What is fear conditioning psychology essay

Emotions are often described as positive or negative feelings brought on by a given situation. Some of the most common emotions seen in everyday life are happiness, sadness, anger and fear. The best said description for emotions is as a multicomponent response that unravels over a short period of time (Fredrinkson, 2001). Emotional responses typically are comprised of three components which are behavioral, autonomic and hormonal responses (Carlson, 2010). Behavioral responses consist of the appropriate physical muscle movements in the given situation. Autonomic responses are the fast reaction and mobilization of energy to facilitate the behavioral response. Lastly, hormonal responses involve the secretion of hormones from the adrenal medulla and adrenal cortex which aid in the availability and conversion of nutrients to glucose. In short, emotions are a chain of responses activated by a specific situation (Carlson, 2010).

Humans often undergo classical conditioning towards a specific emotion. In Pavlov’s classical conditioning, an association between the sound of a ringing bell, a neutral stimulus, and food, a conditioned stimulus, was formed within a dog’s mind. Fear conditioning, much like Pavlov’s classical conditioning, involves a neutral conditioned stimulus is paired aversive unconditioned stimulus (Phillips & LeDoux, 1992). In order to fully comprehend how fear conditioning works, the emotion of fear has to be initially understood. As mentioned before emotional responses comprise of three components, behavioral, autonomic and hormonal responses. Each of these specific components is controlled by a separate neural system. The amygdala is the main mechanism in the brain responsible for the integration of the components responsible for fear.

The amygdala plays a role in behavioral and physiological responses towards a specific stimulus. In the circumstances where aversive stimuli are present, the amygdala is responsible for organizing the emotional response formed by the stimuli (Carlson, 2010). The amygdala is located within the temporal lobes of the brain and consist of several different groups of nuclei which are each responsible for separate input and output responses. The amygdala has been divided into roughly twelve divisions in which each division contain subdivisions (Carlson, 2010). In order to understand the amygdala’s role in the emotion of fear, focus will be placed on the major regions known as the lateral nucleus, the basal nucleus and the central nucleus.

When the body is exposed to an emotional response evoking stimulus, the lateral nucleus receives sensory information from the neocortex, thalamus and hippocampus. Consequently, the lateral nucleus delivers projections to the basal nucleus and other parts of the brain. The lateral nucleus and basal nucleus proceeds to send information to the central nucleus and ventromedial prefrontal cortex which then transmits projections to various regions in the brain which are involved in administering emotional responses (Carlson, 2010).

Philips and LeDoux (1992) conducted an experiment to study the effects of fear conditioning on rats. In the unconditional stimulus phase of the experiment, the rats were exposed to an 800Hz auditory stimulus which was then followed by a short electrical shock to their feet through the base of enclosure. Consequently after the shock, the rats responded with an increase of heart rate and blood pressure, rapid breathing and secretion of catecholamine and steroid stress hormones from the adrenal glands. This

condition was repeated several times to allow the rats to be conditioned. The experiment was then repeated in the conditional stimulus phase with the absence of the shock following the auditory stimulus. The rats in this phase proceeded to react similarly as in the unconditional phase. The rats also displayed the behavioral response known as “ freezing” which showed that the rats anticipated a shock. Phillips and LeDoux (1992) furthered their study by studying the effects of electrolytic lesions on the amygdala and hippocampus. It was found that electrolytic lesions on the amygdala disordered the freezing response cue in a fear conditioned situation. On the other hand, lesion to the hippocampus proceeded to not interfere of the obtaining of the freezing response to the cued condition stimulus but to the contextual stimuli. The information obtained through this experiment suggest that amygdala plays an essential role in fear conditioning regardless of the type of stimulus used as the cued conditioned stimulus while the hippocampus is not necessary for conditioning in the conditioned stimulus but is necessary for fear conditioning responses to contextual stimuli (Philips & LeDoux, 1992).

Humans similarly to the rats in the experiment conducted by Phillips and LeDoux (1992), are able to acquire conditioned emotional response. When exposed to an emotional evoking stimulus, humans will have both specific responses such as a defensive reflex and non-specific response such was increase in heart rate and blood pressure (Carlson, 2010). Studies were conducted on humans to study the effects of removal of parts of the brain to severe seizure disorders. It was found that the stimulation of certain areas of the brain produced autonomic responses which were often linked with fear

and anxiety. However, it was found that only when the amygdala of the brain was stimulated did the patients report actually feeling afraid (Carlson, 2010).

The neural system responsible behind fear conditioning can be divided into two distinct subsystems within the amygdala (LeDoux, 1995; Maren & Fanselow, 1996; as cited in Maren, 1992). The initial subsystem, referred to as the basolateral complex, consist of the lateral nucleus (LA), basolateral nucleus (BL) and the basomedial nucleus of the amygdala (BLA) (Maren, 1992). The LA assumes an essential role in fear conditioning (Amorapanth et al., 2000, as cited in Maren, 1992). Lesions made on the BL does not seem to disrupt fear conditioning, however, does weaken the avoidance behavior (Amorapanth et al., 2000, as cited in Maren, 1992). The second subsystem within the amygdala comprises of the CE is primarily responsible for the amygdala's interface to fear response systems (Maren, 1992). In the condition where lesions were made on the CE, it was found to form a distinct shortage of acquisition and expression of conditional fear (Hitchcock & Davis, 1986; Iwata et al., 1986, Kim & Davis, 1993; Roozendaal et al., 1991; Young & Leaton, 1996; as cited in Maren, 1992). Lesions made in the CE were found to weaken either cardiovascular or somatic conditional fear responses. Based on this it is suggested that the CE is the final common route for the formation of learned fear responses (Maren, 1992).

Based on the model proposed by Paré, Quirck and Ledoux (2004), auditory input is received by the LA from the medial geniculate nucleus (MGm) and the posterior intralaminar nucleus (PIN) which is inferior coliculus projections targets (LeDoux et al. 1990b, Turner & Herkenham, 1991, as cited in Paré et al. 2004). The auditory cortex also transmits information to the LA (Romanski

& LeDoux 1993, as cited in Paré et al., 2004). Auditory inputs from all these various areas of the brain converge at the LA with somatosensory information from the same posterior thalamic regions (LeDoux et al., 1990b, as cited in Paré et al., 2004) Secondly, during conditioning, the lesion or temporary inactivation of the LA disrupts the obtaining of conditioned fear responses (Amorapanth et al., 2000; LeDoux et al., 1990a; Maren et al., 2001; Muller et al., 1997; Sacchetti et al., 1999; Wilensky et al., 1999; as cited in Paré et al., 2004). Third, associative plasticity is displayed by neurons during fear conditioning at latencies regularly found with potentiation of thalamic inputs (Collins & Pare´, 2000; Maren, 2000; Ono et al., 1995; Quirk et al., 1995; Repa et al., 2001; Rogan et al., 1997, as cited in Paré et al., 2004). Fourth, molecular-signaling mechanism is disrupted in the LA which proceeds to prevent long-term memory for fear conditioning (Paré et al., 2004). Based on this, Pare et al. (2004) proposed that this proves that LA is a critical location of plasticity in fear conditioning.

When discussing fear conditioning there are major afferent and efferent information transmission systems in that amygdala that are relevant. The BLA is the location where afferents from the subcortical and cortical sensory systems regions meet (Maren, 1994). The transmission of auditory information from the auditory thalamus or auditory cortex to the BLA is essential for conditioning to auditory CSs ((McDonald, 1998; Pitkanen et al., 1997; Swanson & Petrovich, 1998; as cited in Maren, 1994). Transmission of information from the hippocampal formation to the BLA plays a role in conditioning to contextual CSs ((Kim & Fanselow, 1992; Maren, 1999c; Maren & Fanselow, 1995; Phillips & LeDoux, 1992; as cited in Maren, 1992). Visual

information is transmitted to the BLA from the perirhinal cortex (Campeau & Davis, 1995a; Rosen et al., 1992; as cited in Maren, 1992). The BLA contains single neurons that respond to auditory, visual and somatic stimuli which support the theory that the amygdala is the gathering location of information about CSs and USs (Maren, 1992). In short, it is suggested that the BLA directs information to the CE to allow association to be formed in the BLA to provoke CE to deliver fear responses (Maren , 1992).

In the case where there is damage to the amygdala, it was found that memory regarding emotion was weakened (Carlson, 2010). Normally, humans undergo an increase in memory about a strong emotional inducing event. Conversely, patients with bilateral degeneration of the amygdala showed a significant difference in memory compared to a regular healthy individual. Cathill, Babinsky, Markowitsch and McGaugh (1995) (as cited in Carlson, 2010) conducted an experiment where participants were presented slides of an emotion evoking story and of a neutral story. Typically, a regular participant would show signs of increased memory of the emotion evoking story. However, participants with damage to the amygdala displayed the opposite with not increase of memory. Mori, Ikeda, Hirono, Kitagaki et al. (1999) (Carlson, 2010) conducted a study on patients with Alzheimer’s disease who had witnessed the 1995 earthquake in Kobe, Japan. A correlation was found between memory and amygdala damage. The more damaged the amygdala the lower the chances the patients would remember the earthquake.

In short, the different nuclei within the amygdala are responsible for specific roles in the responses in the emotion of fear. When lesions were made on

the amygdala different effects on fear responses are seen while damage or degeneration of the amygdala effects the memory association with fear. The studies and models mentioned above support that the amygdala is the main area in the brain that is responsible for fear conditioning.