**Biological Explanations of Schizophrenia**

**Research Worksheet**

*Start by identifying which biological theory the research relates to* ***(either genetics, the dopamine hypothesis, or neural correlates)****. Then decide whether it is supporting evidence or challenging evidence. Lastly, match the correct link back statement to each piece of research by writing the number of the study in the link back box*

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|  | **The Research** | **Which Theory?** | **Does it Support or Challenge?** | **Match the link back statement to the correct piece of research** |
| **1** | **Lloyd et al. (1984)** believe that if dopamine is a causative factor, it may be an indirect factor mediated through environmental factors, because abnormal family circumstances can lead to high levels of dopamine, which in turn trigger schizophrenic symptoms |  |  | This suggests brain damage increase in schizophrenics over time and therefore could be a symptom of the disorder rather than the initial cause of the disorder |
| **2** | **Tilo et al. (2001)** used fMRI scans to investigate the level of activity in the Wernicke brain area (an area associated with coherent speech) when schizophrenic and non-schizophrenic patients were asked to talk about a Rorschach ink-blot.  They found that in schizophrenic patients the severity of their though disorder was negatively correlated with the level of activity in Wernicke’s area. |  |  | suggesting that genetics play a greater role than environmental factors |
| **3** | **Ho et al. (2003)**  performed MRI scans on recent-onset schizophrenics and re-scanned them 3 years later. They found evidence of brain damage in the recent-onset patients, which worsened over time, especially in the frontal lobes, which correlated with an increase in the severity of their symptoms. |  |  | Suggesting that high levels of dopamine could be a causal factor in schizophrenia |
| **4** | **Curren et al. (2004)** found that when amphetamines, which activate dopamine production (agonists), are given to non-sufferers it can produce schizophrenia-like symptoms and make symptoms worse in those already suffering from schizophrenia.  Equally, **Kessler et al. (2003)** used PET and MRI scans to compare people with schizophrenia with non-sufferers, finding that schizophrenics had elevated dopamine receptor levels in certain brain areas and differences in levels of dopamine in the cortex were also found. |  |  | This supports the idea of abnormal functioning in specific brain areas being related to schizophrenic symptoms e.g. speech disorganisation |
| **5** | **Kety and Ingraham (1992)** found that prevalence rates of schizophrenia were 10 x higher among genetic than adoptive relatives of schizophrenics |  |  | Suggesting that genetics play a role in schizophrenia but that environmental factors must also contribute |
| **6** | **Gottesman (1991)** found a 48% concordance rate in MZ twins in comparison to 17% in DZ twins |  |  | Suggesting that the differences in the biochemistry of schizophrenics could just as easily be an effect rather than a cause of the disorder and therefore we are unable to draw any firm conclusions |