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ROLE OF NEUROPEPTIDES IN HUMAN BEHAVIOUR

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ABSTRACT: Neuropeptides are chemical messengers that are comprised with small chain of amino acid. Neuropeptides are synthesized and released by neurons. Neuropeptides belongs to class of neurotransmitters. Human Behaviour is dependent on social interaction and it is simply modulated by a human's internal state. In humans, we observe different social contexts that can alter anxiety levels and modulate social behaviour. As like dopamine, serotonin, adrenaline these neuropeptides also modulate behaviours such as addiction, intelligence, habits, social behaviour, reproductive system, moods, pain, states of brain like excitement, sadness, happiness, anxiety, hallucination, depression etc. This review is also outlines about the neuropeptide CRF and NPY for their action on addiction, NPY for effect on memory function. Oxytocin is a birth hormone but it also controls major behavioural activities in male and female but mainly in female it increases social behaviour it gives powers to women hence that she can analyse social clues, identity faces, increase memory. In this review we discuss neuropeptides like Orexin, Dynorphin, Enkephalin, Galanin, CGRP, Oxytocin, CRF (corticotropin releasing factor), NPY (Neuropeptide Y), Phoenixin, Kisspeptin and their effects on behaviour on human with some experimental animal proof.

INTRODUCTION: Neuropeptides are involved in homeostatic regulation. They are secreted only from hypothalamic neurons. Neuropeptides are also recognised as neurotransmitters and neuropeptides are secreted from brain areas that are associated with a myriad of motivated, psychopathological behaviours. Neuropeptides were discovered in the mid-1960s by David de Wied. he first reported influence of stress hormones that are involved in homeostatic regulation.

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CRH is the principal mediator of the stress response of the hypothalamic–pituitary–adrenal axis, as well as its function in neuroplasticity and adaptations in chronic stress exposure. Various neuromodulators and neuropeptides are employed in stress. Astressin, a CRF receptor 1 antagonist that restore sleep to normal levels after CRF blocked controllable stress.

Homeostatic processes, social recognition and fear conditioning these all things are regulated by OT neurons, which project to other brain areas. OT reduces anxiety and depression-like behaviours by inhibiting the neuroendocrine stress signalling pathway. Neuronal plasticity, synaptic strength, and anatomical changes all influence the function of neuropeptides and hormones. Understanding the role of neuropeptides and hormones, in the

regulation of motivation and behaviour that is important for both physical and mental health. Research in this field is now growing [1].

Examples of Neuropeptide:

- **Hypothalamic Releasing Hormones:** TRH, LHRH, GHIH (Somatostatin).
- **Pituitary Peptides:** ACTH, β-Endorphin, α-MSH, PRL, LH, TSH, GH, Vasopressin, Oxytocin.
- **Peptides Acting on Gut & Brain:** Leucin enkephalin, Methionine enkephalin, Substance-P, Gastrin, CCK, VIP, Nerve GF, Brain derived neurotropic factors, Neurotensin, Insulin, Glucagon.
- **From other Tissues:** Ag-II, Bradykinin, Carnosine, Sleep peptides, Calcitonin.

Neuropeptides have a far higher molecular weight than neurotransmitters and they are 1000 times more potent than neurotransmitter. They are autolyzed and have a sluggish action and they operate on receptor proteins in a different way. This review outlines role of few neuropeptide mainly on human behaviour with some experimental animal researches.

Neuropeptides' and Their Role on Human Behaviour:

Galanin: Galanin is neuropeptide, widely located in the central and peripheral nervous systems and endocrine system. Galanin (GAL) has an effect on the GAL1, GAL2, and GAL3 receptors. These are GPCRs receptor- The galaninergic family also includes galanin, GALP/GALP/GALP/GALP/GALP/GALP/GALP/ GA, GMAP (galaninmessage-associated peptide), alarin, galanin receptor types 1–3 (GALR1–3). GAL is found in close proximity with a small number of neurotransmitters and it associated with mood disorders such as anxiety and depression. GAL3 is present with neurotransmitters hence GAL3 is a neuromodulator that inhibits acetylcholine, GABA, 5HT, DA, NA, and glutamate. Humans, monkeys, rats, mice, guinea pigs, sheep, and fish all produce galanin [12]. "In women (but not in males) who has major depressive disorder is noticed with plasma levels of galanin that are positively linked with depression severity". GAL is hypothesised as to control stress-related reactions and coexists with NA. Extracellular 5-HT levels are reduced by the GAL peptide. This action has effect on mood and the modification of addiction-related brain circuits. Anxiety and depression-like behaviours are induced in rats by i.c.v. injection of 3 nmol GAL, as evaluated by the Vogel punished drinking test. Furthermore, the activation of 5-HT1A receptors in the dorsal raphe nucleus (DRN) is modulated by intraventricular GAL, suggesting that GAL causes depressive processes in the DRN. GAL may mediate dopamine induced rewarding element of alcohol. "Humans have been linked to alcoholism in two ethnically and geographically disparate communities." GAL appears to be protective against opiate abuse and nicotine dependence. galanin seems not to play role in physiological control of body weight in humans [20].

Orexin: Orexins, also known as hypocretins. Orexins are neuropeptides produced in the HYP neurons (hypothalamus). Orexinergic neurons are found all across the neuroaxis, especially in areas related to reward, emotion, learning, and memory. Orexins are made up of two neuropeptides, orexin A and B, as well as they have two receptors, orexin receptor 1 (OX1) and orexin receptor 2 (OR2). Chronic ethanol drinking caused a 3-fold increase in pre-pro-orexin mRNA expression in the lateral HYP in alcohol preferring (iP) rats during in a 70-day period.

These various studies also encourage the development of Ox1R blockers for human use in the treatment of addiction and other motivational disorders. Furthermore, Ox1Rs have different effects on open field anxiety-related behaviour depending on age and sex, and there are sex differences in orexin markers in adults with depression. These findings reveal that factors that are contributed to enhance cocaine addiction increase the number of LHA orexin cells and Ox1R expression in the lateral hypothalamic area [15].

Orexin plays a role in wakefulness (as evidenced by narcolepsy). Narcolepsy is caused by a lack of orexin. Orexin has a role in obesity resistance by boosting SPA (spontaneous physical activity) and regulating energy expenditure. As a result, if

shortage orexin occur that may cause obesity. Narcolepsy affects the brain's reward system. Physical activity boosts orexin levels [19].

Dynorphin: Dynorphins are neuropeptides derived from the prodynorphin precursor protein. It is an endogenous ligand of the kappa opioid receptors (KOR), dynorphins (A and B) mediate reward and stress. Rat's experimental behavioural study: In rats, microinjections of dynorphin into the PVN and the ventral medial HYP have been demonstrated to improve food intake [16].

Dynorphin has also been shown to play a functional role in nicotine addiction.KOR antagonist (LY2456302, now known as CERC-501) was developed by Eli Lilly scientists and it has been licenced to treat depression [18].

Enkephalin: The neuropeptide enkephalin is derived from pre proenkephalin and exists in two forms, met-enkephalin and leu-enkephalin, which contain the amino acids methionine and leucine respectively. Fat intake has been linked to enkephalin with increase in high fat diet increases in neuropeptide enkephalin concentration in hypothalamus. This increase was observed after both short and long term (1 week) consumption of the high fat diet. High fat content appears to be a crucial determinant in enhanced enkephalin expression. Alcohol, nicotine has been demonstrated to stimulate c-Fos expression in enkephalin specially in cells within the central nucleus of the amygdala (CeA) and PVN. They have different effects on addiction also [16].

Kisspeptin: It regulates one's behaviour, mood and reproductive system. Kisspeptin was discovered in human melanoma cell lines as a metastasis suppressor gene (previously known as metastin). Reproduction is coordinated by a variety of neuronal, endocrine, and associated behaviours. Kisspeptin (encoded by the KISS1/kiss1 gene), is a reproductive hormone orchestrator that acts up stream of gonadotrophin releasing hormone (GnRH) at the apex of the hypothalamic-pituitary-gonadal (HPG) reproductive axis, is now well-established [11]. Kisspeptin is also found in the limbic and paralimbic brain regions, which have been linked to sexual and emotional behaviour. Kisspeptin signalling and its complicated involvement in upstream reproductive behaviour control including olfactory-driven mate preference, copulatory behaviour, audition, mood, and emotion [3]. Recent research on the role of kisspeptin in relationship with preferences: - A single peripheral injection of kisspeptin restores robust male preference, as seen by the female test mouse spending more time putting her nose through the partition holes in front of the male stimulus throughout the course of a 10-minute observation.

Auditions: Male tactile impulses initiate the lordosis posture in non-primate female mammals, which is critical for reproductive success. The predominant manifestation of nitric oxide is for lordosis. This information comes from mouse experiments. Kisspeptin-mediated lordosis is independent of GnRH signalling, but it is dependent on NO signalling downstream [4].

CGRP: Calcitonin gene-related peptide is also known as CGRP. Anxiety and depression-like behaviours are linked to epigenetic control of the CGRP gene (reduced methylation of the CGRP promoter). The CGRP receptor is a G-protein coupled receptor complex made up of three subunits of the calcitonin receptor-like receptor (CLR) protein, which has 55 percent sequence homology with the calcitonin receptor. This homology is needed for CGRP binding, selectivity and cell surface receptor expression.

The intracellular membrane protein, CGRP-receptor component protein (RCP) affects cAMP signalling coupling. The cAMP-PKA signalling pathway is activated by CGRP receptors. Nociception and emotional processing are linked by CGRP in the amygdala. CGRP delivery stereotaxically into the CeA that boost rat's emotional responses (audible and ultrasonic vocalisations) and induced mechanical hypersensitivity according to behavioural studies (decreased hindlimb withdrawal thresholds). "Behavioural effects of CGRP were prevented by a PKA inhibitor but not by a PKC inhibitor, which was consistent with studies linking NMDA receptors and PKA in CGRP facilitation [16]."

Neuropeptide S (NPS): Neuropeptide S (NPS) is a 20-amino-acid protein with a serine residue at the N-terminus that is conserved across species,

including humans. Neuropeptide S was first discovered to be an asthma susceptibility gene (formerly GPR154). Various activities such as Food intake, alcohol and drug addiction, social behaviour, locomotor activity, arousal, wakefulness, memory processes, fear and anxiety are influenced by NPS. This thing is based on the results of direct injections of NPS into the amygdala, the anxiolytic and fear-extinction effects of NPS have been attributed to activity in the amygdale [16].

Oxytocin: The neuropeptide and peptide hormone oxytocin are generated in the hypothalamus and released by the posterior pituitary gland. OXT (oxytocin) has anxiolytic and anti-stress effects in nursing moms. They experience pleasant mood states, lower anxiety levels, and improved calmness. When compared to bottle-feeding lactating mothers, breast-feeding mothers are reported to have lesser stress and emotional effects. Increased plasma OXT levels were discovered with warm social interaction (*e g.* embracing) and orgasm. Sexual activity is linked to drowsiness, enhanced relaxation and peacefulness in the post-coital phase. These things are in accordance with anecdotal and experimental evidence. According to this concept, intranasal OXT makes humans more trustful.

Also, the subject that is treated with intranasal OXT had lower levels of anxiety and psychosocial stress. Magnetic resonance imaging study demonstrated that intranasal OXT decrease neuronal responses in the amygdala to fearful social stimulus in healthy men. Importantly, OXT improves your capacity to understand other people's minds by identifying social indicators in their facial expressions. On the other hand, intra nasal AVP increased perception of rage and threat in neutral human facial expressions while decreasing impression of friendly faces. As a result, AVP and OXT play distinct roles in social communication. In this respect, it's a worth noting that there is a link between autism spectrum diseases and OXT receptor gene variants related to changes in OXT availability. In conclusion, OXT's effects in the brain have a widerange of impact on the intricate regulation of emotionality, stress coping, and social behaviour in both men and women. As a result, the brain OXT system has been identified as a potential target for the development of novel treatment techniques to treat anxiety and depression [8]. The capacity to recall neutral word pairs after learning is decrease in men treated with 15 IU of OXT, according to the first study. Similarly, administering OXT (10 IU) during the memory retrieval phase rather than encoding phase that reduces memory performance.

When oxytocin is given before learning a word list then OXT (20 and 24 IU, respectively) was found to diminish the quantity of correctly remembered wordsfollows the initial presentation to impair the efficacy of storage. Acute intra nasal OXT increased facial recognition in a very specific setting, suggesting that it may have a biological function in preventing the acquisition of nasty experiences during labour.

After 3 weeks of daily therapy, patients with schizophrenia reported improved long-term verbal memory on non-emotional stimuli. Effects of intranasal OXT have capacity to increase long term memory of emotional stimuli on faces with angry, happy or fearful emotions, aversive visuals and emotional words. OXT can detect social cues (such as faces), but not detect non-social cues (colours, objects).

This difference was found to be independent of gender and emotional expression of the faces in certain experiments. In conclusion, intranasal OXT selectively boosts processes of attention, recognition, and memory in socially important and emotional information, while it has a negative impact on non-emotional, non-social information processing. Patients with Prader-Willi syndrome, who suffer from morbid obesity due to excessive hyperphagia, provide preliminary evidence for an OXT role in food intake in humans. In patients with less oxytocin in hypothalamus, that low oxytocin is found in post mortem tissue. oxytocin is for controlling and reducing food intake within physiological limitations. Findings in mice was that deletion of genes linked to the Prader-Willi syndrome, which causes hyperphagia and late-onset obesity, corroborate this hypothesis. The role of oxytocin in addiction: - The question is if OXT also inhibits drug relapse after sobriety. Then addiction in human is can be triggered by pharmacological signals, and oxytocin has an opportunity in

addiction treatment [7]. OXT has an effect on addiction via acting on the CRF system in the hypothalamus and amygdala. Another theory is that OXT encourages people to transition from seeking object-based incentives (such as drugs) to seeking social reinforcement. AUTISM is a neurodevelopmental disease characterised by difficulty in communicating with others and confined repetitive activity. The presence of a specific OXTR SNP in autism spectrum disorder individuals is not always linked to the etiologic of the illness (autism spectrum disorder). Variation in the OXTR gene (rs75775, rs1488467, rs4564970, rs4686302, rs237897, rs53576, rs2254298, rs2268493, rs237887) is associated with aggressive behaviour in competitive video games. However, the emerging picture of individual SNPs underpinning aggression or antisocial behaviour is complicated; a link between SNP rs237885AA and high levels of aggression or antisocial behaviour has been discovered.

G Allele Carriers Association with:

1. Increased trusting (men, not women).
2. Higher scores on symptoms of emotional withdrawal.
3. Higher susceptibility for bulimia nervosa in Korean women.
4. Increased intergenerational transmission of depression.
5. Increased reward in social interactions (men).
6. Greater level of loneliness if social network is perceived negatively.
7. Increased susceptibility to social ostracism.
8. Decrease in hypothalamic gray matter, increase in amygdala gray matter in males.
9. Impaired effect on recognition.
10. Higher social support seeking at times of distress.
11. Maltreated children perceive lower social support in adolescence.
12. ADHD children have better cognitive ability.
13. Higher general psychopathology scores.
14. Decreased empathy.
15. Increased symptoms of depression in maltreated children.
16. Decreased maternal sensitivity.
17. Higher trust behaviour than A allele carriers.
18. Emotional dysregulation in urban African American children

Unknown Mechanisms of Psychological Trait Association with OXTR SNPs:

The functional impact of relationships between SNPs in the OXTR gene is contribute to numerous behavioural qualities such as loneliness, emotional withdrawal symptoms, sociability, empathy, maternal sensitivity, aggression and antisocial behaviour [8].

Phoenixin (PNX): Phoenixinis a neuropeptide that is formed when the tiny integral membrane protein is broken. This tiny protein is known as Smim20 protein. The activation of the GPR173 receptor is responsible for PNX's biological effects. PNX is involved in the central nervous system (CNS) as well as the female reproductive system, where it regulates the estruses cycle and potentiates LH secretion. It also increases the number of ovulated oocytes by stimulating oocyte maturation. Despite this, PNX has anxiolytic, anti-inflammatory, and cell-protective properties in addition it regulates the reproductive system. It also has a role in behaviour, food consumption, sensory perception, memory, and energy metabolism. PNX has effects on the heart, ovaries, adipose tissue, and pancreas other than CNS. This presents all the currently available studies demonstrating the pleiotropic effects of PNX.

The expression of phoenixin-like immunoreactivity (PNX-li), Smim20, and GPR173 mRNA in humans was estimated semi-quantitatively. In ovary, skin, adipocyte maturation, the expression of Smim20 rises [12]. PNX-14 was discovered to mimic human granulosa HGrC1 cell proliferation and ovarian follicle development, as well as enhance the quantity of ovulated oocytes. In human granulosa cells, PNX also increases the expression of

follicular development-related genes such FshR, LhR and Kitl. This peptide also stimulates CREB phosphorylation and increases oestradiol synthesis in granulosa cells by activating the cAMP/PKA pathway. The role of PNX in appetite regulation in humans is poorly understood. It is worth mentioning however, that blood PNX levels were lower in malnourished anorectic patients and increased after body weight was restored.

When PNX-14 was injected into the hippocampus, it also had memory-enhancing benefits. When a GnRHR antagonist (Cetrorelix) was given, however, these effects were minimised. Alzheimer's disease is accompanied by memory loss and cognitive difficulties (AD). The accumulation of amyloid (A) in the brain is one of the leading hypotheses on the causes of Alzheimer's disease. PNX-14 effectively improved memory deficiency and location recognition memory in mice, in addition to scopolamine-induced cognitive dysfunctions and memory impairment generated by icv injection of A.

Furthermore, plasma PNX levels in Alzheimer's patients did not correlate with any of the cognitive or metabolic indicators. Plasma PNX concentration is adversely correlated with logical memory in moderate cognitive impairment, although it is positively correlated with metabolic parameters such as BMI, systolic blood pressure, and high-density lipoprotein level. It should be mentioned, however, that gender disparities were not taken into account in the study. Hofmann *et al.* found that plasma PNX levels were inversely linked with anxiety scores in obese males in human research. Given that PNX-20 mimics vasopressin secretion but not oxytocin secretion, the antianxiety benefits of PNX are surprising. Both hormones play a role in anxiety and social behaviour, although their effects are diametrically opposed [13]. We show that central phoenixin injection enhanced water drinking in both males and females under ad libitum settings, increased water drinking after nocturnal fluid deprivation, and increased both water and 1.5% NaCl intake under fed and hydrated conditions. Importantly, losartan pre-treatment inhibited phoenixin's effect on water consumption. The knocking of Gpr173 gene with short interfering RNA constructs dramatically reduced water consumption in response to overnight fluid restriction [6]. Kisspeptin (Kiss) and GnRH neurons coordinate reproductive function. The peptide phoenixin-amide (PNX) has been discovered to boost GnRH-stimulated LH production in the pituitary. The GPR173 receptor is responsible for this activation [7].

Vasopressin: Cowley and Liard (1987); Valtin (1987) identified vasopressin as an antidiuretic and pressor hormone produced by the posterior hypophysis. Its endocrine effects are mediated by receptors on target organs, such as vasopressin V receptors on 1A smooth muscle cells lining blood vessels associated with the pressor response, and vasopressin V receptors in 2 the kidney, which are required for vasopressin's antidiuretic renal action. Vasopressin neuromodulates memory in the hippocampus. The mnemonic environment influences the action of vasopressin administered into the ventral hippocampus. Another intriguing discovery was that the mnemonic context is crucial in understanding the effect of vasopressin on memory processes in the ventral hippocampus. Studies of the passive avoidance reaction, in which the animal tends to stay in one spot to avoid an adverse stimulus, reveal that vasopressin has long-term behavioural effects. Vasopressin can increase the release of endogenous ACTH, which can help aged animals and people with attention and arousal. Vasopressin promotes the preference for a familiar (opposite-sex) partner, improves memory, generates anxiety, and affects happiness/anger perception [10].

Tachykinin 2 (TAC2): Chronic social isolation stress has a broad impact on a variety of defensive behaviours. In both people and animal models, prolonged SIS has been shown to produce a variety of behavioural outcome sit include increased aggression and persistent responses to threats. Multiple behavioural alterations in mice were generated by 2 weeks (but not 24 hours) of social isolation stress (SIS), as well as brain-wide overexpression of the neuropeptide tachykinin 2 (Tac2)/neurokinin B(NkB). The ability of osanetant to prevent and mitigate a detrimental brain state created by SIS suggests that Nk3R antagonists should be reconsidered as potential therapies for mood disorders caused by extended periods of social isolation (or other stressors) in humans and domesticated animals. We can consider hat

Tac2/NkB have action to increase in violence in human [17].

Actionsof Neuropeptide Y and Corticotropin Releasing Factor on Alcohol Addiction and Human Behaviour: Neuropeptides are particularly potent in the CeA (central nucleus of the amygdala), where they regulate anxiety and alcohol-related behaviours. The effects of CRF and NPY on alcoholism are diametrically opposed [9].

These peptides have been conceptually divided as: pro-stress peptides (promote), anti-stress peptides (rescue). These peptides reveal unfavourable emotional abnormalities during drug cessation. The CeA contains a large number of pro- and anti-stress peptides. CRF, dynorphin, hypocretin/orexin, and vasopressin are all pro-stress peptides. Neuropeptide Y (NPY) and nociception are two antistress peptides. Opposite behavioural action seen between CRF and NPY.

For Example:
CRF: Promotes increases in anxiety-like behaviour.

NPY: NPY promotes decreases in anxiety-like behaviour, decreases in arousal, increases in feeding. Alcohol withdrawal causes an increase in CRF synthesis and release from CeA and BNST in the central amygdala, which is then normalised by alcohol consumption.

Downregulation of dopamine signalling in the meso-corticolimbic reward system, hyperactivity of glutamate signalling, and dysregulation of brain stress systems these all things characterise the transition from low/moderate to heavy alcohol intake. CRF increases presynaptic GABA release, while antagonism of CRF1Rs decreases it. On acute alcohol consumption, this antagonist also blocks GABAnergic transmission. When pharmacological agonists of Y1Rs and Y2Rs are given into CeA, they reduce anxiety-like behaviour, while Y1R agonists have a smaller effect. In CeA, NPY inhibits and reverses acute increases in GABAnergic transmission caused by alcohol [12]. In depression and suicidal behaviour, NPY has a role. On post-mortem brains of subjects who died by suicide and normal controls. The person who died by natural death, changes in NPY at transcriptional and translational levels were investigated. We see that's the expression of NPY4R and NPY5R in brain samples of depressed people. We found a significant decrease in NPY protein and mRNA expression in both the prefrontal cortex (PFC) and the hippocampus of mental stress patients as compared to healthy controls, this implying that this decrease is linked to depression and suicide.

The NPY mRNA level in the hippocampus of distress participants was considerably lower ($p = 0.001$) than in normal control subjects, according to a univariate analysis utilising the Bonferroni post-hoc test. In depressed suicide participants, univariate comparisons of NPYR mRNA levels revealed substantial increases in NPY1R ($p = 0.004$) and NPY2R ($p = 0.007$) in depressed suicide subjects. NPY4R and NPY5R in the hippocampus indicated a trend of greater expression in DS participants compared to NC subjects, although the differences were not statistically significant. After FDR analysis, changes in NPY ($p = 0.01$), NPY1R ($p = 0.01$), and NPY2R ($p = 0.01$) mRNA also passed the level of significance [5]. The high levels of NPY expression in learning and memory-related brain regions, as well as its neuromodulators and neurotrophic effects, indicating that NPY plays an regulatory function in memory processes. As a result, it's not unexpected that a growing number of studies have found NPY to be a regulator of neuroplasticity, neuro-transmission and memory [14].

CONCLUSION: As previously stated, neuropeptides play an important role in human behaviour. As a result, we can conclude that neuropeptide regulates human behaviour, and that neuropeptide could be used for mental treatment in the future. Because of this significant advantage over neurotransmitters, like they have autolyzed capacity and are 1000 times more potent and slow acting.

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