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Realities and Delusions[1]

To gain a better understanding of what reality is and how the brain translates perception into reality we examine where in the brain delusion arises. A delusion is generally defined as a contradictory or false belief or impression, which is held regardless of its falsehood and proof to the contrary.[2] The world of the delusion is a derivative world that is normally based on a warping or reconfiguring of the real world within the mind. Towards determining the brains creation of reality I will compare affect and brain area activation in two cases: between those who have a psychiatric condition and those who do not, and between those who have a psychiatric condition and experience delusions and those who have a psychiatric condition but do not experience delusions. I have chosen these two cases because they provide extreme contrast in the perceived reality of the subject. Additionally, within the group diagnosed with psychiatric conditions there are times when the individuals are in altered states caused by these conditions and times when the individuals are not in altered states – this allows for further comparisons to be made. By using functional magnetic resonance imaging (fMRI) analysis to study the brains of the individuals mentioned in the cases above, the authors of these studies have been able to isolate which regions of the brain are active during certain delusions as well as which parts of the brain contribute to the overall experienced reality of those not in a delusional state. Through an examination of where in the mind emotional affect arises and where in the mind altered states of emotional

[1] Originally written for Patrick Suppes' "Philosophical Foundation of Neuroscience", Winter 2005.
[2] As it is used here, false denotes something that is not accepted by others and contradictory refers to something inconsistent as accepted by others to be so. The belief or impression that is part of the delusion is held idiosyncratically in so far as it is peculiar to the group of individuals with the delusion not necessarily the individual.

affect arise I investigate how the mind creates the emotional realities which it perceives. I conclude by arguing that that these neurophysiological studies support eliminative materialism and an identity theory of mind.

First, I will present an overview of the neuronal circuitry related to emotion. Gin S. Malhi et al. in the European Journal of Neuroscience describe this emotional circuitry of the brain (to the extent to which it is understood today). The brain consists of a ventral and a dorsal emotional system.[3] The ventral system comprises the amygdala, insula, ventral striatum and the ventral regions of the anterior cingulate gyrus and prefrontal cortex. I will define these terms and other neurological terminology throughout the paper as it becomes necessary. It is significant that in fMRI studies the amygdala, insula and cingulate gyrus of bipolar subjects have altered blood flow patterns, structural abnormalities, or altered blood or oxygen metabolism when compared with normal control subjects. The ventral emotional system is believed to be involved in the appraisal of emotional input, the production of affective state (the external expression of emotional state) and the regulation of autonomic responses. For example consider someone who becomes scared upon seeing a spider. Becoming scared occurs involuntarily, therefore the ventral system responds to this external emotional stimulus.

The dorsal emotional system includes the hippocampus, and dorsal regions of both the anterior cingulate gyrus and prefrontal cortex. We will see later that in studies of schizophrenic subjects the anterior cingulate gyrus is activated at abnormal times (such as during the viewing of neutral facial expressions) and deactivated at abnormal times (such as during the viewing of angry facial expressions) when compared with normal control

[3] The system I describe is essentially the limbic system of the brain, which will be briefly summarized when it becomes relevant. The research I have done includes some areas of the brain that are outside of the limbic system which is why I refrain from using that term here.

subjects (Malhi et al. *European Journal of Neuroscience* 741-742). The dorsal system is thought to be involved in the intentional or effortful regulation of emotional state. For example consider someone calming ones self down after having seen a spider. The dorsal system responds to this stimulus because the calming down is done intentionally and through a process involving active effort of the one who becomes calm.

It is believed that dysfunction in the ventral and dorsal neuronal systems leads to disorders such as major depressive disorder, schizophrenia, and bipolar disorder. Psychopharmacological remedies for these disorders typically attempt to modify these neuronal emotional systems (some work by blocking chemical reuptake in the brain, some by promoting discharge of neuronal chemicals, some in more complex ways, and most in ways that are only partially understood or unknown). Through a better understanding of the emotional networks that underlie major depressive and bipolar disorders pharmacologists can create better psychopharmacological remedies. We will learn the general workings of the human emotional system, which in turn applies to a general understanding of the mind.[4]

To explore the emotional system of the brain and how the mind creates the affective realities it inhabits,[5] we will examine bipolar disorder and schizophrenia. Researchers have found in repeated investigations that the size of specific brain areas in patients with these disorders differs from the size of those brain areas in the general population. These investigations inform us of an important discovery: that structure leads to function and that structure is function. If we look at brain structure across a wide range of the

[4] Throughout this paper I take a physicalist view assuming that the real world consists of the physical world.
[5] Here I use "inhabits" to denote "realities that are emergent from the physical constitution of the brain and that are being experienced by the individual in that constitution". I do not use "experience" because to experience something is a partial attitude. I can *experience* pain yet be happy. I can *experience* a delusion yet be sane. Whereas *inhabits* describes how one operates from a specific physiological state which in turn dictates experiences.

population we find commonalities in regional size and are able to hypothesize the standard, average, or normal brain, giving rise to normal thought, function, behavior, emotions and affect. If the population is divided into those with certain psychiatric disorders, such as bipolar disorder and schizophrenia, we encounter a similar situation. Regional size remains constant in these populations but differs from that found in the general population. If we look at the general population and contrast it with those with disorders we can also draw commonalities in terms of other brain attributes. The attributes researchers have studied include: increased or decreased brain region activity; hyper-metabolism or hypo-metabolism in various brain regions; increased or decreased blood flow and increased or decreased blood oxygen level dependent (BOLD) responses (a measure which is used to determine how much oxygen certain brain areas are using). Such studies have been performed attempting to identify precise differences in brain structure that give rise to psychiatric disorders and altered mental functioning. We will look at studies that explicitly concern emotions and/or fMRI imaging.

I SCHIZOPHRENIA

Schizophrenia is a condition that involves the disintegration of the relationships between thought, emotion, and behavior. It is generally characterized by a sense of mental fragmentation which may involve delusions and hallucinations. It is believed that it has a genetic basis. In one study of schizophrenia, patients and controls viewed emotionally salient stimuli and described the content of the stimuli.[6] These stimuli were pictures of a human face with an expression of disgust, anger, or fear, alternated with the

[6] That is, an event or situation which is notable emotionally or important to the emotions of the individual and will stimulate an emotional response because of this.

presentation of a facial expression of mild happiness, which served as a neutral facial expression. In this study M.L. Phillips et al. found that subjects who are diagnosed with schizophrenia exhibit poorer performance in facial recognition tasks than those without schizophrenia. That is, the group with schizophrenia correctly identified fewer facial expressions than the control group. An example of an incorrect identification would be identifying (a), in figure 1 below, as fear and not correctly as anger. In the study the subjects were divided into three groups. One group contained controls, the second paranoid schizophrenics and the third non-paranoid schizophrenics. Both subject groups performed poorer than the normal control subjects in the experiment. We can hypothesize that there exists some structural difference between the subject and control groups that is causing the poorer performance of the subject groups. We will examine what these structural differences may be.

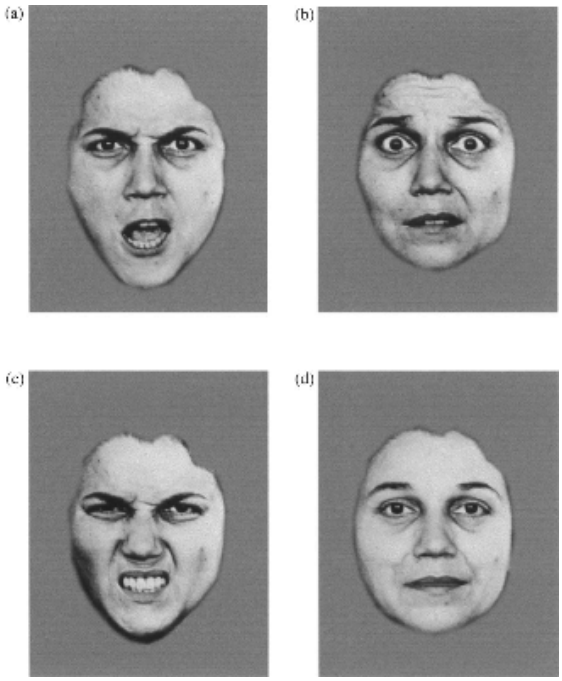

Figure 1: Face from the standard series of Eckman Friesen (1976) appearing in M.L. Phillips et al. (M. L. Phillips et al. 1999). They depict (a) 100% anger; (b) 100% fear; (c) 100% disgust; (d) 75% neutral and 25% happy (used as a neutral baseline).

In non-paranoid schizophrenic patients, brain areas that are normally activated during perception of the stimuli were not activated. Non-paranoid schizophrenic patients identified disgust (c) as either fear (b) or anger (a) more frequently than paranoid schizophrenic patients. Returning to the initial idea that brain structure and activation, etc. (etc. in this context will from now on stand for increased or decreased brain region activation, hyper-metabolism or hypo-metabolism in various brain regions, increased or decreased blood flow, and increased or decreased BOLD responses) properties lead to brain function we can hypothesize (as research suggests) that

(1) both the non-paranoid and paranoid subjects have similar brain structure, or more precisely, similar brain structure when compared to the control subjects.

Given (1), we can make the further hypothesis that

(2) the difference in function that causes a misidentification of facial expressions within the paranoid and non-paranoid schizophrenic subgroup of subjects is expected to be one of activation, etc. and not brain structure.[7] (Although, strictly speaking activation, etc. is a result of brain structure and may, on a fundamental level, be considered part of brain structure. I make the distinction between structure and activation, etc. only as a convenience.)

Hypothesis (2) is marginally justified through the results of Phillips et al. which showed that non-paranoid schizophrenics displayed activation in the amygdala while

[7] There may be brain structure differences in within the schizophrenic subgroup, however these differences are expected to be less significant than the difference between the control group and those diagnosed with schizophrenia. Also of note (and to be explored further below) is how medication administered over a long term can have an impact on brain structure (lithium administered for bipolar disorder is what has been studied). Possibly medications administered to paranoid schizophrenics, which help them to become non-paranoid, may also lead to changes in brain structure in the long term.

viewing pictures of disgust in contrast to paranoid schizophrenics who did not display this activation. The activation of the amygdala is uncharacteristic for viewing pictures of disgust. The amygdala is part of the ventral neuronal emotional circuitry. It is an area of the brain currently associated with the perception of fearful faces and the perception of fear. The activation of the amygdala in non-paranoid schizophrenics distinguishes between the non-paranoid and paranoid schizophrenics, and additionally provides evidence for an altered worldview that affects the perception of those with schizophrenia.

The poor accuracy exhibited by paranoid schizophrenic subjects in the labeling of the threat related negative emotions anger and fear in the above investigation also provides evidence for the mental creation of a worldview different from that of the controls. The types of misidentification include identification of threat where none exists, avoidance of threat appraisal, and incorrect labeling of negative emotions. The perception of these threat related emotions has been dealt with in past neurophysiological studies. In these studies it was shown that the orbitofrontal cortex and anterior cingulate (part of the dorsal portion of the emotional neuronal system) play a role in the perception of anger, although the exact role they play has not been determined.

This leads to the hypothesis that the paranoid subjects who appraise threat related stimuli incorrectly, such as labeling an angry face as one of disgust, will have altered function in some combination of the amygdala, orbitofrontal cortex,[8] anterior cingulate, anterior insula and striatum – when compared with control subjects. Altered function in the anterior cingulate gyrus may produce an altered perception of emotion and thereby lead to unusual perception of threat related facial expressions. The anterior cingulate gyrus is a part of the limbic system. The limbic system is a complex system of nerves in

[8] The orbitofrontal cortex is involved in emotion through its influence upon reward and punishment related behavior.

the brain and body that control mood, instinct, drives and basic emotions. The anterior cingulate gyrus communicates between the prefrontal cortex[9] and the subcortical areas of the limbic system. It plays an important role in the emotional circuitry of the brain (it is responsible for relaying emotions from the cognitive center to the emotional center and vice versa) and it is because of this that we suspect altered function in it. The results showed that in paranoid schizophrenic subjects the anterior cingulate gyrus was activated during the viewing of neutral expressions and deactivated during the viewing of angry expressions. Similar responses were seen in non-paranoid subjects. Because the anterior cingulate gyrus plays a role in the perception of anger through its communication with the subcortical areas of the limbic system, this unusual activation of brain areas fits our expectations. The study concluded that the control subjects had more recognition of and did more processing of anger than the schizophrenic subjects. The dysfunction of the anterior cingulate gyrus appears to lead to this.

The study's results show that in comparison to patients, controls generally exhibited greater activation in the inferior frontal gyrus, putamen, and cerebellum. Additionally, controls exhibited significantly greater activation in the superior temporal gyrus, medial frontal gyrus and anterior cingulate gyrus. The change in the putamen is note worthy because the putamen sends information to the thalamus, which in turn controls the perception of sensory stimuli. The alteration of putamen function, combined with brain function alteration in other areas, leads to changes in the perception of expressions and this leads to changes in the actual perceptual experience. We could hypothesize that if we made a comparison of the information sent by the putamen between schizophrenic subjects and control subjects we would find that the difference in information sent

[9] The prefrontal cortex is involved in planning complex cognitive behaviors, social behaviors and in personality.

corresponds to a difference in sensory realities. The visual stimulation experienced by the schizophrenic subjects may be altered therefore altering their reality. The dysfunction in the anterior cingulate gyrus also serves to alter the individual's affective reality, modifying what information is sent to the prefrontal cortex and possibly altering the individual's personality.

With regard to the fearful facial expressions used in the experiment, controls showed significantly greater activation than schizophrenic subjects in the superior temporal gyrus, amygdala and putamen. Once more the involvement of the putamen – which contains information that gets transmitted to the thalamus and is then involved in sensory perception – may indicate that there is an alteration in sensory perception leading to the altered worldview of schizophrenics. A sensory perception, in which the putamen has been found to be specifically involved in, is that of disgust. With regard to disgust, control subjects showed significantly greater activation in the globus pallidus. The globus pallidus is a part of the brain responsible for transmitting information between the putamen and the thalamus and therefore through an alteration to the globus pallidus' function there is an alteration in how the putamen transmits information. In control subjects in the study performed by M.L. Phillips et al. an alteration was also found in the anterior insula during the viewing of disgust. This all correlates with previous studies, which have linked the viewing of disgust to changes in the anterior insula and striatum.

Paranoid patients exhibited greater overall activation to expressions of disgust and fear than non-paranoid patients. They also exhibited greater activation in the visual cortex and the insula while viewing of images of fear and disgust. This greater activation in the insula while viewing images of disgust may show that increased insular activation

leads to more accurate identification. Additionally, this provides evidence in support of hypothesis (2) that an essential difference between paranoid and non-paranoid schizophrenics is in terms of brain area activation and not unequivocally structural.

Facial expressions expected to induce fear do not appear to activate one of the emotional centers of the brain – the amygdala – when shown to schizophrenic subjects. The amygdala, as said before, is part of the limbic system and is involved in the perception of fear (it additionally plays a large role in motivation and other emotional behaviors). This deficit in the response of the amygdala to emotional cues may lead to the incorrect appraisal of fear.

In the study above we have seen that, in their neurophysiological response to facial expressions, there are fundamental differences between the brain activation occurring in those with schizophrenia and those not diagnosed with schizophrenia. We have seen evidence in support of both hypotheses (1) and (2). More importantly, we have been confronted with a recurring theme as neurophysiology advances, where does structure end and function begin? The evidence above has indicated that on many levels, if not fundamentally, the neurophysiology of the brain leads to the function of the brain: structure *is* function.

II BIPOLAR DISORDER

Bipolar disorder is characterized by abnormalities in mood that can be grouped into three categories: depression, in which the patient experiences unusually low mood and lack of energy; manic and hypomanic periods, which are generally characterized by periods of over activity, delusions and excitement; and euthymic periods, in which the

patient is at a baseline and neither in the state of hypomania/mania or depression.[10] Similar to schizophrenia, genetics is believed to predispose individuals to bipolar disorder, with situational factors leading to its onset. M.P. Caligiuri et al., in an fMRI study they conducted concerning bipolar disorder, found that abnormal activity occurred in the globus pallidus of both manic and depressed bipolar subjects. As said before, the globus pallidus is responsible for transmitting information between the putamen and the thalamus. The thalamus in turn is responsible for relaying auditory, somatosensory and visual sensory information to the cerebral cortex.

Abnormalities in the form of higher BOLD responses in this area were seen in manic patients and lower BOLD responses in depressed patients. The over-stimulation in mania and hypomania is likely to require an unusually high metabolism of oxygen. On the other hand, for depressed bipolar subjects the lack of energy would be expected to require a lower than baseline metabolism of oxygen (where a baseline oxygen metabolism would be the oxygen metabolism seen during a euthymic mood). Yet these differences in oxygen metabolism, expected to result from the differences in mood, do not account for the abnormal activity occurring within the globus pallidus in whole. Hypothetically depressed feelings lead to less energy and therefore lead to a lower BOLD response. This in turn leads to less energy and so the cycle repeats. Or, if depression begins in a neurochemical manner, a lower BOLD response creates a need for less energy and again the same cycle is entered into. The process described above may be one which is more spiral like, involving upward spirals into mania and downward spirals into depression (an often referred to phenomenon).

[10] Baseline is used in the customary psychiatric and psychological way to refer to a normal mood used as the starting point for comparisons.

The study performed by Caligiuri et al. hypothesized that

(3) antipsychotics and/or mood stabilizers normalize cortical and subcortical hyperactivity (Caligiuri 171).

The results of the study showed that patients *not* taking antipsychotic and/or mood stabilizing medication exhibited higher BOLD responses in the motor cortex, basal ganglia and thalamus when compared with patients who were taking these medications. These results are clearly in support of (3). The results show a definite relationship where structure (structure is here defined as the presence of antipsychotics and/or mood stabilizers) determines function (the BOLD response).

The higher BOLD responses in the globus pallidus point to problems with the regulation of brain metabolic function. In recent studies on bipolar disorder it was hypothesized that mania arises from a failure in the function of inhibitory processes which regulate emotional and motor behavior. This regulatory failure then leads to excessive activity and other symptoms typical of bipolar mania. It has been found that in tests of manual reaction time, manic subjects display higher cortical and subcortical activity in motor areas than depressed subjects. Hypothetically, this relates to the increased motor activity in mania and the decreased motor activity in depression. With both this and hypothesis (3) above informing us of the effects of antipsychotics and/or mood stabilizers on BOLD levels, we suppose that regulating neuronal emotional circuitry with antipsychotics and/or mood stabilizers will normalize motor area hyperactivity. Indeed, recent studies on bipolar disorder have shown that patients on mood stabilizing and/or antipsychotic medication exhibit lower levels of cortical and subcortical motor activity than patients not on these medications.

Now, with the information above, we can draw a preliminary map revealing the relationships we see between parts of the brain and how the interactions between these parts of the brain cause the abnormal functioning that is believed to cause bipolar mania. An increased BOLD response, or increased activity in the motor cortex, leads to symptoms of mania such as unusually high activity levels, excitement and elation. This may in turn exacerbate the BOLD response levels and lead to further increases in the motor cortex. Antipsychotics and/or mood stabilizers work by renormalizing activity levels, at least in the motor cortex, and possibly with regard to BOLD responses. Which is essentially hypothesis (3) for which Caligiuri et al. provide evidence for in their study. Additional evidence they found is that subjects with bipolar disorder and not taking antipsychotics displayed higher levels of motor activation throughout the brain including the primary motor cortex, the supplementary motor cortex, the globus pallidus and thalamus.[11]

The increase in brain activity among manic subjects was exclusively in the globus pallidus and primary motor cortex. For those with bipolar depression, this increase was restricted to the primary motor cortex only. This indicates that the globus pallidus is implicated in the expression of mania in bipolar subjects. In addition, activity increases in the thalamus and caudate were seen in bipolar depressed patients when contrasted with manic subjects. The activity increases seen in the thalamus and caudate indicate that these brain areas may be associated with bipolar depression.

[11]Further evidence for the involvement of the thalamus in the expression of mania is provided by researcher findings that individuals who have suffered brain damage localized to the thalamus sometimes experience mania. This occurs in individuals who have *not* been previously diagnosed with bipolar disorder (Caligiuri 180).

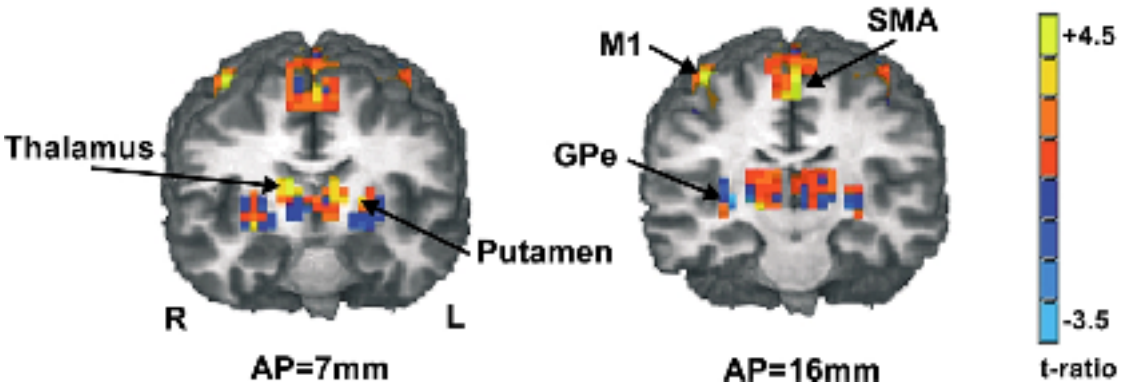

Figure 2: "3-D images portraying the differences (in t-ratios) between bipolar disorder subjects off vs. on antipsychotics and mood stabilizing medications. Yellow voxels[12] indicate greater BOLD responses for unmedicated, while the blue voxels indicate greater BOLD responses for medicated subjects" (Caligiuri et al. 178).

The overall results from the above Caligiuri et al. study showed abnormalities in both hemispheres of the brain and a correlation was seen between the severity of mania and the increase in activity in the external segment of the right section of the globus pallidus. This increase in activity is indicated in figure 2 by the arrow labeled GPe (the abbreviation for external globus pallidus). This increase in activity may cause a decrease in the ability of the globus pallidus to function correctly. As the activity in the globus pallidus increases the ability of it to function correctly decreases and the severity of mania increases. This was earlier hypothesized by Alexander et al. (1986) and Swerdlow and Koob (1987) who suggested that the decreased functioning of the external globus pallidus (manifested as an increase in activity) results in a loss of inhibitory flow to the subthalamic nucleus and that this results in increased excitation in the thalamo-cortical projection. This increased excitation in the thalamo-cortical projection then in turn leads to the symptoms of mania.

In an additional fMRI study on bipolar subjects Malhi et al. refer to research which has shown that projections from various parts of the brain to the thalamic nuclei play a

[12] Voxel is a termed used in fMRI studies to indicate the resolved area being looked at. A useful analog for voxel is pixel.

role in regulating cognitive, emotional and social behavior. It is exactly cognitive, emotional, and social behavior that are affected by bipolar mania. Here we are once more confronted with a relationship in which structure leads directly to function. In this case we see that the globus pallidus is structurally altered by an increase in activation. This activation leads to structural alteration in the thalamo-cortical projection, which then leads to functional differences that result in a deficit in the regulation of cognitive, emotional, and social behavior.

The findings presented by Malhi et al. in their study showed that hypomanic patients, as compared with control subjects, activate additional brain structures in order to process emotionally salient events or situations (Malhi et al. Bipolar Disorders 2004). In the experiment an individual responds to an emotionally salient event or situation by producing an affective state and subsequent emotional behavior in response. While the patient is performing this experiment their brain is imaged using an fMRI. The researchers hypothesize that there exists dysfunction in the limbic system which leads to affective disorders, such as bipolar disorder. It is this dysfunction that the fMRI is attempting to locate.

Past studies have shown that the basal ganglia and amygdala structures are enlarged in patients with bipolar disorder when compared with controls. These structures, the amygdala in particular, are involved in the regulation of affect. It is suspected that abnormalities in their structures are related to abnormalities in affect.

Researchers have also observed that subgenual cingulate volume is less in those with bipolar disorder than in controls. The subgenual cingulate is a curved bundle of nerve fibers that occurs in both hemispheres of the brain and is involved in autonomic

responses and reward mechanisms (Grady, Keightley). Long-term lithium treatment, a bipolar disorder treatment that has proven itself successful with minimal side effects over decades of use, has led to a normalization of subgenual cingulate size. If we hypothesize that the success of lithium treatment is due at least in part to the normalization of subgenual cingulate size we are given evidence that abnormalities in the subgenual cingulate affect bipolar disorder. The study at hand provides additional evidence showing that hypomanic subjects exhibit *no* activation of the subgenual cingulate. If we take into account that researchers have seen subgenual cingulate activation correlated with depression and note that the moods of depression and hypomania are opposite affective states we should expect the lack of activation of the subgenual cingulate in hypomanic subjects. We may hypothesize that the subgenual cingulate activation we are seeing in depression would not be seen in hypomania because the state of hypomania is related to mood excitation and over activity while the state of depression is related to lack of energy and low mood.

In a study of reciprocal limbic-cortical function[13] and negative mood (Mayberg et al.) it was found that with sadness exhibited in depression, there is increased blood flow to the subgenual cingulate. Additionally, a decrease was seen in blood flow to the neocortical region. Although this study does not directly mention size as it relates to depressive and hypomanic states, it does indicate that the subgenual cingulate is intricately involved in the regulation of affect.

In their study of generation of affect in hypomania Malhi et al. found that in hypomanic subjects cortical brain regions were stimulated only when the patients viewed

[13] This refers to the functioning of the subgenual cingulate, anterior insula and neocortical regions (specifically this study involved the right dorsolateral prefrontal and inferior parietal neocortical regions) (Mayberg et al. 675).

stimuli that were congruent to the mood they were experiencing (Malhi et al. Bipolar Disorders 2004). For example, if someone was in a happy mood and they were presented with a picture of a happy face their cortical brain regions would be stimulated. Alternatively, if they were presented with a sad face or if they were experiencing a sad mood and were presented with a happy face, then their cortical brain regions would not be stimulated. This suggests that the hypomanic subjects, in their symptomatically euphoric state, have a cognitive inability to experience negative emotions – or – may experience what normally stimulates a negative affective response as positive affect. In the study it was seen that the hypomanic subjects' responses to negative affect induction approximated what was seen for positive affect induction. This is an indication that in their hypomanic state the cognitive abilities of the subjects enable them to reshape their world. Additionally, hypomanic patients showed a lack of activation in the occipital-visual cortex – an area of the brain responsible for the appraisal of emotionally salient visual stimuli – when compared with other test subjects. This suggests that hypomanic patients are limited in their ability to visually appraise negative emotional affect.

Malhi et al. concluded from this study that hypomanic subjects activated additional brain structures when processing emotional stimuli. These brain structures included the thalamus (responsible for relaying sensory information and serving as a center of pain information) and caudate (responsible for regulating, filtering and organizing information).[14] The hypomanic subjects appear to activate these additional subcortical brain structures in order to process negative affect, while at the same time processing positive affect in a similar manner as the normal subjects. This is apparently a functional

[14] This definition is vague because neurologist knowledge of the function of the caudate is not definitive.

(and hence a structural) compensation for the change exhibited in the manner in which subjects process negative affect.

In an additional study performed by Malhi et al. and reported in the European Journal of Neuroscience (Malhi et al. *European Journal of Neuroscience* 2004) subjects with bipolar disorder were found to have unusually large amounts of activation in emotional centers of the brain. Activation was seen in both the prefrontal and the anterior cingulate cortices. In subjects with bipolar disorder, activation was seen in areas of the brain that were not activated in normal control subjects. This included subcortical areas such as the amygdala, thalamus, hypothalamus, and medial globus pallidus. Similarly to the study published in *Bipolar Disorder 2004* the findings showed that bipolar subjects recruit additional subcortical brain regions to process emotionally salient stimuli during affect induction.

III CONCLUSION

These results suggest that the world of delusions is a very real world. Necessary to the definition of delusion is that the individual undergoing the delusion believes their delusion to be true, regardless of inconsistencies and/or contradictory evidence. Considering what we now know it seems logical that what appears to someone not experiencing the delusion (such as a psychiatrist) as contradictory evidence would not dissuade the individual of their delusion. They are living in a world that positively reinforces their delusion from the ground – the neuronal activity – up to the visual activity. The world enters the brain and is warped to suit the delusion in all fashions. The

brain presents, to the consciousness of the deluded, a version of the world that has gone through abnormal neural processing.

In all the cases presented we see an immediate structure to function relationship. The structure of the brain has determined the functioning of the brain and subsequently the conscious reality that is experienced by the individual. If the individual has a structurally altered brain, such as an abnormally small subgenual cingulate or an enlarged amygdala, then they are more likely to experience mania. However if they then take medication, such as lithium, the structure of their brain will change and they will be less likely to experience mania. Through the uses of fMRI imaging, researchers are beginning to unravel the tangled mystery of how structure leads to function in the brain and are often seeing that function leads back again to structure (an example seen involved spiraling into mania and depression).

Throughout this paper I have at some points made a distinction between structure and function. What seems to be at stake as neurophysiology progresses is precisely this distinction between structure and function. We may even take the studies presented in this paper as a significant vindication of some of the claims made by eliminative materialism. Eliminative materialism is the doctrine that our current understanding of the mind and that commonsense psychology are fundamentally wrong and that mental states do not exist. Beliefs and desires as well the relationship between behavior, internal states, and external conditions is theory to be superseded by a physicalist description of the mind. For example, in the future we will not be in *pain* but will experience 'activation of C-fibers 1 and 4' – or whatever the correct neurophysiological terminology happens to be. The studies at hand vindicate eliminative materialism in so far as we are

now able to reduce qualia to the neurological regions that create them. If someone is experiencing the phenomenon of fear it is because there is a specific pattern of neuronal signals that is moving through their amygdala and interacting with the rest of their brain in such a way as to give rise to the emotion fear. The reality that a person experiences is not shrouded in any mysterious "folk psychology" of beliefs and desires but is a result of the processing of the world by the brain.

Furthermore, when the brain functions in an altered way the reality that is perceived by the brain is altered. We have seen fMRI studies which expose the inner workings of the brain while it is interpreting the real world. We may suppose that each person, control or subject, has an altered view of the world when they perceive it. The view of the world that the controls have is closer to the normal view of the world experienced by those in the general population because they have brains that are structurally (and therefore functionally) similar to the brains of those in the general population. Those with bipolar disorder and schizophrenia receive the same input world yet, because they have altered brain structure (and therefore function), they do not experience the same world.

Herein when I refer to the difference between structure and function, and mention structure leading to function, I am purposely blurring the distinction between structure and function. I believe that this paper presents a significant amount of evidence in support of the argument that the function of the brain is nothing more than the structure of the brain. Further, the evidence supports an identity theory of mind (which I believe fits well beside a doctrine of eliminative materialism) and makes clear the distinction between an identity theory of mind and a functionalist theory of mind. In a functionalist

theory of mind it is not the internal workings of the mind that determine the role a thought plays, but it is the task of the thought in the cognitive system that determines the thought's role. A functionalist may say that *pain* is identified with 'stimulation of C-fibers 1 and 4'. She would hold that *pain* and 'stimulation of C-fibers 1 and 4' do not have the same referent but that they have the same meaning and truth-value.[15] The distinction between functionalism and identity theory enters here. An identity theorist (and an eliminative materialist) would conclude that *pain* is (refers to a thing identical to) 'stimulation of C-fibers 1 and 4'.[16] The structure of the brain (for example, activation of the orbitofrontal cortex and anterior cingulate in such and such a way) *is* the function (perception of anger).

[15] In functionalism these terms are substitutable *salva vertitate* but not *salva significante*. This follows from the supposition that a Martian can be in *pain* and not be experiencing stimulation of 'C-fibers' because they are made of silicon and we might say their 'S-fibers' are stimulated. In this case *pain* would be codesignative with 'C-fibers' and 'S-fibers' hence it is not substitutable *salva significante*.

[16] Assuming an identity theory of mind the terms are substitutable both *salva veritate* and *salva significante*. This begs the question what state, if not *pain*, is the Martian (from 15) in?

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