Module 4:Hormone-Behaviour Relationship

Lecture 26:Most sought after issues in psychology

The Lecture Contains:

- Most sought after issues in psychology
- Hunger

Previous Next

Most sought after issues in psychology

If you closely observe the topics predominantly discussed and researched in psychology and the description given above, almost every psychological response seems to have a chemical basis. Neurobiology literature suggests involvement of large number of bodily chemicals in regulating most of the human behaviour. For long the focus within psychobiology was on biogenic behaviour such as eating and sexual behaviour. A large number of bodily chemicals have been found to be involved along with certain regions of the brain. For instance, bodily chemicals implicated for sexual behaviour includes serotonin, dopamine, prolactin, androgens, neuropeptide transmitters of the hypothalamus, intracellular dihydrotestosterone and testosterone receptors. In the due course of time those topics have also been studied which cross-cuts the boundaries of areas within psychology. Dominant and subordinate positions in a social system, leadership, power are few such examples. We shall now look at the details of two widely studied topics in psychology and try to sketch their chemical roots.

Hunger

Earlier studies based on electrolytic lesion suggested that the ventromedial hypothalamic nucleus triggers voracious tendency and such rats have become obese due to this propensity. Lesion of the lateral hypothalamic nucleus encouraged anorexic tendency forcing the animal to starve. When energy storage of the body is depleted, adipose tissuerelease leptin which gives feedback to the central nervous system about this. The pro-opiomelanocortin (POMC) neurons are implicated in the regulation of energy homeostasis in the hypothalamus and leptin influences POMC. The deficiency of POMC increases appetite making us obese.

With the recent trend of studies we know that eating behaviour is influenced by many peptides and hormones. Orexigenic peptides increase whereas anorectic or anorexigenic peptides inhibit eating. Some of the orexigenic peptides are NPY, ghrelin, melanocortin, PYY3-36, and galanin. Anorexigenic peptides include cholecystokinin (CCK), melatonin, neurotensin, and alphamelanocyte-stimulating hormone. Let us take the example of NPY. Neuropeptide Y (NPY) stimulates eating and arcuate nucleus is the site for NYP in the brain. Leptin is a hormone produced by fat cells. It plays a significant role in maintaining normal body weight. Accumulation of fat in the body increases the size of adipose cells. In cases of excessive fat, the adipose cells secrete leptin which, in turn, inhibits NPY in the brain thus reducing eating behaviour. Absence of leptin triggers overfeeding leading to obesity whereas rats without leptin receptor starve. Leptin acts on specific receptors in the arcuate nucleus of the hypothalamus. Cells containing NYP are also found here. Leptin inhibits the release of neuropeptide Y, thus restraining eating behaviour.

Previous Next

contd..

The levels of peripheral peptides such as leptin, cholecystokinin (CCK) and glucagon-like peptide-1 (GLP-1) rise after meal signaling the brain to stop food intake. Postprandial cholecystokinin (CCK) level is low in individuals with binge eating. Binge eating is a state when one consumes larger amount of food in a discrete period of time and have a lack of control over this behaviour. Ghrelin is a gut peptide that is down-regulated by excess growth hormone. It increases our food intake. It rises before meal and falls after having it. High energy value meals lead to sharp decline in ghrelin. Under normal conditions, peripheral peptide such as ghrelin influences food intake. Its presence facilitates intake whereas decline terminates the intake process. Other peptides such as CCK, leptin, amylin and GLP-1 terminate food intake and rise after meals.

Cholecystokinin is a hormone that signals the brain processes for satiety function, thus helping us stop from eating any more. Decreased cholecystokinin level leads to increase in the amount of food eaten. It is also influences anxiety, depression, psychosis, cognition, nociception, and feeding behavior. There is a likelihood that the release of cholecystokinin is modulated neurotransmitters such as dopamine, serotonin, opioids, glutamate, and GABA receptor activation (Ghijsen et al., 2001). Cholecystokinin is present in the anterior cingulate cortex in abundance and plays significant role in the affective component of pain. It also modulates the nigrostriatal and mesolimbic dopaminergic pathways. As mesolimbic dopaminergic pathway is crucial for motivation and rewarding processes, it gets altered in depression, thus affecting mood disorders.

Postmortem studies of suicide victims have shown elevated preproCCK mRNA level in them as compared to healthy people. Increased density of CCK containing neurons in the dorsolateral prefrontal cortex and CCK receptors in the frontal cortex has also been reported (Harro et al., 1992). CCK in also supposed to facilitate learning and memory processes. It is present in limbic structure and cortical areas concerned with the control of cognitive processes, and motivational and emotional behaviour. CCK1 receptor modulates CCK-stimulated dopamine release in the posterior nucleus accumbens. Alteration in CCK1 receptor results into increased dopamine release, thus predisposing the individual to schizophrenia.

Previous Next