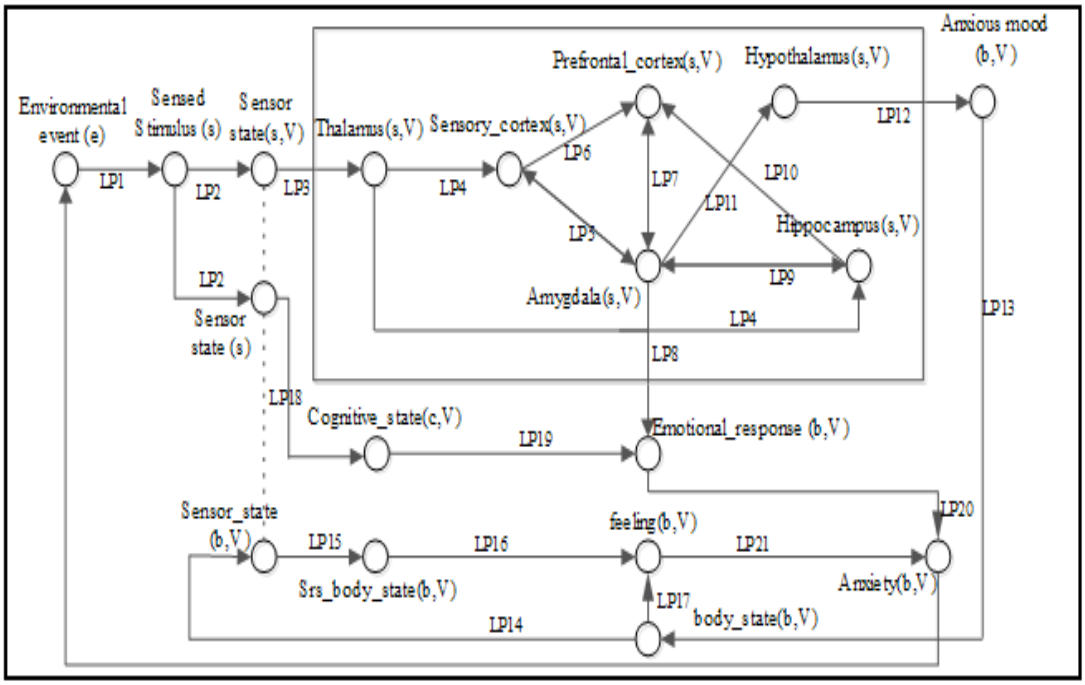

Figure 3.2. Neurocognitive model

The Figure 3.2 is an overview of the neurocognitive model that shows graphical representation of the neuro-cognitive properties. It shows how the various neural properties interacts to influence an emotional property known as Anxiety and how this important property relate with individual cognitive ability and capability to

enhance the body state which often resulted in feeling representing individual psychological state towards a perceived stimulus.

The circles indicated in the figure denote the states properties of each element in the model while the arrows show the corresponding coordinate relationships. The capital letters as specified in the model were used for assumed universally quantified variables while the lower case letters represent instances of certain actions. In the figure, it is assumed that *b* and *c* are corresponding body and cognitive state instances induced by stimulus instance *s*.

The first three properties LP1, LP2 and LP3 are specifically the input from the environment. LP1 describes the sensing property of the stimulus from the environment to the body sense organs. LP2 is the property of the stimulus when it moves from the sensor_state to the thalamus. Thalamus, then send it inform of signal to the sensory cortex with a state property LP3 describing the sensing process to the neural transformation for all instances of the sensed stimulus *s*. Meanwhile, it should be noted that the actual numerical relations between the indicated activation variable level *V* and the corresponding state properties were not provided; details will be given during the analysis of the local properties labeled from LP1 to LP21 in Chapter Four.

3.4 Formalization

Figures 3.3 - 3.17 below are the formalization phases that include transformation of the identified factors into local and non-local dynamics properties. The local dynamic properties are those factors formally represented through the literatures and

expert opinions. The non-local dynamic properties on the other hand, describe the instance factors generated when one or more of the local dynamics properties combined in the process described by the model. The output of this phase yields a logical / mathematical (differential equation) specification of the various executable properties. Meanwhile, the formal model is used to show the dynamic relationship between the various properties needed for simulation to establish the periodic variance of the conditions underlying the process.

The formalization phase is important in the sense that, it unveils the hidden problems that were not apparent during the textual description of the domain model. It as well removes from the process, any traces of ambiguity to provide vivid understanding of how the final model works. Thus, the important aspect of this process is to present facts in formal language settings that could determine the implementation of the final model either as an embedded entity in artificial intelligent systems or as knowledge for automated reasoning process. Table 3.2 shows a formal specification of the factors that can enhance the generation of appropriate equations.

Table 3.2: *Formalization of neurocognitive mechanisms*

No.	Neurocognitive Mechanisms	Formal Specification	Description
1.	Environmental Event	*Ev*	This is the actual event generating stimulus.
2.	Sensed Stimulus	*Ss*	Stimulus produced from the environment

			to the external body organs.
3.	Sensor_State Environment – Neural	*Sn*	Input to the neural process from the environment.
4.	Sensor_State Environment - Cognitive	*Sg*	Input to the cognitive process.
5.	Thalamus	*Hm*	Neural components that receives input from the sensory nerves cells
6.	Sensory Cortex	*Sc*	Neural component that transfer the signal for processing.
7.	Amygdala	*Ag*	Neural component that enhance emotional reaction and also serve as a developmental stage for anxiety.
8.	Prefrontal Cortex	*Pc*	Neural component that support treatment of anxiety.
9.	Hypothalamus	*Hp*	Neural component that receives input from the amygdala.

10.	Hippocampus	*Hc*	Neural component that influence the activities of both amygdala and the prefrontal cortex.
11.	Emotional_Response	*Er*	Part of the output from the amygdala activation.
12.	Cognitive_State	*Cs*	Mental state for signal evaluation.
13.	Anxious Mood	*Am*	An effector of the activity of the neural process.
14.	Body_State	*Bs*	The state of the body when it receives signal from the brain.
15.	Sensor State Body	*Sb*	The internal body sensor for the signal received from the brain.
16.	Sensory Representation	*Sr*	Representation of the signal by the sensor.
17.	Feeling	*Fn*	Feeling generated by the signal after been represented by some body mechanisms.

18.	Anxiety	*Ax*	The output of the global process of the stimulus representing emotion that may lead to maladaptive behavior.

The generation of the executable dynamics properties related to formalization of the corresponding relationship in the model is discussed as follows:

3.4.1 Sensed stimulus

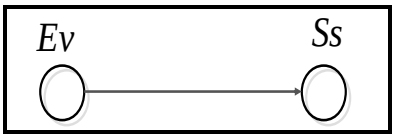

Figure 3.3. Sensed stimulus

From Figure 3.3, it is shown that, at any occurrence of event in the environment such as disaster, the situation generates stimulus which is sensed by the various body organs at a particular point in time. This relationship is represented in equation (1) and the underlying condition is presented in Table 3.3:

$Ss\ (t) = \phi * Ev\ (t)$..... *(1)*

ϕ is constant used to regulate the relationship during programming.

Table 3.3: *Variable conditions for stimulus representations in Ss*

Conditions	Local Dynamic Property	Non-Local Dynamic Property	Descriptions
Condition1	*Ev* High	*Ss* High	*Ss* is high if the impact from the environment is high and vice versa
Condition2	*Ev* Low	*Ss* Low	

3.4.2 Sensor state

Figure 3.4. Sensor state

Figure 3.4 shows that the state of the sensor *Sn* is realized by the input stimulus *Ss* receives through the environment. The relationship is linear and can then be formalized as follows:

$Sn(t) = \gamma * Ss(t)$ ..... *(2).*

γ is a constant that is used to regulate the relationship while *t* is the period of transmission of the signal from the source end to the receiving end.

Assuming the transmission rate is high due to the characteristics possessed by the individual (i.e. High anxiety trait). The corresponding value of V for the signal will be sufficient enough to enhance the development of anxiety, otherwise, the experience does not have a devastating effect on the individual or such possess a good coping strategy to deal with future situation. Hence, in this study, we are considering a situation whereby the transmission rate of the signal is high due to the

characteristics possess by the victims with disastrous experience of disaster. Table 3.4 presents the underlying conditions:

Table 3.4: *Variable conditions for stimulus representations in Sn*

Conditions	**Local Dynamic Property**	**Non-Local Dynamic Property**	**Descriptions**
Condition1	*Ss* High	*Sn* High	*Sn* is high if the input stimulus is high and vice versa
Condition2	*Ss* Low	*Sn* Low	

3.4.3 Thalamus

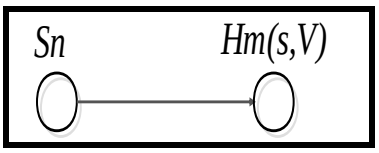

Figure 3.5. Thalamus

Figure 3.5 shows that thalamus receives input signals from the sensory cells at a certain activation level *V*. Below is the formalization:

$Hm\ (t) = \lambda * Sn\ (t)$..... *(3).*

Signals with higher activation level *V* is received due to the interaction of both Hm and Sn and transmitted to both amygdala and the sensory cortex. λ is a constant used to regulate the relationship while the *t* specifies the instances for the reactions. Table 3.5 shows the underlying condition in the relationship.

Table 3.5: *Variable conditions for stimulus representations in Hm*

Conditions	**Local Dynamic Property**	**Non-Local Dynamic Property**	**Descriptions**
Condition 1	*Sn* High	*Hm* High	Signal representation in *Hm* is high if such is high in *Sn* and vice versa.
Condition 2	*Sn* Low	*Hm* Low	

3.4.4 Sensor cortex

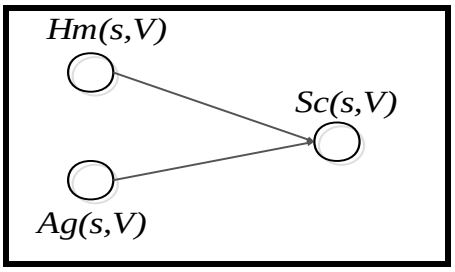

Figure 3.6. Sensory cortex

Figure 3.6 shows that sensory cortex takes its own input from the thalamus at a certain activation level *V*, and it is in turn transmitted to both amygdala and the prefrontal cortex. It also received a partial feedback from the amygdala and this increases the level of signal transmitted to the prefrontal cortex to enhance the top down control of the activation of the amygdala. This relationship is represented below and the underlying condition is presented in Table 3.6:

$Sc\ (t) = \alpha\ *Hm\ (t) + (1-\alpha)*Ag\ (t)$..... *(4)*

α is a constant and *t* the periodic variance.

Table 3.6: *Variable conditions for stimulus representations in Sc*

Conditions	Local Dynamic Property	Non-Local Dynamic Property	Descriptions
Condition 1	*Hm* High *Ag* High	*Sc* High	Signal representations will be high in *sc*, only if it high in both *hm* and *ag* and or hm is high, otherwise it will be low
Condition 2	*Hm* High *Ag* Low	*Sc* High	
Condition 3	*Hm* Low *Ag* High	*Sc* Low	
Condition 4	*Hm* Low *Ag* Low	*Sc* Low	

3.4.5 Prefrontal cortex

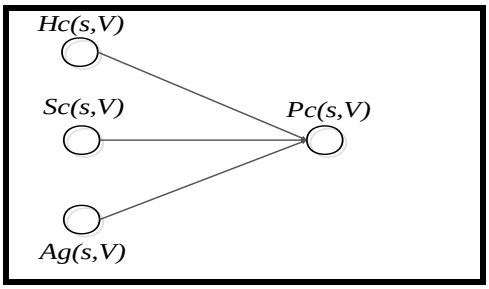

Figure 3.7. Prefrontal cortex

Figure 3.7 shows that prefrontal cortex receives input from Amygdala, sensory cortex and the hippocampus at a certain activation level (between 0 and 1) which is influenced by the corresponding input from both the sensory cortex and the amygdala based on the ability of individual processing the stimulus. If such an individual posses a good cognitive ability to regard the situation generating the stimulus as non-threatening and harmful, the individual will in turn control the level

of attention given to such situation and this empower the activation of the prefrontal cortex to suppress the activation of the amygdala, therefore, controlling the development of anxiety. The relationship is formalized as follows and the underlying conditions are presented in Table 3.7:

$$Pc(t+\Delta t) = Pc(t) + \mu_p * ((g(t) - Pc(t)) * (1-Pc(t)) * Pc(t)) * \Delta t \quad \text{.....(5a)}$$

$$g(t) = \mu_p * (Ag(t) - Hm(t)) \quad \text{.....(5b)}$$

μ_p is a constant for regulating the relationship and the period of activation. An output for this section will be shown on signal graph in the simulation section.

Table 3.7: *Variable conditions for stimulus representations in Pc*

Conditions	**Local Dynamic Property**	**Non-Local Dynamic Property**	**Descriptions**
Conditions 1	*Sc* High *Ag* High *Hc* High	*Pc* High	Activation of *Pc* will be high only if representations of signal is high in *Sc*, processing is low in *Ag* but high in *Hc* or *Sc* high and *Hc* high *Ag* high and also when it is high in *sc* but low in both *Ag* and *Hc* being the primary source from the environment otherwise, it is low
Conditions 2	*Sc* High *Ag* Low *Hc* High	*Pc* High	
Conditions 3	*Sc* High *Ag* Low *Hc* Low	*Pc* High (Inhibition Level)	
Conditions 4	*Sc* Low *Ag* High *Hc* High	*Pc* Low	

Conditions 5	*Sc* Low *Ag* High *Hc* Low	*Pc* Low
Conditions 5	*Sc* High *Ag* Low *Hc* Low	*Pc* High

3.4.6 Amygdala

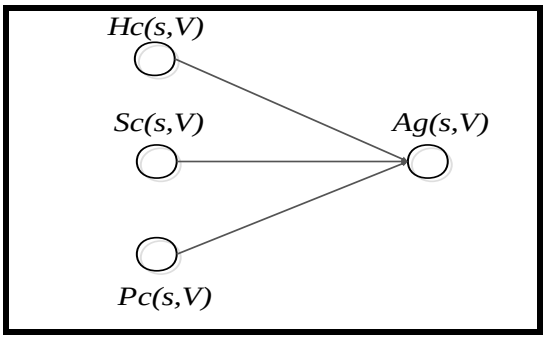

Figure 3.8. Amygdala

Figure 3.8 above, indicates that amygdala receives its own input from the sensory cortex, prefrontal cortex as well as the hippocampus. Meanwhile, the most significant input comes from the sensory cortex as well as the prefrontal cortex that may also receive inputs through other components of the brain.

The activation of the amygdala is influenced by the poor cognitive ability of such individual (victims with post disasters experience) to interpret the present environmental situation as threatening and harmful, thereby committing much attention to such situation. This, however, enhances the development of emotional reactions and anxious mood that later developed to the complex behavioral pattern

known as anxiety. Equations 6a & 6b represent the mathematical formalization of the relationships:

$$Ag(t+\Delta t) = Ag(t) + \gamma * (q(t) - Ag(t)) * Ag(t) * (1 - Ag(t)) * \Delta t \quad \text{(6a)}$$

$$q(t) = w1 * Sc(t) + w2 * (1 - Pc(t)) + w3 * Hc(t) \quad \text{(6b)}$$

The relationship shows that the activation *Hc* may not have much significant impact on the activation of amygdala unlike the other components. The result of this section will be shown as well in the simulation section on a signal graph to demonstrate the level of activation with varying inputs that shall be assigned between 0 and 1. Meanwhile, Table 3.8 presents the underlying conditions.

Table 3.8: *Variable conditions for stimulus representations in Ag*

Conditions	**Local Dynamic Property**	**Non-Local Dynamic Property**	**Descriptions**
Conditions 1	*Sc* High *Pc* High *Hc* High	*Ag* Low (Suppression Stage)	Activation of *Ag* is high only if representations is high in *Sc*,
Conditions 2	*Sc* Low *Pc* High *Hc* High	*Ag* Low	processing low in *Pc* and *Hc* or high in *Sc*, *Hc* but low in *pc* and also when it is high
Conditions 3	*Sc* High *Pc* Low *Hc* Low	*Ag* High (Activation Level)	in *Sc* but low in both *Pc* and *Hc* being the primary source from the environment
Conditions 4	*Sc* Low *Pc* Low *Hc* High	*Ag* High (Extreme Condition	otherwise, it is low

Conditions 5	*Sc* High *Pc* High *Hc* Low	*Ag* Low(Suppression Stage)
Conditions 5	*Sc* Low *Pc* Low *Hc* Low	*Ag* High (High Trait Stage)

3.4.7 Hippocampus

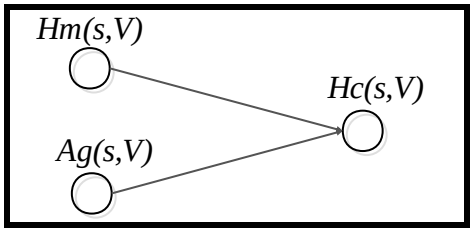

Figure 3.9. Hippocampus

Figure 3.9 above shows signal inputs into hippocampus are provided through the thalamus and the amygdala at a certain activation level. It in turn sends part of this to the amygdala to influence emotional reaction. The result of this relation is shown in equation (7) below and the underlying conditions presented in Table 3.9:

$$Hc(t) = \omega * Hm(t) + (1-\omega) * Ag(t) \quad (7)$$

Table 3.9: *Variable conditions for stimulus representations in Hc*

Conditions	**Local Dynamic Property**	**Non-Local Dynamic Property**	**Descriptions**
Condition 1	*Hm* High *Ag* High	*Hc* High	Signal Processing will be High in *Hc*, only if it High in
Condition 2	*Hm* High	*Hc* High	both *Hm* and *Ag*

	Ag Low		and or Hm is High, otherwise it will be
Condition 3	*Hm* Low *Ag* High	*Hc* Low	Low.
Condition 4	*Hm* Low *Ag* Low	*Hc* Low	

3.4.8 Hypothalamus

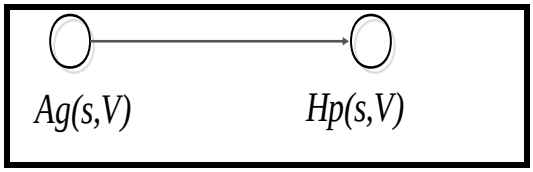

Figure 3.10. Hypothalamus

Figure 3.10 shows the input stage to the hypothalamus. This relationship is represented in equation (8) and the underlying condition is presented in Table 3.10:

$Hp\ (t) = \beta * Ag\ (t)$..... *(8).*

β is a constant used to regulate the relationship while *t* is the periodic variance.

Table 3.10: *Variable conditions for stimulus representations in Hp*

Conditions	Local Dynamic Property	Non-Local Dynamic Property	Descriptions
Condition 1	*Ag* High	*Hp* High	Signal representations will be High In *Hp*, only if it is High in *Ag* ,otherwise It will be Low
Condition 2	*Ag* Low	*Hp* Low	

3.4.9 Emotional response

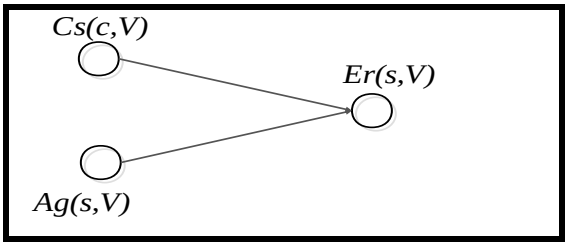

Figure 3.11. Emotional response

Figure 3.11 indicated that emotional response is one of the factors that develop as a result Cognitive activities and the activation of amygdala. This forms one of the fundamental stages for the development of anxiety. This relationship is represented in equations 9a & 9b and the underlying condition is presented in Table 3.11:

$Er\ (t+\Delta t) = Er\ (t) + \tau\ *(k\ (t)-Er\ (t))*(1-Er\ (t))*Er\ (t)*\Delta t$..... *(9a).*

$K\ (t) = w4*Ag\ (t) + w5*Cs\ (t)$..... *(9b)*

τ (tau) is a constant to regulate the relationship and *t* is the periodic variance while *w4* and *w5* are weight of contribution.

Table 3.11: *Variable conditions for stimulus representations in Er*

Conditions	Local Dynamic Property	Non-Local Dynamic Property	Descriptions
Condition 1	*Ag* High *Cs* High	*Er* High	Emotional response will be High, only if the activation of *Ag* Is High and *Cs* is High or *Ag* is High, otherwise it will be Low.
Condition 2	*Ag* High *Cs* Low	*Er* High	
Condition 3	*Ag* Low *Cs* High	*Er* Low	

Condition 4	*Ag* Low *Cs* Low	*Er* Low	

3.4.10 Anxious mood

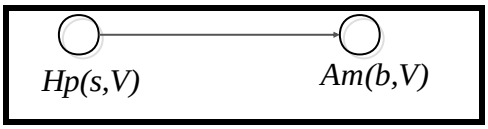

Figure 3.12. Anxious mood

Figure 3.12 shows the initial mood experience by the individual when responding to the circumstances emanating from the occurrence of event within the environment. This state specifies variability in individual personal traits. Some people after processing the stimulus develops high mood while some will develop low mood. The variations in this behaviour will determine the level of anxiety to be experienced by those categories of individual. This relationship is represented by equations 10a & 10b and the underlying condition is presented in Table 3.12:

$Am\ (t+\Delta t) = Am\ (t) + \Theta_m * (Hp\ (t)-Am\ (t))*(1-Am\ (t))*Am\ (t)*\Delta t$ ..... *(10a)*

$Hp\ (t) = \Theta_m * Ag\ (t)$ ..... *(10b)*

Θ_m is a constant to specify changes within the relationship and *t* is periodic variation.

Table 3.12: *Variable conditions for stimulus representations in Am*

Conditions	**Local Dynamic Property**	**Non-Local DynamicProperty**	**Descriptions**
Condition 1	*Hp* High	*Am* High	Anxious mood will high,
Condition 2	*Hp* Low	*Am* Low	if the activation of Amygdala triggers the

	activation of other components as well ,otherwise it will be low.

3.4.11 Body state

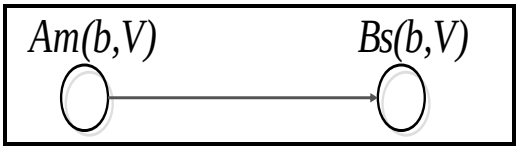

Figure 3.13. Body state

Figure 3.13 shows the body state which is the actual stage through which the body receives response from the brain, if the response received is negative, it influences the development of anxiety, but if positive the body will develop coping skill and the individual will be able to manage the situation. This relationship is represented in equation 11 and the underlying condition is presented in Table 3.13:

$Bs\,(t) = \Theta * Am\,(t)$ ..... *(11).*

Table 3.13: *Variable conditions for stimulus representations in Bs*

Conditions	Local Dynamic Property	Non-Local Dynamic Property	Descriptions
Condition 1	*Am* High	*Bs* High	Body state will be high, only if *Anxious Mood* is high or *Am* is high, otherwise it is low.
Condition 2	*Am* Low	*Bs* Low	

3.4.12 Sensor state for body state

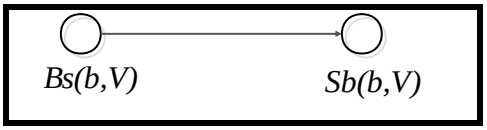

Figure 3.14. Sensor state for body state

The relationship denoted by Figure 3.14 is the state of the internal organs sensing the response received by the body and the formalization is represented by the equation (12). Meanwhile, the corresponding condition underlying this relationship is represented by Table 3.14 below.

$Sb\ (t) = \Phi * Bs\ (t)$..... *(12).*

Table 3.14: *Variable conditions for stimulus representations in Sb*

Conditions	Local Dynamic Property	Non-Local Dynamic Property	Descriptions
Condition 1	*Bs* High	*Sb*High	Sensory representation of body state will be high, only if *Body state* is high or *Bs* is high, otherwise it is low.
Condition 2	*Bs* Low	*Sb* Low	

3.4.13 Sensor state for body response

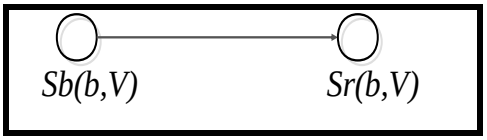

Figure 3.15. Sensor state for body response

Figure 3.15 shows how the response sensed by the body is actively represented by the internal organs and felt by the individual. This relationship is represented by equation (13) and the underlying condition is presented in Table 3.15:

$Sr(t) = \delta * Sb(t)$..... *(13)*

Table 3.15: *Variable conditions for stimulus representations in Sr*

Conditions	Local Dynamic Property	Non-Local Dynamic Property	Descriptions
Condition 1	*Sb* High	*Sr* High	*Sr* will high, if *Sb* is High ,otherwise it will be low
Condition 2	*Sb* Low	*Sr* Low	

3.4.14 Feeling

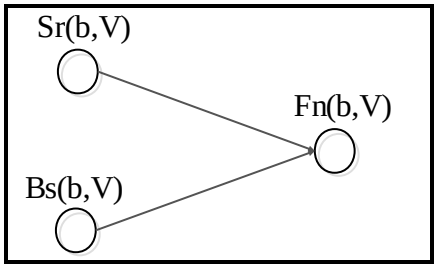

Figure 3.16. Feeling

Figure 3.16 shows the fundamental emotion exhibited by the victims with post disaster experience when responding to environmental events with certain content of fear or seen as a threat. This relationship is represented by the equations 14a & 14b and the underlying condition is presented in Table 3.16:

$Fn(t+\Delta t) = Fn(t) + \sigma_f * (r(t)-Fn(t))*(1-Fn(t))*Fn(t)* \Delta t$..... *(14a)*

$r(t) = v1*Sr(t)+v2*Bs(t)+v3*(1-Ax(t))$.....*(14b)*

σ_f is a constant to regulate the relationship and the periodic variance.

Table 3.16: *Variable conditions for stimulus representations in Fn*

Conditions	Local Dynamic Property	Non-Local Dynamic Property	Descriptions
Condition 1	*Sr* High Bs High	*Fn* High	Feeling will high, if response representation is high, otherwise it is low.
Condition 2	*Sr* Low Bs Low	*Fn* Low	

3.4.15 Anxiety

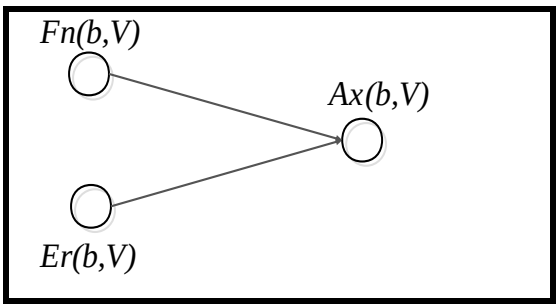

Figure 3.17. Anxiety

Figure 3.17 represent anxiety which is the global behaviour representing the resultant behavior to be exhibited by individuals with high anxiety trait while responding to further environmental stimulus. Its emergence is instatanoeus but often takes time to form part of individual recursive behaviour. Thus, it is often different from fear that occur within little period and die away when such individual became separated from the situation generating it. This relationship is represented by the equations 15a &15b and the underlying conditions are presented in Table 3.17:

$$Ax\,(t+\Delta t) = Ax\,(t) + \xi * (Pq\,(t) - Ax\,(t)) * Ax\,(t) * (1-Ax\,(t)) * \Delta t \quad \text{.....(15a)}$$

$y(t) = \mu_a * Fn(t) + (1- \mu_a) * Er(t)$.....(15b).

Table 3.17: *Variable conditions for stimulus representations in Ax*

Conditions	Local Dynamic Property	Non-Local Dynamic Property	Descriptions
condition 1	*Fn* High *Er* High	*Ax* High	Anxiety will be high, only if feeling is high and emotional
Condition 2	*Fn* High *Er* Low	*Ax* Low	response is high or *er* is high, otherwise it is low.
Condition 3	*Fn* Low *Er* High	*Ax* High	
Condition 4	*Fn* Low *Er* Low	*Ax* Low	

Since, the brain coordinate the entire activities taking place within the human body, this study therefore argue that assessing the developmental stage of anxiety at only the cognitive and somatic level (body) may not provide a convergent measure as the neural components are responsible any instantiation of actions that can lead to such a behaviour. Therefore, this study shows the efficacy of assessing the neural components for the development of anxiety, most significantly through amygdala and prefrontal cortex activation.

3.5 Simulation

The formal model above was used to simulate the executable dynamic properties using Matlab as a programming language to specify both the temporal and the

instantaneous properties in order to generate simulation traces. The Simulation performed is done in two steps. The first step describes the response of the neural components (majorly, Amygdala and Prefrontal cortex) to the stimulus received from the environment. The second is performed against two scenarios.

At the first step, EEG dataset of emotional behavior with a single trial (Liu, 2013) was used to show the response rate at time step 1000hours for each of the trial for both the neural components. The activation level was ranged between 0 and 1. Meanwhile, for spiking neurons, response rate always progresses along both positive and negative value of the potential electrodes. Therefore, the changes are shown between -1 and 1. Results for this simulation is shown in Chapter Four (section 4.3.1 & 4.3.2) respectively.

The second step of the simulation is performed against two distinct scenarios. The scenarios describe the entire neurocognitive activities on the development of anxiety. In this case different types of outcome were obtained by setting out parameters for the most significant factors within the model.

The factors whose outcomes were displayed are the amygdala, prefrontal cortex, emotional response, anxious mood, feeling and anxiety. These factors were assigned values ranging from 0-1 to see the changes at both stages of behavior as analyzed in the model. This range of value is a standard for computational modeling.

Hence, to show relationships with higher tendencies, values ranging between 0.5 - 0.99 are usually assigned as evidence from various other studies as well (e.g. (Bin

Ab Aziz, 2011; Bosse, Memon & Treur 2009; Bosse, Pointier & Treur, 2010; Treur, 2011). Similarly, any relationship with lower tendencies are usually assigned values ranging between 0 – 0.499 (e.g. (Bin Ab Aziz, 2011; Bosse, Memon & Treur 2009; Bosse, Pointier & Treur, 2010; Treur, 2011). Hence, the outcome of simulation base on this concept are presented Chapter Four (Figure 4.1c & 4.1d) respectively.

3.6 Evaluation

The evaluation phase involved the verification of the model and the domain problem through Mathematical analysis. The analysis were done using differential equations specified against the simulation scenarios and cases indicating varying level of individual experience and how the experiences could transform to one another. Details and description are provided in Chapter Four; section 4.4.1 and 4.4.2 respectively. However, further verification analysis using Laplace Transformation is left for future work.

3.7 Summary

This section summarizes the approach implemented in this study to investigate the various conditions needed to explain the development of a formal model for high anxiety trait both at the cognitive and the neural levels. Factors obtained through literatures and expert opinions were formalized into a model to represent the executable properties needed during the programming and simulation process. The relationships among these factors were established with formal equations implemented during simulations. Hence, the result of the simulation is presented in the next Chapter.

CHAPTER FOUR
RESULTS AND DISCUSION

4.1 Introduction

In this chapter, results from Logical analysis (section 4.2) and Simulation (section 4.3) are presented.

4.2 Logical Analysis of Factors Relationships

In this section, the relationships between the various nodes as indicated in the Fig. 3.3 are discussed below:

LP1 Propagating stimulus

If Event *e* occurs in the environment

then a stimulus *S* will occur

Environmental event *(e)* → Sensed stimulus *(s).*

LP2 Sensing a stimulus

If stimulus *s* occurs at an activation level *V,*

then, a sensor state for *s* will occur.

Sensed stimulus *(s)* → sensor_state (s,V)

Hence, it is assumed that the same stimulus activates both the neural and the cognitive state and as such, equal generic dynamic properties applied to both.

LP3 Generating stimulus representation at the Thalamus

If a sensor state with certain activation level *V* occurs,

then, a sensory representation of stimulus *s* will occur at the thalamus at certain activation level *V*: sensor_state (s,V) → thalamus(s,V).

The direct interaction between the thalamus and the sensory cortex enhances further representation of the stimulus in preparation for the neural processing at both the amygdala and the prefrontal cortex. Thus, the dynamic property LP3, determines the specific instance of this stimulus between the thalamus and the sensory cortex at an activation level of *V*.

LP4 Generating an option for a sensory representation of the stimulus

If a sensory representation for the stimulus *s* occur at certain activation level *V*,

then, a representation for option o will take place at the sensory cortex which serve as a gate way to both amygdala and the prefrontal cortex at certain level of activation *V*.

thalamus (s,V) → sensory_cortex (s,V).

The dynamic properties LP4, LP5 and LP6 describe the units of the neural process that determines individual response to stimulus and the corresponding reactions exhibited inform of preparation for a specific bodily action. The unit of the mental component comprises of a number of neural cells, specifically, amygdala and the prefrontal cortex which directly influence the state of emotion towards a particular situation. These two components are being activated majorly by the sensory cortex

and the degree of activation depends on the context of the situation portrayed by the stimulus.

Meanwhile, for any instance of the stimulus, there is an active instantiation of both cognitive and the affect state which determines the options activated by the sensory cortex and the results of neural process directed towards possible actuation of body effect (Anxiety).

This situation gives a clear representation of the part of the recursive loop which occurs between individual anxious state and the possible outcome of behaviour. Mathematically, this affect property interacting with the cognitive ability is expressed based on the following relationship as denoted by the function $a_1\ (\beta_1, \beta_2, V_1, V_2, V_3, \varpi_1, \varpi_2, \varpi_3)$ including a threshold function. This could be further expressed as: $a_1(\beta_1, \beta_2, V_1, V_2, V_3, \varpi_1, \varpi_2, \varpi_3) = a_2\ (\beta_1, \beta_2, \varpi_1 V_1, \varpi_2 V_2, \varpi_3 V_3)$ where V_1, V_2, V_3 are the strength (level) of activation and $\varpi_1, \varpi_2, \varpi_3$ are the corresponding weights of the connections while the threshold function $a_2\ (\beta_1, \beta_2, V) = 1/(1+e^{-\beta 2(V-\beta 1)})$ with threshold β_1 and steepness β_2.

LP5 From sensory representation to option1 mental preparation

If representation of stimulus at option1 occurs at the sensory_cortex with level V

and preparation for neural processing occurs at amygdala at a certain at activation level of V_1

then, a recursive loop occur;

amygdala (n_option1, V_1) → $a_1\ (\beta_1, \beta_2, V_1, \varpi_1) = a_2\ (\beta_1, \beta_2, \varpi_1 V_1)$.

LP6 From sensory representation to option2 mental preparation

If representation of stimulus at option2 occurs at the sensory_cortex with level V

then preparation state for mental processing occurs at the prefrontal cortex at activation level of V_2

sensory_cortex (s, V) → prefrontal cortex (n_option2, V_2)

Dynamic property **LP6** shows the mental processing occurring between the amygdala and the prefrontal cortex to influence Anxiety, Anxious mood and emotional response to the input from the sensory_cortex. The sensory_cortex provides two optional inputs to both the amygdala and the prefrontal cortex and this is expressed mathematically as follows:

LP7a From option1 activation and representation to preparation for mental processing (bottom up reaction at the Amygdala)

If option o with certain level of activation V_1 occurs

and preparation bottom up reaction has activation at level V_2

and representation for o with respect to the stimulus occurs with level V_3

and the preparation for neural process n has level V_4

then, a bottom up reaction for the amygdala will occur with the strength of activation $V_4+\gamma\,(a_1\,(\beta_1, \beta_2, V_1, V_2, V_3, \omega_1, \omega_2, \omega_3) - V_4)\,\Delta t$.

amygdala (s, V_1) & emotional reaction (b, V_2) & sensory_cortex (rep, V_3) & anxious mood for body state (b, V_4) → bottom up reaction ($V_4+\gamma\,(a_1\,(\beta_1, \beta_2, V_1, V_2, V_3, \omega_1, \omega_2, \omega_3) - V_4)\,\Delta t.$).

For the top down control of the prefrontal cortex, inhibiting/ suppressing connections from one of the represented option to the other option relative to the mental state is

considered and this is shown in the expression below with ω_{21} representing the weight of the suppressing connections: $a_1 (\beta_1, \beta_2, V_1, V_2, V_3, V_{21}, \omega_1, \omega_2, \omega_3, \omega_{21}) = a_2 (\beta_1, \beta_2, \omega_1 V_1 + \omega_2 V_2 + \omega_3 V_3 - \omega_{21} V_{21})$.

LP7b From option2 activation and representation to preparation for mental processing (top down control reaction at the prefrontal cortex)

If option1 with activation level V_1 occurs

and option2 with activation level V_2 occurs

and sensory representation for option1 occurs with level V_3

and the preparation state for b_1 has level V_4

then, a preparation for body_state b_1 will occur with level

$V_4 + \gamma (a_1 (\beta_1, \beta_2, V_1, V_2, V_3, V_{21}, \omega_1, \omega_2, \omega_3, \omega_{21}) - V_4) \Delta t.$

sensory_cortex (rep1, V_3) & amygdala (option1, V_1) & prefrontal cortex (option2, V_2) & preparation state (mood) for b_1 (b_1, V_4) → preparation for body_state (b_1, $V_4 + \gamma (a_1 (\beta_1, \beta_2, V_1, V_2, V_3, V_{21}, \omega_1, \omega_2, \omega_3, \omega_{21}) - V_4) \Delta t.$).

However, it should also be noted that, the activation level or strength of activation is a real number that is considered between the interval of [0 and 1] corresponding with the general principle of computational modeling. Thus, the degree of the assigned values determines the actual variations in the above mathematical specification whose output could be observed on the simulation outlines that will be presented later.

LP8 From mental processing to preparation for effector state

If processing of stimulus occurs at the amygdala at certain level of activation V

then, preparation for certain action at the effector will occur at a particular level V

amygdala (s, V_1) → Emotional_response (b, V).

LP9 From sensory representation to preparation for mental processing

If sensory representation occurs at the hippocampus certain level V

then, preparation for mental processing will occur at the prefrontal cortex an activation level V

hippocampus (s, V) → prefrontal_cortex (s, V_2).

LP10 From mental processing to sensory representation and vice versa

If mental processing of stimulus occurs at the amygdala at level V

and representation of part of the processing occurs at the hippocampus at certain level V

then, preparation for anxious mood will be influence at certain level V.

LP11 From sensory processing by the amygdala to preparation for anxious mood

If high neural processing occurs at the amygdala due to greater optional inputs at level V

then, preparation for effector_state (anxious mood) will occur at the hypothalamus

amygdala (s, V_1) → hypothalamus (s, V).

LP12 From preparation to effector_state (Anxious mood) to body modification

If preparation state for effector_state *a* occurs at the hypothalamus with level V

then, effector_state (Anxious mood) for body_state *b* with level V will occur

hypothalamus (s, V) → anxious mood (b, V).

LP13 From effector_state (Anxious mood) to modified body_state

If effector_state *a* occurs with a certain level of activation from the neural processes

then, the body_state *b* with level V will occur

anxious mood (b, V) → body_state (b, V).

LP15 Sensing a body state

If body_state *b* with level V occurs

then, the body_state will be sensed by the internal organs at a certain level V

body_state (b, V) → sensor_state (b, V).

LP16 From sensor state to sensory representation of body state

If sensation of body_state occurs at level V

then, sensory representation of the body_state with level V will occur

sensor_state (b, V) → Srs_body_state (b, V).

LP17 From sensory representation of body state to feeling

If sensory representation of body_state occurs at level V

then, the body_state will be felt with level V

Srs_body_state (b, V) → feeling (b, V)

LP18 From body reaction to preparation state for feeling

If body reaction occurs at certain level of activation V

then, preparation for feeling will occur with level V

body_state (b, V) → feeling (b, V).

LP19 From sensor state to preparation for cognitive state

If stimulus *s* occurs

and sensor state for *s* occurs at level V_s

then, preparation for cognitive state *c* will occur with certain level of activation V_c

sensor_state (s, V) = cognitive_state (c, V).

LP20 From cognitive state to emotional response

If cognitive state *c* occurs at a certain activation level V_c

then preparation for emotional responses will be influenced at a particular level of activation V at body state *b*

cognitive_state (c, V) = emotional_response (b, V).

LP20 & 21 From emotional response and feeling to anxiety development

If and feeling *f* occur at level V

and preparation for certain emotional response *q* with level V_4 will occur

then the development of anxiety will occur at (q, $V_4 + \gamma$ *(*a_1 *(*β_1, β_2, V_1, V_2, V_3, V_{21}, ω_1, ω_2, ω_3, ω_{21}*)* - V_4*)* Δt*.).*

4.3 Simulation Results

4.3.1 Results for first simulation

At this stage, the result of simulation using EEG data set is present and the observable values representing variable activation of both amygdala and prefrontal cortex to received environmental signal are shown in Table 4.1a and Table 4.1b respectively. Similarly, the outputs after simulation are shown by Figure 4.1a and Figure 4.1b respectively. Hence, these values represent possible changes over time.

Table 4.1a: *Value assigned for neural activation*

Stimulus	Prefrontal Activation	Amygdala Activation	Anxiety
0hz	[0.8,-0.95]	[0.1,-0.1]	Low

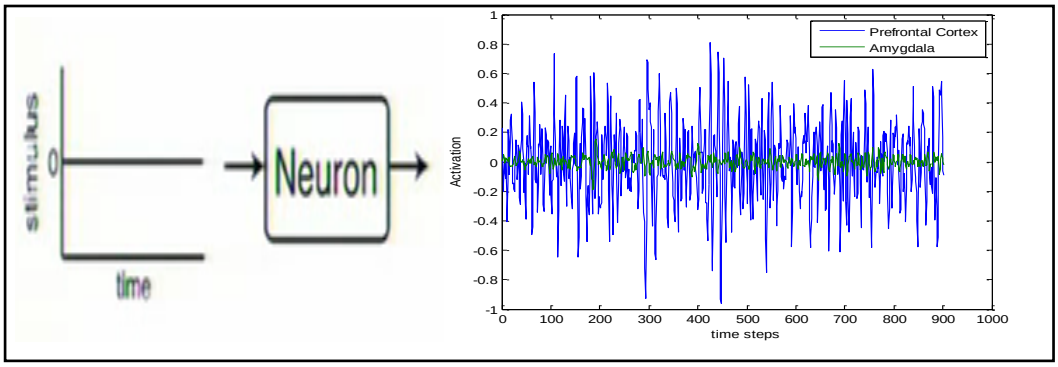

Figure 4.1a. *Prefrontal cortex activation*

In Figure 4.1a, it was demonstrated according to the assumptions made in the study that, if the processing rate of stimulus received from the environment is low or tend to zero, this means that the individuals possess the mechanisms that aid positive interpretation of the event received through the environment, as such prefrontal activation will be enhanced and is assigned value of 0.8 while amygdala activation will be affected and assigned a value of 0.1. This situation will have an inhibitory effect on anxiety development.

Table 4.1b: *Value assigned for neural activation*

Stimulus	Prefrontal Activation	Amygdala Activation	Anxiety
2hz..Nhz	[0.1,-0.05]	[0.9,-0.95]	High

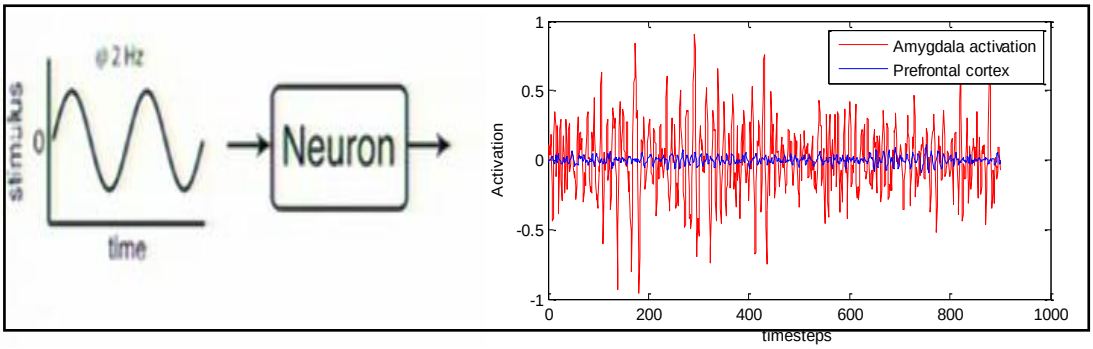

Figure 4.1b. Amygdala activation

Similarly, in Figure 4.1b, it is shown that, if the frequency of stimulus changes either from 0Hz-2Hz and so on, as the case may be due to the poor attentional control mechanisms possess by the individuals as result of the unsupported experiences they have had during the course of exposure to disastrous events. The activation of amygdala play significant role which in turns influences other body mechanisms to enhance the development of anxiety. In that case, a higher value is assigned.

4.3.2 Results for second simulation

4.3.2.1 Scenario #1: High anxious state

This scenario represents a vulnerable stage for victims with post disasters experience to develop anxiety. In this scenario, individuals who are highly anxious normally have high activation of amygdala and low activation of the prefrontal cortex while facing threat related events. The table below presents the simulation outcome and the Matlab code is presented in Appendix A.

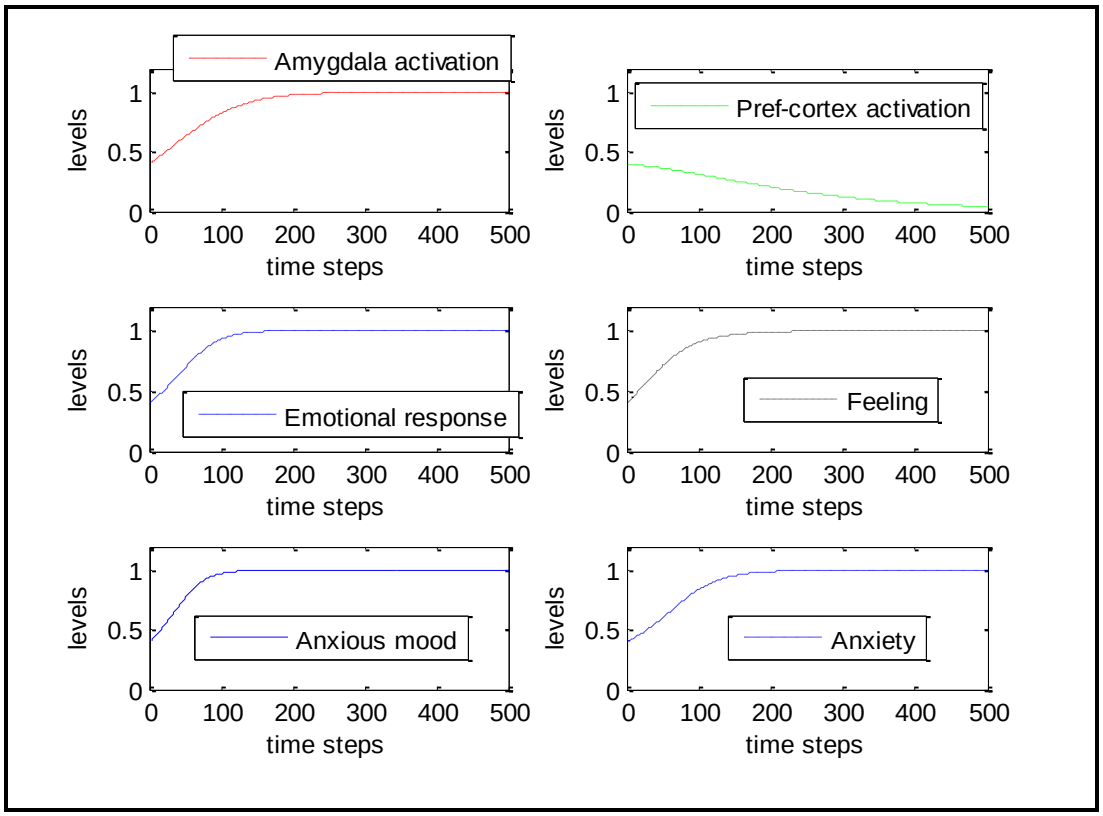

Figure 4.1c. Result of scenario #1

Based on the result presented in Figure 4.1c, it is shown that individuals with high anxious state when encountered threat related events, leads to the gradual response of the amygdala resulting to the development of emotion, mood and feeling which final lead to anxiety if the individual does not received any support**.**

Hence, based on the simulation parameters that were specified for various constants used to regulate the relationships, 0 and 1 were chosen (standard for computational modeling). Evidence from previous studies in the domain of computational modeling shows that, 0 (zero) is considered as a value where no significant changes are possible. One (1) is a maximum value where possible changes within a system can occur. Hence, most studies considered values between 0 & 0.499 ($0 < x < 0.5$) as low

for conditions under investigation (as implemented in (Bn Aziz, 2011; Bosse, Memon & Treur 2009; Bosse, Pointier & Treur, 2010; Treur, 2011). Values between 0.499 & 0.599 were considered for moderate conditions (as implemented in (Bn Aziz, 2011; Bosse, Memon & Treur 2009; Bosse, Pointier & Treur, 2010; Treur, 2011) and those greater than 0.5 but less than or equal to 1 ($0.5 < x <\_ 1$) as the case may be are considered for high and extreme conditions (as implemented in (Bin Ab Aziz, 2011; Bosse, Memon & Treur 2009; Bosse, Pointier & Treur, 2010; Treur, 2011).

4.3.2.2 Scenario #1: Low anxious state

This scenario assumed that individuals with post disasters experience but with low anxious state due to support they have received after their experience in life. These individuals will have tendency to exhibit greater prefrontal cortex activation while responding to threat related event compare to the categories of individuals in the first scenario. This tendency will however, lead to the inhibition of the amygdala which resulted in gradual disintegration of aversive activities such as emotional response, anxious mood and feeling that can aid the development of anxiety. The outcome after simulation is presented below and the Matlab code is provided in Appendix B.

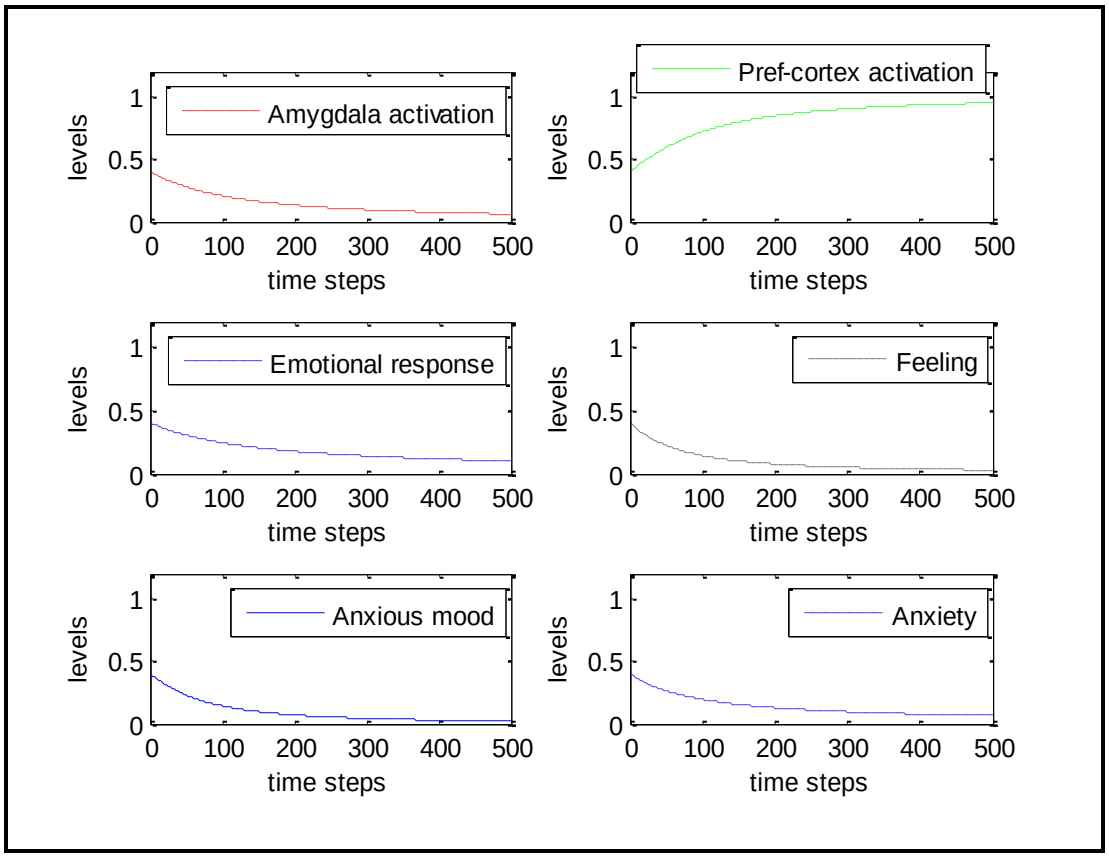

Figure 4.1d. Result of scenario #2

Based on the result presented in Figure 4.1d, it is could be seen that, immediately the individual mechanisms develop resistance to threat received, prefrontal cortex gets activated which causes gradual suppression of the activation of the amygdala. This however resulted in a gradual inhibition of all other body mechanisms that get activated whenever amygdala is activated. Thus, this scenario favours the prevention of anxiety development.

Meanwhile, it should be noted that, this condition can be greatly influence through support from intelligent systems. This system could provide assistance after sensing the state of the individuals through instructions as well emotional support. Therefore,

the implementation of this concept as provided by the model can aid the development of such system that could serve people with post disasters experience.

4.4 Evaluation

4.4.1 Mathematical verification of model

One of the aspects in the model that can be explored using mathematical analysis is to find which of the factor (s) have a direct resultant effect on anxiety development and prevention. To this end, equation for the equilibrium point can be determined (that is, a point where exact variability in the factor(s) will have direct result on the output (anxiety)). In this case, equilibrium points are analyzed based on the certain condition earlier specified in the scenarios. These points describe the situation on which changes in one or more of the factors have a direct resultant effect on changes in the output condition. This is important, as it easily provide justification for problem that is being modeled. As such, realizing a reasonable equilibrium point is a good indication for the correctness of the model.

Meanwhile, this is done by assuming constant values for the variables that were considered as having direct implication on anxiety development. Then, the corresponding relationships earlier specified in differential form can be considered with respect to time *t,* and neglect any possible long time changes (i.e. ignoring Δt). Similarly, assuming all exogenous variables are non-zero, this lead to the following equations where the equilibrium state is characterized: [Refer to Chapter Three, section 3.3-Equations: 6a/6b, 5a/5b, 9a/96, 10a/10b, 14a/14b and 15a/15b respectively].

$dAg(t)/dt = \gamma *(q\ (t)-Ag\ (t))*Ag\ (t)*(1-Ag\ (t))$	**1**
$dPc(t)/dt = \mu_p * ((g\ (t) - Pc\ (t)) * (1-Pc\ (t)) * Pc\ (t))$	**2**
$dEr(t)/dt = \tau *(k\ (t)-Er\ (t))*(1-Er\ (t))*Er\ (t)$	**3**
$dAm(t)/dt = \theta_m *(Hp\ (t)-Am\ (t))*(1-Am\ (t))*Am(t)$	**4**
$dFn(t)/dt = \sigma_f *(r(t)-Fn(t))*(1-Fn(t))*Fn(t)$	**5**
$dAx(t)/dt = \xi * (y\ (t) – Ax\ (t)) * Ax\ (t) * (1-Ax\ (t))$	**6**

Hence, if the adaptation rate for those conditions is assumed to be 0, the equations become

$dAg(t)/dt=0,\quad dPc(t)/Dt=0,\quad dEr(t)/Dt=0,\quad dAm(t)/Dt=0,\quad dFn(t)/dt=0,\quad dAx(t)/dt=0$

Meanwhile, if the adaptation rate for both conditions is assumed to be 1, the relationship will become;

$(q=Ag) \vee (Ag=0) \vee (Ag=1)$	**7**
$(g =Pc) \vee (Pc=0) \vee (Pc=1)$	**8**
$(k=Er) \vee (Er=0) \vee (Er=1)$	**9**
$(Hp=Am) \vee (Am=0) \vee (Am=1)$	**10**
$(r=Fn) \vee (Fn=0) \vee (Fn=1)$	**11**
$(y=Ax) \vee (Ax=0) \vee (Ax=1)$	**12**

From here, the first conclusion can be arrived at. That is to say that, the equilibrium can only occur when *(q=Ag)* ∨ *(Ag=0)* ∨ *(Ag=1),..... (y=Ax)* ∨ *(Ax=0)* ∨ *(Ax=1)* (refer to 7-12 above). By combining these three similar conditions, the relationships could be re-written into a set of alphabetically quantified distributed property, that is informs of:

(A ∨ B ∨C) ∧ (D ∨ E ∨ F) expression, where A = *(q=Ag),* B = *(Ag=0),* C = *(Ag=1),* D = *(g =Pc), E = (Pc=0),* F = *(Pc=1).* All in the first column, the same can be repeated for the rest of the conditions.

[(q=Ag) ∨(Ag=0) ∨(Ag=1)] ∧ [(g =Pc) ∨(Pc=0) ∨(Pc=1)] ∧	**13**
[(k=Er) ∨(Er=0) ∨(Er=1)] ∧ [(Hp=Am) ∨(Am=0) ∨(Am=1)] ∧	
[(r=Fn) ∨(Fn=0) ∨(Fn=1)] ∧ [(y=Ax) ∨(Ax=0) ∨(Ax=1)]	

These relationships can be further expressed using distributive law. That is,

[(A ∧ D) ∨ (A ∧ E) ∨ (A ∧ F) ∨ (B ∧ D) ∨ (B ∧ E) ∨ (B ∧ F) ∨ (C ∧ D) ∨ (C ∧ E) ∨ (C ∧ F)] ≡ [*(q=Ag)* ∧ *(g =Pc)* ∨ *(q=Ag)* ∧ *(Pc=0)* ∨ *(q=Ag)* ∧ *(Pc=1)* ∨..... ∨ *(Ag=1)* ∧ *(Pc=1)*] and so on for the remaining conditions. These however, provide the possible combinations for the equilibrium points which could be further analyzed.

Meanwhile, due to the enormous possibilities for the distribution (i.e. 3^6= 729), it may be difficult to come up with an adequate classification for the equilibrium point that will determine the state of the output. Therefore, in line with the previous

section, two scenarios were analyzed; these could be further expatiated using the current analytical approach.

Scenario #1: High anxious state

At this scenario, the adaptation rate for all the condition is assumed to be 1 except *Pc*.

That is: *Ag=1, Pc=0, Er=1, Am=1, Fn =1, Ax=1*

For this scenario, from equations (4), (7) and (8) in Chapter Three, it follows that:

$Sc\ (t) = \alpha\ *Hm\ (t) + (1-\alpha)$

$Hc\ (t) = \omega\ *\ Hm\ (t) + (1-\omega)$

$Hp\ (t) = \beta$

Similarly, at

Scenario #2: Low anxious state

The adaptation rate for all condition gradually tend to zero except for *Pc*

Ag=0, Pc=1, Er=0, Am=0, Fn =0, Ax=0

Therefore, the corresponding equations become:

$Sc\ (t) = \alpha\ *Hm\ (t)$

$Hc\ (t) = \omega\ *\ Hm\ (t)$

$Hp\ (t) = 0.$

In sum, it could be inferred that the equilibrium point exist *Ag* (Amygdala) whose variability corresponding to the expected variability within the model, this suggests that the assumption made with the model is able to explain the intended purpose. If the output from the neural process which is determine by $Hp= \beta, \beta$ could represent

possible values that can be taking between 0 and 1. This will trigger anxious mood which will contribute to other processes that influences anxiety development.

On the contrary, if *Hp (t) = 0* as shown in the second conditions, it means the body is not receiving output from the neural process, so, the development of anxiety will not be triggered. Meanwhile, it could be argued form the model outline that how about the contribution of *Er*, it should be noted from the result of simulation provided above that immediately, the activities of the amygdala is suppressed, all other conditions gradually deteriorates and this favours the prevention of anxiety. Therefore, this aspect has provided an additional evidence for the assumption made in designing the model.

4.4.2 Mathematical verification of domain problem

At any instance of disasters occurrence, four categories of individual experiences could manifest due to the impact the event may have on the affected population. The first category could be that possessed by victims within the environment where the disaster occurs. These individuals could be referred to as those that experience the highest impact due to the psychological pressure the event might have propagated in them. Therefore, this state could be represented as *H (t)* (i.e. the level of impact of the disaster that varied with time). This may be instantaneous (instant) or temporal (vary with time) to support future behavior.

Another category that might emerge is moderate experience (i.e. individuals that felt the impact of the disasters but can cope to a certain extent). This category could be

represented as *M (t).* Similarly, other categories could be distinguished from these events, that is, those that experience less or no impact of the disaster. This could be represented as *L (t) and N (t)* respectively.

However, each of these states could transit to one another over time, if such individuals received or lack supports. The state of experience of individuals with H(t) could transform to either M(t), L(t) or N(t) [H(t) → M(t) ⟶L(t)→ N(t)]as the case may be depending on the kind of support and the continuity with respect to time. Meanwhile, in the absence of support, the individuals' experience could undergo transformation from [N(t)→L(t) →M(t)→H(t)].

Hence, the stages of transformation of each categories could be represented in a four dimensional dynamic system as adopted from (Ding, 2007).

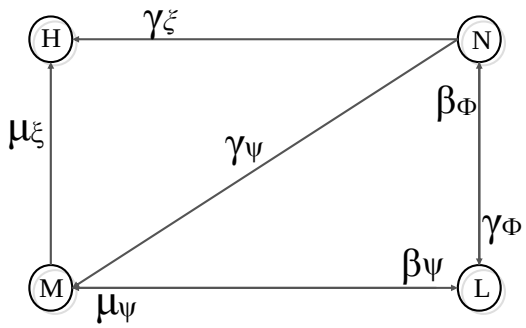

Figure 4.2. Transition stage

As shown in Figure 4.4, four stages of experience are indicated as a condition that transform with respect to some parametric functions. So, these are expressed in form of differential equations to show the actual variability with respect to time.

The corresponding constants β_Φ and β_Ψ denotes the average trajectory rate at which an individual recover from low to neutral state and moderate to low states respectively. Also, μ_Ψ and μ_ξ are the average deterioration rate at which an individual condition deteriorate from low to moderate state or from moderate to a severe state that can further manifest to maladaptive behaviour.

Meanwhile, γ_Φ, γ_Ψ and γ_ξ are the corresponding transmission rate that enhances various other processes that influences this transformation stages and this is likened to High anxiety trait described in this study. For the initial condition,

when t=0; H(0) = 0, M(0) = 0, L(0) = 0 , N(0) = 0 and H(t) + M(t) +L(t) + N(t) = q, q is a constant representing the total population with disasters' experience. Therefore, to establish relationship among the stages, each of the stage is taken as a case on its own.

Case #1: High level of experience

Individuals with high level of disasters' experience but have received interventions and vice versa can have transformation expressed as:

$H(t + \Delta t) = H(t) + [\mu_\xi * M(t) + \gamma_\xi * N(t)] \Delta t.$

so,at $\lim \Delta t \rightarrow 0$

$$\frac{dH(t)}{dt} = \mu\xi * M(t) + \gamma\xi * N(t) \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \ (a)$$

This denote that, at a certain stage, the state of individuals with M(t) and L(t) can deteriorate to H(t) and vice versa.

Case #2: Moderate level of experience

Individuals with moderate level of experience can have the following transformation relationship: $M(t+\Delta t) = M(t) + [\mu_{\psi} * L(t) - (\beta\psi + \mu_{\xi}) * M(t) + \gamma_{\Phi} * N(t)]\ \Delta t$.

At $\lim \Delta t \to 0$

$$\frac{dM(t)}{dt} = \mu\psi * L(t) - (\beta\psi + \mu\xi) * M(t) + \gamma\Phi * N(t) \dots\dots\dots\dots\dots\dots\dots\dots (b)$$

The individual state with low and neutral experience can progress and deteriorate overtimetomoderatelevelandviceversa.

Case #3: Low level of experience

The relationship in the low level of experience is given below:

$L(t + \Delta t) = L(t) + [(-(\beta_{\Phi} + \mu_{\psi}) * L(t) + \beta_{\psi} * M(t) + \gamma_{\Phi} * N(t))]\ \Delta t$.

At $\lim \Delta t \to 0$

$$\frac{dL(t)}{dt} = -(\beta\Phi + \mu\psi) * L(t) + \beta\psi * M(t) + \gamma\Phi * N(t) \dots\dots\dots\dots\dots\dots\dots (c)$$

Individuals with low level of experience can recover over time due to support or progresses to moderate stage without support.

Case #4: Neutral level of experience

This category of individual experience could be represented as follows:

$N(t + \Delta t) = N(t) + [\beta_{\Phi} * L(t) - (\gamma_{\Phi} + \gamma_{\psi} + \gamma_{\xi}) * N(t)]\ \Delta t$.
At $\lim \Delta t \to 0$

$$\frac{dN(t)}{dt} = \beta\Phi * L(t) - (\gamma\Phi + \gamma\psi + \gamma\xi) * N(t) \dots\dots\dots\dots\dots\dots\dots\dots (d)$$

However, the corresponding relationships among the equations (a), (b), (c) and (d) can be further analyzed using Transformation analysis to show the quantitative information relating to the changing behaviour of the victims' experience and the outcome of such experiences on the behaviour.

4.5 Summary

This section provides adequate information and necessary analysis to describe the basis of this study. Graphical illustrations of the result of simulation for both neural and the neurocognitive process has been provided. The analysis and discussions shows the significance of the neurocognitive process in the development of anxiety. Therefore, observing the development of anxiety only at the level of cognition and somatosensory (body) mechanisms alone may not provide sufficient evidence for the investigation of anxiety.

CHAPTER FIVE
CONCLUSION AND RECOMMENDATION

This chapter concludes the findings and presents the summary of the project by specifying (i) the Contribution (ii) Limitations and (iii) Recommendation for Future works.

5.1 Conclusion

As specified in Chapter One, this study has three (3) Objectives; i) to identify the factors or neurocognitive mechanisms associated with the development of high anxiety trait ii) to develop a model (neurocognitive) that formalizes the representation of those mechanisms and iii) to evaluate the model and the domain problem described by the model using mathematical analysis. Therefore, based on the outcome presented in both Chapters (Three and Four), this study has successfully accomplished all the set objectives. The result for objectives (i) and (ii) are shown in section 3.2, section 3.3 and section 4.2. Results of objective (iii) is presented in section 4.3 (sub-section 4.3.1 and 4.3.2) and section 4.4 (sub-sections 4.4.1 and 4.4.2) respectively.

Based on neuropsychological and cognitive theories related to anxiety, several factors related to the neural and cognitive mechanisms for the development of anxiety and high anxiety traits were identified. These have been presented in Chapter Three, section 3.2.

At the neural section, thalamus, sensory cortex, amygdala, prefrontal cortex, hippocampus and hypothalamus were identified from literatures and experts opinion. Meanwhile, abstractions of cognitive and sensory mechanisms were used as suggested by a renowned neurologist Antonio Damasio in some of his work and colleagues in 1999 and 2004 respectively. Damasio stated that the process of generating emotional related behaviour roughly proceeds through a body loop according to the following causal chain: Cognitive state ⟶preparation for bodily response ⟶ bodily response ⟶sensing the bodily response ⟶ Sensory representation of bodily response⟶ feeling.

This approach has been widely explored by several experts such as; Treur (2011, 2013), Bosse, Memon and Treur (2009), Bosse, Pointier and Treur (2010) and so on. Emotional response and anxious mood were derived through literature and expert opinion respectively. Hence, these factors provided the fundamental properties used for the Neurocognitive model designed in this study.

The model is strictly based on the identified factors. The relationships among the factors were established and coded using Matlab as a programming language. This generated simulation traces against two selected scenarios; High anxious state and Low anxious state. This gave insight to the expected behaviour of the mechanisms under investigation for the development of anxiety.

Hence, the design of the model is based on theoretical knowledge. Further investigation using EEG to validate the concept introduced in this study is still required.

5.1.1 Contribution

This study has contributed to a greater extent in the domain of investigation, most significantly by providing a clear justification on the role of the neural components in the development of anxiety. It has identified several factors that can suggest the global trend in the future investigation of this behaviour. The activity of these factors has been simulated to provide evidence for their contributions.

Furthermore, real abstractions of the neural component has been conceptually formalized which has not been done in any of the previous studies. Lastly, the model produced is suitable as domain model when validated for the realization of support systems that can support individuals with devastating experience of disasters so as to prevent future emergence into psychological problem.

5.1.2 Limitations

Meanwhile, despite greater efforts expended on this research, several areas still need be verified and validated in future studies and this includes:

1. Experimentation of the approach using EEG (Electroencephalogram) on the sample of concerned population.
2. Extension of the model through the use of real time cognitive properties.
3. Further Transformation analysis of the domain problem using either Fourier or Laplace transformation analysis.

5.2 Recommendation and Future work

5.2.1 Verification

Ample opportunity can still be explored by analyzing the electrical properties of the various neural components especially, the amygdala and the prefrontal cortex to the varying values of input stimulus through Fourier transformation analysis. The variations can be justified on the convolution signal curves to explain how the activation of prefrontal cortex affects that of the amygdala and vice versa. In addition, this process could be validated with sample of individuals with adverse event of disastrous events to study their response rate to aversive stimulus and how this play significant role in the activation and deactivation of the neural components that in turn influences their resultant behaviour.

5.2.2 Simulation

Meanwhile, as an alternative approach to the one implemented in this research, experimentation using EEG for simulation can be conducted to enhance collection and documentation of efficient/ reliable data that can support real time implementation of the model.

5.2.3 Implementation

This process could enhance validation of the model. Future research can ensure the enhancement of this model into support model as well as intelligent agent model that will be used to design intelligent system that can render assistive support for victims with post disasters experience, doing this, will validate the purpose of the study.

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