EMOTIONS & MOTIVATIONS

- Emotion – Cognitive interpretation of subjective feelings
- Motivation – goal directed behavior
- Neuroanatomy of emotion and motives
    - Hypothalamus, limbic system, frontal lobes
- What causes behaviour?
    - Brain needs stimulation?
        - Sensory deprivation – subject is allowed only restricted sensory input
        - May display hallucinations; low tolerance
        - Hebb: after 4-8 hours ppl become distressed; rarely last 24 hours +
        - Brain has inherent need for stimulation; behaviours stimulate brain
- Drives and behaviour
    - Drives – hypothetical states of arousal that motivates an organism to engage in a particular behaviour
    - Drive theories: assume brain is storing energy for behaviour
        - Flush Model: Behaviour is continued until all the energy in its reservoir is used
        - Separate stores of energy for different behaviours
- Neural circuits and behaviour
    - Behavioural changes correlate with changes in hormones and cellular activity e.g. In men, sex drive linked to androgens
- Evolutionary theory
    - Innate releasing mechanism: mechanism that detects stimuli and directs to take a particular action
        - Brain must match stimuli to set of norms to trigger proper response; e.g. Blind babies display facial expressions, and babies respond to facial expressions
        - Can be modified with experience eg sexual stimuli
    - Behaviours have persisted through natural selection
- Chemical senses
    - Play central role in behaviours
        - Smell: Mark territory, identify group members (urine, sweat)
        - Taste: avoid poisons
- Olfaction
    - Odorants enter air; enter olfactory empithelium, contains receptor cells; chemicals dissolve in olfactory mucosa and interact with cilia; activate metabotrophic receptors and opens sodium channels and influence membrane

potential; olfactory cells project to olfactory bulb, which ends in tufts of dendrites (glomeruli) – synapses formed with mitral cells which project to areas of forebrain
    - Pathways: pyriform cortex, enterhinal cortex, amygdala, hypothalamus, thalamus and orbitofrontal cortex; no connection through thalamus from amygdala or pyriform cortex – strong emotional and memory association with smell
    - Pheromone: acts as chemosignal and can affect physio and behaviour; voneronasal organ- made of small group of receptors to detect pheromones and projects to amygdala and hypothalamus
    - Lundstrom – brain analyzes body odors differently, activate structures associated with visually emotional stimuli; smelling stranger activates area assoc with fearful stimuli
- Gustation
    - Taste receptors found in taste buds
        - 5 types: sweet, sour, salty, bitter, unami
    - Stimuli interacts with microvilli on tips of receptors to open ion channels and alter membrane potential
    - Pathway: cranial nerves 7(facial), 9(glossopharyngeal) and 10(vagus) form main solitary tract; 2 paths emerge; 1. Splits at thalamus and projects to primary somatosensory and gustatory cortex on insula, then goes to orbito cortex – responsible for flavor; 2. Hypothalamus and amygdala – affects feeding behaviour
- Environmental influences
    - Skinner: operant conditioning; experience shapes behaviour, pair stimuli and rewards ; reinforce – strengthens behaviours it follows; e.g. learned taste aversion; Garcia – coyotes stopped eating sheep because meat was poisoned and made them sick; preparedness: predisposition to respond to certain stimuli differently than others
- Neuroanatomy of motivated behaviour
    - Critical – hypothalamus and pituitary gland; sends info to brain stem circuits to produce behaviour; also limbic system; frontal lobe- critical for homeostasis – maintains critical body functions in narrow range
- Regulatory behaviours – important for survival; controlled by homeostatic mechanism, involves hypothalamus; eg. Eating, body temp
    - Hypothalamus maintains homeostasis by acting on endocrine and autonomic NS; Lateral hypothalamus – contains nuclei and nerve tracts that connect lower brainstem to forebrain ; medial forebrain bundle: tract that connects structures

in brainstem with various parts of limbic system, forms activating projections from brainstem to basal ganglia and frontal cortex
    - Hypothalamic circuit – controls pituitary gland
    - Pituitary gland: posterior pituitary: peptides released and enter bloodstream, neural tissue, continuation of hypothalamus; anterior pituitary: synthesizes various hormones, controlled by releasing hormones; releasing hormones: peptides that are released by the hypothalamus and act to manage amount of hormones from anterior pituitary
- Non regulatory – unnecessary for survival; involves frontal lobes; influenced by external stimuli; eg. Sex, parental behaviour, food preference
- Factors involved in controlling hypothalamic hormone related activity
    - Feedback loops: control amount of hormone released; controls own release
    - Neural regulation: other brain regions influence hormone release
    - Experimental responses: experience can alter structure and function of hypothalamic neurons
- Electrical stimulation of different nuclei in hypothalamus will produce goal directed behaviours eg. Digging, displaying fear, eating; requires presence of electing stimuli
- Organizing function of the limbic circuit
    - Primitive limbic cortex comprised of: cingulate gyrus, Hippocampal formation (in medial temporal region of temporal lobe, 3 layers, plays role in species specific behaviours, memory, and spatial navigation, vulnerable to stress), and parahippocampal cortex
- Amygdala: almond shaped collection of nuclei in limbic system; plays role in emotions and species specific behaviours; neurons are multimodal(respond to 1+ senses); sends projection primarily to hypothalamus and brainstem
- Executive function of frontal lobes
    - 3 main regions: motor cortex(fine movements), premotor cortex(select movement sequence), and prefrontal cortex(specific goal movement should lead to; dorsal and lateral regions); motivated by external and internal behaviour; size related to social behaviour
- Prefrontal cortex inputs
    - Receives connections from amygdala, dorsomedial thalamus, posterior pariental(sensory appreciation) cortex, and dopaminergic cells of ventral tegrental area
        - Dopaminergic input influences how prefrontal neurons react to stimuli, esp emotionally arousing stimuli
- Frontal cortex: outputs

    - Inferior region projects to amygdala an hypothalamus; influences nervous system
    - Dorsaletal region projects to posterior pariental cortex, cingulate cortex, basal ganglia and premotor cortex; influences movement and memory
- Emotion
    - 3 components: autonomic response, hypothalamus and associated structures eg. Increased heart rate; subjective feelings, amygdala and frontal lobe eg. Fear; cognitions, cerebral cortex eg thoughts about experience
- Amygdala and emotional behaviours
    - Influences conscious awareness of consequences of events and objects via connections with prefrontal cortex
    - Kluver Bucy Syndrome: characterized by hypoersexuality, results from bilateral injury to temporal lobe
- James – Lange Theory: brain concocts story to explain bodily reactions
    - Somatic marker hypotheses: posits that marker signals arising from emotions and feelings act to guide behaviour and decision making, usually unconsciously
- Prefrontal cortex and emotional behaviours
    - Damage has severe effects; cant understand or use emotions or facial expressions, disorganized, loss of drive, cant plan
- Eating
    - Lipids, amino acids, glucose extracted
    - Aphagia: failure to eat, follows lesions to lateral hypothalamus; hyperahagia: overeat, follows lesions to ventromedial hypothalamus or paraventricular nuclus of hypothalamus
    - Amygdala – projects to hypothalamus, damage alters food preferences and abolishes taste aversion
    - Inferior prefrontal cortex – receives input from olfactory bulb, damage diminished sensory responses to food = decreased eating
- Drinking
    - 2 kids of thirst: 1. Osmatic – want to restore ideal solute, receptors in hypothalamus detect changes; 2. Hypovolemic – loss of fluid volume, restore nutrients, different hypothalamic circuit
- Sex hormones
    - Estrodial masculinizes brain; aromatase converts testosterone to estradiol; in females, enzyme alpha fetoprotein converts estrogen from entering neurons
    - Ventromedial hypothalamus controls female mating posture; preoptic area of medial hypothalamus controls copopulatory behaviour in males; amygdala controls sexual motivation

    - Sexual identity and orientation: compared to hetero females, hetero males have 2X neurons in preoptic area, 2-5X longer bed nucleus of stria terminals, $INAH_2$ region is 2X bigger, suprachiasmic nucleus contains 2X neurons
        - Hypothalamus of homo males differences from male and female brain
        - Genetic basis? Hamir – increased incidence of male homo on maternal side of men's families; potential involvement of X chromosome; but genes specify proteins not behaviour
    - Cortex male have role; not understood; damage to frontal lobes can result in loss of inhibition of sexual behaviours or libido
- Motivation and reward
    - Reward may be mechanism that evolved to increase adaptive fitness
    - Olds and Milner – rats press bar to stimulate sites of brain eg. Lateral hypothalamus and medial forebrain bundle epec effective
    - Mesolimbic system: dopamine released during intercranial self-stimulation
    - Wanting (incentive) and Liking (evaluation of pleasure) theory: usually, but not always, occur together; both have susceptible neural circuits (Robinson and Berrridge); wanting involves dopamine, liking involves GABA systems (opoid and benzodiazepine)

LEARNING & MEMORY

- Learning: a change in an organisms behaviour as a result of experience
- Memory: ability to recall or recognize previous experience
- Memory trace: a mental rep of a previous experience
- Pavlovian conditioning aka classical or respondent conditioning: learning procedure whereby a neutral stimulus comes to elicit a response because of repeated pairing; stimulus + response
    - Eye blink conditioning: pair puff of air with tone; blink in response to tone
    - Fear condition: learn to associate stimulus with noxious event (eg shock); involves amygdala
- Instrumental (operant) conditioning: learned procedure in which consequences of a particular behaviour increase or decrease probability of behaviour occurring again
    - Thorndikes puzzle box
- Reinforcement: organisms tend to repeat responses that are followed by favourable consequences; + reinforcement: rewarding stimulus; - reinforcement: remove aversive stimulus
- Implicit (procedural) memory: unconscious; can demonstrate knowledge (eg skill) on prompting, but can explicitly retrieve info; processed bottom up – sensory systems to cortex; passive role

    - Proposed circuit – Mishkin *KNOW DIAGRAM*
        - Basal ganglia, ventral thalamus, substantia niagra (use dopamine systems), premotor cortex
        - Have a lot of inputs, not a lot of connections going back
        - Unconscious b/c connections between basal ganglia and cortex are unidirectional; must be feedback to cortex to be conscious
- Explicit (declarative) memory: conscious; can retrieve item and indicate that they know retrieved item is correct item; processed top down; active role
    - Neural circuit: prime structures – medial temporal region (hippocampus, amygdala, entorhinal cortex, parahippocampal cortex, perihinal cortex), also frontal lobe; parahippocampal cortex believed to be involved in visuospatial processing; parahinal cortex: receives connections from visual regions of ventral streams believed to be involved in visual object memory
    - Reciprocal connections: neocortex projects to enterhinal cortex. Which projects back to neocortex; keeps sensory regions alive in brain; pathway back to neocortex means it is kept appraised of info being processed in medial temporal region
    - Proposed neural circuit – Mishkin *KNOW DIAGRAM*
        - Temporal lobe structures (LTM), frontal lobe structures (important for STM and order of events), medial thalamus, basa forebrain (activating systems, keeps activity in forebrain high to pay attention)
        - Medial temporal lobe project back to cortex
- Priming: using a stimulus to sensitize NS to a later presentation of same or similar stimulus; often used to measure implicit memory
- Processing Memories
    - STM: few mins, involved frontal lobe; LTM: indefinite duration, involves temporal lobe
- Info from each sensory modality is processed and stored in different areas
    - Martin: subjects show black and white drawings; asked to generate words describing either colours or actions of drawings; recalling colours activated neural temporal lobe; recalling actions activated middle of temporal gyrus
- Episodic memory: autobio memory for events; episodic amnesia: inability to recall any personally experienced events, associated with frontal lobe injuries of decreased blood flow
- Patient HM: following surgery by Scotville for severe epilepsy; lost explicit memory (amnesia)
- Patient JK: impaired implicit memory; developed memory problems at old age; damage to basal ganglia; couldn't perform tasks he had done all his life

- Hippocampus and spatial memory
    - Monkeys with hippocampal lesions have difficulties with visuospatial learning; hippocampal function in food storing animals is larger
- Frontal lobe and STM
    - All sensory systems project to frontal lobe; monkeys who must keep task in STM over a delay have firing in frontal lobe throughout delay
- Korsakoffs syndrome: lack of vitamin B1 (thiamine) from malnutrition and alcohol consumption (compromises B1 absorption; causes neurotoxicity; leads to amnesia (retrograde and antegrade); memory disturbances display confabulation to compensate for memory loss; mammillary body atrophy – dies, also damage to medial thalamus and frontal lobes
- Neural circuit for emotional memories
    - Emotional memory – memory for affective properties of stimuli or events
    - Damage to amygdala abolishes emotional memory; little effect on implicit or explicit; also Kluver Bucy
    - Circuitry: amygdala, medial temporal cortex (EM), brainstem (autonomic arousal), hypothalamus (hormones), periaqueductal gray matter (pain), basal ganglia (IM)
    - Important for survival and learning
- Structural basis of brain plasticity
    - Changes correspond to behavioural change
    - Memory – changes at synapse
    - 2 research approaches: 1. Synaptic changes in NS of simple organism 2. Mammalian brain
    - Measuring synaptic change: 1.modifying existing circuit – neurons change structure in response to experiences, changes # of dendrites 2. Creating new circuits – neurogenesis may occur in olfactory bulb, hippocampal formation, and possibly neocortex
- Rats raised in enriched enclosures had heavier brains, more dendrites, astrocytes, blocked capillaries, synapses per neuron, and increased micochondrial volume (marker of increase metabolic activity)
- Change and Greenough – places patches over 1 eye of rats so contralateral hemisphere was deprived of visual stimuli; put in maze; side connect to visible eye had more dendrites
- Nudo – monkeys took food from big or small well; monkeys with small well had larger digit rep on motor cortex

- Ramachandron – measured cortical maps in those with limb amputations; stroked face with cotton swab softly, reported sensations in amputated hand; may explain phantom limb pain
- Wernicke's area has more dendritic branching with more education
    - Schiebel – life experience alters complexity of dendritic branching and morphology; differences in finger vs. chest in neurons based on how much fingers used
- Hormones and plastic
    - Increased estrogen = more dendritic spines in hippocampus; decreased estrogen = more dendritic spines in neocortex, less in hippocampus (memory decline); lower levels of testosterone = more dendritic spines in neocortex (low spatial ability)
    - Glucocorticoids – released from adrenal cortex in times of stress; assist in metabolism of proteins and carbohydrates and sugar levels in blood; steady levels over prolonged stress can be neurotoxic
- Neurotropic factors and plasticity
    - Nerve Growth factor: stimulates neurons to grow dendrites and synapses and in some cases survival of neurons; brain derived neurotropic factor: may enhance plastic changes such as growth of dendrites and synapses
- Psychoactive drugs and plasticity
    - Drug induced behavioural sensitization: escalating behavioural response to repeated admin of a psychomotor stimulant; associated with an increase in number of receptors, synapses, and dendrites; changes related to regions that receives large dopamine projection
- Recovery from brain injury
    - Learn new ways to solve problems: 3 legged cat solution; learn to compensate
    - reorganize brain to do more with less: brain can form new connections and do more with less; recovery increased if person also engages in form of intervention (behavioural therapy, pharm therapy)
    - generate new neurons to produce new circuit: fetal tissue implantation: more suited for when a small # of cells are needed; replace lost cells - epidermal growth factor: NTF that stimulates subventricular zone to generate cells that migrate into striatum and eventually differentiate

BRAIN ABNORMAILIES

- Causes of abnormal behaviour
    - Evidence for brain abnormalities in organic neurological disease is straight forward and causes are generally known

        - Genetic errors (HD), progressive cell death (AD), rapid cell death (stroke), loss of neural connections (MS)
    - Etiology – cause: far less is known about causes of behavioural psychiatric disorders
- Pheylketonuria (PKU): behavioural disorder caused by elevated levels of amino acid phenylakone in blood; results from defect in gene for enzyme phenylalanine hydroxylaise; mental retardation
- Challenges to diagnosis
    - Difficult to diagnose behavioural disorders ; ppl are seldom objective observers of own behaviour or that of loved one; may be selective in what they notice; ppl are seldom specific in identifying symptoms: evaluations have own conceptual basis, which may influence questions they ask and info they gather; brains plasticity can cover up symptoms
- Investigating neurobio of behavioural disorder
    - Heningter – no clear evidence of single receptor system with a specific relation to a specific behaviour
    - Animal models are useful, but provide oversimplified view
- identifying and classifying mental disorders
    - epidemiology: study of distribution and causes of diseases in human pops
    - DSM: diagnostic and statistical manual of mental disorders
    - Psychiatric disorders are to an extent arbitrary and depend on prevailing cultural views
- Use of brain imaging
    - Tools must be sensitive enough to detect unique features and specific enough to rule out similar conditions
- Treatment for disorders
    - Neurosurgical treatments: either damage or stimulate some dysfunctional area of brain; DBS: electrodes implanted in brain stimulate targeted area with electrical current to facilitate behaviour (eg PD); stem cell therapy: return dysfunctional brain region to embryonic state and regrow normal region, mulitpotent cells proving more effective source
    - Electrophysio treatments: electroconvulsive therapy: use electrical current to produce seizures to treat depression, stimulates production of NTF, must medicate person to avoid effects cause by electrical stimulation, can lead to memory loss which can show cumulative effects; transcranial magnetic stimulation: less drastic the ECT with fewer side effects, can be applied narrowly rather than diffusely

    - Pharm Treatments: neuroleptics, antianxiety agents, SSRIs, L Dopa; behavioural disorders can be reduced to single chemical abnormality; pill is not a skill; can have side effects (eg. Taroine Dyskinesia: inability to stop tongue from moving, motor side effect of neuroleptics, can last long period)
    - Behavioural treatments: psychotherapy; behavioural therapy: applies learning principles to eliminate unwanted behaviours; cognitive therapy: change maladaptive patterns of thinking
    - Often useful to combine treatments
- Traumatic brain injury
    - 2 important factors: age and sex; can disrupt brains blood supply, induce bleeding, cause swelling, expose brain to infection, and scar brain tissue
    - 2 behavioural effects: Specific impairment may be result of coup(site of impact) and countercoup (opposite side) lesion; more generalized impairments may be due to widespread damage throughout brain
    - often misdiagnosed; no abnormal MRI or CT scan; most cognitive recovery occurs in first 6-9 months; cognitive abilities tend to make a good recovery, but social skills and normal personality typically don't show significant change
- stroke: interruption of blood flow either from blockage of blood vessel or from bleeding from a vessel: ischemia: lack of blood to brain as a result of stroke – sets off a cascade of cellular events that causes damage to initial site and surrounding areas; diaschisis: natural shock that follows damage in areas connected site of damage show temporary arrest in function
    - treatment: admin anti-clot drug (e.g. t-PA-tissue plasminogen activator), must be given within 3 hours of stroke; neuroprotectant: drug used to try and block cascade of post stroke neural events (no good ones yet); therapies (speech or physical) are often used to facilitate plastic changes in the brain; constraint induced therapy: force patient to use impaired limb
- epilepsy: recurrent seizures accompanied by loss of consciousness; symptomatic seizure: identified with a specific cause (e.g. Infection, trauma, vascular formation, toxic chemicals, very high fever, neuro disorders); idiopathic seizure: appears spontaneously and in absence of other disease of CNS, variety of factors may precipitate seizure e.g. stress, fatigue, alcohol
    - 3 common symptoms: an aura, or warning of impending seizure; loss of consciousness often followed by perceived amnesia that includes seizure; a motor component that can vary from shaking to autonomic movements such as hand rubbing or chewing
    - Focal seizures: begin locally and at focus, and then spreads out to adjacent areas, Jacksonian March; Complex partial seizure: type of focal seizure, originate mostly

in temporal lobe, characterized by subjective experiences that presage attack, automatisms or repetitive stereotypical movements, postural changes, not necessarily accompanied by loss of consciousness; generalized seizures: lack of focal onset. Occur on both sides of the body; grand mal seizure: characterized by loss of consciousness and stereotyped motor activity, 3 stages: tonic stage (body stiffens and breathing stops), clonic stage (rhythmic shaking); postictal depression: post seizure state of confusion; petite mal: of brief duration, characterized by loss of awareness with no motor activity except for blinking, turning the head, or rolling the eyes

    - Treatment: anti convulsant medication: eg. Diphenylhydantain or phenobarbital, work by stabilizing neuronal membrane, esp inhibitory neurons, eg stabilize GABA; surgical removal of abnormal tissue that is focus of seizures is some times performed in severe cases

- Multiple Sclerosis: characterized by loss of myelin, largely in motor tracts but also sensory nerves; relapses and remissions are common; brain imaging reveals discrete lesions; cause is unknown, but proposed causes include bacterial infection, a virus, environmental factors (e.g. pesticides), and an immune response if the CNS
- Dementia: acquired and persistent syndrome of intellectual impairment; diagnostic features (DSM): memory and other cognitive deficits, impairment in social and occupational functions; 2 broad categories: nondegerative: heterogeneous group of disorders with diverse etiology's e.g. Vascular dementia, karsacofs syndrome; degenerative: presumed to have a degree of genetic transmission e.g. HD, AD
- Parkinson's Disease: related to degeneration of substantia nigra and to the loss of NT dopamine; despite a common site of damage, symptoms vary enormously, resemble regular signs of aging; progressive disorder
    - + symptoms: increased incidence, symptoms appear; tremor at rest, muscular ridgity (increased muscle tone), involuntary movements (akathesia – small involuntary movements or changes in posture, restlessness, oculogyric crisis – involuntary turns of head and eyes to one side)
    - – symptoms: behaviours you lose; disorders of posture (fixation and equilibrium), disorders of righting (e.g., standing), disorders of locomotion (festination: tendency to engage in a behaviour, such as walking, at faster and faster speeds), speech disturbances (e.g. Loss of prosody), akinesia (slowness of movement)
    - Also cognitive symptoms: in improvishment of feeling, libido, motive, and attention; cognitive slowing

    - Causes: loss of cells in substantia nigra, which may be caused by diseases such as encephalitis, syphilis, drugs (e.g. MPTP), or unknown causes; environmental pollutants? Insecticides?
    - Treatment: treatment is symptomatic; important how patient copes behavioural e.g. Physical therapy is often helpful; pharm: increase levels of dopamine (e.g. Dopamine agonists such as L Dopa), supress activity of structures that show heightened activity in absence of adequate dopamine action (e.g. Anticholinergic drugs), become less evective as disease progresses; surgical: electrical stimulation or lesioning of internal global pallidus decrease rigidty and tremor; transplantation: transplant embryonic dopamine producing cells or multipotential stem cells into basal ganglia
- AD: approx. 65% of dementia cases; cause is unknown, but proposed causes include: genetic predisposition, environmental toxins (eg, aluminum), autoimmune response, slow acting virus, decreased blood flow to hemispheres
    - Anatomical correlates: neuritic (amyloid) plaques: located mostly in cerebral cortex, precursor to make proteins, also found in non AD patients; Neurofibrillary tangles: paired helical filaments found in cerebral cortex and hippocampus, also found in down syndrome, PD, and other dementias; cortical degeneration: most affected areas are limbic system, influence temporal cortex, and posterior premotor cortex; the entorhinal cortex (link between neotcortex and hippocampus) shows clearest evidence for cell loss, which may explain why memory problems occur early in disease; primary sensory and motor areas are spared; cerebral atrophy may be due in large part to loss of dendrite arborisation; loss of NT (AcH, noradrenaline, dopamine, serotonin, NMDA, AMPA glutamate receptors)
- Understanding and treating psychiatric disorders
    - 3 categories in DSM: psychiatric disorders, mood disorders, anxiety disorders
- Schizophrenia: 6 diagnostic symptoms: delusions, hallucinations, disorganized speech and behaviour, catatonic behaviour, negative symptoms (blunted emotions)
    - Type 1: + symptoms (hallucinations, agitated movements, delusions); likely due to dopaminergic dysfunction; assoc. with acute onset, good prognosis, or a favourable response to neuroleptics; Type 2: - symptoms (cataonia, - affect), enlarged ventricles and cortical atrophy, particularly in the frontal cortex, assoc. with chronic affliction, poor prognosis, poor response to neuroleptics, cognitive impairments; 20-30% of patients show mixed
    - Neuroanatomical correlates: memory deficits; large ventricles and thinner cortex in medial temporal regions and abnormal dendritic fields in dorsal prefrontal area, hippocampus and entorhinal cortex; deficits in executive functioning,

abnormal blood flow in dorsolateral prefrontal cortex; auditory hallucinations: abnormalities in auditory regions of temporal lobes; thought disorders: abnormalities in Wernicke's area; dopamine: are one of the first NT to be implicated in schizophrenia, neuroleptics are dopamine antagonists, dopamine agonists can produce psychotic symptoms; dopamine theory appears too simple; many other NC also associated

- Mood disorder
    - Depression, mania, bipolar
    - Depression: AD drugs act to increase levels of norepinephrine and serotonin; there is low evidence that low levels of these NT cause depression, low levels don't in normal ppl, AD alters levels of these NT within days, but take weeks to start relieving depression
    - Dumon – suggest depression may involve low levels of NTF; BDNTF is down regulated by stress and up regulated by AD; AD may increase levels of BDNF altering intracellular signals
    - Stress and Depression: HPA Axis: hypothalamic-pituitary-adrenal circuit that controls the production and release of hormones related to stress; chronic stress can lead to over secretion of cortisol, assoc. with depression in adulthood; 45% of adults with depression lasting 2+ years experienced abuse, neglect or parental loss as children
    - Treatment: CBT: problem focused, action oriented, structured treatment for eliminating dysfunctional thoughts and maladaptive behaviour; at least as effect as medication
- Anxiety Disorders: 6 different types: generalized AD, panic disorders, PTSD, social phobia, specific phobias, OCD
    - Neural correlates: brain imaging studies: increased activation in cingulate cortex and parahippocampal gyrus; enhanced response to anxiety provoking stimuli in amygdala and prefrontal cortex; excessive excitatory NT may enhance anxiety, GABAergic drugs are effective at reducing anxiety; AD may be caused by stressful experiences early in life
    - Treatment: pharm: benzodiazepines were once primary treatment for AD, but SSRIs are now common; CBT: focuses on challenging reality of patients obsessions and behavioural necessity for their compulsions

SLEEP AND DREAMING

- Biorhythm: inherent timing mechanism that controls or initiates various bio processes; linked to cycle of days and seasons produced by Earths rotation around sun; animals living near poles are more affected by seasonal changes

- Bio clocks: neural system that time behaviour; behaviour not simply driven by external cues; rhythms are endogenous (in body)
- Bio rhythms: period: time required to complete a cycle of activity; circonnual rhythm: yearly (migratory cycles); infradian rhythm: less than a year (menstrual cycle); circadian rhythm: daily (sleep cycle): ultradian rhythm: less than a day (eating cycle)
- Free running: rhythm of body's own devising in the absence of all external cues; can depend on what external cues are removed
- Zeitgebers: environmental events that entrains bio rhythms; time giver; e.g. light resets bio clock; entrainment: determination or mod of period of a biorhythm; jet lag: fatigue and disorientation from a rapid travel through time zones and exposure to a different light dark cycle
- Neural basis of bio clock: suprachiasmatic nucleus (SCN): main pace maker of circadian rhythms located just above optic chiasm, acts as bio clock; retinohypothalamic pathway: neural route from a subset of cone receptors in the retina to the SCN of the hypothalamus, allows light to entrain the rhythmic activity of SCN
- Immortal time: endogenous rhythm not learn
    - Martin – transplantation studies in hamsters; after lesions to SCN, eat and sleep normal amount but rhythmic nature of these behaviours disappears; if SCN cells from embryos are transplanted into the lesioned animal, they will establish circadian rhythms
- Neural basis bio clock: at least half a dozen genes and proteins seem to produce circadian rhythm of SCN cells; not understood
- Model for circadian timing system: light entrains SCN pacemaker; SCN pacemaker devises a number of slave oscillators, each of which controls rhythmic occurrence of one behaviour; SCN pacemaker may drive the slave oscillators via hormones, proteins or NT
- Circannual rhythms: melatonin: hormone secreted by pineal gland during night cycle; influences daily and seasonal biorhythms; e.g. Hamsters: smaller melatonin levels and gonads during winter, larger gonads and increased melatonin during summer
- Measuring sleep in lab: polygraph used to measure electrical activity of brain and body; electroencephalogram (EEG): record brain wave activity; electromyogram (EMG): record of muscle activity; electroculogram (EOG): record of eye movements
- Stages of waking and sleeping
    - Waking State: small amplitude waves with fast frequency; beta rhythm
    - Drowsy: amplitude of EEG increases, lower frequency; alpha rhythm
    - Sleeping: slower, larger EEG waves; delta waves; assoc. with NREM – slow wave sleep: 4 stages of NREM (1-shallow, 4-deep); pass through stages through night; each stage about 90 mins; dreaming not as vivid; possibly sleepwalking, talking, night terrors; large range of activates take place (e.g. Lower body temp)

    - Dreaming: assoc. with REM sleep – fast brain wave pattern displayed by neocortical EEG record during sleep; atonia: no tone, complete muscle inactive produced by inhibition of motor neurons; mechanism that regulate body temp stop working; vivid dreams
- Dreams
    - Psychoanalytic theories: Freud – dreams are symbolic fulfillment of unconscious wishes, manifest content: loosely connected series of bizarre images and actions, latent content: true meaning of dreams; Jung – dreams are expressions of our collective unconscious = history of human race
    - Hobson: Activation-Synthesis Theory: cortex is bombarded with signals from brainstem, and in response cortex generates images, actions and emotions from personal memory store, dreams are personal but have no meaning; dreamer attempts to create story from fragments
    - Revonsuo: evolutionary hypothesis: dreams are biologically important b/c they lead to enhanced performance in dealing with threatening life events; dreams are coping strategies; dreams more threatening images
- Purpose of Sleep
    - Early explanation: sleep is a passive process that takes place as a result of decreased sensory stimulation
    - Sleep as adaptation: sleep is an energy conserving strategy – save energy when not hunting; animals with nutrient rich diets sleep more, e.g. predators; nocturnal or diurnal animals sleep when they can't travel easily
        - Basic rest activity cycle: recurring cycle of temporal packets, about 90 mins, during which animals level of arousal waxes and wanes; e.g. meals, sleep cycles
    - Sleep as restorative process: chemical events that provide energy to cells may be decreased during waking and are replenished during sleep
- Sleep Deprivation: long periods without sleep produce physio and behavioural problems, grow worse over time; Randy Gordner – stayed awake for 264 hours (11 days) for a science fair project, cognitive capabilities declined, did not regain lost sleep; microsleeps: 2-3 sec, lids droop, less responsive; partial deprivation or sleep restriction has 3 consistent effects: increased sleepiness, disturbances in mood, perform poorly on tests of vigilance, impaired attention, reactive, coordination and decision making
    - REM sleep deprivation: little effect on daytime function; with each successive night of deprivation, increased tendency to initiate REM sequences, rebound effect, have more REM for few nights
- Sleep and memory storage: sleep plays a role in solidifying and organizing events in memory; place cell: hippocampal neuron that fires when a rat is in a certain location in

an environment; Wilson & McNaughten – groups of place cells that fired during a food search task also fired during subsequent sleep period; NREM important for memory storage; used PET imaging to record brain activity while humans performed a serial reaction time task, PET imaging during subsequent sleep revealed that the same brain regions that were active during task were also active during REM

- Reticular Activating System: large reticulum (mixture of cell nuclei and nerve fibers) that runs through center of brain stem; assoc. with sleep/wake behaviour and arousal; stimulation of RAS produces waking EEG; damage produces slow wave EEG
- Coma: prolonged state of unconsciousness resembling sleep; can result from brainstorm damage (including RAS)
- Neural basis of EEG changes assoc. with waking: 2 brainstem systems influences waking: basal forebrain – contain cholinergic cells that secrete AcH into neocortex neurons that stimulate EEG (beta rhythm), animal is still alert; median rople nucleus (midbrain): contains serotonin nucleus that project diffusely to neocortex, also stimulate beta rhythms, animal is alert and moving
- Neural basis for REM sleep: peribrachial area: cholinergic nucleus In the dorsal brainstem having a role in REM sleep behaviours; projects to the medial pontine reticulum; initiates REM sleep; Medial pontine reticular formation: nucleus in pons participating in REM sleep; projects to several other brain areas that produce REM related behaviours; e.g. Produce atonia of REM sleep
- Disorders of NREM sleep: insomnia: disorder of slow wave sleep resulting in prolonged inability to sleep, multiple causes; narcolepsy: SWSD, person uncontrollably falls asleep, may be due to mutations in gene that produces hypocretin/onexin peptide; sleep apnea: stop breathing while sleeping
- Disorders of REM sleep: sleep paralysis: inability to move during sleep owing to brains inhibition of motor neurons; cataplexy: from of narcolepsy linked to strong emotional stimulation in which an animal loses all muscle tone as if in REM sleep, while awake; hypnogogic hallucination: dreamlike event at beginning of sleep or while a person is in state of cataplexy