**Discuss neural explanations of OCD (16 marks)**

Low serotonin levels are thought to be a cause of OCD. This is because if a person has a low level of serotonin then normal transmission of mood-relevant information does not take place, which means that mood, and sometimes other mental processes are affected. This can lead to the obsessive thoughts and compulsions that are part of OCD. In addition, research has found that sufferers of OCD have elevated levels of activity in the orbitofrontal cortex and the caudate nucleus (located in the basal ganglia). The orbital frontal cortex, which receives sensory information, will send a message of panic to the caudate nucleus. In a normal brain, the caudate nucleus with decide if this information is important and if so, pass it on to the thalamus which would then take action. If it is not an important issue the information will be filtered out. However, in an OCD brain, the caudate nucleus is faulty and sends the message of panic to the thalamus, whether or not it is an important issue. The thalamus then carries out the action (e.g. washing hands). This process will keep repeating on a loop, which is why OCD sufferer tend to perform repetitive rituals (compulsions).

There is research supporting the biochemical explanation of OCD. For example, Hu (2006) found that serotonin levels were lower in OCD sufferers than in non-sufferers. Also, Zohar et al (1987) found that when a drug which lowers serotonin levels was given to OCD sufferers and non-OCD sufferers, it worsened symptoms in the sufferers. These studies both support the view that low levels of serotonin are a linked to the development of OCD. However, there are issues with the research that might lower their support for the explanation. For example, Hu’s study only shows an association between OCD and low levels of serotonin, meaning that it is not possible to establish whether the low serotonin levels were a cause or an effect of the disorder. Furthermore, Zohar’s study does not explain why the non-sufferers did not develop symptoms of OCD having had their serotonin levels lowered. The study suggests that low serotonin levels only have an effect when the disorder is already present, indicating that it can only be considered a contributory factor, rather than a causal one.

However, the biochemical explanation has led to practical applications such as the development of drug therapies which enhance levels of serotonin. These have been found to be effective at reducing the symptoms of OCD in many sufferers. This has benefits, not only for the individual, but also to the economy, as drug therapies are an inexpensive and often highly effective treatment for OCD which means less pressure on NHS resources. It also means that the individual can quickly regain functioning, thus boosting productivity in the workplace.

There is also research supporting the neurophysiological explanation of OCD. For example, brain-imaging research shows elevated activity in the orbital region and the caudate nucleus which has been found consistently in OCD patients compared to healthy controls. After treatment, activity in these brain areas reduces to a level comparable to that of controls as found by Saxena and Rauch (2000). This supports the neurophysiological explanation as it shows that these areas of the brain are linked to OCD.