The symptoms of mental illness health and social care essay

Student number 200623579PSYC 334Although many decades of research have been invested into the pathology and aetiology of many categories of mental illness such as schizophrenia and the psychosis that can often accompany them, these disorders and their symptoms remain largely misunderstood. It was the arrival of chlorpromazine in the treatment of metal health illness that signalled the beginning of highly promising psychopharmacological revolution. However as the promise of this revolution grew, so did the number of mentally ill. Mental illness is such a vast area to try and cover in an entire lifetime of research, so for a single essay this task must abandoned, for this reason the focus of this essay will attempt to utilise research conducted with schizophrenia exploring both genetic and environmental determinants. From the available research, its history and science, it has been suggests that there may be a need to develop new models of care which emphasize psychosocial support using psychiatric medications in a selective, more limited manner. Mental illness more often than not will present with paranoid beliefs, which have been said to be the most common form of symptom involved in severe mental illness (Bentall & Fernyhough, 2008). These symptoms with often accompany normal functioning, with less severe forms of suspicion about the intentions common place which can appear to be assisting normal mechanisms involved in the detection and avoidance of social threat (Bentall & Fernyhough, 2008). Furthermore there are major differences between the paranoid delusions found in acute psychosis and less severe paranoid states, which are only discovered by attention to the phenomenology of beliefs and their relationship to life experiences (Bentall & Fernyhough, 2008). Suggesting that the psychological mechanisms underlying paranoid beliefs appear to

fluid and highly dynamic. Research exploring these mechanisms along with any determinants will be discussed, leading to a biopsychosocial model and conclusions to any implications of these studies, for psychological intervention for those with psychosis (Bentall & Fernyhough, 2008). Accordingly, a schizophrenia psychotic phenomenon is thought to develop in three phases (Ciompi, 1984). The first phase is the premorbid phase, whereby combined biological and psychosocial influences can suggest a premorbid vulnerability, manifesting in a low tolerance of cognitive and emotional stress which in turn can lead to an insufficient capacity to process complex information (Ciompi, 1984). Secondly, stressful life events can lead to repeated acute psychotic episodes (Ciompi, 1984). Finally, the third phase which is dependant on long term evolution, influenced by psychosocial influences more than on biological factors (Ciompi, 1984). Therefore, during stressful and difficult conditions susceptible individuals could proceed into unproductive residual states, largely understood as defensive mechanisms for the organism to restrict stressful over stimulation (Ciompi, 1984). Consequently, this suggests there is no disease entity of schizophrenia with causes, psychopathological picture, or predictable direction. However a multi conditioned life process, developing in individuals with a particular vulnerability, exacerbated by complex and stressful life events and circumstances. If this paradigm is to be truly embraced surely a different concept and approach to schizophrenia would result in positive consequences for both therapy and prevention (Ciompi, 1984). Moreover, in order redress the approach taken towards schizophrenia it must be studied further and in greater depth than ever before, endeavouring to discover possible correlations or indicators. Such research has often suggested that

conditions that one would not associate with schizophrenia have been continuously linked, such as metabolic symptoms including hyperinsulinaemia, type 2 diabetes, dyslipidaemia and obesity (Peters & Langemann, 2009; Venkatasubramanian et al., 2007). These symptoms have shown to be side effects of some antipsychotic medications that are prescribed to treat schizophrenia, maybe coincidentally these side effects or symptoms of metabolic dysfunction have been reported before the chlorpromazine era and also reported in first onset patients before the administration of antipsychotic treatment (Herberth et al., 2011). Using molecular analysis research on post mortem brain tissue, alterations in glucose metabolism and insulin signalling pathways was reported along with blood based molecular profiling indicating hyperinsulinaemia and faults with the secretion of insulin including released factors at first presentation of symptoms (Regenold et al., 2004; Herberth et al., 2011). These observations are not displayed by all with the disorder and not everyone displaying these symptoms become schizophrenic. With the indication of any underlying metabolic weakness in subjects, environmental or genetic factors may progress to the onset of the more overt symptoms of schizophrenia (Regenold et al., 2004; Herberth et al., 2011). Schizophrenia is a psychiatric disorder causing long term disability characterised by cognitive, perceptual and behavioural disturbances, if left unchecked can result with impaired function within social scenarios such as interpersonal relationships, parenting and self-care. Schizophrenia is considered to be a polygenic disorder which has a mixture of environmental risk factors, however the complete aetiology and pathology has not, as yet, been unveiled. Most research into the neuropathological level of schizophrenia has not found a definitive diagnostic

feature, although research has found links with alterations in glutamate, synaptic deficits and dopamine neurotransmission (Zhang et al., 2004). In addition to the findings of the central nervous system effects, peripheral abnormalities have also been recorded including impaired skin flush reaction to the administration of niacin, increased nailfold plexus visibility and other minor physical abnormalities (Compton & Walker, 2009), along with a variety of immune system defects. In addition, promising theories on the aetiology of schizophrenia must include the systemic nature of the disorder. Schizophrenia patients tend to have a reduced lifetime and disproportionate mortality resulting from causes such as accidental death and suicide. Brown, (1997) suggests many deaths in schizophrenia are ascribed to effects of physical illness or metabolic syndrome. Physical effects of schizophrenia can include increased risk of cardiovascular disease, hypertension, dyslipidaemia, insulin resistance, high levels of visceral fat deposition, type 2 diabetes mellitus and impaired glucose tolerance (Bushe & Holt, 2004; Peters & Langemann, 2009; Brown, 1997). The increased occurrence of metabolic syndrome related with mental disorders tends to be reliant on factors such as age, average body mass index and ethnicity of the population in question (Peters & Langemann, 2009). Nevertheless, study of the metabolic syndrome in schizophrenia has become increasingly difficult due to the increased use of second generation antipsychotics such as Clozapine and Olanzapine which can induce similar metabolic conditions such as type 2 diabetes mellitus and weight gain (Peters & Langemann, 2009). With these side effects potentially masked by the use of antipsychotic leading to an increased rate of treatment discontinuation (Peters & Langemann, 2009). There have been frequent incidences of impaired

glucose tolerance and insulin resistance in schizophrenia from research studies conducted before the widespread use of antipsychotics (Bushe & Holt, 2004). In addition, current research has suggested that metabolic abnormalities may occur in the first episode of antipsychotic patients, thus signifying such abnormalities could contribute to the pathology and onset of the disease. Further study into the importance of metabolic perturbations, its role in schizophrenia and their connection with psychiatric symptoms could only result in a much improved treatment and diagnostic strategies based on a personalised medicine approach (Guest et al., 2011). Although glucose homeostasis is largely controlled by the central nervous system mechanisms directed through leptin and insulin signalling and the effects of other components in the neuroendocrine system (Zhang et al., 2004). Thus the only conclusion can be that peripheral and central nervous system metabolic events must be connected at a fundamental level (Zhao et al., 2006). Therefore evidence for peripheral disturbance in glucose metabolism in schizophrenia, with particular focus on studies concerning production of insulin and other molecules related to insulin secretion and insulin action in schizophrenia and evidence for metabolic disturbance in the brain itself (Zhang et al., 2004). Consequently understanding and evaluating research into how underlying metabolic dysfunction could result in psychotic symptoms and thus any implications this will have for the future of diagnosis and treatment strategies for schizophrenia (Zhao et al., 2006). Consequently, how metabolic dysfunction is linked to schizophrenia is of great importance, with hyperinsulinaemia and metabolic abnormalities implicated in other neuropsychiatric conditions such as major depressive disorder (Bushe & Holt, 2004). Upon identification of a panel of analytes

comprised of insulin, growth hormone, cortisol and leptin that was capable of distinguishing first onset schizophrenia patience's (Khaitovich et al., 2008; Zhang et al., 2004). Interestingly, this same panel of metabolic markers was less strongly related to major depressive disorder and bipolar disorder (Regenold et al., 2004). As there is a wide continuum of symptoms between many of the psychotic disorders, further investigation of this would help for improving diagnosis (Venkatasubramanian et al., 2007). Therefore after finding impaired insulin signalling in schizophrenia this suggests that agents which can reverse this effect might have restorative benefit (Elman et al., 2003). This is not surprising as nearly all cells of the body, such as neurons and glia, rely on insulin signalling and regulation of metabolic pathways for normal function (Regenold et al., 2004). Thereby, drugs which improve insulin signalling may in turn provide an improved treatment strategy (Elman et al., 2003). Antipsychotic drugs are capable of inducing metabolic side effects including insulin resistance, weight gain and type 2 diabetes (Peters & Langemann, 2009). The amount of weight gain induced by Clozapine and Olanzapine has been connected with enhancement of psychopathology, signifying that the two effects may be functionally connected (Zhang et al., 2004). Metabolic adjustment such as body weight, blood glucose levels and leptin levels were establish that they accompany improvement in core positive and negative symptoms and another study found that the pre-existing metabolic abnormalities in CSF were normalized by atypical antipsychotic treatment (Venkatasubramanian et al., 2007; Zhang et al., 2004). Early intervention emerges to be crucial for a positive outcome (Brown, 1997) as well as for the normalisation of metabolic abnormalities in schizophrenia (Elman et al., 2003). Rather than treating the traditional

endpoint of the disorder associated with abnormal neurotransmitter signalling, therapeutic strategies which target the underlying metabolic dysfunction may provide an effective alternative, particularly for patient stratification and personalised medicine strategies (Guest et al., 2011). Interestingly the insulinsensitizing agents Metformin and Rosiglitazone have been used to correct the insulin resistance induced by antipsychotic agents without compromising the psychotropic effects (Zhang et al., 2004). Moreover, insulin related molecules may have use as biomarkers not only for patient stratification but also for recording responses to therapeutic treatment strategies (Cohn et al., 2006). Glucose metabolism could be of great benefit for treating cognitive symptoms in schizophrenia, such as memory deficits (Cohn et al., 2006). Consequently, study of the association between peripheral metabolic indices and cognition is justified and may lead to sub typing of schizophrenia on the molecular level (Zhang et al., 2004). With the negative symptoms of schizophrenia difficult to treat, therapeutic strategies that aim at glucose metabolism should be of great benefit (Guest et al., 2011; Kirkpatrick et al., 2009). In addition, these strategies are already proving successful in the treatment of memory deficits in Alzheimer's disease. Cognitive enhancers used for Alzheimer's disease based on the inhibition of insulin regulated aminopeptidase has also been proposed (Khaitovich et al., 2008). This enzyme has effects on both CNS and peripheral systems and provides an instance of how peripheral biomarker discovery can provide additional therapeutic strategies for the treatment of CNS disorders (Holmes et al., 2006). An increased understanding of metabolic dysfunction in schizophrenia may help improve the quality of similar strategies thus improving cognitive function (Perkins, 2007). Given

the potential of this line of research to improve diagnosis and create alternative treatment strategies with pre-existing compounds, it would only be prudent that more research is completed (Perkins, 2007). Future study should include investigations of the metabolic dysfunction in the cognitive subtypes of schizophrenia with additional longitudinal studies to examine the evolution of insulin resistance during the time of the disorder in addition to antipsychotic treatment (Herberth et al., 2011). Further investigation into the peripheral cellular models of schizophrenia, particularly targeting the insulin signalling pathway, should shed light on the underlying metabolic component (Guest et al., 2011). Additional study of preclinical models based on metabolic perturbation, such as prenatal protein deprivation, in combination with standard paradigms in psychiatry research, including psychoactive drug administration and environmental stressors would continue to provide insight (Perkins, 2007). In conclusion, schizophrenia following the publication of DSM-III and DSM-IV continues to be unresolved with the coming of DSM-V, with the difficulty arising in the deconstruction of schizophrenia for this next revision involving an ongoing tension between conflicting approaches such as top-down versus bottom which tend to be best suited for different research or clinical aims. Therefore, the DSM can only continue its function as a rough guide for clinical work and continue to focus on what is visible to clinicians. Persevering with the categorical diagnosis of schizophrenia, continues to add to the stagnation in the etiologic and therapeutic development of schizophrenia. However, incorporating symptom dimensions, including cognitive deficits within schizophrenia for DSM-VI may permit operation outside the strict confines of the categorical limitations in place, thus gaining a greater understanding of what

schizophrenia really is, through breaking the disorder down to its essential components. Finally making it apparent and relevant to clinicians and patients alike. Further study into the metabolic symptoms of schizophrenia could provide crucial insight into schizophrenia which in turn may lead to improvement in both future diagnosis and treatment.