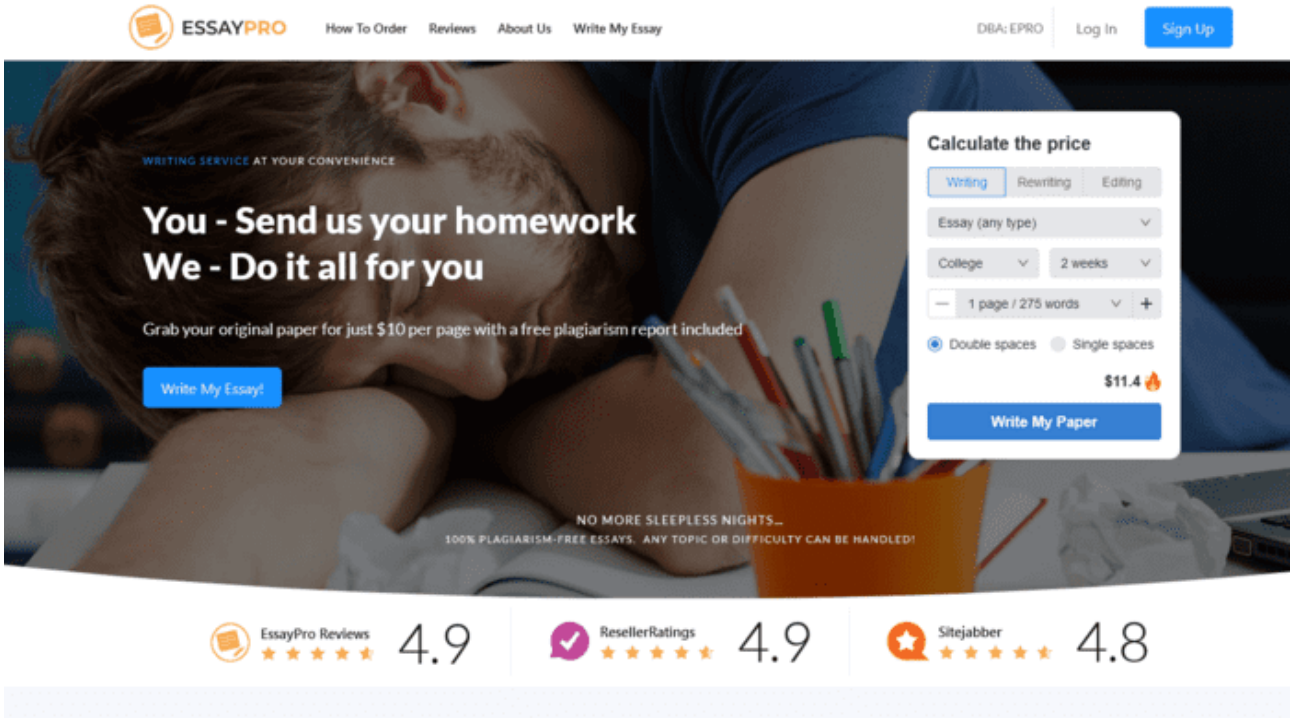
LSD



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LSD

Lysergic Acid Diethyl amide (LSD) has been implicated in a variety of studies to determine its potential for influence on certain neural activities. To date, little that can be certified as concrete fact has been found, though a number of theories with considerable support exist. Although dopamine, epinephrine, and norepinephrine may be implicated in some LSD studies, serotonin seems to be the main focus of scientific inquiry with respect to LSD. Leicht (1996), postulates four theories concerning serotonin (5-HT) pre- and post-

synaptic transmitter sites and the potential for LSD to affect these sites, in particular. All of these theories point to the synaptic neuronal dendrites and terminal buttons as the main suspects with regard to LSD and its particular target area on the neurons themselves. After considerable dialogue which analyses studies by Aghajanian and colleagues, Leicht came to the conclusion that the evidence points toward certain types of activities on particular pre- and post-synaptic serotonergic neurons. The theories are as follows:

1: LSD Pre-synaptically inhibits 5-HT neurons.

2: LSD Post-synaptically antagonizes 5-HT2 receptors.

3: LSD Post-synaptically partially agonizes 5-HT receptors.

4: LSD Post-synaptically agonizes 5-HT receptors.

Neural clusters in the Raphe Nuclei, which spread out from there, mainly into the frontal and prefrontal cortices have been identified as serotonergic. They are also auto-reactive, and LSD appears to inhibit the spontaneous firing of the neurons at that site, when the drug is systemically administrated. 5-HT2 receptors have been identified as pH dependent, while LSD molecules have been identified as pH independent. 5-HT2 receptors are connected to a second messenger system (phosphatidyloniitol, or PI). PI turnover has been found to be affected by 5-HT2 in an antagonistic fashion, but is stimulated by 5-HT. LSD, in micrometric doses, can inhibit 1000 times that amount of 5-HT, which supports theory #2, as well as supporting, partially, theory #3;

when LSD is administered in a variety of doses, it apparently acts as a partial agonist. Though LSD and 5-HT are highly compatible, 5-HT is more effective at the serotonin receptor site, but LSD can compete with it at the 5-HT2 site. The conclusion is, "…since 5-HT is a more potent agonist than LSD, the effects of LSD would appear antagonistic." Finally, for theory #4, Leicht cites Dr. Glennon's explanation of LSD's relationship with post-synaptic 5-HT receptors.

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