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14th & 15th October 2006*



DEPARTMENT OF ANATOMY

ORAL PRESENTATION

Abstract – Anat - 01

Eagle's syndrome

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Eagle syndrome or styloid–carotid artery syndrome is a rare condition where an elongated temporal styloid process (more than 30mm) is in conflict with the adjacent anatomical structures. Two forms of eagle syndrome exists: the classic form and the vascular one. The styloid process is a slender outgrowth at the base of the temporal bone, immediately posterior to the mastoid apex. According to Balcioglu (2009), the mean length of the styloid processes of the subjects reporting Eagle syndrome is reported to be 40 +/- 4.72 mm. Patients with this syndrome tend to be between 30 and 50 years of age but it has been recorded in teenagers and in patients > 75 years old. It is more common in women with a male: female ratio ~ 1:2. Patients with the classic "Eagle Syndrome" can present with unilateral sore throat, dysphagia, tinnitus, unilateral facial and neck pain, and otalgia. In patients with the vascular form of "Eagle syndrome", the elongated styloid process is in contact with the extracranial internal carotid artery. This can cause a compression (while turning the head) or a dissection of the carotid artery causing a transient ischemic event or a stroke. Symptoms tend to be worsened on bimanual palpation of the styloid through the tonsillar bed. They may be relieved by infiltration of lidocaine into the tonsillar bed. Because of the proximity of several large vascular structures in this area this procedure is not be considered to be risk free.



Abstract – Anat - 02

Mandibular fractures & reconstruction

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Mandibular fracture, also known as fractures of the jaw, are breaks through the mandibular bone. They usually occur due to trauma and are often associated with other facial trauma. The types of mandibular fractures include fractures at the symphyseal area, horizontal ramus, mandibular angle and condylar neck. Mandibular fractures are also classified according to categories that describe the condition of the bone fragments at the fracture site and also the presence of communication with the external environment as Greenstick, Simple, Comminuted, Compound and involvement of dentition. By far, the two most common symptoms described are pain and the feeling that teeth no longer correctly meet (traumatic malocclusion, or disocclusion). Like all fractures, consideration has to be given to other illnesses that might jeopardize the patient, then to reduction and fixation of the fracture itself. Except in avulsive type injuries, or those where there might be airway compromise, a several day delay in the treatment of mandible fractures seems to have little impact on the outcome or complication rates. Since mandible fractures are usually the result of blunt force trauma to the head and face, other injuries need to be considered before the mandible fracture. First and foremost is compromise of the airway. While rare, bilateral mandible fractures that are unstable can cause the tongue to fall back and block the airway. Fractures such as a symphyseal or bilateral parasymphiseal may lead to mobility of the central portion of the mandible where genioglossus attaches, and allow the tongue to fall backwards and block the airway in larger fractures, or those from high velocity injuries, soft tissue swelling can block the airway. In addition to the potential for airway compromise, the force delivered to break the jaw can be great enough to either fracture the cervical spine or cause intra-cranial injury (head injury). It is common for both to be assessed with facial fractures. Finally, vascular injury can result (with particular attention to the internal carotid and jugular) from high velocity injuries or severely displaced mandible fractures. Loss of consciousness combined with aspiration of tooth fragments, blood and possibly dentures mean that the airway may be threatened.



Abstract – Anat - 03

Metopic Suture

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The metopic suture (also known as the median frontal suture) is a type of calvarial suture. This suture runs through the midline across the frontal bone from the nasion to the bregma, although it may often be incomplete. It usually fuses by around 9 months of age. A premature fusion of the suture is termed metopic synostosis (type of craniosynostosis) which can then result in trigonocephaly. It is present in a fetal skull so that the skull can bend and is very elastic at the time of birth. The baby's head bends when coming out of the mother's womb. The space is filled as the child grows older. In some individuals the suture can persist (totally or partly) into adulthood, and in these cases it is referred to as a persistent metopic suture. The suture can either bisect the frontal bone and run from nasion to bregma or persist as a partial metopic suture (where part of the suture survives and is connected to either bregma or nasion) or as an isolated metopic fissure. Persistent frontal sutures are of no clinical significance, although they can be mistaken for cranial fractures. As persistent frontal sutures are visible in radiographs, they can be useful for the forensic identification of human skeletal remains. Persistent frontal sutures should not be confused with supranasal sutures (a small zig-zag shaped suture located at and/or immediately superior to the glabella).



Abstract – Anat - 04

Development of Mandible

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The mandible, the largest and strongest bone of the face, serves for the reception of the lower teeth. It consists of a curved, horizontal portion, the body, and two perpendicular portions, the rami, which unite with the ends of the body nearly at right angles. The ossification of the mandible refers to the Human mandible laying down new bone material in the fibrous membrane covering the outer surfaces of Meckel's cartilages. These cartilages form the cartilaginous bar of the mandibular arch, and are two in number, a right and a left. Their proximal or cranial ends are connected with the ear capsules, and their distal extremities are joined to one another at the symphysis by mesodermal tissue. They run forward immediately below the condyles and then, bending downward, lie in a groove near the lower border of the bone; in front of the canine tooth they incline upward to the symphysis. From the proximal end of each cartilage the malleus and incus, two of the bones of the middle ear, are developed; the next succeeding portion, as far as the lingula, is replaced by fibrous tissue, which persists to form the sphenomandibular ligament. Between the lingula and the canine tooth the cartilage disappears, while the portion of it below and behind the incisor teeth becomes ossified and incorporated with this part of the mandible. Ossification takes place in the membrane covering the outer surface of the ventral end of Meckel's cartilage, and each half of the bone is formed from a single center which appears, near the mental foramen, about the sixth week of fetal life. By the tenth week the portion of Meckel's cartilage which lies below and behind the incisor teeth is surrounded and invaded by the membrane bone. Somewhat later, accessory nuclei of cartilage make their appearance: a wedge-shaped nucleus in the condyloid process and extending downward through the ramus; a small strip along the anterior border of the coronoid process; smaller nuclei in the front part of both alveolar walls and along the front of the lower border of the bone. These accessory nuclei possess no separate ossific centers, but are invaded by the surrounding membrane bone and undergo absorption. The inner alveolar border, usually described as arising from a separate ossific center (splenial center), is formed in the human mandible by an ingrowth from the main mass of the bone. At birth the bone consists of two parts, united by a fibrous symphysis, in which ossification takes place during the first year.



Abstract – Anat - 05

Stem Cell Challenges

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Stem cell therapy encompasses new technologies and therapies that aim to replace damaged cells with healthy new ones. Cells may be dysfunctional due to any number of reasons such as genetics, disease, injury or aging. Currently, stem cells offer the potential to treat cancer, Parkinson's disease, spinal cord injuries and diabetes, among other serious diseases. Unfortunately, there are several challenges faced by researchers that must be overcome before stem cell therapies can become a successful reality for those suffering from disease. Researchers do expect to eventually move beyond these challenges but the unfortunate reality is that those suffering from disease often have little time to wait for treatment. A major difficulty that scientists continue to encounter during their research is the identification of stem cells in adult tissues. These tissues contain many different types of cells and an attempt to locate the often scarce numbers of stem cells in tissues that could contain thousands of different cells is difficult at best. The research involved is complex and even after cells are isolated, the process to successfully trigger differentiation into the desired cell type is another challenge for researchers. This requires an understanding of stem cell control and regulation that has yet to be fully gained. In addition, researchers must also use the correct laboratory medium, or solution, to coax the growth and this has proven to be difficult. The first challenge researchers face when considering stem cell treatment is to understand the mechanisms by which stem cells function in the injured microenvironment using animal models, and then to translate the results of these studies to humans. Another challenge is how to identify and isolate stem cells from tissue, and then induce their differentiation into the desired cell types. The third challenge is how to prevent immunorejection after stem cell transplantation. Immunological rejection is a major barrier to successful stem cell transplantation. A person's immune system may also recognize the transplanted cells as foreign bodies and this can trigger an immune reaction that results in the rejection of the transplanted cells. Recipients of the transplant usually have to take strong immunosuppressive drugs to reduce the chances of rejection but these drugs induce infection by viruses or microbes in the environment



Abstract – Anat - 06

Cardiac Pain in Mandibular Region

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Pain referred to the orofacial structures can sometimes be a diagnostic challenge for the clinician. In some instances, a patient may complain of tooth pain that is completely unrelated to any dental source. This poses a diagnostic and therapeutic problem for the dentist. Cardiac pain most commonly radiates to the left arm, shoulder, neck, and face. In rare instances, angina pectoris may present as dental pain. When this occurs, an improper diagnosis frequently leads to unnecessary dental treatment or, more significantly, a delay of proper treatment. This delay may result in the patient experiencing an acute myocardial infarction. It is the dentist's responsibility to establish a proper diagnosis so that the treatment will be directed toward the source of pain and not to the site of pain. One of the most important sources of pain referral to the jaws comes from symptoms generated during attacks of angina in ischemic heart disease. Typically, pain or other unpleasant symptoms develop in the jaws during actual ischemic episodes with remission of pain when the crisis is over. Symptoms are usually felt in the left body of the mandible or left ramus, but pain may also occur within the mandibular teeth on the left side. It has generally been suggested that the pain is located at the angle of the mandible, but pain can occur over the entire left side of mandible. Craniofacial pain commonly is induced by cardiac ischemia. This must be considered in differential diagnosis of toothache and orofacial pain. Because patients who have AMI without chest pain run a higher risk of experiencing a missed diagnosis and death, the dentist's awareness of this symptomatology can be crucial for early diagnosis and timely treatment.



Abstract – Anat - 07

Disappearance of sutures

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An infant's skull is made up of six separate cranial bones: Frontal bone, Occipital bone, Two parietal bones, Two temporal bones. These bones are held together by strong, fibrous, elastic tissues called sutures. The spaces between the bones where the sutures are called fontanelles. Sometimes, they are called soft spots. These spaces are a part of normal development. The cranial bones remain separate for about 12-18 months. They then grow together as part of normal growth. They stay connected throughout adulthood. Two fontanelles usually appear on a newborn's skull. On the top of the middle head, just forward of center (anterior fontanelle). In the back of the middle of the head (posterior fontanelle). The posterior fontanelle usually closes by age 1 or 2 months. It may already be closed at birth. The anterior fontanelle usually closes sometime between 9 months and 18 months. The sutures and fontanelles are needed for the infant's brain growth and development. During childbirth, the flexibility of the sutures allows the bones to overlap so the baby's head can pass through the birth canal without pressing on and damaging his or her brain. During infancy and childhood, the sutures are flexible. This allows the brain to grow quickly and protects the brain from minor impacts to the head (such as when the infant is learning to hold his head up, roll over, and sit up). Without flexible sutures and fontanelles, the child's brain could not grow enough. The child would develop brain damage. Feeling the cranial sutures and fontanelles is one way that doctors and nurses follow the child's growth and development. They are able to assess the pressure inside the brain by feeling the tension of the fontanelles. The fontanelles should feel flat and firm. Bulging fontanelles may be a sign of increased pressure within the brain. In this case, doctors may need to use imaging techniques such as CT scan or MRI scan. Surgery may be needed to relieve the increased pressure. Sunken, depressed fontanelles are sometimes a sign of dehydration.



Abstract – Anat - 08

Congenital Midline Evaluation Of Nasal Masses

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The nasal encephalocele, the glioma, and the dermoid are the most common of the congenital midline nasal masses. Due to similar embryologic development, each of these lesions may be associated with bony cranial defects and intracranial abnormalities, as well as CSF leakage and the potential for fatal meningitis if not handled properly. Preoperative manipulation should be avoided. Radiologic studies are instructive only if they are positive. If intracranial attachments are identified radiologically or suspected clinically, neurosurgical consultation should be obtained, and intracranial exploration and resection should be carried out as the initial procedure. Extracranial resection of the remaining mass may be performed immediately after intracranial resection, may be postponed, or may become unnecessary.



Abstract – Anat - 09

Trigeminal neuralgia

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Trigeminal neuralgia, is a neuropathic disorder of the trigeminal nerve that causes episodes of intense pain in the eyes, lips, nose, scalp, forehead, and jaw. Trigeminal neuralgia is considered by many to be among the most painful of conditions and has been labeled the suicide disease, due to the significant numbers of people taking their own lives because they were unable to have their pain controlled with medications or surgery. An estimated one in 15,000 people suffers from trigeminal neuralgia, although numbers may be significantly higher due to frequent misdiagnosis. It usually develops after the age of 40, although there have been cases with patients being as young as three years of age .



Abstract – Anat - 10

Cleft lip & Cleft palate

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Cleft palate, with or without cleft lip, is one of the more common congenital malformations in man. A cleft is a fissure or opening—a gap. It is the non-fusion of the body's natural structures that form before birth. Approximately 1 in 700 children born have a cleft lip or a cleft palate or both. In decades past, the condition was sometimes referred to as harelip, based on the similarity to the cleft in the lip of a hare, but that term is now generally considered to be offensive. Congenital cleft lip and cleft palate have been the subject of many genetic studies, but until recently there has been no consensus as to their modes of inheritance. In fact, claims have been made for just about every genetic mechanism one can think of. Recently, however, evidence has been accumulating that favors a multifactorial basis for these malformations.



Abstract – Anat - 11

Larynx- Gift of voice

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The larynx , commonly called the voice box, is an organ in the neck of amphibians, reptiles, and mammals involved in breathing, SOUND PRODUCTION, and protecting the trachea against food aspiration. It manipulates pitch and volume. The larynx houses the vocal folds, which are essential for phonation. The vocal folds are situated just below where the tract of the pharynx splits into the trachea and the esophagus. The vocal folds can be held close together so that they vibrate .The muscles attached to the arytenoid cartilages control the degree of opening. Vocal fold length and tension can be controlled by rocking the thyroid cartilage forward and backward on the cricoid cartilage (either directly by contracting the cricothyroids or indirectly by changing the vertical position of the larynx), by manipulating the tension of the muscles within the vocal folds, and by moving the arytenoids forward or backward. This causes the pitch produced during phonation to rise or fall.



Abstract – Anat - 12

Importance of infra temporal fossa

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The infratemporal fossa is an irregularly shaped cavity, situated below and medial to the zygomatic arch. Boundaries defined by: anteriorly, by the infratemporal surface of the maxilla and the ridge which descends from its zygomatic process. posteriorly, by the articular tubercle of the temporal and the spina angularis of the sphenoid. superiorly, by the greater wing of the sphenoid below the infratemporal crest, and by the under surface of the temporal squama, containing the foramen ovale, which transmits the mandibular branch of the trigeminal nerve, and the foramen spinosum, which transmits the middle meningeal artery. inferiorly, by the medial pterygoid muscle attaching to the mandible. medially, by the lateral pterygoid plate. laterally, by the ramus of mandible, which contains the mandibular foramen, leading to the mandibular canal through which the inferior alveolar nerve passes. This also contains the lingula, a triangular piece of bone that overlies the mandibular foramen antero-medially. Finally, the mylohyoid groove descends obliquely transmitting the mylohyoid nerve the only motor branch of the posterior division of the trigeminal nerve.



Abstract – Anat - 13

Paranasal Air Sinuses

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Paranasal sinuses are air filled hollow sacs seen around the skull bone. These sacs precisely surround the nasal cavity. There are four paired sinuses surrounding the nasal cavity. Common complications following endoscopic sinus surgery are caused by inadequate understanding of the highly complex and variable anatomy of paranasal sinuses. The four paired sinuses include frontal, maxillary, ethmoidal and sphenoidal air complex cells. They lie within the facial bones, in a complex arrangement, and group around the nasal cavities. Minimally-invasive interventions of the paranasal sinus are frequent surgical procedures. While in many cases an extensive pre-operative intervention planning is not really necessary, it becomes desirable in complicated patient cases, and is currently done based on the inspection of the acquired CT data of the paranasal sinus. For a more accurate access planning including 3D measures of the cavity, especially in complex cases of tumor diseases, a segmentation of the paranasal sinus is a useful tool.



Abstract – Anat - 14

Freys syndrome

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Frey's syndrome is a complication of parotidectomy that occurs as a result of aberrant regeneration of the postganglionic parasympathetic nerve fibres supplying the parotid gland to severed postganglionic sympathetic fibres which innervate the sweat glands of the face. Frey's syndrome consists of gustatory discomfort, sweating and flushing of the skin overlying the parotid area (hemifacial flushing) which may be associated with pain in the auriculotemporal nerve distribution. Frey's syndrome is difficult to treat but is a preventable phenomenon and surgeons must be aware of the available preventative methods during the initial surgery. Frey's syndrome can be socially debilitating and because of the difficulty in its management, preventive measures should be instituted during the initial surgery. Until now the longest latency of Frey's syndrome after parotidectomy recorded in the literature is 50 years. Diagnostic methods, preventive measures and management options are briefly discussed. Frey syndrome is usually secondary to traumatic injury in the parotid region and is thought to be the result of misdirected re-sprouting of damaged autonomic nerve fibres. It is treated using butolinium neurotoxins.



Abstract – Anat - 15

Movement of eye ball

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Eye movement refers to the voluntary or involuntary movement of the eyes, helping in acquiring, fixating and tracking visual stimuli. Specific systems are used in maintaining fixation, when reading and in music reading. A special type of eye movement, rapid eye movement, occurs during REM sleep. The eyes are the visual organs of the human body, and move using a system of six muscles. The retina, a specialised type of tissue containing photoreceptors, senses light. These specialised cells convert light into electrochemical signals. These signals travel along the optic nerve fibers to the brain, where they are interpreted as vision in the visual cortex. Primates and many other vertebrates use three types of voluntary eye movement to track objects of interest: smooth pursuit, vergence shifts and saccades. These movements appear to be initiated by a small cortical region in the brain's frontal lobe. This is corroborated by removal of the frontal lobe. In this case, the reflexes (such as reflex shifting the eyes to a moving light) are intact, though the voluntary control is obliterated.



Abstract – Anat - 16

Goitre

Gitambika & Marina

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A goitre or goiter, is a swelling of the neck or larynx resulting from ENLARGEMENT of the thyroid gland (thyromegaly), associated with a thyroid gland that is functioning properly or not. Goitre which is associated with hypothyroidism or hyperthyroidism may be present with symptoms of the underlying disorder. For hyperthyroidism, the most common symptoms are associated with adrenergic stimulation: tachycardiac, palpitations, nervousness, tremor, and increased blood pressure. Clinical manifestations are often related to hypermetabolism, including increased metabolism, excessive thyroid hormone, an increase in oxygen consumption, metabolic changes in protein metabolism, immunologic stimulation of diffuse goiter, and ocular changes (exophthalmos).



**DEPARTMENT OF ANATOMY
POSTER PRESENTATION**

Abstracts – Anat - 01

Stages of tooth development

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The developing tooth represents a suitable model for understanding the molecular mechanisms involved in induction, morphogenesis and differentiation of organs. It is conceivable that the developmental changes could be reflected in the distribution of different cytoskeletal components and in this report we analyze the expression of the intermediate filament nestin during rodent tooth development at the protein and mRNA levels (by immunofluorescence and electronmicroscopy, and by in situ hybridization). Tooth development is under strict genetic control, and during recent years an increasing number of genes have been identified that are involved in the regulation of tooth morphogenesis. One of the organs in which development is now beginning to be understood at the gene level, the tooth is an example of a typical vertebrate organ starting as an epithelial bud and undergoing complex morphogenesis, regulated by interactions between epithelial and mesenchymal tissue layers. It has become evident that developmental regulatory genes have been conserved to a high degree during evolution and that similar gene networks regulate the development of teeth as of other vertebrate organs.



Abstract – Anat - 02

Cleft lip and cleft palate

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Cleft lip and cleft palate is a congenital deformity caused by abnormal facial development during gestation. A cleft is a fissure or opening—a gap. It is the non-fusion of the body's natural structures that form before birth. The birth of a child with a cleft lip or a cleft palate, or both, can be traumatic to the family. Although referral to a multidisciplinary team experienced in craniofacial abnormalities is essential, the family physician can reduce the impact on the family by providing antenatal diagnosis and continued care of the entire family after diagnosis, during initial feeding and bonding difficulties and throughout the many years of surgical and speech therapy. Numerous investigators have reported on a low frequency of other anomalies in patients with cleft lip, cleft palate, or both. The data have been somewhat inconsistent, ranging from a 3% to over 30% frequency of associated malformations. The high frequency of associated anomalies has obvious implications for the genetic counselling offered to all patients at cleft palate and craniofacial centres.



Abstract – Anat - 03

Maxillary sinus and oral antral fistula

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Oroantral fistula, an abnormal communication between the oral cavity and the maxillary sinus, is infrequently diagnosed radiologically. The tooth most frequently involved was the upper first molar, followed by the second and third molars. The sockets of the palatal roots of the first and the second molars were most frequently involved. There have been many expanded applications for the use of endosseous implants in the reconstruction of partially and totally edentulous patients. The posterior maxilla, which frequently has inadequate quality and quantity of bone, and the contiguous maxillary sinus often provide poor recipient sites for endosseous implants. However, innovative procedures using autogenous, allogeneic, and alloplastic graft materials have enabled clinicians to place implants in the reconstructed resorbed maxilla. These techniques often violate the anatomic integrity and interfere with the physiologic mechanisms of the maxillary sinus, creating potential complications.



DEPARTMENT OF BIOCHEMISTRY

ORAL PRESENTATION

Abstract - Bio - 01

Vitamins In Oral Health

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An unhealthy diet has been implicated as risk factors for several chronic diseases that are known to be associated with oral diseases. Nutritional intake influences the oral tissues to which bacteria bind (i.e.epithelium, collagen, bone, teeth), as well as saliva. Teeth in a preeruptive phase are influenced by the nutritional state. Secretory proteins (mucin) found within saliva provide an effective barrier against desiccation, penetration, physical and chemical irritants, and bacteria. Synthesis of glycoproteins, such as mucin, requires vitamin A. Retinol deficiency can reduce mucin production, leading to compromised salivary flow, weakened tooth integrity, and a marked increase in risk for caries. Vitamin A deficiency also causes Gingivitis, Periodontitis and Hyperplasia of the Gingiva. A lack of vitamins D and A and protein-energy malnutrition have been associated to hypoplasia of the enamel and atrophy of the salivary glands, conditions that determine a greater susceptibility to caries. Some hypoplasia and pits on the surface of the enamel correlate to a lack of vitamin A . A lack of vitamin D is associated to the more diffused hypoplastic forms . A lack of vitamin B3 can cause bad breath and canker sores in the mouth. To boost your B3 levels, eat chicken and fish.You also can develop mouth sores when you do not consume enough of the vitamins B12 and B2. Red meat, chicken, liver, pork, fish, as well as dairy products like milk, yogurt, and cheese, are good sources of vitamin B12. Vitamin B2 is found in foods like pasta, bagels, spinach, and almonds. The relationship between vitamin C and periodontal disease may be due to vitamin C's role in maintaining and repairing healthy connective tissue along with its antioxidant properties. It is very important to consume enough vitamin D because it helps your body absorb calcium. A diet People with vitamin K deficiency may have excessive bleeding after a tooth is extracted, or even after a tooth cleaning.

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Abstract - Bio - 02

Biochemical Alterations In In Lead Poisoning

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Lead is a heavy metal and important environmental toxicant and nerve poison that can destruct many functions of the nervous system. Lead poisoning is a medical condition caused by increased levels of lead in the body. Lead interferes with a variety of body processes and is toxic to many organs and tissues including the heart, bones, intestines, kidneys, and reproductive and nervous systems. It interferes with the development of the nervous system, causes changes in neuro transmitter levels and is therefore particularly toxic to children, causing potentially permanent learning and behaviour disorders. Lead inhibits several enzymes, particularly Alpha aminolevulinate Synthase and dehydrogenase and ferrochelatase of heme synthesis leading to anaemia. Lead causes impairment in vitamin D metabolism, lymphocyte and antibody production. Exposure to lead can be through contaminated air, water, soil, food, and consumer products. Occupational exposure is a common cause of lead poisoning in adults. Symptoms include abdominal pain, confusion, headache, anaemia, irritability, and in severe cases seizures, coma, and death. So this article mainly throws light on the biochemical effects of lead toxicity. Elevated lead in the body can be detected by the presence of changes in blood cells visible with a microscope and dense lines in the bones of children seen on X-ray, but the main tool for diagnosis is measurement of the blood lead level. When blood lead levels are recorded, the results indicate how much lead is circulating within the blood stream, not the amount being stored in the body. Humans have been mining and using this heavy metal for thousands of years, poisoning themselves in the process. Although lead poisoning is one of the oldest known work and environmental hazards, the modern understanding of the small amount of lead necessary to cause harm did not come about until the latter half of the 20th century. No safe threshold for lead exposure has been discovered—that is, there is no known sufficiently small amount of lead that will not cause harm to the body.



Abstract - Bio - 03

Biochemical Parameters In Saliva

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Saliva is a watery substance located in the mouths of organisms, secreted by the salivary glands. Human saliva is 99.5% water, while the other 0.5% consists of electrolytes, mucus, glycoproteins, enzymes, and antibacterial compounds such as secretory IgA and lysozyme. Saliva is supersaturated with various ions. The enzymes found in saliva are essential in beginning the process of digestion of dietary starches and fats and also play a role in breaking down food particles entrapped within dental crevices, protecting teeth from bacterial decay. Saliva contains the enzyme amylase, also called ptyalin, which is capable of breaking down starch into simpler sugars. Salivary glands also secrete salivary lipase (a more potent form of lipase) that plays a large role in fat digestion, especially in newborn infants as their pancreatic lipase still needs some time to develop. Saliva contains hormone gustin, which is thought to play a role in the development of taste buds. Certain salivary protein prevents precipitation, which would form salts. These ions act as a buffer, keeping the acidity of the mouth within a certain range, typically ph 6.2 - 7.4. This prevents minerals in the dental hard tissues from dissolving. Furthermore, saliva serves a lubricative function, wetting food and permitting the initiation of swallowing, and protecting the mucosal surfaces of the oral cavity from desiccation.



Abstract - Bio - 04

PH Of Saliva

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Saliva is a watery substance located in the mouths of organisms, secreted by the salivary glands. The pH of saliva is 7.4, which is equal to the pH of blood that is 7.4, & pH of spinal fluid that is also 7.4. The pH of saliva is almost constant but it varies depending on the measurement and various Investigators determine the pH of saliva in range of pH 5.0 to pH 8.0. This varies depending on the type of meal, if you have taken acidic meal then pH is slightly low, if you have taken alkaline meal then pH is slightly high. Therefore, it is advisable to wash your mouth before the measurement of pH of saliva. Additionally, the pH test of saliva represents the most consistent and most definitive physical sign of the ionic calcium deficiency syndrome. The pH of the non-deficient and healthy person is in the 7.5 (dark blue) to 7.1 (blue) slightly alkaline range. The range from 6.5 (blue-green) which is weakly acidic to 4.5 (light yellow) which is strongly acidic represents states from mildly deficient to strongly deficient, respectively. Most children are dark blue, a pH of 7.5. Over half of adults are green-yellow, a pH of 6.5 or lower, reflecting the calcium deficiency of aging and lifestyle defects. Cancer patients are usually a bright yellow, a pH of 4.5. The elaboration of extracellular proteolytic activity by *Candida albicans* during growth in laboratory broth or in human whole salivary supernatant was investigated. Growth of the organism in broth at pH 3 to 7 followed by assay of culture supernatants at pH 4 (optimum for activity) indicated protease was only present in cultures grown at a pH of less than 5. In contrast, growth in sterile human whole salivary supernatant over the pH range of 3 to 7 uniformly failed to result in production of protease. Growth of the organism at pH 4 in broth supplemented with saliva resulted in a saliva-dependent inhibition of protease production. Although the addition of up to 16% (vol/vol) saliva had essentially no effect on growth, 4% saliva caused a 50% reduction in proteolysis of substrate protein. Due to the low pH requirement for protease production and activity and the demonstration that saliva is a potent inhibitor of protease synthesis, we conclude *C. albicans* most likely does not produce extracellular protease in the human oral cavity. Test strips purchased retail can be very expensive for patients and may not be the quality required to separate the pH ranges adequately.



Abstract - Bio – 05

Dental Fluorosis

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Dental fluorosis, also called mottling of tooth enamel, is a developmental disturbance of dental enamel caused by excessive exposure to high concentrations of fluoride during tooth development. The risk of fluoride overexposure occurs at any age but it is higher at younger ages. In its mild forms, fluorosis often appears as unnoticeable, tiny white streaks or specks in the enamel of the tooth. In its most severe form, tooth appearance is marred by discoloration or brown markings. The enamel may be pitted, rough and hard to clean. The spots and stains left by fluorosis are permanent and may darken over time. The greatest concern in dental fluorosis is aesthetic changes in the permanent dentition. Many well-known sources of fluoride may contribute to overexposure including dentifrice/fluoridated mouth rinse (which young children may swallow), bottled waters which are not tested for their fluoride content, inappropriate use of fluoride supplements, ingestion of foods especially imported from other countries, and public water fluoridation. The severity of dental fluorosis depends on the amount of fluoride exposure, the age of the child, individual response, weight, degree of physical activity, nutrition, and bone growth. The differential diagnosis for this condition may include Turner's hypoplasia, some mild forms of amelogenesis imperfecta, and other environmental enamel defects of diffuse and demarcated opacities. H.T. Dean's fluorosis index became the most universally accepted classification system for dental fluorosis. Tooth bleaching, micro abrasion, and conservative composite restorations or porcelain veneers are commonly used treatments.



Abstract - Bio - 06

Recombinant Dna Tecnology

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Recombinant DNA (rDNA) technology is a field of molecular biology in which scientists "edit" DNA to form new synthetic molecules, which are often referred to as "chimeras". Recombinant DNA (DNA) molecules are DNA molecules formed by laboratory methods of Transformation, Phage Introduction, and Non-Bacterial Transformation, with the goal of introducing recombinant genes into a host cell along with expression factor, so that the host cell expresses the desired protein. Recombinant chymosin found in rennet, is an enzyme required to manufacture cheese. It was the first genetically engineered food additive used commercially..Recombinant DNA technology has led to powerful diagnostic procedures useful in both medicine and forensics. Recombinant human insulin almost completely replaced insulin obtained from animal sources (e.g. pigs and cattle) for the treatment of insulin-dependent diabetes. Recombinant human growth hormone (HGH, somatotropin) administered to patients whose pituitary glands generate insufficient quantities to support normal growth and development. Before recombinant HGH became available, HGH for therapeutic use was obtained from pituitary glands of cadavers. Several transgenic plants that kill insects have been introduced in several species, including corn and cotton . commercial varieties of important agricultural crops (including soy, maize/corn, sorghum, canola, alfalfa and cotton) have been developed that incorporate a recombinant gene that results in resistance to the herbicide glyphosate (trade name *Roundup*), and simplifies weed control by glyphosate application. Recombinant blood clotting factor VIII, a blood-clotting protein that is administered to patients with forms of the bleeding disorder hemophilia, who are unable to produce factor VIII in quantities sufficient to support normal blood coagulation. Hepatitis B infection is controlled through the use of a recombinant hepatitis B vaccine, which contains a form of the hepatitis B virus surface antigen that is produced in yeast cells. each of the three widely used methods for diagnosing HIV infection has been developed using recombinant DNA Golden rice a recombinant variety of rice that has been engineered to express the enzymes responsible for β -carotene biosynthesis. This variety of rice holds substantial promise for reducing the incidence of vitamin A deficiency in the world's population.



Abstract - Bio - 07

Operon concept

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An Operon is a co-ordinated unit of genetic expression and a functional unit of genomic DNA containing a cluster of genes under the control of a single regulatory signal or promoter. The genes are transcribed together into an mRNA strand and either translated together in the cytoplasm, or undergo trans-splicing to create monocistronic mRNAs that are translated separately, i.e. several strands of mRNA that each encode a single gene product. The result of this is that the genes contained in the Operon are either expressed together or not at all. Several genes must be both *co-transcribed* and *co-regulated* to define an Operon. Operons were thought to occur primarily in prokaryotes but they were also found in eukaryotes. Operons have also been found in viruses such as bacteriophages. rRNA genes often exist in operons that have been found in a range of eukaryotes including chordates. An Operon is made up of several structural genes arranged under a common promoter and regulated by a common operator, regulator and structural genes. It is defined as a set of adjacent structural genes, plus the adjacent regulatory signals that affect transcription of the structural genes. Control of an Operon is a type of gene regulation that enables organisms to regulate the expression of various genes depending on environmental conditions. Operon regulation can be either negative or positive by induction or repression. Negative control involves the binding of a repressor to the operator to prevent transcription. Operons can also be positively controlled. With positive control, an activator protein stimulates transcription by binding to DNA (usually at a site other than the operator). The *lac* operon of the model bacterium *Escherichia coli* was the first operon to be discovered and provides a typical example of operon function. The number and organization of operons has been studied most critically in *E. coli*. As a result, predictions can be made based on an organism's genomic sequence. An alternative method to predict operons is based on finding gene clusters where gene order and orientation is conserved in two or more genomes.



Abstract - Bio - 08

Gene Therapy In Cancer

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Gene therapy is the use of DNA as a drug to treat disease by delivering therapeutic DNA into a patient's cells. The most common form of gene therapy involves using DNA that encodes a functional, therapeutic gene to replace a mutated gene. Other forms involve directly correcting a mutation, or using DNA that encodes a therapeutic protein drug (rather than a natural human gene) to provide treatment. In gene therapy, DNA that encodes a therapeutic protein is packaged within a "vector", which is used to get the DNA inside cells within the body. Once inside, the DNA becomes expressed by the cell machinery, resulting in the production of therapeutic protein, which in turn treats the patient's disease. The broad field of gene therapy promises a number of innovative treatments that are likely to become important in preventing deaths from cancer. In this review, we discuss the highlights and future of three different gene therapy treatment approaches: immunotherapy, oncolytic virotherapy and gene transfer. Immunotherapy uses genetically modified cells and viral particles to stimulate the immune system to destroy cancer cells. Recent clinical trials of second and third generation vaccines have shown encouraging results with a wide range of cancers, including lung cancer, pancreatic cancer, prostate cancer and malignant melanoma. Oncolytic virotherapy, which uses viral particles that replicate within the cancer cell to cause cell death, is an emerging treatment modality that shows great promise, particularly with metastatic cancers. Initial phase I trials for several vectors have generated excitement over the potential power of this technique. Gene transfer is a new treatment modality that introduces new genes into a cancerous cell or the surrounding tissue to cause cell death or slow the growth of the cancer. This treatment technique is very flexible, and a wide range of genes and vectors are being used in clinical trials with successful outcomes. As these therapies mature, they may be used alone or in combination with current treatments to help make cancer a manageable disease.



Abstract - Bio – 09

DNA Finger Printing

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DNA fingerprinting (also called DNA profiling) is the present day genetic detective employed by forensic scientists to assist in the identification of individuals by their respective DNA profiles. It is an analysis of the nitrogenous base sequences in the DNA. DNA profiles are encrypted sets of numbers that reflect a person's DNA makeup, which can also be used as the person's identifier. Although 99.9% of human DNA sequences are the same in every person, enough of the DNA is different to distinguish one individual from another, unless they are monozygotic twins. DNA profiling uses repetitive ("repeat") sequences that are highly variable, called variable number tandem repeats (VNTRs), particularly short tandem repeats (STRs). VNTR loci are very similar between closely related humans, but so variable that unrelated individuals are extremely unlikely to have the same VNTRs. DNA finger printing uses the techniques of RFLP, PCR and VNTR analysis. The first methods for finding out genetics used for DNA profiling involved restriction enzyme digestion, followed by Southern blot analysis. The process, the polymerase chain reaction (PCR), mimics the biological process of DNA replication, but confines it to specific DNA sequences of interest. In this process, the DNA sample is denatured into the separate individual strands. Two DNA primers are used to hybridize to two corresponding nearby sites on opposite DNA strands in such a fashion that the normal enzymatic extension of the active terminal of each primer (that is, the 3' end) leads toward the other primer. The method of DNA profiling used today is based on PCR and uses short tandem repeats (STR). This method uses highly polymorphic regions that have short repeated sequences of DNA (the most common is 4 bases repeated, but there are other lengths in use, including 3 and 5 bases). Because unrelated people almost certainly have different numbers of repeat units, STRs can be used to discriminate between unrelated individuals. These STR loci (locations on a chromosome) are targeted with sequence-specific primers and amplified using PCR. The DNA fragments that result are then separated and detected using electrophoresis. There are two common methods of separation and detection, capillary electrophoresis (CE) and gel electrophoresis.



Abstract - Bio - 10

Test Tube Teeth

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Teeth develop from a series of reciprocal interactions that take place between epithelium and mesenchyme during development of the mouth that begin early in mammalian embryogenesis. The molecular control of key processes in tooth development such as initiation, morphogenesis and cytodifferentiation are being increasingly better understood, to the point where this information can be used as the basis for approaches to produce biological replacement teeth (BioTeeth). This review outlines the current approaches, ideas and progress towards the production of BioTeeth that could form an alternative method for replacing lost or damaged teeth. By carefully manipulating intercellular communication, they forced cells to rearrange themselves, forming a tooth. Surprisingly, the signaling pathways can be used to assemble teeth from adult stem cells as well as existing dental cells. Best of all, bioengineering teeth from an individual's own tissues avoids immune rejection and allows for a more realistic replacement, since tooth size, shape, and color are genetically determined. Unfortunately, the challenges of growing roots and identifying ideal raw materials remain. Even so, scientific progress can be fast, and teeth may be the first successfully engineered organs. Scientists have observed how nature engineers a tooth and have combined this understanding with advances in stem cell biology and tissue engineering technology to get closer to and understand biological replacement teeth. The construction of a tooth takes about 14 months to complete in a developing human. Two different types of embryonic tissue combine to produce a tooth, and an ongoing molecular interaction between them leads the process. Tissue engineers are studying these signals and steps to understand the signals they need to replicate as they manufacture living bioengineered replacement teeth. Scientists are connecting stem cells to create the tooth buds that form in the early embryo. The idea is to implant these "primordial teeth" (that are in their early stage) into human jaws and let the cells take it from there. By using a patient's own stem cells avoids the problems that often happen in ordinary transplants. Tissue engineers working toward creating living replacement teeth take cues from nature as they coax disparate cell types to form a functional organ. Alternative methods include building teeth from existing dental cells or growing them from progenitor tissues. Both approaches have already produced structurally correct teeth.



Abstract - Bio - 11

Applications of Genetic Engineering

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Genetic engineering, also called genetic modification, is the direct manipulation of an organism's genome using biotechnology. New DNA may be inserted in the host genome by first isolating and copying the genetic material of interest using molecular cloning methods to generate a DNA sequence, or by synthesizing the DNA, and then inserting this construct into the host organism. Genes may be removed, or "knocked out", using a nuclease. Genetic engineering techniques have been applied in numerous fields including research, agriculture, industrial biotechnology, and medicine. Plants, animals or micro organisms that have changed through genetic engineering are termed genetically modified organisms or GMOs. Plants have been modified for insect protection, herbicide resistance, virus resistance, enhanced nutrition, tolerance to environmental pressures and the production of edible vaccines. Most commercialised GMO's are insect resistant and/or herbicide tolerant crop plants. Genetically modified animals have been used for research, model animals and the production of agricultural or pharmaceutical products. They include animals with genes knocked out, increased susceptibility to disease, hormones for extra growth and the ability to express proteins in their milk. In medicine genetic engineering has been used to mass-produce insulin, human growth hormones, follistim (for treating infertility), human albumin, monoclonal antibodies, antihemophilic factors, vaccines and many other drugs. Vaccination generally involves injecting weak, live, killed or inactivated forms of viruses or their toxins into the person being immunized.^[71] Genetically engineered viruses are being developed that can still confer immunity, but lack the infectious sequences Genetic engineering is used to create animal models of human diseases. They have been used to study and model cancer (the oncomouse), obesity, heart disease, diabetes, arthritis, substance abuse, anxiety, aging and Parkinson disease.^[77] Potential cures can be tested against these mouse models. Gene therapy is the genetic engineering of humans by replacing defective human genes with functional copies.



Abstract - Bio - 12

Polymerase Chain Reaction

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The polymerase chain reaction (PCR) is a biochemical technology in molecular biology to amplify a single or a few copies of a piece of DNA across several orders of magnitude, generating thousands to millions of copies of a particular DNA sequence. PCR is now a common and often indispensable technique used in medical and biological research labs for a variety of applications. These include DNA cloning for sequencing, DNA-based phylogeny, or functional analysis of genes; the diagnosis of hereditary diseases; the identification of genetic fingerprints (used in forensic sciences and paternity testing); and the detection and diagnosis of infectious diseases. The method relies on thermal cycling, consisting of cycles of repeated heating and cooling of the reaction for DNA melting and enzymatic replication of the DNA. Primers (short DNA fragments) containing sequences complementary to the target region along with a DNA polymerase (after which the method is named) are key components to enable selective and repeated amplification. As PCR progresses, the DNA generated is itself used as a template for replication, setting in motion a chain reaction in which the DNA template is exponentially amplified. PCR can be extensively modified to perform a wide array of genetic manipulations. Almost all PCR applications employ a heat-stable DNA polymerase, such as Taq polymerase (an enzyme originally isolated from the bacterium *Thermus aquaticus*). This DNA polymerase enzymatically assembles a new DNA strand from DNA building-blocks, the nucleotides, by using single-stranded DNA as a template and DNA oligonucleotides (also called DNA primers), which are required for initiation of DNA synthesis. The vast majority of PCR methods use thermal cycling, i.e., alternately heating and cooling the PCR sample through a defined series of temperature steps. In the first step, the two strands of the DNA double helix are physically separated at a high temperature in a process called DNA melting. In the second step, the temperature is lowered and the two DNA strands become templates for DNA polymerase to selectively amplify the target DNA. Many variations have been developed in the basic PCR techniques recently. PCR allows isolation of DNA fragments from genomic DNA by selective amplification of a specific region of DNA. This use of PCR augments many methods, such as generating hybridization probes for Southern or northern hybridization and DNA cloning, which require larger amounts of DNA, representing a specific DNA region. PCR permits early diagnosis of malignant diseases such as leukemia and lymphomas, which is currently the highest-developed in cancer research and is already being used routinely.



Abstract - Bio - 13

Leptin

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Leptin is a 16-kDa adipokine that plays a key role in regulating energy intake and expenditure, including appetite and hunger, metabolism, and behavior. It is one of the most important adipose-derived hormones. Leptin functions by binding to the leptin receptor. The *Ob(Lep)* gene (Ob for obese, Lep for leptin) is located on chromosome 7 in humans. Leptin is a hormone made by fat tissue that acts on the brain to regulate food intake and body weight. Human leptin is a protein of 167 amino acids. It is manufactured primarily in the adipocytes of white adipose tissue, and the level of circulating leptin is proportional to the total amount of fat in the body. In addition to white adipose tissue—the major source of leptin—it can also be produced by brown adipose tissue, placenta (syncytiotrophoblasts), ovaries, skeletal muscle, stomach (the lower part of the fundic glands), mammary epithelial cells, bone marrow, pituitary, and liver. Leptin has also been discovered to be synthesized from gastric chief cells and P/D1 cells in the stomach. This hormone circulates in blood and acts on the hypothalamus to regulate food intake and energy expenditure. When fat mass falls, plasma leptin levels fall stimulating appetite and suppressing energy expenditure until fat mass is restored. When fat mass increases, leptin levels increase, suppressing appetite until weight is lost. This physiological system ensures that total energy stores are stably maintained within a relatively narrow range. The identification of a physiologic system that controls energy balance also establishes a biologic basis for obesity. Leptin also regulates many other physiologic systems and plays a critical role in the adaptive response to starvation. Leptin circulates in blood in free form and bound to proteins. Serum leptin levels are higher between midnight and early morning which could have an effect in suppressing appetite during the night while sleeping. The diurnal rhythm of plasma leptin can be modified by meal-timing indicating that plasma leptin is entrained to meal timing. Leptin binds to neuropeptide Y (NPY) neurons in the arcuate nucleus in such a way as to decrease the activity of these neurons. Leptin signals the brain that the body has had enough to eat, producing a feeling of satiety. Moreover, this fullness hormone may make it easier for people to resist the temptation of foods high in calories. The role of leptin/leptin receptors in modulation of T cell activity in immune system was shown in experimentation with mice. It modulates the immune response to atherosclerosis, of which obesity is a predisposing factor. Leptin's ability to regulate bone mass was also recognized. Leptin has traditionally been regarded as a link between present fat storage, food intake, and energy expenditure.



Abstract - Bio - 14

Sickle Cell Haemoglobin

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Approximately 5% of the world's population carries trait genes for haemoglobin disorders, mainly, sickle-cell disease. Sickle-cell disease is a hereditary blood disorder, characterized by a modification in the shape of the red blood cell from a smooth, donut-shape into a crescent or half moon shape. The misshapen cells lack plasticity and can block small blood vessels, impairing blood flow leading to shortened red blood cell survival, and subsequent anaemia, often called sickle-cell anaemia. Poor blood oxygen levels and blood vessel blockages in people with sickle-cell disease can lead to chronic acute pain syndromes, severe bacterial infections, and necrosis (tissue death). Sickle-cell anaemia can lead to various complications, including: Overwhelming post-(auto)splenectomy infection (OPSI), stroke, Cholelithiasis (gallstones) and cholecystitis and Decreased immune reactions due to hyposplenism. The sickling occurs because of a mutation in the haemoglobin gene. Sickle hemoglobin differs from normal hemoglobin by a single amino acid: valine replaces glutamate at position 6 on the surface of the beta chain. This creates a new hydrophobic spot, which stick to each other causing deoxygenated hemoglobin to aggregate into chains. The polymerized hemoglobin distorts red blood cells into an abnormal sickle shape. Heterozygotes have a mixture of normal hemoglobin A and mutant hemoglobin S. The hemoglobin A stops polymerization, preventing serious sickling. The pure hemoglobin S in homozygotes polymerizes to a greater degree. Red cells lyse in homozygotes, producing the disease 'sickle cell anemia. Sickle hemoglobin, even in heterozygotes, confers resistance to one type of malaria. Through evolution, this selective advantage has led to a 40% incidence of sickle hemoglobin in regions of the world where malaria is endemic.



Abstract - Bio - 15

Biochemical Alterations In AIDS

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Human immunodeficiency virus infection / acquired immunodeficiency syndrome (HIV/AIDS) is a disease of the human immune system caused by infection with human immunodeficiency virus (HIV). HIV continues to be a major global public health issue, having claimed more than 36 million lives so far. There were approximately 35.3 [32.2–38.8] million people living with HIV in 2012. Infection with HIV-1 primarily involves a subgroup of T-lymphocytic cells, but other cell types are also invaded by the virus, including cell lines within the haematopoietic system. During the initial infection, a person may experience a brief period of influenza-like illness. This is typically followed by a prolonged period without symptoms. As the illness progresses, it interferes more and more with the immune system, making the person much more likely to get infections, including opportunistic infections and tumours that do not usually affect people who have working immune systems. Abnormalities in blood lipids (elevated cholesterol and triglyceride fats) and glucose (elevated blood sugar, as in diabetes) changes are observed in HIV patients and those on highly active antiretroviral therapy. A significant increase in liver marker enzymes (AST, ALP and ALT) and decrease in CD4+ cell count is observed in HIV infected persons. Most people infected with HIV develop specific antibodies (i.e. seroconvert) within three to twelve weeks of the initial infection.^[13] Diagnosis of primary HIV before seroconversion is done by measuring HIV-RNA or p24 antigen.^[13] Positive results obtained by antibody or PCR testing are confirmed either by a different antibody or by PCR. Antibody tests in children younger than 18 months are typically inaccurate due to the continued presence of maternal antibodies. Thus HIV infection can only be diagnosed by PCR testing for HIV RNA or DNA, or via testing for the p24 antigen.



Abstract - Bio - 16

Thalassemia

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Thalassemia is a blood disorder passed down through families (inherited) in which the body makes an abnormal form of hemoglobin, the protein in red blood cells that carries oxygen. The disorder results in excessive destruction of red blood cells, which leads to anemia. Thalassemia is caused by variant or missing genes that affect how the body makes hemoglobin. The majority of adult hemoglobin (HbA) is composed of four protein chains, two α and two β globin chains arranged into a heterotetramer. In thalassemia, patients have defects in either the α (encoded by a single gene on chromosome 11) or β globin chain (encoded by a single gene on chromosome 16) causing production of abnormal red blood cells. The α thalassemias involve the genes HBA1 and HBA2. There are two gene loci and so four alleles. It is also connected to the deletion of the 16p chromosome. α Thalassemias result in decreased alpha-globin production, therefore fewer alpha-globin chains are produced, resulting in an excess of β chains in adults and excess γ chains in newborns. The excess β chains form unstable tetramers (called Hemoglobin H or HbH of 4 beta chains), which have abnormal oxygen dissociation curves. Beta thalassemias are due to mutations in the HBB gene on chromosome 11,^[10] also inherited in an autosomal-recessive fashion. The severity of the disease depends on the nature of the mutation. Mutations are characterized as either β^0 or β thalassemia major if they prevent any formation of β chains, the most severe form of β thalassemia. Also, they are characterized as β^+ or β thalassemia intermedia if they allow some β chain formation to occur. In either case, there is a relative excess of α chains, but these do not form tetramers: Rather, they bind to the red blood cell membranes, producing membrane damage, and at high concentrations they form toxic aggregates. About 3% of adult hemoglobin is made of alpha and delta chains. Just as with beta thalassemia, mutations that affect the ability of this gene to produce delta chains can occur. Thalassemia can cause significant complications, including iron overload, bone deformities and cardiovascular illness. Thalassemia will be present as microcytic anemia which may be differentiated from iron deficiency anemia using the mentzer index calculation. However this same inherited disease of red blood cells may confer a degree of protection against malaria, which is or was prevalent in the regions where the trait is common. This selective survival advantage on carriers (known as heterozygous advantage) may be responsible for perpetuating the mutation in populations.



Abstract - Bio - 17

Alzheimers Disease

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Alzheimer's disease is a neurological disorder in which the death of brain cells causes memory loss, confusion, hallucinations and cognitive decline. A neurodegenerative type of dementia, the disease starts mild and gets progressively worse, as it has no cure. Most often, AD is diagnosed in people over 65 years of age, although the less-prevalent early-onset Alzheimer's can occur much earlier. In 2006, there were 26.6 million people worldwide with AD. Alzheimer's is predicted to affect 1 in 85 people globally by 2050. The accumulation of amyloids, namely β -amyloid which is prone for self aggregation is believed to be the cause of the disease. The cause and progression of Alzheimer's disease are not well understood. Research indicates that the disease is associated with plaques and tangles in the brain. Alzheimer's disease has been identified as a protein misfolding disease (proteopathy), caused by plaque accumulation of abnormally folded beta amyloid and tau amyloid proteins in the brain. Plaques are made up of small peptides, 39–43 amino acids in length, called beta-amyloid ($A\beta$). Beta-amyloid is a fragment from a larger protein called amyloid precursor protein (APP), a transmembrane protein that penetrates through the neuron's membrane. APP is critical to neuron growth, survival and post-injury repair.^{[80][81]} In Alzheimer's disease, an unknown process causes APP to be divided into smaller fragments by enzymes through proteolysis. One of these fragments gives rise to fibrils of beta-amyloid, which form clumps that deposit outside neurons in dense formations known as senile plaques. Exactly how disturbances of production and aggregation of the beta-amyloid peptide gives rise to the pathology of AD is not known. The amyloid hypothesis traditionally points to the accumulation of beta-amyloid peptides as the central event triggering neuron degeneration. Accumulation of aggregated amyloid fibrils, which are believed to be the toxic form of the protein responsible for disrupting the cell's calcium ion homeostasis, induces programmed cell death (apoptosis). It is also known that $A\beta$ selectively builds up in the mitochondria in the cells of Alzheimer's-affected brains, and it also inhibits certain enzyme functions and the utilisation of glucose by neurons. Current treatments only help with the symptoms of the disease. There are no available treatments that stop or reverse the progression of the disease. As of 2012, more than 1,000 clinical trials have been or are being conducted to test various compounds in AD. Mental stimulation, exercise, and a balanced diet have been suggested as ways to delay cognitive symptoms (though not brain pathology) in healthy older individuals, but there is no conclusive evidence supporting an effect.



Abstract -Bio -18

Agression, Suicide And Sertonin

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Serotonin or 5-hydroxytryptamine (5-HT) is a monoamine neurotransmitter. Biochemically derived from tryptophan, serotonin is primarily found in the gastrointestinal (GI) tract, platelets, and the central nervous system (CNS) of animals, including humans. It is popularly thought to be a contributor to feelings of well-being and happiness. Serotonin secreted from the enterochromaffin cells eventually finds its way out of tissues into the blood. There, it is actively taken up by blood platelets, which store it. When the platelets bind to a clot, they release serotonin, where it serves as a vasoconstrictor and helps to regulate hemostasis and blood clotting. Serotonin also is a growth factor for some types of cells, which may give it a role in wound healing. Serotonin is a neurotransmitter and is found in all bilateral animals, where it mediates gut movements and the animal's perceptions of resource availability. When humans smell food, dopamine is released to increase the appetite, the serotonin released while consuming activates 5-HT_{2C} receptors on dopamine-producing cells. This halts their dopamine release, and thereby serotonin decreases appetite. In humans, levels of 5-HT_{1A} receptor activation in the brain show negative correlation with aggression, and a mutation in the gene that codes for the 5-HT_{2A} receptor may double the risk of suicide for those with that genotype. Serotonin in the brain is not usually degraded after use, but is collected by serotonergic neurons by serotonin transporters on their cell surfaces. Studies have revealed nearly 10% of total variance in anxiety-related personality depends on variations in the description of where, when and how many serotonin transporters the neurons should deploy. In humans, defective signaling of serotonin in the brain may be the root cause of sudden infant death syndrome (SIDS). If neurons that make serotonin — serotonergic neurons — are abnormal in infants, there is a risk of sudden infant death syndrome (SIDS). Depletion of serotonin is common between disorders such as obsessive-compulsive disorder, depression, and anxiety. Drugs that alter serotonin levels are being used in treating depression, generalized anxiety disorder and social phobia.



Abstract -Bio -19

Lipoprotein Dysfunction In Atherosclerosis

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Atherosclerosis is a complex disease characterised by thickening of arterial wall as a result of the accumulation of calcium and fatty materials such as cholesterol and triglyceride. It reduces the elasticity of the artery walls and therefore allows less blood to travel through. This also increases blood pressure. It is a syndrome affecting arterial blood vessels, a chronic inflammatory response in the walls of arteries, caused largely by the accumulation of macrophages and white blood cells and promoted by low-density lipoproteins (LDL, plasma proteins that carry cholesterol and triglycerides) without adequate removal of fats and cholesterol from the macrophages by functional high-density lipoproteins. Atherosclerosis is a chronic disease that remains asymptomatic for decades. Atherosclerotic lesions, or atherosclerotic plaques are separated into two broad categories: Stable and unstable (also called vulnerable). These complications of advanced atherosclerosis are chronic, slowly progressive and cumulative. Most commonly, soft plaque suddenly ruptures (see vulnerable plaque), causing the formation of a thrombus that will rapidly slow or stop blood flow, leading to death of the tissues fed by the artery in approximately 5 minutes. This catastrophic event is called an infarction. One of the most common recognized scenarios is called coronary thrombosis of a coronary artery, causing myocardial infarction (a heart attack). The same process in an artery to the brain is commonly called stroke. Another common scenario in very advanced disease is claudication from insufficient blood supply to the legs, typically caused by a combination of both stenosis and aneurysmal segments narrowed with clots. Atherosclerosis affects the entire artery tree, but mostly larger, high-pressure vessels such as the coronary, renal, femoral, cerebral, and carotid arteries. These are termed "clinically silent" because the person having the infarction does not notice the problem and does not seek medical help, or when they do, physicians do not recognize what has happened. Lipoprotein a (Lp-a) is similar to LDL, but has an additional apoprotein. It inhibits fibrinolysis and is thought to reduce the breakdown of blood clots and triggers heart attack. Atherosclerosis is initiated by inflammatory processes in the endothelial cells of the vessel wall in response to retained low-density lipoprotein (LDL) molecules. Once inside the vessel wall, LDL particles get stuck and their content becomes more prone to oxidation. The damage caused by the oxidized LDL molecules triggers a cascade of immune responses which over time can produce an atheroma.



DEPARTMENT OF BIOCHEMISTRY

POSTER PRESENTATION

Abstract - Bio - 01

Prion Proteins

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The Prion Protein (PrP) belongs to the class of amyloid-forming proteins which are, in some cases, associated with certain diseases. The *PRNP* gene provides instructions for making the prion protein (PrP), which is active in the brain and several other tissues. The cellular prion protein (PrP^C) is a membrane associated protein occurring in a wide range of eukaryotic cells. The wide distribution among mammalian species and the high conservation of PrP^C indicates a role of general importance. The cellular isoform (PrP^C) is expressed widely in the immune system, in haematopoietic stem cells and mature lymphoid and myeloid compartments in addition to cells of the central nervous system. PrP^C is highly expressed in the CNS, and as this is the major site of prion pathology most interest has focused on defining the role of PrP^C in neurones. PrP^C has been detected on human T and B lymphocytes, natural killer (NK) cells, platelets, monocytes, dendritic cells and follicular dendritic cells. The prion protein (PrP) is involved in neurodegeneration via its conversion from the normal cellular form, PrP^C, to the infectious form, PrP^{Sc}, which is the causative agent of the transmissible spongiform encephalopathies (TSEs) including Creutzfeldt-Jakob disease (CJD). Conformational change of the normal (cellular) form of prion protein (PrP^C) to a pathological, disease-associated form (PrP^{TSE}) is considered central to pathogenesis and formation of the infectious agent or prion. The PrP^{TSE} features a predominantly β -pleated structure, whereas PrP^C is α -helix dominant. PrP^{TSE} can be distinguished from PrP^C by its resistance to protease treatment, although a protease-sensitive (PrP^{Sen}) but disease-associated transitional form has also been described. Possible functions comprise roles in neurogenesis and differentiation of neural stem cells, neuritogenesis, involvement and interaction with signal transduction pathways, synaptogenesis, neuronal survival via anti- or pro-apoptotic functions, copper binding, redox homeostasis, long-term renewal of hemopoietic stem cells, activation and development of T cells, differentiation and modulation of phagocytosis of leukocytes, and altering leukocyte recruitment to sites of inflammation.

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Abstract - Bio – 02

Translation

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Translation is the process in which cellular ribosomes create proteins. It is part of the process of gene expression. In translation, messenger RNA (mRNA) produced by transcription is decoded by a ribosome complex to produce a specific amino acid chain, or polypeptide, that will later fold into an active protein. In bacteria, translation occurs in the cell's cytoplasm, where the large and small subunits of the ribosome are located, and bind to the mRNA. In eukaryotes, translation occurs across the membrane of the endoplasmic reticulum in a process called vectorial synthesis. In bacteria, translation happens in the cell cytoplasm as they have no nucleus. The ribosome facilitates decoding by inducing the binding of tRNAs with complementary anticodon sequences to that of the mRNA. The tRNAs carry specific amino acids that are chained together into a polypeptide as the mRNA passes through and is "read" by the ribosome. Translation proceeds in four phases: initiation, elongation, translocation and termination (all describing the growth of the amino acid chain, or polypeptide that is the product of translation). Amino acids are brought to ribosomes and assembled into proteins. In activation, the correct amino acid is covalently bonded to the correct transfer RNA (tRNA). The amino acid is joined by its carboxyl group to the 3' OH of the tRNA by an ester bond. When the tRNA has an amino acid linked to it, it is termed "charged". Initiation involves the small subunit of the ribosome binding to the 5' end of mRNA with the help of initiation factors (IF). Termination of the polypeptide happens when the A site of the ribosome faces a stop codon (UAA, UAG, or UGA). No tRNA can recognize or bind to this codon. Instead, the stop codon induces the binding of a release factor protein that prompts the disassembly of the entire ribosome/mRNA complex. In many instances, the entire ribosome/mRNA complex binds to the outer membrane of the rough endoplasmic reticulum and releases the nascent protein polypeptide inside for later vesicle transport and secretion outside of the cell. Many types of transcribed RNA, such as transfer RNA, ribosomal RNA, and small nuclear RNA, do not undergo translation into proteins. A number of antibiotics act by inhibiting translation; these include anisomycin, cycloheximide, chloramphenicol, tetracycline, streptomycin, erythromycin, and puromycin, among others. Prokaryotic ribosomes have a different structure from that of eukaryotic ribosomes, and thus antibiotics can specifically target bacterial infections without any detriment to a eukaryotic host's cells.



Abstract - Bio – 03

Isoenzymes In Myocardial Infarction

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Serial monitoring of the serum isoenzyme patterns of creatine phosphokinase (CPK) and lactate dehydrogenase (LDH) in patients suspected of acute myocardial infarction has become a highly sensitive and specific diagnostic method at present. The predictable evolution of isoenzyme patterns following infarction permits diagnosis and recognition of early stages, recovery stages and extension of infarction in the individual. Usual therapeutic and resuscitative manipulations do not interfere with evaluation of patients with angina or following cardiopulmonary arrest without infarction. Despite significant elevations of serum enzyme levels following general and cardiac operative procedures, the occurrence of myocardial necrosis in the surgical population can be recognized by detection of the specific CPK-MB isoenzyme. Myocardial infarction results in death of myocardial cells served by the effected artery after about 30 minutes of anoxia. The acute inflammatory response, as a consequence of cell death, results in fever, leukocytosis, and increased concentrations of acute phase reactant proteins in the circulation (determined in the clinical laboratory most commonly by the ESR but better by measurement of C-reactive protein). An earlier and more specific consequence of myocardial death is the liberation of intracellular contents and their appearance in the circulation several hours later following diffusion through the interstitium into patent vessels and lymph ducts. Intracellular markers, routinely determined by laboratory testing, are certain enzymes present at high activity in the tissue. The enzymes routinely measured in the clinical laboratory for the purpose of diagnosing and monitoring myocardial infarction include creatine kinase (CK), aspartate amino transferase (sGOT or AST), and lactate dehydrogenase (LDH). These enzymes are present in sufficiently high content in myocardial tissue so that the death of a relatively small amount of tissue results in a substantial increase in measured enzyme activity in serum. Plasma and serum enzyme-activity alterations have proved useful as laboratory parameters in the diagnosis of myocardial disease. Among the enzymes employed to reflect myocardial necrosis are aldolase, glutamic oxalacetic transaminase, lactic dehydrogenase and malic dehydrogenase. Although the quantitative and serial changes in blood enzymes observed in myocardial infarction are characteristic, they are not specific in that other states such as those of liver, biliary and pancreatic tract and skeletal muscle may result in similar changes. In addition, the administration of drugs such as chlorpromazine, promazine, bishydroxycoumarin, pyrazinamide and opiates may result in elevations of serum enzymes similar to those observed .



Abstract - Bio - 04

Mutations

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In genetics, a mutation is a change of the nucleotide sequence of the genome of an organism, virus, or extra chromosomal genetic element. Mutations result from unrepaired damage to DNA or to RNA genomes (typically caused by radiation or chemical mutagens), errors in the process of replication, or from the insertion or deletion of segments of DNA by mobile genetic elements. Mutations may or may not produce discernible changes in the observable characteristic of an organism. Mutations play a part in both normal and abnormal biological processes including: evolution, cancer, and the development of the immune system. Mutation can result in several different types of change in sequences. Mutations in genes can either have no effect, alter the product of a gene, or prevent the gene from functioning properly or completely. Mutations can also occur in nongenic regions. Mutations can involve the duplication of large sections of DNA, usually through genetic recombination. These duplications are a major source of raw material for evolving new genes, with tens to hundreds of genes duplicated in animal genomes every million years. Most genes belong to larger families of genes of shared ancestry. Novel genes are produced by several methods, commonly through the duplication and mutation of an ancestral gene, or by recombining parts of different genes to form new combinations with new functions. Here, domains act as modules, each with a particular and independent function, that can be mixed together to produce genes encoding new proteins with novel properties. For example, the human eye uses four genes to make structures that sense light: three for color vision and one for night vision; all four arose from a single ancestral gene. Another advantage of duplicating a gene (or even an entire genome) is that this increases redundancy; this allows one gene in the pair to acquire a new function while the other copy performs the original function.



Abstract - Bio - 05

Alzheimers Disease

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Alzheimer's disease is a neurological disorder in which the death of brain cells causes memory loss, confusion, hallucinations and cognitive decline. A neurodegenerative type of dementia, the disease starts mild and gets progressively worse, as it has no cure. Most often, AD is diagnosed in people over 65 years of age, although the less-prevalent early-onset Alzheimer's can occur much earlier. In 2006, there were 26.6 million people worldwide with AD. Alzheimer's is predicted to affect 1 in 85 people globally by 2050. The accumulation of amyloids, namely β -amyloid which is prone for self aggregation is believed to be the cause of the disease. The cause and progression of Alzheimer's disease are not well understood. Research indicates that the disease is associated with plaques and tangles in the brain. Current treatments only help with the symptoms of the disease. There are no available treatments that stop or reverse the progression of the disease. As of 2012, more than 1,000 clinical trials have been or are being conducted to test various compounds in AD. Mental stimulation, exercise, and a balanced diet have been suggested as ways to delay cognitive symptoms (though not brain pathology) in healthy older individuals, but there is no conclusive evidence supporting an effect.



Abstract - Bio - 06

Hybridoma Technology

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Hybridoma technology is a technology of forming hybrid cell lines (called hybridomas) by fusing a specific antibody-producing B cell with a myeloma (B cell cancer) cell that is selected for its ability to grow in tissue culture and for an absence of antibody chain synthesis. The antibodies produced by the hybridoma are all of a single specificity and are therefore monoclonal antibodies (in contrast to polyclonal antibodies). The production of monoclonal antibodies was invented by César Milstein and Georges J. F. Köhler in 1975. They shared the Nobel Prize of 1984 for Medicine and Physiology with Niels Kaj Jerne, who made other contributions to immunology. The term hybridoma was coined by Leonard Herzenberg during his sabbatical in César Milstein's laboratory in 1976/1977. Laboratory animals (mammals, e.g. mice) are first exposed to an antigen against which we are interested in isolating an antibody. Usually this is done by a series of injections of the antigen in question, over the course of several weeks. These injections are typically followed by the use of *in vivo* electroporation, which significantly enhances the immune response. Once splenocytes are isolated from the mammal's spleen, the B cells are fused with immortalized myeloma cells. The fusion of the B cells with myeloma cells can be done using electro fusion. Electrofusion causes the B cells and Myeloma cells to align and fuse with the application of an electric field. The myeloma cells are selected beforehand to ensure they are not secreting antibody themselves and that they lack the hypoxanthine-guanine phosphoribosyltransferase (HGPRT) gene, making them sensitive to the HAT medium. Fused cells are incubated in HAT medium (hypoxanthine-aminopterin-thymidine medium) for roughly 10 to 14 days. Aminopterin blocks the pathway that allows for nucleotide synthesis. Hence, unfused myeloma cells die, as they cannot produce nucleotides by the *de novo* or salvage pathways because they lack HGPRT. Removal of the unfused myeloma cells is necessary because they have the potential to outgrow other cells, especially weakly established hybridomas. Unfused B cells die as they have a short life span. In this way, only the B cell-myeloma hybrids survive, since the HGPRT gene coming from the B cells is functional. These cells produce antibodies (a property of B cells) and are immortal (a property of myeloma cells). The incubated medium is then diluted into multi-well plates to such an extent that each well contains only one cell.



Abstract - Bio - 07

Natural Antioxidants

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An antioxidant is a molecule that inhibits the oxidation, a chemical reaction that can produce free radicals. In turn, these radicals can start chain reactions. When the chain reaction occurs in a cell, it can cause damage or death to the cell. Antioxidants terminate these chain reactions by removing free radical intermediates, and inhibit other oxidation reactions. They do this by being oxidized themselves, so antioxidants are often reducing agents such as thiols, ascorbic acid, or polyphenols. Multiple types of natural antioxidants, such as glutathione, vitamin C, vitamin A, and vitamin E as well as enzymes such as catalase, superoxide dismutase and various peroxidases exist. Insufficient levels of antioxidants, or inhibition of the antioxidant enzymes, cause oxidative stress and may damage or kill cells. "Antioxidant" is the collective name for the vitamins, minerals, carotenoids, and polyphenols that protect the body from harmful free radicals. The recent growth in knowledge of free radicals and reactive oxygen species (ROS) in biology is producing a medical revolution that promises a new age of health. Free radicals are highly unstable molecules that are naturally formed when you exercise and when your body converts food into energy. Free radicals can cause "oxidative stress," a process that can trigger cell damage. Antioxidants are widely used in dietary supplements and have been investigated for the prevention of diseases such as cancer, coronary heart disease and even altitude sickness. Endogenous antioxidant defense (H_2O_2 -removing enzymes, metal binding proteins) are inadequate to prevent damage completely, so diet derived antioxidants are important in maintaining health. Antioxidants are used as food additives to help guard against food deterioration. Antioxidants are frequently added to industrial products. A common use is as stabilizers in fuels and lubricants to prevent oxidation, and in gasolines to prevent the polymerization that leads to the formation of engine-fouling residues.



Abstract - Bio – 08

Diabetic Cataract

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Cataract in diabetic patients is a major cause of blindness in developed and developing countries. The pathogenesis of diabetic cataract development is still not fully understood. Recent basic research studies have emphasized the role of the polyol pathway in the initiation of the disease process. This paper provides an overview of the pathogenesis of diabetic cataract, clinical studies investigating the association between diabetes and cataract development, and Worldwide more than 285 million people are affected by diabetes mellitus. This number is expected to increase to 439 million by 2030 according to the International Diabetes Federation. A frequent complication of both type 1 and type 2 diabetes is diabetic retinopathy, which is considered the fifth most common cause of legal blindness in the United States. In 95% of type 1 diabetics and 60% of type 2 diabetics with disease duration longer than 20 years, signs of diabetic retinopathy occur. More severe cases of proliferative diabetic retinopathy are seen in patients suffering from type 1 diabetes. Tight control of hyperglycaemia, blood lipids, and blood pressure has been shown to be beneficial to prevent its development or progress. Cataract is considered a major cause of visual impairment in diabetic patients as the incidence and progression of cataract is elevated in patients with diabetes mellitus. The association between diabetes and cataract formation has been shown in clinical epidemiological and basic research studies. Due to increasing numbers of type 1 and type 2 diabetics worldwide, the incidence of diabetic cataracts steadily rises. Even though cataract surgery, the most common surgical ophthalmic procedure worldwide, is an effective cure, the elucidation of patho mechanisms to delay or prevent the development of cataract in diabetic patients remains a challenge. Furthermore, patients with diabetes mellitus have higher complication rates from cataract surgery. Both diabetes and cataract pose an enormous health and economic burden, particularly in developing countries, where diabetes treatment is insufficient and cataract surgery often in accessible.



Abstract - Bio – 09

Collagen

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Collagen is the main structural protein of the various connective tissues in animals. As the main component of connective tissue, it is the most abundant protein in mammals, making up from 25% to 35% of the whole-body protein content. Collagen, in the form of elongated fibrils, is mostly found in fibrous tissues such as tendons, ligaments and skin, and is also abundant in corneas, cartilage, bones, blood vessels, the gut, and intervertebral discs. The fibroblast is the most common cell which creates collagen. In muscle tissue, it serves as a major component of the endomysium. Collagen constitutes one to two percent of muscle tissue, and accounts for 6% of the weight of strong, tendinous muscles. Collagen is a part of the connective tissue that in the skin helps in firmness, suppleness and constant renewal of skin cells. Collagen is vital for skin elasticity. Collagen is a protein made up of amino-acids, which are in turn built of carbon, oxygen and hydrogen. Collagen contains specific amino acids – Glycine, Proline, Hydroxyproline and Arginine. These are tough and strong structures found all over the body: in bones, tendons and ligaments. In nature, collagen is found exclusively in animals, especially in the flesh and connective tissues of mammals. These are used widely for reconstruction of bone and a wide variety of dental, orthopedic and surgical purposes. Gelatin, which is used in food and industry, is collagen that has been irreversibly hydrolyzed. One thousand mutations have been identified in twelve out of more than twenty types of collagen. These mutations can lead to various diseases at the tissue level. The diseases like Osteogenesis imperfecta – Caused by a mutation in type 1 collagen, Chondrodysplasias – Skeletal disorder believed to be caused by a mutation in type 2 collagen, further research is being conducted to confirm this, Ehlers-Danlos Syndrome – Ten different types of this disorder which lead to deformities in connective tissue, some types can be lethal that lead to the rupture of arteries, each syndrome is caused by a different mutation, for example type four of this disorder is caused by a mutation in collagen type 3, Alport syndrome – Can be passed on genetically, usually as X-linked dominant, but also as both an autosomal dominant and autosomal recessive disorder, sufferers have problems with their kidneys and eyes, loss of hearing can also develop in during the childhood or adolescent years, Osteoporosis -brought on with age, associated with reduced levels of collagen in the skin and bones and Knobloch syndrome – Caused by a mutation in the collagen XVIII gene. Collagen has a wide variety of applications, from food to medical.



Abstract –Bio 10

Insulin Resistance

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Insulin resistance (IR) is a physiological condition in which cells fail to respond to the normal actions of the hormone insulin. The body produces insulin, but the cells in the body become resistant to insulin and are unable to use it as effectively, leading to hyperglycemia. Beta cells in the pancreas subsequently increase their production of insulin, further contributing to hyperinsulinemia. This often remains undetected and can contribute to a diagnosis of Type 2 Diabetes. One of insulin's functions is to regulate delivery of glucose into cells to provide them with energy. Insulin resistant cells cannot take in glucose, amino acids and fatty acids. Thus, glucose, fatty acids and amino acids 'leak' out of the cells. A decrease in insulin/glucagon ratio inhibits glycolysis which in turn decreases energy production. The resulting increase in blood glucose may raise levels outside the normal range and cause adverse health effects, depending on dietary conditions. Certain cell types such as fat and muscle cells require insulin to absorb glucose. When these cells fail to respond adequately to circulating insulin, blood glucose levels rise. The liver helps regulate glucose levels by reducing its secretion of glucose in the presence of insulin. This normal reduction in the liver's glucose production may not occur in people with insulin resistance. Insulin resistance in muscle and fat cells reduces glucose uptake (and also local storage of glucose as glycogen and triglycerides, respectively), whereas insulin resistance in liver cells results in reduced glycogen synthesis and storage and also a failure to suppress glucose production and release into the blood. Insulin resistance normally refers to reduced glucose-lowering effects of insulin. However, other functions of insulin can also be affected. For example, insulin resistance in fat cells reduces the normal effects of insulin on lipids and results in reduced uptake of circulating lipids and increased hydrolysis of stored triglycerides. Increased mobilization of stored lipids in these cells elevates free fatty acids in the blood plasma. Elevated blood fatty-acid concentrations (associated with insulin resistance and diabetes mellitus Type 2), reduced muscle glucose uptake, and increased liver glucose production all contribute to elevated blood glucose levels. High plasma levels of insulin and glucose due to insulin resistance are a major component of the metabolic syndrome. If insulin resistance exists, more insulin needs to be secreted by the pancreas. If this compensatory increase does not occur, blood glucose concentrations increase and type 2 diabetes occurs.



Abstract - Bio - 11

Ageing

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Ageing is the accumulation of changes in a person over time. Ageing in humans refers to a multidimensional process of physical, psychological, and social change. Some dimensions of ageing grow and expand over time, while others decline. Reaction time, for example, may slow with age, while knowledge of world events and wisdom may expand. Research shows that even late in life, potential exists for physical, mental, and social growth and development. Ageing is an important part of all human societies reflecting the biological changes that occur, but also reflecting cultural and societal conventions. Roughly 100,000 people worldwide die each day of age-related causes. Age is measured chronologically and a person's birthday is often an important event. However the term "ageing" is somewhat ambiguous. Distinctions may be made between "universal ageing" (age changes that all people share) and "probabilistic ageing" (age changes that may happen to some, but not all people as they grow older including diseases such as type two diabetes). Chronological ageing may also be distinguished from "social ageing" (cultural age-expectations of how people should act as they grow older) and "biological ageing" (an organism's physical state as it ages). There is also a distinction between "proximal ageing" (age-based effects that come about because of factors in the recent past) and "distal ageing" (age-based differences that can be traced back to a cause early in person's life, such as childhood poliomyelitis). Differences are sometimes made between populations of elderly people. Divisions are sometimes made between the young old (65–74), the middle old (75–84) and the oldest old (85+). However problematic this may be, chronological age does not correlate perfectly with functional age, i.e. two people may be of the same age, but differ in their mental and physical capacities. Each nation, government and non-government organisation has different ways of classifying age. Population ageing is the increase in the number and proportion of older people in society. Population ageing has three possible causes: migration, longer life expectancy (decreased death rate) and decreased birth rate. Ageing has a significant impact on society. Young people tend to commit most crimes, they are more likely to push for political and social change, to develop and adopt new technologies and to need education, the latter of which tend to lose political significance for people in the ageing process. Older people have different requirements from society and government as opposed to young people and frequently differing values as well, such as for property and pension rights.

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Abstract - Bio - 012

Multiple Myeloma

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Multiple myeloma is a plasma cell cancer, constitutes about 1% of all cancers affecting the population. It usually occurs in the age group of 45-60 years. Multiple myeloma is due to the malignancy of a single clone of plasma cells in bone marrow, causing over production of abnormal immunoglobulins. As the production of normal immunoglobulins is reduced, it causes depressed immunity. Most cases of myeloma also feature the production of a paraprotein—an abnormal antibody which can cause kidney problems. Bone lesions and hypercalcemia (high blood calcium levels) are also often encountered. The underlying pathophysiologic phenomena of the clinical features include suppression of humoral- and cell-mediated immunity, elevation of IL-6, abnormalities of the bone marrow microenvironment, and increased osteoclastic activity. Overwhelming predictors of prognosis include albumin, β_2 -microglobulin, and chromosomal karyotype. Myeloma is diagnosed with blood tests (serum protein electrophoresis, serum free kappa/lambda light chain assay), bone marrow examination, urine protein electrophoresis, and X-rays of commonly involved bones. The plasma of these patients shows a characteristic electrophoretic pattern with sharp and distinct band (M band) between β and γ globulins. The patients release Bence Jones proteins in the urine. These patients show symptoms of amyloidosis. Some symptoms of the multiple myeloma are bone pain, renal failure, recurring infections and anemia. Radiation therapy is sometimes used to reduce pain from bone lesions. With modern, intensive therapy including autologous hematopoietic stem cell transplantation, the median survival is approximately 5 yr. The disease is incurable and eventually relapses; requiring salvage therapy. It is treated with chemotherapy. A commonly used chemotherapy regimen is the Vincristine, Adriamycin, and Dexamethasone.



Abstract - Bio - 13

Lipoprotein Metabolism

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The handling of lipoproteins in the body is referred to as lipoprotein metabolism. It is divided into two pathways, exogenous and endogenous, depending in large part on whether the lipoproteins in question are composed chiefly of dietary (exogenous) lipids or whether they originate from epithelial cells lining the small intestine readily absorb lipids from the diet. These lipids, including triglycerides, phospholipids, and cholesterol, are assembled with apolipoprotein B-48 into chylomicrons. These nascent chylomicrons are secreted from the intestinal epithelial cells into the lymphatic circulation in a process that depends heavily on apolipoprotein B-48. As they circulate through the lymphatic vessels, nascent chylomicrons bypass the liver circulation and are drained via the thoracic duct into the bloodstream. In the bloodstream, HDL particles donate apolipoprotein C-II and apolipoprotein E to the nascent chylomicron; the chylomicron is now considered mature. Via apolipoprotein C-II, mature chylomicrons activate lipoprotein lipase (LPL), an enzyme on endothelial cells lining the blood vessels. LPL catalyzes the hydrolysis of triacylglycerol (i.e. glycerol covalently joined to three fatty acids) that ultimately releases glycerol and fatty acids from the chylomicrons. Glycerol and fatty acids can then be absorbed in peripheral tissues, especially adipose and muscle, for energy and storage. The hydrolyzed chylomicrons are now considered chylomicron remnants. This interaction causes the endocytosis of the chylomicron remnants, which are subsequently hydrolyzed within lysosomes. Lysosomal hydrolysis releases glycerol and fatty acids into the cell, which can be used for energy or stored for later use. VLDL is synthesized in the liver (endogenous). As in chylomicron metabolism, the apolipoprotein C-II and apolipoprotein E of VLDL particles are acquired from HDL particles. Once loaded with apolipoproteins C-II and E, the nascent VLDL particle is considered mature. Again like chylomicrons, VLDL particles circulate and encounter LPL expressed on endothelial cells. Apolipoprotein C-II activates LPL, causing hydrolysis of the VLDL particle and the release of glycerol and fatty acids. A receptor and apolipoprotein B-100 or E on the LDL particle.



Abstract - Bio - 14

Insulin Inhalers

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Inhalable insulin was available from September 2006 to October 2007 in the United States as a new method of delivering insulin, a drug used in the treatment of diabetes, to the body. After the withdrawal of the only inhalable formulation, all currently available insulin formulations are administered by subcutaneous or intravenous injection. The first such product to be marketed was Exubera, a powdered form of recombinant human insulin, delivered through an inhaler into the lungs where it is absorbed. Once it has been absorbed, it begins working within the body over the next few hours. Type 1 diabetics still need to take a longer acting basal insulin by injection. A systematic review concluded that inhaled insulin "appears to be as effective, but no better than injected short-acting insulin. The additional cost is so much more that it is unlikely to be cost-effective. In October 2007, Pfizer announced that it would be discontinuing the production and sale of Exubera due to poor sales. Several other companies are developing inhaled forms of the drug to reduce the need for daily injections among diabetics. Diabetes sufferers may soon be able to inhale life-saving drugs rather than having to give themselves several daily injections, according to research published today. An American study of a new inhaler device for insulin found that it worked as well as the traditional method of injections. The findings could offer hope to diabetes sufferers who have to go through the discomfort and inconvenience of two or three daily jabs to maintain their blood glucose levels. Around 1.4 million people in the UK are diagnosed with diabetes. An estimated million more have the condition but are not aware of it. Half a million diabetics have to give themselves daily injections of insulin to maintain their glucose levels every day. Diabetes sufferers cannot convert the glucose in their blood into energy because the hormone insulin is either not produced or does not work properly. Sufferers say the gruelling regime affects their everyday life, social relations and even their own self-image. Insulin inhalers have been tried from as early as 1925, but the devices have not been effective enough in getting the insulin into the body's system.



**DEPARTMENT OF MICROBIOLOGY
ORAL PRESENTATION**

Abstract – Micro - 01

Microbial Ecology In The Root Canal

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Understanding of microbial ecology of the pulp-dentine complex is important because it provides a rational basis for disease prevention and treatment. Endodontic microorganisms can be acquired via traumatic damage, failing pulpal restorations, the apical foramen and a multitude of dentinal tubules and accessory/lateral canals. Survival within the endodontium is determined by the microenvironment; for example, Eh, pH, absence of sugars, other microbes and host defences. Compared with other sites in the mouth, the endodontium is a highly selective environment so that the flora differs from that seen in other sites in the oral cavity. Very little is known presently of effects of localised endodontic microenvironments on specific species. The endodontic flora affects other environments such as the periodontium and leads to periapical and more far-reaching infection. Treatment is designed to render the root canal and surrounding dentine less hospitable to infecting microorganisms. Some species are closely associated with endodontic infection, e.g. *Mitsuokella dentalis*, *P. endodontalis*, *P. nigrescens*. However these particular species are not always present by any means. Other bacteria have been associated with endodontic disease features, including *P. melaninogenica* and *Peptostreptococcus micros* with pain and swelling. In refractory cases, other species may achieve importance in the altered environment of the treated dental root canal; the facultative and highly chemically resistant bacterium *Enterococcus faecalis* is one such microorganism. Fundamental to our understanding of oral microbial disease is the important role of microbial ecology within the oral cavity. The relevance to clinical disease of endodontic microbial ecology is clear. 'The principles of endodontic treatment require the control of microorganisms and potential nutrients by the microbial decontamination of teeth...and the sealing of dentine to prevent recolonisation'. This review examines the endodontic environment from the point of view of factors affecting microbiota as well as the effect of microorganisms on the host.



Abstract – Micro – 02

Antibiotic Policy

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Antibiotic policy is a key issue for both better care of patients and combating antimicrobial resistance. Antibiotic use is complex and requires coordination of the activities of health care authorities, institutions and individual practitioners. Furthermore, on a community basis it involves restriction of non-human usage of antibiotics, awareness of society about both the useful and harmful effects of antibiotics and on a national basis, participation of governmental and non-governmental organizations. Institutional, regional, national and global aspects of antibiotic policies should be considered. Antibiotic policies should be established in each country. They should consider both hospital and primary care settings as well as veterinary and agriculture use. Guidelines should be widely discussed through professional meetings of multidisciplinary groups, involving clinicians, microbiologists and pharmacologists/pharmacists. Some important issues to be included are : - Existing laws should be enforced to prevent non-prescription over the counter (OTC) sale of antibiotics - A national antimicrobial resistance surveillance system should be established and co-ordinated with international surveillance systems - A national control of infections programme should be implemented - Educational programs should be elaborated: - for health care practitioners including veterinarians, junior doctors, nurses, medical students - for the wider audience (antibiotic consumers) - Collaboration should be established with appropriate International organizations like WHO, APUA, the Medical Council of the European Commission (EC), ESGAP and pharmaceutical companies. An effective antibiotic policy also provides and ensures education on the use of antibiotics at undergraduate and postgraduate level for medical and nursing staff. The educational programme should teach how to critically evaluate and assess new drugs and provide education on the use and mis-use of antibiotics to hospital staff and practising physicians. This will reduce inappropriate prescribing. The programme will instruct in correct dosage, route and frequency from the point of view of cost effectiveness, and provide information to prescribers on the impact of their decisions on both economics and bacterial ecology.



Abstract – Micro - 03

Adhesins

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Adhesins are cell-surface components or appendages of bacteria that facilitate adhesion or adherence to other cells or to surfaces. Adhesins are a type of virulence factor. The majority of bacterial pathogens exploit specific adhesion to host cells as their main virulence factor. “A large number of bacterial adhesins with individual receptor specificities have been identified.” Many bacterial pathogens are able to express an array of different adhesins. Expression of these adhesins at different phases during infection play the most important role in adhesion based virulence. Numerous studies have shown that inhibiting a single adhesin in this coordinated effort can often be enough to make a pathogenic bacterium non-virulent. This has led to the exploration of adhesin activity interruption as a method of bacterial infection treatment.

Bacteria are typically found attached to and living in close association with surfaces. During the bacterial lifespan, a bacterium is subjected to frequent shear-forces. In the crudest sense, bacterial adhesins serve as anchors allowing bacteria to overcome these environmental shear forces, thus remaining in their desired environment. They act as specific surface recognition molecules, allowing the targeting of a particular bacterium to a particular surface such as root tissue in plants, lacrimal duct tissues in mammals, or even tooth enamel. Most fimbria of gram-negative bacteria function as adhesins, but in many cases it is a minor subunit protein at the tip of the fimbriae that is the actual adhesin. In gram-positive bacteria, a protein or polysaccharide surface layer serves as the specific adhesin. To effectively achieve adherence to host surfaces, many bacteria produce multiple adherence factors called adhesins.

Bacterial adhesins provide species and tissue tropism. Adhesins are expressed by both pathogenic bacteria and saprophytic bacteria. This prevalence marks them as key microbial virulence factors in addition to a bacterium’s ability to produce toxins and resist the immune defenses of the host.



Abstract – Micro - 04

Mitochondrial Dna- A Surprise

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Mitochondria are sub cellular organelles that are found in the cytoplasm of the eukaryote cells and their principal function is the production of cellular energy. Mitochondria provide about 90 percent of the energy that cells-and thus tissues, organs and the body as a whole-need to function. They convert the energy from food into a form that cells can use, and are hence referred to as the "powerhouses". Mitochondria produce energy through a process called oxidative phosphorylation. This process uses oxygen and simple sugars to create adenosine triphosphate (ATP), the cell's main energy source. Each cell contains hundreds to thousands of mitochondria. One of the particularities of these organelles is that they have a genetic system of their own with all the machinery necessary for their expression; that is, to replicate, transcribe and translate the genetic information they contain. This genetic material is known as mitochondrial DNA or mtDNA

Mitochondrial genome consist of a single circular molecule of DNA that resembles that of bacteria not that of the nuclear genome. Mitochondria have striking similarities to bacteria cells. This fact can be explained by endosymbiotic theory. The endosymbiosis theory postulates that the mitochondria of eukaryotes were evolved from aerobic bacteria living within their host cell. Symbiosis occurs when two different species benefit from living and working together. When one organism actually lives inside the other it's called endosymbiosis. The endosymbiotic theory describes how a large host cell and ingested bacteria could easily become dependent on one another for survival, resulting in a permanent relationship. Over millions of years of evolution, mitochondria have become more specialized and today they cannot live outside the cell

A double membrane that surrounds mitochondria further provides evidence that they were ingested by a primitive host. The organelles also reproduce like bacteria, replicating their own DNA and directing their own division. Mitochondrial metabolism is a critical component in the functioning and maintenance of cellular organs. The presence of mitochondrial DNA (mtDNA) is responsible for the encoding of proteins required for oxidative phosphorylation. Mitochondrial DNA (mtDNA) variations have been implicated in a broad spectrum of disease. Mitochondrial



disorders may be caused by mutations, acquired or inherited, in mitochondrial DNA (mtDNA) or in nuclear genes that code for mitochondrial component.

Abstract – Micro – 05

Prions

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Prions are unprecedented infectious pathogens that cause a group of invariably fatal neurodegenerative diseases by an entirely novel mechanism. Prion diseases may present as genetic, infectious, or sporadic disorders, all of which involve modification of the prion protein (PrP). Bovine spongiform encephalopathy (BSE), scrapie of sheep, and Creutzfeldt–Jakob disease (CJD) of humans are among the most notable prion diseases. Prions are transmissible particles that are devoid of nucleic acid and seem to be composed exclusively of a modified protein (PrP^{Sc}). The normal, cellular PrP (PrP^C) is converted into PrP^{Sc} through a posttranslational process during which it acquires a high sheet content. The species of a particular prion is encoded by the sequence of the chromosomal PrP gene of the mammals in which it last replicated. In contrast to pathogens carrying a nucleic acid genome, prions appear to encipher strain-specific properties in the tertiary structure of PrP^{Sc}. While knowledge about prions has profound implications for studies of the structural plasticity of proteins, investigations of prion diseases suggest that new strategies for the prevention and treatment of these disorders may also find application in the more common degenerative diseases. Transmissible spongiform encephalopathies (TSEs) are inevitably lethal neurodegenerative diseases that affect humans and a large variety of animals. The infectious agent responsible for TSEs is the prion, an abnormally folded and aggregated protein that propagates itself by imposing its conformation onto the cellular prion protein (PrP^C) of the host. PrP^C is necessary for prion replication and for prion-induced neurodegeneration, yet the proximal causes of neuronal injury and death are still poorly understood. Prion diseases such as Creutzfeldt–Jakob disease (CJD) are incurable and rapidly fatal neurodegenerative diseases. Because prion protein (PrP) is necessary for prion replication but dispensable for the host, we developed the PrP–FRET-enabled high throughput assay (PrP–FEHTA) to screen for compounds that decrease PrP expression. The prion-degrading reagents identified in this study are readily available, inexpensive, non-corrosive to instruments, non-hazardous to staff and compatible with current equipment and procedures used in hospital sterilization units.

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Abstract – Micro - 06

Resurgence Of Chikungunya

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Chikungunya virus (CHIKV) is an arthropod-borne virus, of the genus Alphavirus, that is transmitted to humans by virus-carrying Aedes mosquitoes. There have been recent breakouts of CHIKV associated with severe illness. CHIKV infection causes an illness with a similar mode of transmission as dengue fever, with an acute febrile phase lasting two to five days, followed by a longer period of joint pains in the extremities. The pain associated with CHIKV infection of the joints may persist for weeks or months, or in some cases years. Prevention is via mosquito control and preventing bite by infected mosquitoes. There is no specific treatment with medications used to help with symptoms. Chikungunya is a specifically tropical disease. It is relatively uncommon and poorly documented. The high morbidity and loss in daily activity associated with CHIKV infection results in considerable economic loss among the affected nations, specifically in India. The incubation period of chikungunya disease ranges from one to twelve days, usually two to three. Its symptoms include a fever up to 40 °C (104 °F), a petechial or maculopapular rash of the trunk and occasionally the limbs, and arthralgia or arthritis affecting multiple joints. Other nonspecific symptoms can include headache, nausea, vomiting, conjunctivitis, slight photophobia and partial loss of taste. Ocular inflammation from Chikungunya may present as iridocyclitis, and have retinal lesions as well. Pedal oedema (swelling of legs) is observed in many patients, the cause of which remains obscure as it is not related to any cardiovascular, renal or hepatic abnormalities. Typically, the fever lasts for two days and then ends abruptly. Patients have complained of joint pains for much longer time periods; some as long as two years, depending on their age. Recovery from the disease varies by age. The severity of the disease as well as its duration is less in younger patients and pregnant women. Chikungunya fever though expected to be related to monsoon and post monsoon seasons is showing its presence throughout the year and involves all age groups. It is also conceivable that chikungunya virus never disappeared entirely from the Indian subcontinent, and that the current outbreak is because of a simple resurgence.



Abstract – Micro - 07

Battle Against HIV

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The human immunodeficiency virus (HIV) is a lentivirus (slowly replicating retrovirus) that causes the acquired immunodeficiency syndrome (AIDS), a condition in humans in which progressive failure of the immune system allows life-threatening opportunistic infections and cancers to thrive. Infection with HIV occurs by the transfer of blood, semen, vaginal fluid, pre-ejaculate, or breast milk. Within these bodily fluids, HIV is present as both free virus particles and virus within infected immune cells. HIV infects vital cells in the human immune system such as helper T cells (specifically CD4+ T cells), macrophages, and dendritic cells. HIV infection leads to low levels of CD4+ T cells through a number of mechanisms including: apoptosis of uninfected bystander cells, direct viral killing of infected cells, and killing of infected CD4+ T cells by CD8 cytotoxic lymphocytes that recognize infected cells. When CD4+ T cell numbers decline below a critical level, cell-mediated immunity is lost, and the body becomes progressively more susceptible to opportunistic infections.

There has been great success on the HIV/AIDS battlefield. Where deaths were once so commonplace that a diagnosis had victims making funeral arrangements and getting their wills in order, the emergence of effective anti-retroviral drugs has made HIV infection a treatable chronic disease. The prevalence of HIV was strongly over-estimated for a long time because of inadequate national epidemiological systems. Improved data led to a sharp downward revision of the extent of the pandemic in 2007. The estimated prevalence of HIV was reduced by 33 per cent globally and 36 per cent for sub-Saharan Africa. The improvement of national surveillance systems in Africa primarily took the form of investment in population studies as a supplement to data from pregnant women attending antenatal clinics. The data were also improved in countries where HIV is concentrated in particular risk groups. HIV prevalence trends amongst young people provide a good indication of trends in incidence (rate of new infections) in epidemics that are generalised, and more reliable prevalence data permit more reliable estimation of incidence.



Abstract – Micro - 08

Infection Control Systems In Hospital

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Children and young people can be at a higher risk of getting an infection when they are ill. The body has natural defence mechanisms to fight off infections, but these may be affected for a variety of reasons when someone is ill. For example, when a child has an operation, the surgical wound means that the natural skin barrier is broken, which could allow bacteria (germs) to enter the body. Bacteria and viruses (germs) may come from other patients, staff, visitors (including siblings), equipment or the environment. Nosocomial infections—known also as hospital-acquired infections, hospital-associated infections, and hospital infections—are infections that are not present in the patient at the time of admission to hospital but develop during the course of the stay in hospital. Healthy people are naturally contaminated. Faeces contain about 10¹³ bacteria per gram, and the number of microorganisms on skin varies between 100 and 10000 per cm². Many species of microorganisms live on mucous membranes where they form a normal flora. None of these tissues, however, is infected. Microorganisms that penetrate the skin or the mucous membrane barrier reach subcutaneous tissue, muscles, bones, and body cavities (e.g. peritoneal cavity, pleural cavity, bladder), which are normally sterile (i.e. contain no detectable organisms). If a general or local reaction to this contamination develops, with clinical symptoms, there is an infection. Early detection of new or novel variants of nosocomial pathogens is a public health priority. We apply recently developed mathematical models to patient admission data from the national healthcare systems of England and The Netherlands. Relatively short detection times are achieved once 10-20% hospitals are recruited as sentinels and only modest reductions are seen as more hospitals are recruited thereafter. Using a heuristic optimisation approach to sentinel selection, the same expected time to detection can be achieved by recruiting approximately half as many hospitals. Our study provides a robust evidence base to underpin the design of an efficient sentinel hospital surveillance system for novel nosocomial pathogens, delivering early detection times for reduced expenditure and effort. The results indicate negligence in various aspects of infection control. Providing staff education and training programs, establishing effective surveillance systems, and enforcing regulations in the hospitals should help improve infection prevention



DEPARTMENT OF MICROBIOLOGY

POSTER PRESENTATION

Abstract – Micro - 01

Bacteremia

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Bacteremia is a life threatening issue. It means the bacteria has invaded the blood stream. Though bacteremia is a frequent issue most of them are transient and undetectable. Bacteremia can occur while brushing the teeth or while defecation. Bacteremia can occur through tooth extraction, tonsilectomy, through surgery, from septic foci externally or internally, by the invasive bacteria like salmonella. catheter-related bloodstream infections, intra-abdominal infections, pneumonia, pyelonephritis and skin and soft-tissue infections. In any study the gram negative bacteria dominates in any population. It is a cause of concern that the organisms isolated are showing drug resistance. Presence of MDR, ESBL, metalloproteases, VRE, HA- MRSA, CA-MRSA are increasing the case fatality rate. Prompt diagnosis and treating the patients with the sensitive drug is very important. Many cases of bacteremia are diagnosed at the end stage of the illness. Prompt antibiotic therapy usually succeeds in clearing bacteria from the bloodstream but the recurrence is a common issue that arises from the untreated septic foci internally. Though the bacteremia is detected by the BACTEC and other methods the treatment is dependent on the antibiogram pattern of the bacteria isolated. Conditions which increase the chances of developing bacteremia include immune suppression, either due to HIV infection or drug therapy, antibiotic therapy which changes the balance of bacterial types in the body, prolonged or severe illness, alcoholism or other drug abuse, malnutrition, diseases or drug therapy that cause ulcers in the intestines. *Acinetobacter baumannii* (30 – 40 %) is the most common among the non fermentors isolated from the bacteremia cases. In many recent studies *Acinetobacter baumannii* dominates all the isolates from the ICU patients and inpatients with medical supportive devices especially in old age or in immunocompromised. Incidence of *Acinetobacter baumannii* can be controlled by strict aseptic procedure, good sanitary control and a controlled antibiotic policy.



Abstract – Micro - 02

Emerging Pathogenic Virus

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Viruses are defined as the smallest infectious agent that do not have a cellular organization and have either RNA or DNA as their genetic element. Viruses are prone to genetic modification because of their host cell dependent replicative cycle. Genetic modifications at times shift their target of attack within their natural host or can even target a newer host. Thus the newer virus emerges. The term became popular in 1990s, but emerging viruses are not new. Certain viruses are natural among non human host. The entry of such viruses in to the human population may establish themselves as a human pathogens. Existence of such viruses in a common pool may also lead to gene contamination and emergence of a new strain. Such modifications are common with influenza virus natural to human, swine and avian. Emerging viruses are known to have existed for centuries with little incidence. Adaptation to new hosts and environments through variation and selection is one of the reasons. The incidence of vaccines contaminated with animal or avian viruses has been documented in the past. The viruses which are natural to other species thus spread to the human population. Large scale changes in ecology due to global warming, deforestation or afforestation, building of dams or canals, changed agricultural practices, rearing of livestock or birds may also contribute to emergence of viral diseases. New viruses and virus-related diseases have threatened the health of humans and many animal species. The viruses will behave differently when entered into different species. It is the slow onset of disease that can be particularly raising query. The delayed onset of chronic debilitating diseases that could be associated with animal viruses finding their way into a new species, e.g., man, are much more challenging. The Ebola virus, Hanta virus or HIV can be mentioned as new. Viral haemorrhagic fever caused by Arbo virus transmitted from rodents frequent outcome of cross-species infection, but not of intraspecies infections often host is killed so quickly that there is little or no transmission to others. Alternatively, the new infected host cannot transmit the infection to others of the same species, contribute little to the spread of a natural infection. An international network of databases of virus infections needs to be instituted



Abstract – Micro - 03

Join Me To Make India Free From Hiv/ Aids – The Onus Is On Us

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Human immunodeficiency virus infection / acquired immunodeficiency syndrome(HIV/AIDS) is a disease of the human immune system caused by infection with human immunodeficiency virus (HIV). During the initial infection, a person may experience a brief period of influenza-like illness. This is typically followed by a prolonged period without symptoms. As the illness progresses, it interferes more and more with the immune system, making the person much more likely to get infections, including opportunistic infections and tumors that do not usually affect people who have working immune systems. HIV infection was identified in the early part of the decade. Since its discovery, AIDS has caused an estimated 36 million deaths . Approximately 35.3 million people are living with HIV globally.HIV/AIDS is considered a pandemic, a disease outbreak which is present over a large area and is actively spreading. HIV is transmitted by three main routes: sexual contact, exposure to infected body fluids or tissues and from mother to child during pregnancy, delivery, or breastfeeding (known as vertical transmission). There is no risk of acquiring HIV if exposed to feces, nasal secretions, saliva, sputum, sweat, tears, urine, or vomit unless these are contaminated with blood. It is possible to be co-infected by more than one strain of HIV a condition known as HIV super infection. HIV is common in the high risk group occurring at Blood transfusion. Consistent condom use reduces the risk of HIV transmission by approximately 80% over the long term. Programs encouraging sexual abstinence do not appear to affect subsequent HIV risk. Evidence for a benefit from peer education is equally poor. Comprehensive sexual education provided at school may decrease high risk behavior. A substantial minority of young people continues to engage in high-risk practices despite knowing about HIV/AIDS, underestimating their own risk of becoming infected with HIV. Universal precautions within the health care environment are believed to be effective in decreasing the risk of HIV. With respect to dietary advice and AIDS, some evidence has shown a benefit from micronutrient supplements. Evidence for supplementation with selenium is mixed with some tentative evidence of benefit. The WHO further states that several studies indicate that supplementation of vitamin A, zinc, and iron can produce adverse effects in HIV positive adults. There is not enough evidence to support the use of herbal medicines



**DEPARTMENT OF PATHOLOGY
ORAL PRESENTATION**

Abstract - Path - 01

Inflammation And Pulp Pathology

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Dental pulp is a connective tissue situated in a low-compliance environment represented by mineralized dentin. The pulp is considered together with the dentin as the pulpo-dentin complex due to anatomic, developmental, and functional relationships. The extracellular matrix is the major constituent of the connective tissue. This is composed of ground substance and fibrillar proteins such as collagen and elastin. Ground substance is mainly composed of macromolecules called proteoglycans and glycosami-noglycans. The main cells of the connective tissue are the fibroblasts. The pulp also contains odontoblasts (the highest differentiated cells), undifferentiated mesenchymal cells, and immunocompetent cells (lymphocytes, macrophages, dendritic cells). The inflammatory process in human dental pulp consists in vascular changes and migration of inflammatory cells to the site of inflammation. No mast cells are normally present in human dental pulp, but they are known as active cells in the inflammatory response. According to the studies of Miller, Sternberg, in 1978, mast cells are occasionally found in inflamed pulp, although degranulation cannot be observed histologically because these cells lose their characteristic features after degranulation. Vascular proliferation in the perinervous space of human dental pulp is an aspect that supplements the description of the angiogenesis model, and remains to be clarified. The presence of calcium carbonate and phosphate deposits in pulpal tissue is frequently observed, and it sometimes represents a major problem of the endodontic treatment. According to Weine, 1989, calcifications are found in both healthy and aging pulps, although their incidence increases with age. False denticles are formed when a degenerating tissue structure serves as a nidus for deposition of concentric layers of calcified tissue. Pulp degeneration - condition seen in older people where persistent and mild irritation to the pulp leads to generation of pulp. It may be induced by irritation, attrition of teeth. Necrosis of pulp is death of pulp which may be partial or complete. It may be either coagulative or liquefaction type of necrosis.



Abstract - Path - 02

Tuberculosis

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Tuberculosis (TB), infectious disease that is caused by the tubercle bacillus, *Mycobacterium tuberculosis*. In most forms of the disease, the bacillus spreads slowly and widely in the lungs, causing the formation of hard nodules (tubercles) or large cheeselike masses that break down the respiratory tissues and form cavities in the lungs. Blood vessels also can be eroded by the advancing disease, causing the infected person to cough up bright red blood. Today, in less-developed countries where population is dense and hygienic standards poor, tuberculosis remains a major fatal disease. The prevalence of the disease has increased in association with the HIV/AIDS epidemic; an estimated one out of every four deaths from tuberculosis involves an individual coinfecting with HIV. In addition, the successful elimination of tuberculosis as a major threat to public health in the world has been complicated by the rise of new strains of the tubercle bacillus that are resistant to conventional antibiotics. Symptoms may include chest pain and a prolonged cough producing sputum. About 25% of people may not have any symptoms (i.e. they remain "asymptomatic"). Occasionally, people may cough up blood in small amounts, and in very rare cases, the infection may erode into the pulmonary artery, resulting in massive bleeding (Rasmussen's aneurysm). Tuberculosis may become a chronic illness and cause extensive scarring in the upper lobes of the lungs. The upper lung lobes are more frequently affected by tuberculosis than the lower ones. The reason for this difference is not entirely clear. It may be due either to better air flow, or to poor lymph drainage within the upper lungs. Treatment of TB uses antibiotics to kill the bacteria. Effective TB treatment is difficult, due to the unusual structure and chemical composition of the mycobacterial cell wall, which hinders the entry of drugs and makes many antibiotics ineffective. The two antibiotics most commonly used are isoniazid and rifampicin, and treatments can be prolonged, taking several months. Latent TB treatment usually employs a single antibiotic while active TB disease is best treated with combinations of several antibiotics to reduce the risk of the bacteria developing antibiotic resistance. People with latent infections are also treated to prevent them from progressing to active TB disease later in life. Directly observed therapy, i.e. having a health care provider watch the person take their medications, is recommended by the WHO in an effort to reduce the number of people not appropriately taking antibiotics. The evidence to support this practice over people simply taking their medications independently is poor.



Abstract - Path - 03

Recent Advances In Cancer Diagnosis

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Malignant transformation of cells into cancer arises due to long term accumulation of genetic and epigenetic events. Early diagnosis of these transformations in cells can improve the prognosis of cancer cases. Cancer screening and surveillance methods include ultrasound, mammography, digital mammography, magnetic resonance imaging, computed tomography, positron emission tomography and magnetic resonance spectroscopy. Other techniques such as immunohistochemistry, in situ hybridization (FISH, CSH), PCR, RT-PCR (real time- PCR), flow cytometry and microarray are used nowadays for diagnosis. Microarray technology is a new and efficient approach to extract data of biomedical relevance for a wide range of applications. In cancer research, it will provide high-throughput and valuable insights into differences in an individual's tumor as compared with constitutional DNA, mRNA expression, and protein expression and activity. This review highlights the recent developments in cancer diagnostic technologies and describes the eventual use of these technologies for clinical and research applications. Tumor markers are biologic or biochemical substances produced by tumors and secreted into body fluids or present on body tissues in higher than normal amounts. Gold *et al.*, isolated a glycoprotein molecule from specimens of human colonic cancer and thus discovered the first "tumor antigen", later identified as carcino-embryonic antigen (CEA). Today there are literally hundreds of tumor markers, although their clinical utility is under investigation. Tumor markers can be detected by various methods including antigen-antibody based techniques (ELISA – enzyme linked immunosorbant assay, radio-immunoassay, precipitin tests, flow-cytometry, immunohistochemistry, immunoscintigraphy) and molecular genetic methods. Measurement of tumormarkers levels, when used along with other diagnostic tests, can be useful in the detection and diagnosis of some type of cancers. However, in most instances tumor marker levels alone are not sufficient to diagnose cancer for example, in patients with cirrhosis or viral hepatitis may have abnormal Alpha-Fetoprotein (AFP) values, although usually less than 500 ng per mL but pregnancy also associated with elevated AFP levels, particularly if the pregnancy is complicated by a spinalcord defect or other abnormality . No simple tests are yet available with sufficient sensitivity and specificity to detect the presence of a cancer. The field of tumor markers is ever expanding with many new candidate markers either in clinical use or under active evaluation.



Abstract - Path - 04

Osteomyelitis

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Osteomyelitis is a common infectious disease among elderly patients. Older adults are predisposed to osteomyelitis either because of an increased incidence of associated disorders that predispose to osteomyelitis e.g., peripheral vascular disease, diabetes mellitus, and poor dentition or because of surgical procedures that are frequently performed in the elderly population e.g., dental extractions, open-heart surgery, and prosthetic joint replacement. As with osteomyelitis in other age groups, osteomyelitis in the elderly population may also be considered in terms of acuteness of the infectious process acute osteomyelitis, subacute osteomyelitis, or chronic osteomyelitis. Osteomyelitis may be caused by a variety of microorganisms, but osteomyelitis in the elderly population is most often caused by pyogenic organisms. In general, microorganisms may infect bone through one or more of three basic methods: via the bloodstream, contiguously from local areas of infection (as in cellulitis), or penetrating trauma, including iatrogenic causes such as joint replacements or internal fixation of fractures or root-filled teeth. Once the bone is infected, leukocytes enter the infected area, and, in their attempt to engulf the infectious organisms, release enzymes that lyse the bone. Pus spreads into the bone's blood vessels, impairing their flow, and areas of devitalized infected bone, known as sequestra, form the basis of a chronic infection. Often, the body will try to create new bone around the area of necrosis. The resulting new bone is often called an involucrum. On histologic examination, these areas of necrotic bone are the basis for distinguishing between acute osteomyelitis and chronic osteomyelitis. Osteomyelitis is an infective process that encompasses all of the bone (osseous) components, including the bone marrow. When it is chronic, it can lead to bone sclerosis and deformity. Osteomyelitis often requires prolonged antibiotic therapy, with a course lasting a matter of weeks or months. A PICC line or central venous catheter is often placed for this purpose. Osteomyelitis also may require surgical debridement. Severe cases may lead to the loss of a limb. Initial first-line antibiotic choice is determined by the patient's history and regional differences in common infective organisms. A treatment lasting 42 days is practiced in a number of facilities. Local and sustained availability of drugs have proven to be more effective in achieving prophylactic and therapeutic outcomes. Hyperbaric oxygen therapy has been shown to be a useful adjunct to the treatment of refractory osteomyelitis. Open surgery is needed for chronic osteomyelitis, whereby the involucrum is opened and the sequestrum is removed or sometimes saucerization can be done.



Abstract - Path - 05

Oral Manifestation Of Systemic Disease

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Careful examination of the oral cavity may reveal findings indicative of an underlying systemic condition, and allow for early diagnosis and treatment. Examination should include evaluation for mucosal changes, periodontal inflammation and bleeding, and general condition of the teeth. Oral findings of anemia may include mucosal pallor, atrophic glossitis, and candidiasis. Oral ulceration may be found in patients with lupus erythematosus, pemphigus vulgaris, or Crohn disease. Additional oral manifestations of lupus erythematosus may include honeycomb plaques (silvery white, scarred plaques); raised keratotic plaques (verrucous lupus erythematosus); and nonspecific erythema, purpura, petechiae, and cheilitis. Additional oral findings in patients with Crohn disease may include diffuse mucosal swelling, cobblestone mucosa, and localized mucogingivitis. Diffuse melanin pigmentation may be an early manifestation of Addison disease. Severe periodontal inflammation or bleeding should prompt investigation of conditions such as diabetes mellitus, human immunodeficiency virus infection, thrombocytopenia, and leukemia. In patients with gastroesophageal reflux disease, bulimia, or anorexia, exposure of tooth enamel to acidic gastric contents may cause irreversible dental erosion. Severe erosion may require dental restorative treatment. In patients with pemphigus vulgaris, thrombocytopenia, or Crohn disease, oral changes may be the first sign of disease. Orofacial symptoms of Crohn disease include diffuse labial, gingival, or mucosal swelling; cobblestoning of the buccal mucosa and gingiva; aphthous ulcers; mucosal tags; and angular cheilitis. Noncaseating granulomas are characteristic of orofacial Crohn disease. Oral granulomas may occur without characteristic alimentary involvement (orofacial granulomatoses). The only manifestation of advanced liver disease visible in the oral mucosa is jaundice, which is the yellow pigmentation that results from the deposition of bilirubin in the submucosa. Jaundice may occur following disorders in bilirubin metabolism, production, or secretion. Mucosal conditions, such as glossitis, recurrent aphthae, candidal infections, and angular stomatitis may be more common in patients with anemia. Glossitis may be the first sign of folate or vitamin B-12 deficiency. The tongue appears reddened, and the papillae are atrophic, producing a smooth ("bald") appearance. Angular stomatitis is commonly caused by a candidal infection. Oral changes in Sjögren syndrome include difficulty in swallowing and eating, disturbances in taste and speech, increased dental caries, and a predisposition to infection, all due to a decrease in saliva. Oral manifestations of sarcoidosis may include multiple, nodular, painless ulcerations of the gingiva, buccal mucosa, labial mucosae, and palate.



Abstract - Path - 06

Premalignant Disorders Of Oral Cavity

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Oral cavity cancer accounts for approximately 3% of all malignancies and is a significant worldwide health problem. Most oral malignancies occur as squamous cell carcinomas (SCCs); despite remarkable advances in treatment modalities, the 5-year survival rate has not significantly improved over the past several decades and still hovers at about 50-60%. Many oral SCCs develop from premalignant conditions of the oral cavity. A wide array of conditions have been implicated in the development of oral cancer, including leukoplakia, erythroplakia, palatal lesion of reverse cigar smoking, oral lichen planus, oral submucous fibrosis, discoid lupus erythematosus, and hereditary disorders such as dyskeratosis congenital and epidermolysis bullosa. Despite the general accessibility of the oral cavity during physical examination, many malignancies are not diagnosed until late stages of disease. In order to prevent malignant transformation of these precursor lesions, multiple screening and detection techniques have been developed to address this problem. The early detection of cancer is of critical importance because survival rates markedly improve when the oral lesion is identified at an early stage. Oral submucous fibrosis (OSF) is a chronic progressive condition found predominantly in people of Asian descent. OSF is considered to be the result of the use of the Areca nut product with resultant disruption of the extracellular matrix. The disease often manifests with diffuse involvement of the oral cavity, pharynx, and upper esophagus that appears clinically as whitish mucosa lacking elasticity. Epithelial dysplasia has been described in 7-26% of OSF tissues, and long-term studies suggest a malignant transformation rate in approximately 7% of these lesions. Lichen planus (LP) is a disease of the skin and/or mucous membranes that resembles lichen. The cause is unknown, but it is thought to be the result of an autoimmune process with an unknown initial trigger. There is no cure, but many different medications and procedures have been used to control the symptoms. There is no cure for lichen planus, and so treatment of cutaneous and oral lichen planus is for symptomatic relief or due to cosmetic concerns. When medical treatment is pursued, first-line treatment typically involves corticosteroids, and removal of any triggers. Without treatment, most lesions will spontaneously resolve within 6–9 months for cutaneous lesions, and longer for mucosal lesions



Abstract - Path - 07

Malignant bony lesions of oral cavity

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Primary malignant tumours of the hard palate are rare. The hard palate has one of the highest concentrations of minor salivary glands in the upper aerodigestive tract. It is therefore not surprising that a large number of malignant neoplasms in this location are tumours of salivary gland origin (adenoid cystic carcinoma and mucoepidermoid carcinoma). As the mucosa of the hard palate is closely applied to the underlying bone, early osseous erosion is often encountered. It is therefore important to obtain coronal CT sections of the oral cavity for adequate staging of hard palate carcinoma. SCCs of the palate manifest as ulcerative surface lesions. Often, patients are asymptomatic in the early stages, but they may experience pain in advanced stages. A palate mass, bleeding, a foul odor, ill-fitting dentures in edentulous patients, or loose teeth may be the presenting symptoms for patients with hard palate cancer. In persons with advanced-stage soft palate cancers, velopharyngeal insufficiency, altered speech, difficulty swallowing, referred otalgia, trismus, or a neck mass may be present. Because the area is easily visualized, tumors are often found at early stages incidentally by the patient or the physician. The alveolar ridge is the area of your mouth just behind the top front teeth. Most alveolar ridge cancers are squamous cell carcinomas, meaning they arise from flat, thin cells in the epidermis lining the region. Unlike most head and neck cancers, alveolar ridge cancer is more common in women than in men. Symptoms can include loose teeth, bleeding, or pain that worsens when you chew. Osteosarcoma of jaw bones represents a distinct group of lesions from the conventional type commonly occurring in long bones. Nonetheless, our present knowledge of the tumor allows us to affirm that its clinical behavior and pathologic features differ markedly from those of its homolog in the long bones. The maxillary tumors show predilection for posterior portion of the alveolar process and the antrum, whereas the body is most commonly involved in the mandible followed, by angle, symphysis, and ascending ramus.



Abstract - Path - 08

Developmental lesions of hard tissues

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Otocephaly - This is a lethal condition in which the primary feature is the total or virtual absence of the lower jaw (a developmental anomaly called agnathia). The “oto” in the name refers to the relationship of the ears to the face in this disorder. The condition is considered lethal because of a poorly functioning airway. In otocephaly, agnathia may occur alone or together with holoprosencephaly. Micrognathism (or Micrognathia) is a condition where the jaw is undersized. It is also sometimes called "Mandibular hypoplasia". It is common in infants, but is usually self-corrected during growth, due to the jaws' increasing in size. It may be a cause of abnormal tooth alignment and in severe cases can hamper feeding. It can also, both in adults and children, make intubation difficult, either during anesthesia or in emergency situations. In some cases, the jaw is small enough to interfere with the infant's feeding. Infants with this condition may need special nipples in order to feed properly. Micrognathia often corrects itself during growth. The jaw may grow a lot during puberty. The problem can be caused by certain inherited disorders and syndromes. Micrognathia is can cause the teeth not to align properly. This can be seen in the way the teeth close. Often there will not be enough room for the teeth to grow. Children with this problem should see an orthodontist when the adult teeth come in. Because children may outgrow the condition, it often makes sense to delay treatment until a child is older. Macrognathia refers to the condition of abnormally large jaws. It is also called 'megagnathia'. Mandibular protrusion (when mandible is affected). Clinical presentation will be "Gummy smile" (when maxilla is affected), Ramus of mandible forms a less steep angle with body of mandible, Mandibular prognathism caused by excessive condylar growth and Chin appears prominent. Cleft lip (*cheiloschisis*) and cleft palate (*palatoschisis*), which can also occur together as cleft lip and palate, are variations of a type of clefting congenital deformity caused by abnormal facial development during gestation. A cleft is a fissure or opening—a gap. It is the non-fusion of the body's natural structures that form before birth. Approximately 1 in 700 children born have a cleft lip or a cleft palate or both. In decades past, the condition was sometimes referred to as harelip, based on the similarity to the cleft in the lip of a hare, but that term is now generally considered to be offensive. Clefts can also affect other parts of the face, such as the eyes, ears, nose, cheeks, and forehead. A cleft lip or palate can be successfully treated with surgery, especially so if conducted soon after birth or in early childhood.



Abstract - Path - 09

Developmental lesions of soft tissues

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Congenital lip pits (also known as "Congenital sinus of the lower lip, "Lip sinus," and "Midline sinus of the upper lip") are a cutaneous condition that are divided into three types based on their location: (1) commissural; (2) upper lip; and (3) lower lip. Double lip is an uncommon oral anomaly, which occurs mostly in the upper lip. It may be congenital or acquired and occurs as an isolated case or in association with other lesions. Cheilitis glandularis is a rare inflammatory disease that affects minor salivary glands and their ducts, predominantly those of the lower lip. Cheilitis granulomatosa (or granulomatous cheilitis) is a chronic swelling of the lip due to granulomatous inflammation. Miescher cheilitis is the term used when the granulomatous changes are confined to the lip. Miescher cheilitis is generally regarded as a monosymptomatic form of the Melkersson-Rosenthal syndrome, although the possibility remains that these may be 2 separate diseases. Oral pigmentation is a relatively common condition that may involve any portion of the oral cavity. Multiple causes are known, and they may range from simple iatrogenic mechanisms, such as implantation of dental amalgam, to complex medical disorders, such as Peutz-Jeghers syndrome. Local irritants, such as smoking, may also result in melanosis of varying degrees. Oral pigmented lesions result from cellular hyperplasia that can range from benign nevi to fatal oral melanoma. Fordyce spots (also termed Fordyce granules, or Fordyce disease), are visible sebaceous glands that are present in most individuals. They appear in the mouth. They appear as small, painless, raised, pale, red or white spots or bumps 1 to 3 mm in diameter that may appear in the inner surface (retromolar mucosa) and vermilion border of the lips of the face. They are not associated with any disease or illness, nor are they infectious but rather they represent a natural occurrence on the body. No treatment is therefore required, unless the individual has cosmetic concern. Macroglossia is the medical term for an unusually large tongue. Severe enlargement of the tongue can cause cosmetic and functional difficulties including in speaking, eating, swallowing and sleeping. Macroglossia is uncommon, and usually occurs in children. Macroglossia may be caused by a wide variety of congenital and acquired conditions. Isolated macroglossia has no determinable cause. The most common causes of tongue enlargement are vascular malformations (e.g. lymphangioma or hemangioma) and muscular hypertrophy (e.g. Beckwith-Weidemann syndrome or hemihyperplasia). Enlargement due to lymphangioma gives the tongue a pebbly appearance with multiple superficial dilated lymphatic channels.



Abstract - Path - 10

Metastasis

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Metastasis, or metastatic disease, is the spread of a cancer from one organ or part to another non-adjacent organ or part. The new occurrences of disease thus generated are referred to as metastases (sometimes abbreviated mets). It was previously thought that only malignant tumor cells and infections have the capacity to metastasize; however, this is being reconsidered due to new research. In origin metastasis is a Greek word meaning "displacement". Cancer occurs after a single cell in a tissue is progressively genetically damaged to produce cells with uncontrolled proliferation. This uncontrolled proliferation, mitosis, produces a primary tumor. The cells which constitute the tumor eventually undergo metaplasia, followed by dysplasia then anaplasia, resulting in a malignant phenotype. This malignant phenotype allows for intravasation into the circulation, followed by extravasation to a second site for tumorigenesis. Some cancer cells acquire the ability to penetrate the walls of lymphatic and/or blood vessels, after which they are able to circulate through the bloodstream (circulating tumor cells) to other sites and tissues in the body. This process is known (respectively) as lymphatic or hematogenous spread. After the tumor cells come to rest at another site, they re-penetrate the vessel or walls and continue to multiply, eventually forming another clinically detectable tumor. This new tumor is known as a metastatic (or secondary) tumor. Metastasis is one of three hallmarks of malignancy (contrast benign tumors). Most neoplasms can metastasize, although in varying degrees (e.g., basal cell carcinoma rarely metastasize). When tumor cells metastasize, the new tumor is called a secondary or metastatic tumor, and its cells are similar to those in the original tumor. This means, for example, that, if breast cancer metastasizes to the lungs, the secondary tumor is made up of abnormal breast cells, not of abnormal lung cells. The tumor in the lung is then called metastatic breast cancer, not lung cancer. Treatment and survival is determined, to a great extent, by whether or not a cancer remains localized or spreads to other locations in the body. If the cancer metastasizes to other tissues or organs, it usually dramatically decreases a patient's likelihood of survival (i.e. the "prognosis"). However, there are some cancers - such as some forms of leukemia, a cancer of the blood, or malignancies in the brain - that can kill without spreading at all.



Abstract - Path - 11

Grading And Staging Of Cancer

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Oral cancer or mouth cancer, a subtype of head and neck cancer, is any cancerous tissue growth located in the oral cavity. Staging: The stage of a cancer means how big it is and whether it has grown or spread. Staging systems for mouth and oropharyngeal cancers There are different ways of staging cancers. The two main systems are the TNM system and number system. TNM stages of mouth and oropharyngeal cancers: TNM stands for Tumour, Node and Metastasis. The system describes The size of a primary tumour (T) Whether the cancer has spread to the lymph nodes (N) Whether the cancer has spread to a different part of the body (M) T stages: There are 4 main T stages of mouth and oropharyngeal cancer T1 means the tumour is contained within the tissue of the mouth or oropharynx and is no larger than 2cm (¾ inch). T2 means the tumour is larger than 2cm, but smaller than 4cm (about 1½ inches). T3 means the tumour is bigger than 4cm. T4a means the tumour has grown further than the mouth or oropharynx and into nearby body tissues such as bone, tongue, the air cavities of the face (sinuses) or the skin. T4b means the tumour has spread into nearby areas such as the space around and behind the jaws, the back of the upper jaw where the large jaw muscles attach, the base of the skull, or the area of the neck that surrounds the main arteries. (carotid arteries) N stages: There are 4 main lymph node stages in cancer of the mouth and oropharynx. One of these, stage N2, is broken down into 3 sub stages. The important points here are whether there is cancer in the lymph nodes in the neck and if so, the size of the node and which side of the neck it is on. N0 means there are no cancer cells in the lymph nodes. N1 means there are cancer cells in 1 lymph node on the same side of the neck as the cancer, but the node is less than 3cm across. N2a means there is cancer in 1 lymph node on the same side of the neck, and the node is more than 3cm across but less than 6cm across. N2b means there is cancer in more than 1 lymph node, but none of these nodes are more than 6cm across. All the affected nodes are on the same side of the neck as the cancer. N2c means there is cancer in nodes on the other side of the neck, or in nodes on both sides, but none of these nodes are more than 6cm across. N3 means that at least 1 node containing cancer is more than 6cm across. M stages - There are two M stages for cancers of the mouth and oropharynx. M0 means there is no cancer spread to other parts of the body. M1 means the cancer has spread to other parts of the body, such as the lungs. Together, the T, N and M stages give a complete description of the stage of your cancer.



Abstract - Path – 12

Tumour Markers

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Tumor markers are substances that can be found in the body when cancer is present. Ideally, a tumor marker would always be found in the blood in higher-than-normal amounts, but only when a certain type of cancer is present. In reality, tumor markers are rarely like that. Some tumor markers are found in blood, but others are found in urine or other body fluids. Still others are found in tumors and other tissues. They may be made by the cancer cells themselves, or by the body in response to cancer or other conditions. Most tumor markers are proteins, but some newer markers are genes or other substances. There are many different tumor markers. Some are linked only to one type of cancer, while others can be found in many cancers. Today, the most widely used tumor marker is the prostate-specific antigen (PSA) blood test. The PSA test is used to screen men for prostate cancer. Men with prostate cancer usually have high PSA levels. But it's not always clear what the test results mean — men without cancer can have high PSA levels, and a normal PSA level does not always mean that no cancer is present. Alpha fetoprotein (AFP) is another example of a tumor marker that may be used to help diagnose cancer. The level of AFP can go up with some liver diseases, but when it reaches a certain high level in someone with a liver tumor, doctors can be fairly sure that the tumor is liver cancer (a biopsy will still be needed, though). Early on in the search for tumor markers, the hope was that someday all cancers could be detected early with a blood test. A simple blood test that could find cancers in their earliest stages could prevent the deaths of millions of people. But very few tumor markers are useful for finding cancer at an early stage. There are a few reasons for this - Almost everyone has a small amount of these markers in their blood, so it's very hard to spot early cancers by using these tests. The levels of these markers tend to get higher than normal only when there's a large amount of cancer present. Some people with cancer never have high tumor marker levels. Even when levels of these markers are high, it doesn't always mean cancer is present.



Abstract - Path - 13

Periodontitis And Atherosclerosis

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The conventional risk factors for atherosclerosis are well understood, but they can account for only about 50% to 70% of atherosclerotic events in the general population. Many other putative risk factors for atherosclerosis have been proposed, including traits related to obesity, inflammation, and infection. Periodontal disease is a candidate risk factor that shares many of these related traits. The periodontal diseases reflect a spectrum of oral pathology from gingivitis (gum inflammation) to severe periodontitis (progressive loss of gum attachment) with alveolar bone and tooth loss. The pathogenesis of periodontal disease is thought to be due to accumulation of dental plaque (bacteria in subgingival biofilms) with consequent mucosal infection and inflammation. Abnormal host responses, with upregulation of matrix metalloproteinases, contribute to a more rapid disease progression in some patients. Periodontal disease is more common with cigarette smoking, obesity, and diabetes, and it affects about 75% of the adult population in the United States, with about 20% to 30% of adults having severe forms. Increasing evidence over the past decade suggests a link between periodontal disease and atherosclerosis. There are several possible explanations for the association between periodontal disease and complications of atherosclerosis. First, it may merely reflect confounding by common risk factors that cause both periodontal disease and atherosclerosis, such as smoking, obesity, and diabetes. All of the observational studies have adjusted statistically for these risk factors, though such adjustments can be problematic when large differences in risk factor burden exist between groups. Additionally, there may be as yet unknown shared risk factors that cannot be taken into account. Second, the association may reflect an individual propensity to develop an exuberant inflammatory response to intrinsic (age, sex, genes) or extrinsic stimuli (diet, smoking, etc) that then predisposes to both periodontal disease and atherosclerosis. Third, the presence of an inflammatory focus in the oral cavity may potentiate the atherosclerotic process by stimulation of humoral and cell-mediated inflammatory pathways. The degree of inflammation in periodontal disease is clearly sufficient to cause a systemic inflammatory response, as evidenced by increases in C-reactive protein. Cross-reactivity of antibodies to periodontal pathogens with antigens present in platelets or endothelial cells might be an additional pro-inflammatory mechanism. Fourth, the presence of periodontal infection may lead to brief episodes of bacteremia with inoculation of atherosclerotic plaques by periodontal pathogens such as *Porphyromonas gingivalis*, *Actinobacillus actinomycetemcomitans*, and *Bacteroides forsythus*. Subsequent growth of these bacteria would cause inflammation and plaque instability. Indeed, there is evidence using immunostaining and polymerase chain reaction for bacterial rDNA that these pathogens are present in 18% to 30% of carotid atheromas.



Abstract - Path - 14

Oral Manifestations Of Leukaemia

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Patients with leukaemia can exhibit signs or complain of symptoms in the oral cavity and oropharynx; indeed, these may be the initial presenting complaints of the disease. Septicaemia is common in patients undergoing treatment, and has been reported as having an oral cause in up to 50% of cases. Signs and local symptoms of leukemia in the oral cavity include paleness of the oral mucosa with gingival bleeding that develops into painless gingival hyperplasia, hemorrhages, and ulcerative necrotic lesions. These findings are common clinical manifestations of leukemias and frequently herald the onset of the disease. Because of their clinical importance, all such lesions deserve the full attention of dentists and physicians. Oral manifestations of leukemia may include mucosal bleeding, ulceration, petechiae, and diffuse or localized gingival enlargement. Gingival infiltration by leukemic cells occurs most often in acute monocytic leukemia and acute myelomonocytic leukemia. The gingiva may feel boggy and appear hemorrhagic with or without concurrent ulceration. Impaired immune function can lead to various secondary oral complications, such as candidiasis, herpes simplex virus infection, and periodontal bone loss. Patients receiving treatment for leukemia may develop opportunistic infection and chemotherapy-related oral mucositis. Various preventive protocols (e.g., acyclovir [Zovirax], nystatin, chlorhexidine [Peridex], oral hygiene care) may be used to minimize these complications.



Abstract - Path - 15

Childhood Tumours

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These cancers can be quite different from cancers affecting adults. They tend to occur in different parts of the body to adult cancers. They also look different under the microscope and respond differently to treatment. Cure rates for children are much higher than for most adult cancers. On average, about 75% (more than 7 in 10) of all children can now be completely cured. For some types of children's cancer, the cure rate is much higher. One-third of all childhood cancers are leukaemia. Approximately three out of four of these cases are acute lymphoblastic leukaemia (ALL). ALL can affect children of any age, but is more common in children aged 1-4. It is more common in boys than in girls. There are four main types of leukaemia: acute lymphoblastic (ALL), acute myeloid (AML), chronic lymphocytic (CLL) and chronic myeloid (CML). Chronic leukaemias usually affect adults and are extremely rare in children and young people. Each type of leukaemia has its own characteristics and treatment. Acute lymphoblastic leukaemia is a cancer of immature lymphocytes, called lymphoblasts or blast cells. Children with certain genetic disorders, such as Down's syndrome, are known to have a higher risk of developing leukaemia. Brothers and sisters of a child with ALL (particularly identical twins) have a slightly increased risk of developing ALL themselves, although this risk is still small. Acute myeloid leukaemia is an overproduction of immature myeloid white blood cells (blast cells). Cells that have started to show some of the features of myeloid cells are said to show differentiation. Cells that do not show signs of becoming a particular type of white blood cell are known as undifferentiated. There are different sub-types of AML, depending upon exactly which type of cell has become leukaemic, the stage of development (maturation) the cells are at and whether the cells are differentiated. Knowing the sub-type of AML is important, as it helps doctors decide on the best treatment.



**DEPARTMENT OF PATHOLOGY
POSTER PRESENTATION**

Abstract - Path - 01

Oral Manifestations Of AIDS

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Oral manifestations of HIV disease are common and include oral lesions and novel presentations of previously known opportunistic diseases. Careful history taking and detailed examination of the patient's oral cavity are important parts of the physical examination, and diagnosis requires appropriate investigative techniques. Early recognition, diagnosis, and treatment of HIV-associated oral lesions may reduce morbidity. Oral candidiasis is most commonly associated with *Candida albicans*, although other species, such as *C. glabrata* and *C. tropicalis*, are frequently part of the normal oral flora. A number of factors predispose patients to develop candidiasis: infancy, old age, antibiotic therapy, steroid and other immunosuppressive drugs, xerostomia, anemia, endocrine disorders, and primary and acquired immunodeficiency. Candidiasis is a common finding in people with HIV infection. Reports describe oral candidiasis during the acute stage of HIV infection, but it occurs most commonly with falling CD4+ T-cell count in middle and late stages of HIV disease. Human papilloma virus lesions - Oral warts, papillomas, skin warts, and genital warts are associated with the human papillomavirus (HPV). Lesions caused by HPV are common on the skin and mucous membranes of persons with HIV disease. Because the HPV types found in oral lesions in HIV-infected persons are different from the HPV types associated with anogenital warts, clinicians should probably not use the term condyloma acuminata to describe oral HPV lesions. Periodontal disease is a fairly common problem in both asymptomatic and symptomatic HIV-infected patients. It can take two forms: the rapid and severe condition called necrotizing ulcerative periodontitis (NUP) and its associated and possibly precursor condition called linear gingival erythema (LGE). The presenting clinical features of these diseases often differ from those in non-HIV-infected persons. Kaposi's sarcoma (KS) may occur intraorally, either alone or in association with skin and disseminated lesions.



Abstract - Path - 2

Radicular Cyst

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It is defined as an odontogenic cyst of Inflammatory origin that is preceded by a chronic periapical granuloma and stimulation of cell rests of malassez present in the periodontal membrane. The periapical cyst (also termed radicular cyst, and to a lesser extent dental cyst) is the most common odontogenic cyst. It is caused by pulpal necrosis secondary to dental caries or trauma. The cyst lining is derived from the cell rests of Malassez. Usually, the periapical cyst is asymptomatic, but a secondary infection can cause pain. On radiographs, it appears a radiolucency (dark area) around the apex of a tooth's root. Radicular cyst is the most common odontogenic cystic lesion of inflammatory origin. It is also known as periapical cyst, apical periodontal cyst, root end cyst or dental cyst. It arises from epithelial residues in periodontal ligament as a result of inflammation. The inflammation usually follows death of dental pulp. Radicular cysts are found at root apices of involved teeth. These cysts may persist even after extraction of offending tooth; such cysts are called residual cysts. Dental cysts are usually caused due to root infection involving the tooth affected greatly by carious decay. The resulting pulpal necrosis causes release of toxins at the apex of the tooth leading to periapical inflammation. This inflammation leads to the formation of reactive inflammatory (scar) tissue called periapical granuloma further necrosis and damage stimulates the Malassez epithelial rests, which are found in the periodontal ligament, resulting in the formation of a cyst that may be infected or sterile (The epithelium undergoes necrosis and the granuloma becomes a cyst). These lesions can grow into large lesions because they apply pressure over the bone causing resorption. The toxins released by the breakdown of granulation tissue is one of the common causes of bone resorption. These cysts are not true neoplasms. It is suggested that insertion of file or other root canal instrument beyond the apical foramen (for 1-2mm) produces transitory acute inflammation which may destroy epithelial lining of radicular cyst and convert it into granuloma. A mucoperiosteal flap over cyst is raised and a window is opened in the bone to give adequate access. The cyst is carefully separated from its bony wall. The entire cyst is removed intact. The edges of bony cavity are smoothed off, free bleeding is controlled and cavity is irrigated to remove debris. Mucoperiosteal flap is replaced back and sutured in place. Marsupialisation- The cyst is opened essentially as for enucleation but the epithelial lining is sutured to mucous membrane at margins of opening. The aim is to produce a self-cleansing cavity, which becomes an invagination of oral tissues. The cavity is initially packed with ribbon gauze and after margins are healed a plug or extension of denture is made to close the openings



Abstract - Path - 3

Ameloblastoma

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Ameloblastoma (ameloblasts, or outside portion, of the teeth during development) much more commonly appearing in the lower jaw than the upper jaw. This type of odontogenic neoplasm was designated as an adamantinoma in 1885 by the French physician Louis-Charles Malassez. It was finally renamed to the modern name ameloblastoma in 1930 by Ivey and Churchill. While these tumors are rarely malignant or metastatic (that is, they rarely spread to other parts of the body), and progress slowly, the resulting lesions can cause severe abnormalities of the face and jaw. Additionally, because abnormal cell growth easily infiltrates and destroys surrounding bony tissues, wide surgical excision is required to treat this disorder. If an aggressive tumor is left untreated, it can obstruct the nasal and oral airways making it impossible to breathe without oropharyngeal intervention. There are three main clinical subtypes of ameloblastoma: unicystic, multicystic, peripheral. The peripheral subtype composes 2% of all ameloblastomas. Of all ameloblastomas in younger patients, unicystic ameloblastomas represent 6% of the cases. A fourth subtype, malignant, has been considered by some oncologic specialists, however, this form of the tumor is rare and may be simply a manifestation of one of the three main subtypes. Ameloblastoma also occurs in long bones, and another variant is Craniopharyngioma (Rathke's pouch tumour, Pituitary Ameloblastoma.) Ameloblastomas are often associated with the presence of unerupted teeth. Symptoms include painless swelling, facial deformity if severe enough, pain if the swelling impinges on other structures, loose teeth, ulcers, and periodontal (gum) disease. Lesions will occur in the mandible and maxilla, although 75% occur in the ascending ramus area and will result in extensive and grotesque deformities of the mandible and maxilla. In the maxilla it can extend into the maxillary sinus and floor of the nose. The lesion has a tendency to expand the bony cortices because slow growth rate of the lesion allows time for periosteum to develop thin shell of bone ahead of the expanding lesion. This shell of bone cracks when palpated and this phenomenon is referred to as "Egg Shell Cracking" or crepitus, an important diagnostic feature. Ameloblastoma is tentatively diagnosed through radiographic examination and must be confirmed by histological examination (e.g., biopsy). Radiographically, it appears as a lucency in the bone of varying size and features—sometimes it is a single, well-demarcated lesion whereas it often demonstrates as a multiloculated "soap bubble" appearance



Abstract - Path - 4

Growth Factors Affecting Wound Healing

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Wound healing, as a normal biological process in the human body, is achieved through four precisely and highly programmed phases: hemostasis, inflammation, proliferation, and remodeling. For a wound to heal successfully, all four phases must occur in the proper sequence and time frame. Many factors can interfere with one or more phases of this process, thus causing improper or impaired wound healing. This article reviews the recent literature on the most significant factors that affect cutaneous wound healing and the potential cellular and/or molecular mechanisms involved. The factors discussed include oxygenation, infection, age and sex hormones, stress, diabetes, obesity, medications, alcoholism, smoking, and nutrition. A better understanding of the influence of these factors on repair may lead to therapeutics that improve wound healing and resolve impaired wounds.

Wound healing is a complex biological process which requires cellular interactions between a variety of cells like fibroblasts, myofibroblasts, smooth muscle cells, endothelial cells, keratinocytes and immune cells. These interactions are mediated by numerous growth factors namely epidermal growth factor (EGF), fibroblast growth factor (FGF), insulin-like growth factor (IGF), keratinocyte growth factor (KGF), platelet-derived growth factor (PDGF), transforming growth factor (TGF) and vascular endothelial growth factor (VEGF). Growth factors are hormone-like molecules that interact with specific cell surface receptors to control the process of tissue repair. Even trace quantities of these growth factors exert a powerful influence in the wound healing process. By the third day tissue macrophages migrate into the wound and serve as the principal cell, controlling and regulating wound healing. These macrophages control wound healing through the production of growth factors such as platelet-derived growth factor (PDGF), transforming growth factor (TGF), interleukin (IL), and tumor necrosis factor (TNF). The phase of wound healing called fibroplasia begins as the number of macrophages and fibroblasts increase in number which enables the process of matrix formation and collagen synthesis. Fibroplasia begins about 5 days after injury and may continue for as long as 2 weeks. Fibroblasts migrate into the wound and replicate in response to mediators released during inflammation. These mediators include C5a, fibronectin, PDGF, fibroblast growth factor (FGF), and TGF. Therefore growth factors play fundamental roles in wound healing process by stimulating chemotaxis and cellular proliferation, by providing signaling among cells of the same and different type, by controlling extracellular matrix formation and angiogenesis, by regulating the process of contraction and by re-establishing tissue integrity.



Abstract - Path - 5

Crigler Najjar Syndrome

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Crigler–Najjar syndrome or CNS is a rare disorder affecting the metabolism of bilirubin, a chemical formed from the breakdown of red blood cells. The disorder results in an inherited form of non-hemolytic jaundice, which results in high levels of unconjugated bilirubin and often leads to brain damage in infants. This syndrome is divided into type I and type II, with the latter sometimes called Arias syndrome. These two types, along with Gilbert's syndrome, Dubin–Johnson syndrome, and Rotor syndrome, make up the five known hereditary defects in bilirubin metabolism. Crigler-Najjar syndrome occurs when the enzyme that normally converts bilirubin into a form that can easily be removed from the body does not work correctly. Without this enzyme, bilirubin can build up in the body and lead to jaundice (yellow discoloration of skin and eyes) and damage to the brain, muscles, and nerves. Crigler-Najjar (type 1) is the form of the disease that starts early in life. Arias syndrome (type 2) starts later in life. The syndrome runs in families (inherited). A child must receive a copy of the defective gene from both parents to develop the severe form of the condition. Parents who are carriers (with just one defective gene) have about half the enzyme activity of a normal adult, but do not have symptoms. Symptoms include Confusion and changes in thinking .Yellow skin (jaundice) and yellow in the whites of the eyes (icterus), which begin a few days after birth and get worse over time. Treatment - Light treatment (phototherapy) is needed throughout a person's life. In infants, this is done using bilirubin lights (bili or 'blue' lights). Phototherapy does not work as well after age 4, because thickened skin blocks the light. A liver transplant can be done in some people with type 1 disease. Blood transfusions may help control the amount of bilirubin in blood. Calcium compounds are sometimes used to remove bilirubin in the gut. The drug phenobarbital is sometimes used to treat Arias syndrome (type 2).



**DEPARTMENT OF PHYSIOLOGY
ORAL PRESENTATION**

Abstract - Physio - 01

Artificial retina

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Artificial Retina Project was a collaborative, multi-institutional effort to develop an implantable microelectronic retinal prosthesis that restores useful vision to people blinded by retinal diseases. The ultimate goal of the project was to restore reading ability, facial recognition, and unaided mobility in people with retinitis pigmentosa and age-related macular degeneration. A spectacle-mounted camera captures image data for Konstantopoulos; that data is then processed by a mini-computer carried on a strap and sent to a neuron-stimulating array of 60 electrodes that was implanted on one of his retinas in 2009. It is the first vision-restoring implant sold to patients. Currently, the system is only approved for patients with retinitis pigmentosa, a degenerative eye condition that strikes around one in 5,000 people worldwide, but it's possible the Argus II and other artificial retinas in development could work for those with age-related macular degeneration, which affects one in 2,000 people in developed countries. In these conditions, the photoreceptor cells of the eye (commonly called rods and cones) are lost, but the rest of the neuronal pathway that communicates visual information to the brain is often still viable. Artificial retinas depend on this remaining circuitry, so cannot work for all forms of blindness. Many groups around the world are working on bionic vision systems to replace lost photoreceptors. Most use a camera that communicates to an implanted chip, but vary in the number of electrodes in the chip and how deep the chip is placed inside the retina. Yet others eschew the camera for light-sensitive diodes in the chip. The LLNL team contributed three major components to the artificial retina program: the thin-film electrode array that contains the neural electrodes; the biocompatible electronics package that contains the electronics for stimulating the retina and wireless power and communication; and an ocular surgical tool that will enable the replacement of the thin-film electrode array. In addition, LLNL was responsible for the system integration and assembly of the next-generation artificial retina system with 240 stimulating electrodes.



Abstract - Physio - 02

Alzheimer's disease

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Alzheimer's disease (AD), also known in medical literature as Alzheimer disease, is the most common form of dementia. There is no cure for the disease, which worsens as it progresses, and eventually leads to death. It was first described by German psychiatrist and neuropathologist Alois Alzheimer in 1906 and was named after him. Most often, AD is diagnosed in people over 65 years of age, although the less-prevalent early-onset Alzheimer's can occur much earlier. In 2006, there were 26.6 million people worldwide with AD. Alzheimer's is predicted to affect 1 in 85 people globally by 2050. Like all types of dementia, Alzheimer's is caused by brain cell death. It is a neurodegenerative disease, which means there is progressive brain cell death that happens over a course of time. The total brain size shrinks with Alzheimer's - the tissue has progressively fewer nerve cells and connections. While they cannot be seen or tested in the living brain affected by Alzheimer's disease, postmortem/autopsy will always show tiny inclusions in the nerve tissue, called plaques and tangles: Plaques are found between the dying cells in the brain - from the build-up of a protein called beta-amyloid (you may hear the term "amyloid plaques") and The tangles are within the brain neurons - from a disintegration of another protein, called tau. For a detailed visualization of what goes on in the Alzheimer's disease process, progressing from the normal brain to increasing dementia changes, the Alzheimer's Association has produced a journey of 16 slides. This sort of change in brain nerves is also witnessed in other disorders, and researchers want to find out more than just that there are protein abnormalities - they also want to know how these develop so that a cure or prevention might be discovered. Although some kinds of memory loss are normal parts of aging, the changes due to aging are not severe enough to interfere with the level of function. Many different diseases can cause dementia, but Alzheimer's disease is by far the most common cause for dementia in the United States and in most countries in the world. Although some kinds of memory loss are normal parts of aging, the changes due to aging are not severe enough to interfere with the level of function. Many different diseases can cause dementia, but Alzheimer's disease is by far the most common cause for dementia in the United States and in most countries in the world.



Abstract - Physio - 03

Cerebral palsy

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Cerebral palsy (CP) is a general term for a group of permanent, non-progressive movement disorders that cause physical disability in development, mainly in the areas of body movement. It is a central motor dysfunction affecting muscle tone, posture and movement resulting from a permanent, non-progressive defect or lesion of the immature brain. CP is neither genetic nor an infectious disease, and thus it is not contagious. Most cases are congenital, arising at or about the time of birth, and are diagnosed at a young age rather than during adolescence or adulthood. The term cerebral palsy refers to any one of a number of neurological disorders that appear in infancy or early childhood and permanently affect body movement and muscle coordination but don't worsen over time. Even though cerebral palsy affects muscle movement, it isn't caused by problems in the muscles or nerves. It is caused by abnormalities in parts of the brain that control muscle movements. The majority of children with cerebral palsy are born with it, although it may not be detected until months or years later. The early signs of cerebral palsy usually appear before a child reaches 3 years of age. The most common are a lack of muscle coordination when performing voluntary movements (ataxia); stiff or tight muscles and exaggerated reflexes (spasticity); walking with one foot or leg dragging; walking on the toes, a crouched gait, or a "scissored" gait; and muscle tone that is either too stiff or too floppy. A small number of children have cerebral palsy as the result of brain damage in the first few months or years of life, brain infections such as bacterial meningitis or viral encephalitis, or head injury from a motor vehicle accident, a fall, or child abuse. Congenital cerebral palsy results from brain injury during a baby's development in the womb. It is present at birth, although it may not be detected for months. It is responsible for about 70% of children who have cerebral palsy. An additional 20% are diagnosed with congenital cerebral palsy due to a brain injury during the birthing process. Cerebral palsy usually is diagnosed by 3 years of age. About 2 to 3 children in 1,000 are affected. About 800,000 children and adults of all ages in the United States have cerebral palsy.



Abstract - Physio - 04

Mitral stenosis and Regurgitation

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Mitral stenosis (MS) refers to narrowing of the mitral valve orifice, resulting in impedance of filling of the left ventricle in diastole. It is usually caused by rheumatic heart disease. Less common causes include severe calcification of the mitral annulus, infective endocarditis, systemic lupus erythematosus, rheumatoid arthritis, and carcinoid heart disease. The leaflets are fused, are too thick, or there is some other structural defect in the valve leaflets. Sometimes, mitral stenosis occurs as a result of surgery to repair an abnormal mitral valve. As a result, the valve is too narrow, and the heart has to work harder to pump blood through the valve. Mitral regurgitation (MR) is leakage of blood from the left ventricle into the left atrium during systole. It is caused by various mechanisms related to structural or functional abnormalities of the mitral apparatus, adjacent myocardium, or both. The most common causes of mitral regurgitation in the United States are myxomatous degeneration, chordal rupture, rheumatic heart disease, infective endocarditis, coronary artery disease, and cardiomyopathy. The valve doesn't close completely and allows blood to leak back (regurgitate) into the atrium (upper chamber) from the ventricle (lower chamber). Common causes of mitral valve insufficiency/regurgitation include rheumatic fever, mitral valve prolapse, mitral annulus calcification, infective endocarditis (an infection that affects the lining of the heart's chambers and the heart valves), congenital causes, a weakened heart muscle caused by a heart attack, rheumatic heart disease, infections of the heart valve, and weakness of the heart muscle that has dilated due to primary heart muscle disease. Rheumatic fever in childhood is by far the most common cause of mitral stenosis in adulthood. It can damage the heart valve and cause scarring, which results in problems later in life, usually in young adulthood. More rarely, mitral stenosis can occur from congenital heart disease or from calcium deposits that accumulate over years. Diagnosis is based on the medical history including symptoms, existence of a characteristic murmur upon examination of the heart, and echocardiography (an ultrasound study of the heart valves and muscle). Treatment of mitral stenosis depends on factors that include the patient's symptoms, severity of the stenosis, and health of the patient. Surgical repair of mitral valve stenosis is called commissurotomy.



Abstract - Physio – 05

Rheumatic diseases

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Rheumatism or rheumatic disorder is a non-specific term for medical problems affecting the joints and connective tissue. The study of, and therapeutic interventions in, such disorders is called rheumatology. Rheumatic diseases (rheumatism) are painful conditions usually caused by inflammation, swelling, and pain in the joints or muscles. Some rheumatic diseases like osteoarthritis are the result of "wear and tear" to the joints. Other rheumatic diseases, such as rheumatoid arthritis, happen when the immune system becomes hyperactive; the immune system attacks the linings of joints, causing joint pain, swelling, and destruction. Almost any joint can be affected in rheumatic disease. There are more than 100 rheumatic diseases but we'll focus on some of the common types. More than 100 diseases are classified as rheumatic diseases, including many types of arthritis. Arthritic conditions are distinguished by red, swollen joints and inflamed connective tissues such as cartilage, synovial tissue, and tendons. Other rheumatic diseases are considered autoimmune diseases, meaning that the body's own immune system is turning on parts of the body. There are roughly 46 million people in the United States living with rheumatic diseases, conditions that affect the joints and bones and cause chronic joint pain, swelling, and stiffness. Some rheumatic diseases also affect other areas of the body, including the heart, kidneys, lungs, and skin. More than 100 conditions fall under the category of rheumatic diseases, including gout, pseudogout, ankylosing spondylitis, reactive arthritis, Reiter's syndrome, enteropathic arthritis, Lyme disease, and infectious arthritis. Our immune system is an amazing network of cells that function from very basic to highly complex levels. The purpose of this system is to protect us from our environment and watch for any early damage in our own cells. Sometimes, however, the system goes awry and misreads signals. As a result, our defenses do not recognize our own body at work, and begin "attacking" cells. This leads to illnesses called autoimmune (self-immune) diseases such as rheumatoid arthritis (inflammation of the joints), systemic lupus erythematosus (commonly known as "lupus," an inflammatory disease of connective tissue), and vasculitis (inflammation of a vessel of the body).



Abstract - Physio - 06

Respiratory function test

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Respiratory Function Tests (RFTs) are a series of tests of lung capacity and function which help to classify the type of respiratory disease in an individual. Components include: Forced Vital Capacity (FVC), Forced Expiratory Volume in 1 second (FEV1), FEV1/FVC ratio, pre- and post-bronchodilator measurements. Respiratory Function Tests (RFTs) involve breathing into a mouthpiece, which is connected to a small machine via some tubing. You will be required to breathe in and out as deeply as possible, and then as fast and as forcefully as possible. The test may make you feel temporarily short of breath, but is otherwise harmless. More sophisticated testing such as gas transfer tests require the patient to breathe into a closed system, via a tight-fitting face mask. Pulmonary function tests (PFTs) are noninvasive diagnostic tests that provide measurable feedback about the function of the lungs. By assessing lung volumes, capacities, rates of flow, and gas exchange, PFTs provide information that, when evaluated by your doctor, can help diagnosis certain lung disorders. A normally-functioning pulmonary system operates on many different levels to ensure adequate balance. One of the primary functions of the pulmonary system is ventilation, the movement of air into and out of the lungs. Some medical conditions may interfere with ventilation. These conditions may lead to chronic lung disease. Conditions that interfere with normal ventilation are categorized as restrictive or obstructive. An obstructive condition occurs when air has difficulty flowing out of the lungs due to resistance, causing a decreased flow of air. A restrictive condition occurs when the chest muscles are unable to expand adequately, creating a disruption in air flow. Pulmonary Function testing measures the function of lung capacity and lung and chest wall mechanics to determine whether or not the patient has a lung problem. Pulmonary Function Tests are commonly referred to as "PFTs". When a patient is referred for PFT's, it means that a battery of tests may be carried-out including: simple screening spirometry, static lung volume measurement, diffusing capacity for carbon monoxide, airways resistance, respiratory muscle strength and arterial blood gases. Spirometry measures airflow. By measuring how much air you exhale, and how quickly, spirometry can evaluate a broad range of lung diseases. In a spirometry test, while you are sitting, you breathe into a mouthpiece that is connected to an instrument called a spirometer. The spirometer records the amount and the rate of air that you breathe in and out over a period of time.



Abstract - Physio - 07

Growing your own replacement teeth

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Growing a replacement tooth from your own cells may be a step closer, according to new research. It is still too early for use in people, but the technique involves taking stem cells and growing more of them to produce a very small, immature tooth, similar to what a tooth would look like when it starts to grow in an embryo. These are transplanted directly into the mouth where they get their blood supply, and they start to grow and gradually form a complete tooth. Although the technique is unlikely to allow scientists to grow a specific type of tooth, dentists would be able to shape the tooth crown according to its position in the jaw. Sharpe's team from the Dental Institute at King's College combined human gum cells with the cells in mice responsible for growing teeth. They transplanted this combination of cells into the mice. The result was hybrid human/mouse teeth with roots. The ability to make a tooth replacement with roots would be a major step forward in dental surgery. Replacing missing or damaged teeth currently involves fixed or removable dental implants. Although implants work well, the impact from chewing can wear down the implant. This is not a problem with natural teeth because they have soft tissue at the root that acts as a shock absorber. The latest advance made by Sharpe and his team brings the prospect of bioengineered teeth with their own root system a step closer. The next step will be finding enough adult sources of human cells to make this new technique a viable alternative to dental implants. When people lose teeth, they have several options - few of them dignified, and most expensive - but scientists hope to persuade the body to 'grow its own' replacements. The scientists are studying pufferfish - a delicacy in Japan, where the fish have to be prepared carefully, or they are deadly poison. Pufferfish have four teeth which are constantly renewed every few weeks throughout the creature's life. Scientists have developed a new method of replacing missing teeth with a bioengineered material generated from a person's own gum cells. Current implant-based methods of whole tooth replacement fail to reproduce a natural root structure and as a consequence of the friction from eating and other jaw movement, loss of jaw bone can occur around the implant.



Abstract - Physio - 08

Telomeres as an aging factor

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Age is the dominant risk factor for cardiovascular diseases. Average telomere length (TL) acts as a biomarker for biological aging and cardiovascular disease (CVD) in particular. We therefore sought to provide baseline TL information and assess the association of prevalent CVD risk factors with TL in subjects free of overt CVD within a small age range. We measured mean telomere restriction fragment length of peripheral blood leukocytes in a large, representative Asklepios study cohort of 2509 community-dwelling, Caucasian female and male volunteers aged approximately 35–55 years and free of overt CVD. We found manifest age-dependent telomere attrition, at a significantly faster rate in men as compared to women. No significant associations were established with classical CVD risk factors such as cholesterol status and blood pressure, yet shorter TL was associated with increased levels of several inflammation and oxidative stress markers. Importantly, shorter telomere length was associated with an increasingly unhealthy lifestyle, particularly in men. All findings were age and gender adjusted where appropriate. With these cross-sectional results we show that TL of peripheral blood leukocytes primarily reflects the burden of increased oxidative stress and inflammation, whether or not determined by an increasingly unhealthy lifestyle, while the association with classical CVD risk factors is limited. This further clarifies the added value of TL as a biomarker for biological aging and might improve our understanding of how TL is associated with CVD. Age-associated telomere damage, diminution of telomere 'capping' function and associated p53 activation have emerged as prime instigators of a functional decline of tissue stem cells and of mitochondrial dysfunction that adversely affect renewal and bioenergetic support in diverse tissues. Constructing a model of how telomeres, stem cells and mitochondria interact with key molecules governing genome integrity, 'stemness' and metabolism provides a framework for how diverse factors contribute to ageing and age-related disorders.



Abstract - Physio – 09

Wrinkles and grey hair

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Skin and hair phenotypes are powerful cues in human communication. They impart much information, not least about our racial, ethnic, health, gender and age status. The hair follicle pigmentary unit is perhaps one of our most visible, accessible and potent aging sensors, with marked dilution of pigment intensity occurring long before even subtle changes are seen in the epidermis. This dichotomy is of interest as both skin compartments contain melanocyte subpopulations of similar embryologic (i.e., neural crest) origin. Whether some follicular melanocyte subpopulations are affected, like epidermal melanocytes, by UV irradiation is not yet clear. A particular target of research into hair graying or canities is the nature of the melanocyte stem compartment and whether this is depleted due to reactive oxygen species-associated damage, coupled with an impaired antioxidant status, and a failure of melanocyte stem cell renewal. Over the last few years, we and others have developed advanced in vitro models and assay systems for isolated hair follicle melanocytes and for intact anagen hair follicle organ culture which may provide research tools to elucidate the regulatory mechanisms of hair follicle pigmentation. Hair pigmentation is one of the most conspicuous phenotypes in humans ranging from black, brown, and blonde to red. Premature graying of hair occurs more commonly without any underlying pathology but is said to be inherited in autosomal dominant pattern. Premature graying has been shown to be associated with a few of the autoimmune disorders. One particular fruitful topic for future study will be to elucidate the function of the amelanotic melanocytes distributed in the outer root sheath of human scalp hair follicles. It will be important to determine if these cells are indeed progeny of so-called stem cells from the bulge area of the hair follicle, and if so, whether they retain some stem cell characteristics themselves. The recruitment of these immature outer root sheath melanocytes for re-pigmentation of the hair follicle (and even the overlying epidermis, especially after wounding) may offer significant clinical gains. Alternatively, these nonpigmenting follicular melanocytes may represent a subpopulation of transient or migrating melanocytes that can only differentiate when in a permissive microenvironment, e.g., melanogenic zone close to the follicular dermal papilla. The reversal of canities after some types of therapy, e.g., radiation/drug, may involve a cytokine-induced activation of these outer root sheath melanocytes.



Abstract - Physio - 10

Physiological effect of Pranayama

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Different types of pranayama produce different physiological cardiovascular responses in normal young individuals. The significant decrease in resting pulse rate, systolic and diastolic blood pressure after the yoga practice in the present study is in accordance with the findings of other studies on physiological effects of yoga practice in healthy individuals. Similar reduction in resting PR and blood pressure after yoga practice was also reported in hypertensive patients, in asthmatic patients and in diabetic patients. In the present study a highly significant reduction in PR, SBP, and DBP can be attributed to modulation of autonomic activity with parasympathetic predominance and relatively reduced sympathetic tone. This autonomic modulation in yoga is mediated through modification of breathing patterns which triggers various central and autonomic mechanisms as well as mechanical and hemodynamic adjustments causing both tonic and phasic changes in cardiovascular functioning. As respiratory and cardiovascular systems have similar control mechanisms, alteration in one system will modify the functioning of the other. During slow and deep breathing lung inflates to the maximum. This stimulates pulmonary stretch receptors which bring about withdrawal of sympathetic tone in skeletal muscle blood vessels leading to widespread vasodilatation and decrease in peripheral resistance and thus decrease diastolic blood pressure. While practicing pranayama one concentrates on the act of breathing which removes attention from worries and “de-stresses” him. This stress-free state of mind evokes relaxed responses in which parasympathetic nerve activity overrides sympathetic activity. Meditation by modifying the state of anxiety reduces stress-induced sympathetic over activity thereby decreasing arterial tone and peripheral resistance resulting in lowering of diastolic blood pressure and heart rate. Regular practice of yoga has showed improvement in baroreflex sensitivity and decrease in the sympathetic tone thereby restoring blood pressure to normal level in patients of essential hypertension. The stress and stress-induced disorders like hypertension and angina are fast growing epidemics and bane of “modern” society. The holistic science of yoga is the best method for prevention as well as management of stress and stress-induced disorders. Numerous studies have shown yoga to have an immediate down-regulating effect on both the HPA axis responses to stress.



DEPARTMENT OF PHYSIOLOGY

POSTER PRESENTATION

Abstract - Physio - 1

Effects of smoking on systemic organs

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Cigarettes contain more than 4000 chemical compounds and at least 400 toxic substances. When you inhale, a cigarette burns at 700°C at the tip and around 60°C in the core. This heat breaks down the tobacco to produce various toxins. As a cigarette burns, the residues are concentrated towards the butt. The products that are most damaging are: tar, a carcinogen (substance that causes cancer), nicotine is addictive and increases cholesterol levels in your body, carbon monoxide reduces oxygen in the body, components of the gas and particulate phases cause chronic obstructive pulmonary disorder (COPD). Cardiovascular disease is the main cause of death due to smoking. Hardening of the arteries is a process that develops over years, when cholesterol and other fats deposit in the arteries, leaving them narrow, blocked or rigid. When the arteries narrow (atherosclerosis), blood clots are likely to form. Smoking accelerates the hardening and narrowing process in your arteries: it starts earlier and blood clots are two to four times more likely. Cardiovascular disease can take many forms depending on which blood vessels are involved, and all of them are more common in people who smoke. It's a leading cause for coronary thrombosis. The vessels to the brain can become blocked, which can lead to collapse, stroke and paralysis. Damage to the brain's blood supply is also an important cause of dementia. If the kidney arteries are affected, then high blood pressure or kidney failure results. Blockage to the vascular supply to the legs may lead to gangrene and amputation. Smokers tend to develop coronary thrombosis 10 years earlier than non-smokers, and make up 9 out of 10 heart bypass patients. Smoking raises blood pressure, which can cause hypertension (high blood pressure) – a risk factor for heart attacks and stroke. Couples who smoke are more likely to have fertility problems than couples who are non-smokers. Heavy smokers are twice as likely to get macular degeneration, resulting in the gradual loss of eyesight. Smokers run an increased risk of cataracts. Smokers take 25 per cent more sick days year than non-smokers. Smoking stains your teeth and gums. It also causes an acid taste in the mouth and contributes to the development of ulcers.



Abstract - Physio - 2

Edema

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Edema is the medical term for swelling. It is a general response of the body to injury or inflammation. It can be isolated to a small area or affect the entire body. Medications, infections, pregnancy, and many medical problems can cause edema. Edema results whenever small blood vessels become "leaky" and release fluid into nearby tissues. The extra fluid accumulates, causing the tissue to swell. Edema is a normal response of the body to inflammation or injury. Increased fluid from the blood vessels allows more infection-fighting white blood cells to enter the affected area. Edema can also result from medical conditions or problems in the balance of substances normally present in blood. Albumin and other proteins in the blood act like sponges to keep fluid in the blood vessels. Low albumin may contribute to edema, but isn't usually the sole cause. Edema is a usual component of most allergic reactions. In response to the allergic exposure, the body allows nearby blood vessels to leak fluid into the affected area. If the drainage of fluid from a body part is blocked, fluid can back up. A blood clot in the deep veins of the leg can result in leg edema. A tumor blocking lymph or blood flow will cause edema in the affected area. Burns, life-threatening infections, or other critical illnesses can cause a whole-body reaction that allows fluid to leak into tissues almost everywhere. Widespread edema throughout the body can result. When the heart weakens and pumps blood less effectively, fluid can slowly build up, creating leg edema. If fluid buildup occurs rapidly, fluid in the lungs (pulmonary edema) can develop. Severe liver disease (cirrhosis) results in an increase in fluid retention. Cirrhosis also leads to low levels of albumin and other proteins in the blood. Fluid leaks into the abdomen (called ascites), and can also produce leg edema. A kidney condition called nephrotic syndrome can result in severe leg edema, and sometimes whole-body edema (anasarca). Due to an increase in blood volume during pregnancy and pressure from the growing womb, mild leg edema is common during pregnancy. However, serious complications of pregnancy such as deep vein thrombosis and preeclampsia can also cause edema. Swelling in the brain can be caused by head trauma, low blood sodium (hyponatremia), high altitude, brain tumors, or an obstruction to fluid drainage (hydrocephalus). Headaches, confusion, and unconsciousness or coma can be symptoms of cerebral edema.



DEPARTMENT OF PHARMACOLOGY

ORAL PRESENTATION

Abstract - Pharma - 01

Pharmacotherapy Of Alzheimer's Disease

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Alzheimer's disease (AD) is the most common cause of memory impairment and dementia in the elderly. AD is pathologically characterized by extracellular deposits of beta-amyloid (A β) peptide, neurofibrillary tangles (NFTs) composed of hyperphosphorylated tau, neuronal loss, and neurotransmitter dysfunction. Clinically, AD is characterized by progressive cognitive decline that usually starts with memory impairment and progresses to cause a more generalized cognitive dysfunction, behavioral dysregulation, and neuropsychiatric symptoms. These symptoms collectively lead to a progressive and relentless decline in the ability to perform functions of daily living, eventually leading to total incapacitation. The incidence and prevalence of AD are expected to exponentially increase with the aging of the population. These drugs, known as acetylcholinesterase inhibitors (AChEIs), were first approved by the U.S. Food and Drug Administration (FDA) in 1995 based on clinical trials showing modest symptomatic benefit on cognitive, behavioral, and global measures. In 2004 the FDA approved memantine, an NMDA antagonist, for treating dementia symptoms in moderate to severe AD cases. In clinical practice, memantine may be co-administered with an AChEI, although neither drug individually or in combination affects the underlying pathophysiology of dementia. Dementia in AD results from progressive synaptic loss and neuronal death. As knowledge of the mechanisms responsible for neurodegeneration in AD increases, it is anticipated that neuroprotective drugs to slow or prevent neuronal dysfunction and death will be developed to complement current symptomatic treatments. This review summarises the pharmacological properties of the main classes of drugs in current use for the symptomatic treatment of Alzheimer's disease. However, a number of drugs are in development which are designed to block the neurotoxic action of amyloid beta peptide and thereby reverse the underlying pathological processes. These include the gamma secretase inhibitors and vaccines against amyloid beta peptide. The limitations of these novel approaches are discussed.



Abstract - Pharma - 02

Geriatric Pharmacology

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The rate of medication errors is high, and these errors can cause adverse drug reactions. Elderly individuals are most vulnerable to adverse drug reactions. One cause of medication errors is the lack of drug knowledge on the part of different health professionals. Adults >65 years old Fastest growing population in US 20% of hospitalizations for those >65 are due to medications they're taking. The geriatric dosing axiom, "start low and go slow" is based on pharmacokinetic considerations and concern for adverse drug reactions, not from clinical trial data. In the absence of generalizable dosage guidelines, individualization via effect titration is required. Adverse drug reactions and polypharmacy represent major linkages to avoidable morbidity and mortality. This, combined with a deficient therapeutic evidence base, suggests that extrapolation of risk-benefit ratios of geriatric populations is not necessarily valid. Pharmacological constitution and regimen for older people is an important topic, one that is related to changing and differing physiology and psychology. Changes in physiology with aging may alter the absorption, the effectiveness and the side effect profile of many drugs. These changes may occur in oral protective reflexes (dryness of the mouth caused by diminished salivary glands), in the gastrointestinal system (such as with delayed emptying of solids and liquids possibly restricting speed of absorption), and in the distribution of drugs with changes in body fat and muscle and drug elimination. Psychological considerations include the fact that elderly persons (in particular, those experiencing substantial memory loss or other types of cognitive impairment) are unlikely to be able to adequately monitor and adhere to their own scheduled pharmacological administration. One study found that 25% of participants studied admitted to skipping doses or cutting them in half. Self-reported noncompliance with adherence to a medication schedule was reported by a striking one-third of the participants. Further development of methods that might possibly help monitor and regulate dosage administration and scheduling is an area that deserves attention. Another important area is the potential for improper administration and use of potentially inappropriate medications, and the possibility of errors that could result in dangerous drug interactions. Polypharmacy is often a predictive factor. Research done on home/community health care found that "nearly 1 of 3 medical regimens contain a potential medication error". The possible care to be taken for the treatment of geriatric patients are presented in this paper.



Abstract - Pharma - 03

Steroid In Health And Disease

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Steroids are commonly prescribed to help reduce inflammation (anti-inflammatory) suppress the immune system (immunosuppressant) replace hormones that are not being produced by the body due to a health condition (replacement therapy) Inflammation occurs when the immune system tries to prevent an infection from spreading. The immune system is the body's natural defence against infection and illness. It sends special chemicals to the site of the infection, causing it to become inflamed and swollen. Corticosteroids are also used to treat a number of allergic skin conditions. Corticosteroid creams and lotions are often used to treat these types of skin conditions, although steroid tablets may be needed if your symptoms are particularly severe. Some people with COPD have periods where their lungs and airways become very inflamed, often as a result of infection. Corticosteroid tablets are routinely prescribed to help reduce the inflammation and improve breathing. Sometimes, joints, muscles and tendons can become persistently inflamed as a result of injury or over-use. In such cases, a corticosteroid injection may be recommended to help reduce the inflammation. In some illnesses, the immune system malfunctions and attacks healthy tissue. These are known as autoimmune conditions. Corticosteroids are similar to the natural hormones produced by the adrenal gland (located above the kidneys). These hormones play an important role in regulating the body's metabolism (the process of converting food into energy). Corticosteroids are often used to treat Addison's disease. This is where the adrenal glands do not produce the correct amount of hormones. Anabolic steroids, technically known as anabolic-androgenic steroids (AAS), are drugs that are structurally related to the cyclic steroid ring system and have similar effects to testosterone in the body. Conditions pertaining to hormonal imbalances such as gynecomastia and testicular atrophy may also be caused by anabolic steroids.



Abstract - Pharma - 04

Pediatric Pharmacology

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In the field of pediatrics, it is vital to understand the developmental pharmacology of infants. This is due to the fact that the pharmacodynamics and pharmacokinetics differ between the adults and pediatric population. Failure to appreciate these differences can result in preventable serious morbidity or even mortality. Many of these differences are particularly important in the neonatal or premature neonatal group. Therefore Scaling adult doses to infants based on body weight or surface area does not account for developmental changes that affect drug disposition or tissue/organ sensitivity. The first year of life is associated with major changes in the processes affecting the absorption, distribution, metabolism and excretion of drugs. Drug absorption by the oral route is affected by reduced gastric emptying so that this route is unreliable in the neonate. The intramuscular route is also unreliable but transdermal absorption is often greater, with risks of toxicity. The volume of distribution of many drugs is often markedly increased in the neonate, partly because of reduced plasma protein binding but also because of an increased volume of extracellular fluid relative to total body water. Many clinical monitoring parameters used in adults for assessing medication efficacy or toxicity are also used in the critically ill child. However, normal values for infants and children may differ. One must be aware of these differences in laboratory parameters and normal vital signs in order to adequately monitor pharmacotherapy in PICU patients. For example, children, especially neonates and young infants, have lower blood pressures and higher respiratory and heart rates, compared to adults. Proper references should be consulted for the normal values for age when providing clinical care to critically ill children. Drug distribution is dependent upon the physicochemical properties of the drug (molecular weight, degree of ionization, solubility in water and lipids) and various patient-specific physiologic factors. These factors include: the composition and size of body compartments (e.g., total body water, intracellular and extracellular water, and adipose tissue), membrane permeability, pH, protein binding, and hemodynamic variables such as cardiac output, tissue perfusion and regional blood flow. 16 Many of these factors, especially body composition and protein binding, are age related and can be influenced by various disease states observed in PICU patients.



Abstract - Pharma - 05

Cosmoceuticals

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Cosmeceuticals represent one of the most promising, yet challenging treatment options available to physicians. They are the fastest growth segment in the skin-care market, and a number of topical cosmeceutical treatments for conditions such as photoaging, hyperpigmentation, and wrinkles have come into widespread use. This comprehensive review attempts to examine the current literature of the more commonly encountered cosmeceutical agents in order to determine their utility in treating various dermatologic conditions, as well as their potential use in the area of wound healing. Each section, dealing with a different agent, provides a brief chemical background, a review of the published research studies, and finally concludes with a prediction about its potential role in skin regeneration. Although further research needs to be conducted, adjuvant cosmeceutical therapy may help in prevention of skin cancer, photoaging, and the rejuvenation of skin during wound healing. Almost all the major cosmetic manufacturers use novel delivery systems in their products. The worldwide cosmetics and perfume industry currently generates an estimated annual turnover of US\$170 billion (according to Eurostat-May 2007). L'Oreal has a number of nanotechnology-related products in the market and ranks 6th in US, which devotes about \$600 million of its annual \$17 billion revenues to research in the number of nanotech related patents. These products cross female and male markets, and male grooming is one of the fastest growing markets. There are number of innovative cosmetic delivery systems used in cosmetic products. A cosmetic delivery system is a composition or a process that can enhance perceptual or measured performance of cosmetic product. One of the reasons for the widespread use of liposomes in the cosmetic industry is their ease of preparation and the ability to improve the absorption of active ingredients by skin. Liposomes are generally utilised in aqueous systems. Recently, water-sensitive 20 to 30 micron-size microspheres of polymer structure have been developed for the delivery of fragrances, botanicals, and vitamins from anhydrous formulations, such as lipsticks, deodorants, antiperspirants and body sprays. Liposomes are unstable due to their susceptibility to oxidation and the breakdown of liposomal structure; it is overcome by optimising the storage conditions and adding chelators and anti-oxidants.



Abstract - Pharma - 06

Drug Administration Through Nanotechnology

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Nanomaterials often have chemical, physical, or biological properties that are different from those of their larger-scale counterparts. Such differences may include altered magnetic, electrical, or optical properties, structural integrity, and chemical or biological activity. Because of researchers' ability to engineer such properties, nanoscale materials have great potential for use in a vast array of products, including FDA-regulated products intended to protect and promote public health. Also, because of some of their special properties, nanomaterials may pose different issues for toxicological, safety, and effectiveness assessments. There is a growing need for scientific information and tools to help better predict or detect potential impacts of such changes on human health. FDA nanotechnology investments have focused on enabling the agency to characterize nanotechnology-based products; develop models for safety and efficacy assessment; and study the behavior of nanomaterials in biological systems and their effects on human health. FDA has long encountered the combination of promise, risk, and uncertainty that accompanies emerging technologies. Nanotechnology is not unique in this regard. The very changes in biological, chemical and other properties that can make nanotechnology applications so exciting also may merit examination to determine any effects on product safety, effectiveness, or other attributes. Understanding nanotechnology remains a top FDA priority. FDA is monitoring the evolving science and has a robust research agenda to help assess the safety and effectiveness of products using nanotechnology. Strong science is critical to FDA's ongoing review of the products it regulates. In general, FDA considers the current framework for safety assessments sufficiently robust and flexible to be appropriate for a variety of materials, including nanomaterials. When a drug enters the body there are certain biological barriers that the drug molecule must first pass to get to its target organ. For example, when a drug is taken orally it must pass barriers in the stomach, the small intestine, the blood and the liver before it reaches the target organ. Barriers which must be overcome in these regions are cell membranes, metabolic enzymes, efflux transporters and binding proteins. Needless to say, there are many criteria which a drug must possess in order to reach its target region..



Abstract - Pharma - 07

Poisonings And Antidotes

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The antidotes for some particular toxins are manufactured by injecting the toxin into an animal in small doses and extracting the resulting antibodies from the host animals' blood. This results in an antivenom that can be used to counteract poison produced by certain species of snakes, spiders, and other venomous animals. A number of venoms lack a viable antivenom, and a bite or sting from an animal producing such a toxin often results in death.[citation needed] Some animal venoms, especially those produced by arthropods (e.g. certain spiders, scorpions, bees, etc.) are only potentially lethal when they provoke allergic reactions and induce anaphylactic shock; as such, there is no "antidote" for these venoms because it is not a form of poisoning and anaphylactic shock can be treated (e.g., by the use of epinephrine). Under the head of poisons, it is intended to include all those substances which exercise pernicious, as distinguished from medicinal, effects upon the human body, tending to disturb its action or organization injuriously, and if not remedied to possibly cause death. Such substances may be swallowed, or taken in by the breath, absorbed through the skin, or the thinner and more delicate mucous membranes, or implanted by bites, stings, or other punctured wounds. In many cases persons are aware almost immediately after the act that they have swallowed a poison; but in many others, also, no suspicion is entertained at first. In a general way, it may be stated that it is reasonable to surmise a person has swallowed some poisonous substance, if, shortly after taking food or drink, he is seized with violent pain in the stomach, with vomiting and purging, especially if convulsions or paralysis are present, or if the individual suffer from marked giddiness or delirium, or should there be a great tendency to sleep. The antidote is required to be adapted to the poison, and therefore an effort should be made, instantly after the emetic is given, to find out what kind of a noxious substance has been swallowed, and the proper remedy should be administered in accordance with the following suggestions. The object of most antidotes is to render the active poison an inert substance, after which treatment may be instituted with a view to remedy the mischief which it has previously done. Antidotes, therefore, are generally chemical agents, which attack or combine with the poison in such a way as to render it insoluble, and so inert.



Abstract - Pharma - 08

Drug Resistance And Management

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Drug resistance is the reduction in effectiveness of a drug such as an antimicrobial or an antineoplastic in curing a disease or condition. When the drug is not intended to kill or inhibit a pathogen, then the term is equivalent to dosage failure or drug tolerance. More commonly, the term is used in the context of resistance that pathogens have "acquired", that is, resistance has evolved. The development of antibiotic resistance in particular stems from the drugs targeting only specific bacterial proteins. Because the drug is so specific, any mutation in these proteins will interfere with or negate its destructive effect, resulting in antibiotic resistance. Resistance to chemicals is only one aspect of the problem, another being resistance to physical factors such as temperature, pressure, sound, radiation and magnetism, and not discussed in this article, but found at Physical factors affecting microbial life. Bacteria and other disease-causing microorganisms have a remarkable ability to mutate and acquire resistant genes from other organisms and thereby develop resistance to antimicrobial agents. When an antimicrobial agent is used, the selective pressure exerted by the agent favors the growth of organisms that are resistant to the agent's action. The inappropriate use of antimicrobial drugs has resulted in drug resistance that threatens to reverse the achievements of the last half century. *Staphylococcus aureus* (*S. aureus*) is an important cause of health-care associated infections. The organism can cause mild skin infections to potentially fatal systemic infections. During the 1980s, the bacteria became increasingly resistant to semisynthetic penicillin, leading to the reliance on vancomycin for treatment when this resistant pattern was noted. Currently, *S. aureus* susceptibility to vancomycin may no longer be assumed. Antimicrobial agents are very important tools for treating infectious diseases. Their proper use is essential for patients to recover from the infectious process, as well as to avoid potential toxic effects, reduce associated costs, and reduce the emergence of resistance. Studies suggest that inappropriate antimicrobial use generally precedes the emergence of antimicrobial resistance. Therefore, it is essential to address this issue as the cornerstone of a program designed to prevent the emergence of MDRO. Antimicrobial management involves a multidisciplinary team approach. This team ideally includes the physician, pharmacist, microbiologist, and infection control practitioner. Additionally, each facility is encouraged to develop guidelines that outline and assist in the appropriate use of antimicrobials, based on national standards and local experience.



Abstract - Pharma - 09

Teratogenicity associated cleft palate

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One in every 250 newborns is exposed to antiepileptic drugs in utero. Various studies have attributed a teratogenic effect to these drugs mainly consisting of major malformations, minor anomalies, intrauterine or postnatal growth failure, and psychomotor retardation. Barbiturates and phenytoin are particularly associated with congenital heart malformations, facial clefts, and some other malformations. Cleft lip, with or without cleft palate, is a congenital malformation that affects about 1 in every 700 live human births in the United States each year. Clefts occur more frequently among Asians (about 1:400) and certain American Indians than Europeans. Clefts are relatively less common among Africans and African Americans (about 1:1500). Generally, facial clefting results when medial, lateral, and maxillary nasal processes on either left, right or both sides of the forming craniofacial complex do not fuse completely. Early embryonic changes during the fourth and tenth weeks of gestation, may result in clefting. Suspected causes include environmental insults like maternal diseases, chemotherapy, radiation, alcohol, excess retinoic acid and anticonvulsant medications; or genetic factors. Ultrasound, amniocentesis and molecular genetic techniques can be used to detect common congenital malformations, including cleft lip, early. Advances in surgical techniques and growth factors also help correct problems associated with cleft lip or cleft palate. Cleft lip is usually less serious than cleft palate. About 22 percent of facial clefting has a genetic origin. Again, most cleft lips with or without cleft palate are produced by environmental insults (teratogens such as alcohol, retinoic acid, maternal illness, protein/calorie malnutrition during pregnancy) interacting with one or more genes. There is increased risk for congenital malformations because of maternal age at the time of pregnancy. Additional risk factors include lack of prenatal care during pregnancy, cigarette smoking, lack of a balanced diet and the chronic use of non-prescribed drugs or substance abuse. If parents without a cleft have a child with a cleft, the chance that a subsequent baby will have a cleft is only two to four percent. If either parent has a cleft, the relative risks become about four to five percent for having a baby with a cleft. If both parents have clefts, the risks are much greater. In addition to the required tissue repair, children with cleft lip and palate may have difficulty hearing or speaking clearly. So, in this paper teratogenicity and cleft palate are highlighted.



Abstract - Pharma - 10

Immunomodulators

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The recent development of inhibitors of key immune response proteins has revolutionized the therapy of autoimmune diseases; these immunomodulator agents include monoclonal antibodies and receptor antagonists. The active agents of immunotherapy are collectively called immunomodulators. Some of these substances, such as granulocyte colony-stimulating factor (G-CSF), interferons, imiquimod and cellular membrane fractions from bacteria. Others including IL-2, IL-7, IL-12, various chemokines, synthetic cytosine phosphate-guanosine (CpG) oligodeoxynucleotides and glucans are currently being investigated extensively in clinical and preclinical studies. Immunomodulatory regimens offer an attractive approach as they often have fewer side effects than existing drugs, including less potential for creating resistance in microbial diseases. A substance that alters the immune response by augmenting or reducing the ability of the immune system to produce antibodies or sensitized cells that recognize and react with the antigen that initiated their production. Immunomodulators include corticosteroids, cytotoxic agents, thymosin, and immunoglobulins. Some immunomodulators are naturally present in the body, and certain of these are available in pharmacologic preparations. The first two immunomodulators to be used widely in IBD are azathioprine and 6-mercaptopurine, drugs that are chemically quite similar. They are used to maintain remission in Crohn's disease and ulcerative colitis. Both have a slow onset of action. Accordingly, they are usually given along with another faster-acting drug (such as corticosteroids). Other immunomodulators to treat IBD are cyclosporine A and tacrolimus (Prograf, both used for organ transplantation as well. Cyclosporine A has a more rapid onset of action (one to two weeks) than azathioprine and 6-MP. It is useful in people with active Crohn's disease, but only when given intravenously and at high doses. Both cyclosporine A and tacrolimus have been more effective in treating people with severe ulcerative colitis, and are generally given until one of the slower-acting immunomodulators begins to work or until the patient undergoes curative surgery. Tacrolimus can be used in Crohn's disease when corticosteroids are not effective or when fistulas develop. Tacrolimus may be applied topically for Crohn's disease that affects the mouth or perineal area. Topical tacrolimus is also used to treat *pyoderma gangrenosum*, an ulcerating skin disorder often associated with IBD. Methotrexate works more rapidly than azathioprine or 6-MP, and is given by weekly injections.



Abstract - Pharma - 11

Nsaid Induced Peptic Ulcer And Management

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Peptic ulcer disease (PUD) refers to a disruption of the mucosal integrity of the stomach, duodenum, or both, caused by local inflammation, which leads to a well-defined mucosal defect. This results from an imbalance between the damaging effects of noxious substances and the ability of the mucosa to defend against them. Non-steroidal anti-inflammatory drugs (NSAIDs) is the major cause of peptic ulcer. Gnawing pain, burning discomfort, and tenderness in the epigastric area are the symptoms and signs most commonly associated with peptic ulcer. Diagnostic procedures and tests used in evaluating peptic ulcer. Pharmacologic treatment involves acid-neutralizing drugs in the form of H₂-receptor antagonists, proton pump inhibitors (PPIs), and antacids and Cytoprotective agents. It involves the use of at least two antibiotics for 10 to 14 days. Ulcer risk reduction after H. pylori eradication therapy appears to be more marked in patients beginning NSAID therapy than in patients already receiving and tolerating NSAID therapy. Nonpharmacologic therapy includes endoscopic treatment. Gastroduodenal ulceration and bleeding are the major limitations to the use of non-steroidal anti-inflammatory drugs (NSAIDs). The development of safer NSAIDs or of effective therapies for the prevention of the adverse effects of existing NSAIDs requires a better understanding of the pathogenesis of NSAID-induced ulcer disease. NSAIDs can cause damage to the gastroduodenal mucosa. The presence of acid in the lumen of the stomach also contributes to the pathogenesis of NSAID-induced ulcers and bleeding, by impairing the restitution process, interfering with haemostasis and inactivating several growth factors that are important in mucosal defence and repair. In recent years, a fuller understanding of the pathogenesis of NSAID-induced ulcer disease has facilitated some new, very promising approaches to the development of stomach-sparing NSAIDs. The various treatment modalities of peptic ulcer using H₂, Proton pump inhibitors and other medications are explained in this paper.



Abstract - Pharma - 12

Myocardial Infarction

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Myocardial infarction is a major cause of death and disability worldwide. Coronary atherosclerosis is a chronic disease with stable and unstable periods. Myocardial infarction may be a minor event in a lifelong chronic disease, it may even go undetected, but it may also be a major catastrophic event leading to sudden death or severe hemodynamic deterioration. A myocardial infarction may be the first manifestation of coronary artery disease. There are a number of imaging tests that are used in the setting of acute myocardial infarction and acute coronary syndrome. It is clear that there is a definite role for imaging. Furthermore, the term myocardial infarction has major psychological and legal implications for the individual and society. It is an indicator of one of the leading health problems in the world, and it is an outcome measure in clinical trials and observational studies. With these perspectives, myocardial infarction may be defined from a number of different clinical, electrocardiographic, biochemical, imaging, and pathological characteristics. Although aspirin is an effective, inexpensive, and safe treatment of acute myocardial infarction. The term *myocardial infarction* pathologically denotes the death of cardiac myocytes due to extended ischemia, which may be caused by an increase in perfusion demand or a decrease in blood flow. AMI falls in the spectrum of acute coronary syndromes (ACS), which includes unstable angina (UA), non-ST-elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction (STEMI). Persistent elevation of the ST-segment on ECG signifies total occlusion of a coronary artery that causes necrosis of the myocardial tissue. This condition is STEMI. ACS without ST-segment elevation may either be NSTEMI or UA. NSTEMI is more severe than UA. In this condition, the ischemia in the cardiac tissue is extensive enough to release cardiac biomarkers (troponin I or T) into the blood, but the occlusion is not as complete enough to cause elevation of the ST-segment.



Abstract - Pharma - 13

Drug Interactions Of Antimicrobials

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As new classes of antimicrobial drugs have become available, and new uses found for older drugs, pharmacokinetic drug interactions with antimicrobials have become more common. Macrolides, fluoroquinolones, rifamycins, azoles and other agents can interact adversely with commonly used drugs, usually by altering their hepatic metabolism. The mechanisms by which antimicrobial agents alter the biotransformation of other drugs is increasingly understood to reflect inhibition or induction of specific cytochrome P450 enzymes. Macrolides inhibit cytochrome P450 IIIA4 (CYP3A4), which appears to be the most common metabolic enzyme in the human liver and is involved in the metabolism of many drugs, including cyclosporin, warfarin and terfenadine. Some quinolones preferentially inhibit CYP1A2, which is partially responsible for methylxanthine metabolism. Azoles appear to be broad spectrum inhibitors of cytochromes P450. Within each of these antibiotic classes, there is a rank order of inhibitory potency towards specific cytochrome P450 enzymes. By contrast, rifampicin (rifampin) and rifabutin induce several cytochromes P450, including CYP3A4, and hence can enhance the metabolism of many other drugs. By using in vitro preparations of human enzymes it is increasingly possible to predict those antibiotics that will adversely affect the metabolism of other drugs. In addition, between-patient variability in frequency of interaction may relate to differences in the activities of these enzymes. Although the mechanisms and scope of these interactions are becoming well characterised, the remaining challenge is how to best inform the clinician so that the undesirable consequences of interactions may be prevented.



Abstract - Pharma - 14

Management Of Hypertension In Dental Practice

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Dentistry has played an important role in the detection of patients with hypertension. Patients found to have high blood pressure at or beyond defined levels should be referred for a medical diagnosis and indicated treatment. Once the hypertensive condition is under control, oral and dental evaluation and treatment can be initiated. A significant number of patients with undetected high blood pressure or uncontrolled hypertension today are seeking dental treatment. These patients are at high risk for significant complications such as stroke, heart disease, kidney disease, and retinal disease. Those with very high blood pressure are at great risk for acute medical problems when receiving dental treatment. For those reasons, dentistry must continue to place an emphasis on the detection and referral of patients with high blood pressure. In addition, increased numbers of medically compromised patients are seeking dental treatment who should have their blood pressure monitored during the more stressful dental procedures, such as oral surgery, periodontal surgery, and placement of dental implants. This article reviews the recent advances in the dental and medical management of hypertension. It is important for dentists to be aware of hypertension in relation to the practice of dentistry. The first step in managing the patient with medical problems is acquiring a thorough health history; the second step is for the clinician to fully understand the significance of the disease that may be endorsed by the patient. Each identified condition can affect dental care in a unique manner. For example, medication prescribed for a medical condition might produce a problem during the administration of a local anesthetic, or it could interact with pain medication prescribed post intervention. The dental clinician needs to understand the potential complications that can occur as a consequence of dental treatment of a medically compromised patient and when pretreatment or post-treatment medication or emergency care is indicated. Dental management of the medically compromised patient requires acquisition of a complete health history of the patient. This should include documentation via questionnaire as well as a verbal history. A comprehensive health history questionnaire should include questions about the patients cardiovascular, hematologic, neural and sensory, gastrointestinal, respiratory, dermal, mucocutaneous, and musculoskeletal, endocrine, and urinary systems as well as questions related to sexually transmitted diseases, drug use (eg, alcohol, tobacco), allergies, x-ray exposure or treatment, medications, and hospitalizations.



Abstract - Pharma - 15

Pharmacological Approach To Migraine

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Migraine is a common episodic disorder, the hallmark of which is a disabling headache generally associated with nausea, and or light and sound sensitivity. The abortive (symptomatic) therapy of migraine ranges from the use of simple analgesics such as nonsteroidal antiinflammatory drugs (NSAIDs) or acetaminophen to triptans, antiemetics, or the less commonly used dihydroergotamine. Abortive treatments are usually more effective if they are given early in the course of the headache; a large single dose tends to work better than repetitive small doses. Many oral agents are ineffective because of poor absorption secondary to migraine-induced gastric stasis. A wide variety of agents are available for the symptomatic treatment of migraine headache, including over-the-counter analgesics, nonsteroidal anti-inflammatory drugs (NSAIDs), combination products, opiates, ergot alkaloids, corticosteroids, dopamine antagonists, and triptans. In the stepped-care approach, simple analgesics and NSAIDs are the recommended first step for the treatment of mild-to-moderate migraine headaches. Patients who do not respond to first-step treatments may be given ergots, combination products, dopamine antagonists, or triptans as the second step. Corticosteroids or opiates may be used as rescue treatment in patients who do not respond to second-step treatment. A stratified approach to care individualizes treatment based on the severity of the headache and other patient-specific factors. In a stratified approach, dihydroergotamine or triptans may be the first-step treatment for patients who present with a history of severe migraines that have responded poorly to previous treatments. Sumatriptan was the first triptan approved for the symptomatic treatment of migraine headache; newer triptans include zolmitriptan, naratriptan, and rizatriptan. Since sumatriptan is rapidly absorbed by the subcutaneous route, its time to onset of effect is shortest. Among triptan drugs that are administered orally, the relative time to onset may be shorter with rizatriptan than sumatriptan. Naratriptan has a longer time to onset but is associated with a lower rate of migraine recurrence than other triptans.



Abstract - Pharma - 16

Gene Therapy

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Gene therapy is an experimental technique that uses genes to treat or prevent disease. In the future, this technique may allow doctors to treat a disorder by inserting a gene into a patient's cells instead of using drugs or surgery. Researchers are testing several approaches to gene therapy, including replacing a mutated gene that causes disease with a healthy copy of the gene, inactivating, or "knocking out," a mutated gene that is functioning improperly, introducing a new gene into the body to help fight a disease. Although gene therapy is a promising treatment option for a number of diseases (including inherited disorders, some types of cancer, and certain viral infections), the technique remains risky and is still under study to make sure that it will be safe and effective. Gene therapy is currently only being tested for the treatment of diseases that have no other cures. Gene therapy is a treatment that involves altering the genes inside your body's cells to stop disease. Genes contain DNA the code that controls much of your body's form and function. Genes that don't work properly can cause disease. Gene therapy replaces a faulty gene or adds a new gene in an attempt to cure disease or improve your body's ability to fight disease. Gene therapy holds promise for treating a wide range of diseases, including cancer, cystic fibrosis, heart disease, diabetes, hemophilia and AIDS. The significance and various uses of gene therapy is explained in this paper.



Abstract - Pharma - 17

Drug Interaction Of Antimicrobials

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Antibiotics can sometimes interact with other medicines or other substances. This means that the effects of one of the medicines can be altered by the other. Some antibiotics, such as rifampicin and rifabutin (which can be used to treat tuberculosis and meningitis) can reduce the effectiveness of the combined oral contraceptive pill. Penicillin at the same time as a medication called methotrexate, which is used to treat some types of cancers and severe autoimmune conditions such as the skin condition psoriasis. This is because combining the two medications can cause a range of unpleasant and sometimes serious side effects. Cephalosporins may not be suitable to take with blood-thinning medications such as heparin and warfarin. Aminoglycosides with antifungals, cyclosporin, diuretics, muscle relaxants can risk of kidney and hearing damage has to be balanced against the benefits of using aminoglycosides to treat life-threatening conditions such as meningitis. Taking a tetracycline medications vitamin A supplements, retinoids such as acitretin, isotretinoin and tretinoin, blood-thinning medication, diuretics, kaolin-pectin and bismuth subsalicylate, insulin, atovaquone, sucralfate used to treat ulcers, lithium, methotrexate. Macrolide with simvastatin, terfenadine, astemizole and mizolastine. Fluoroquinolones with theophylline, ibuprofen, cyclosporin, glibenclamide. Some fluoroquinolones can intensify the effects of caffeine (a stimulant found in coffee, tea and cola), which could make you feel irritable, restless and cause problems falling asleep (insomnia). Need to avoid taking medication that contains high levels of minerals or iron as this can block the beneficial effects of fluoroquinolones. The various antibiotics and their interactions are included in this paper.



**DEPARTMENT OF PHARMACOLOGY
POSTER PRESENTATION**

Abstract - Pharma - 1

Hallucinogenic Drugs

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Hallucinogens are a type of drug that change a person's perception of reality. Also known as 'psychedelic drugs', hallucinogens make a person see, feel and hear things that aren't real, or distort their interpretation of what's going on around them. Some are quick acting, others take longer to take effect. Some hallucinogens are synthetically manufactured, like LSD (lysergic acid diethylamide), PCP (phencyclidine, or 'angel dust') and ketamine. Naturally occurring compounds found in particular plants. For example the peyote cactus produces the hallucinogen mescaline, while psilocybin is found in certain mushrooms, known as 'magic mushrooms'. They target specific centres of the brain to alter the interpretations of sensory input. For instance, a person might be looking at a blank wall, but their hallucinating brain will interpret the blank wall as moving and swirling, or perhaps covered in insects. The effects of hallucinogens depend on the type of drug, the strength of the dose, the physiology of the person taking them and their state of mind. Some of the common effects of hallucinogens include hallucinations of sight, sound, taste and touch a blurring of the senses, such as sounds being 'felt' or colours being 'heard' feeling detached from the body distortions of time, direction and distance, relaxation, accelerated heart rate, dilated pupils, nausea and loss of appetite. Like many other drugs, it is possible to build up a tolerance to hallucinogens. This means larger and larger doses need to be taken to achieve the same effect. Physical dependence on hallucinogens like PCP or ketamine is possible. If a person stops taking the drug, they may experience withdrawal symptoms. There is no safe level of drug use. Use of any drug always carries some risk. It's important to be careful when taking any type of drug. Hallucinogens affect everyone differently, based on: Size, weight and health, Whether the person is used to taking it, Whether other drugs are taken around the same time, The amount taken, The strength of the drug (varies from batch to batch). The effects of hallucinogens can last for 4 to 12 hours and can be different depending on which type of hallucinogen is used. The following may be experienced during this time: Feeling happy and relaxed, Seeing and hearing things that aren't there, Confusion and trouble concentrating, Dizziness, Blurred vision, Clumsiness, Fast or irregular heart beat, Breathing quickly, Vomiting, Sweating and chills. Common hallucinogens and complications are highlighted in this poster

14th & 15th October 2006



Abstract - Pharma - 2

Newer Trends In Insulin Administration

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Insulin therapy is effective at lowering blood glucose in patients with diabetes mellitus (DM)]. Insulin is a key player in the control of hyperglycemia for type 1 diabetes patients while it is required at later stage or in selective individuals in patients of type 2 diabetes. The major advances achieved in this area include the synthesis of human insulin analogues by recombinant technology. Insulin delivery systems that are currently available for the administration of insulin include insulin syringes, insulin infusion pumps, jet injectors and pens. The traditional and most predictable method for the administration of insulin is by subcutaneous injections. The ultimate goal would be to eliminate the need to deliver insulin exogenously and regain the ability of patients to produce and use their own insulin. The major drawback of current forms of insulin therapy is their invasive nature. In type 1 diabetes, good glycemic control usually requires at least three or more daily insulin injections. To decrease the suffering and improve the adherence in insulin regimens, the use of supersonic injectors, infusion pumps, sharp needles and pens has been adopted. The newer methods explored include the artificial pancreas with closed-loop system, transdermal insulin, and buccal, oral, pulmonary, nasal, ocular and rectal routes. This review focuses on the new concepts that are being explored for use in future. Use of syringes for insulin delivery is the most common method in use and it offers a wide choice of products that are easy to read and operate glycated haemoglobin, in adult patients without a higher rate of hypoglycaemia. Insulin pens more discreet compared with vials and syringes. Insulin pens combine the insulin container and the syringe into a single modular unit. Insulin pens eliminate the inconvenience of carrying insulin vials and syringes and are more accurate and less painful. Insulin pens are user-friendly, with decreased discomfort of injection, ease of cartridge replacement, insulin-dose setting dial use and prominence of audible clicks can all affect overall dose accuracy. These are the advantages over syringes and needles. The various insulin preparations are explained in a brief way in this paper.



Abstract - Pharma - 3

Drug Abuse And Drug Addiction With Reference To Alcohol

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The goal of treatment for alcoholism is abstinence. Among alcoholics with otherwise good health, social support, and motivation, the likelihood of recovery is good. Approximately 50% to 60% remain abstinent at the end of a year's treatment and a majority of those stay dry permanently. Those with poor social support, poor motivation, or psychiatric disorders tend to relapse within a few years of treatment. For these people, success is measured by longer periods of abstinence, reduced use of alcohol, better health, and improved social functioning. Treatment for alcoholism can begin only when the alcoholic accepts that the problem exists and agrees to stop drinking. He or she must understand that alcoholism is curable and must be motivated to change. Treatment has three stages: Detoxification may be needed immediately after discontinuing alcohol use and can be a medical emergency, as detox can result in withdrawal seizures, hallucinations, delirium tremens, and in some cases may result in death. Rehabilitation: This involves counseling and medications to give the recovering alcoholic the skills needed for maintaining sobriety. This step in treatment can be done inpatient or outpatient. Both are equally effective. Maintenance of sobriety: This step's success requires an alcoholic to be self-driven. The key to maintenance is support, which often includes regular Alcoholics Anonymous (AA) meetings and getting a sponsor. Because detoxification does not stop the craving for alcohol, recovery is often difficult to maintain. For a person in an early stage of alcoholism, discontinuing alcohol use may result in some withdrawal symptoms, including anxiety and poor sleep. Withdrawal from long-term dependence may bring the uncontrollable shaking, spasms, panic, and hallucinations. Treatment may involve one or more medications. Benzodiazepines are anti-anxiety drugs used to treat withdrawal symptoms such as anxiety and poor sleep and to prevent seizures and delirium. These are the most frequently used medications during the detox phase, at which time they are usually tapered and then discontinued. They must be used with care, since they may be addictive. There are several medicines used to help people in recovery from alcoholism maintain abstinence and sobriety. One drug, disulfiram may be used once the detox phase is complete and the person is abstinent. It interferes with alcohol metabolism so that drinking a small amount will cause nausea, vomiting, blurred vision, confusion, and breathing difficulty. This medication is most appropriate for alcoholics who are highly motivated to stop drinking or whose medication use is supervised, because the drug does not affect the motivation to drink.



Abstract - Pharma - 4

Oral Manifestations Of Poisoning

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Poisons are harmful substances to our body. They can be found in everyday products such as medicines (if overdosed), carbon monoxide and household cleaning products. The symptoms of poisoning are varied and their severity depends on the type, the amount and how the poison was taken. Poisons (also called toxins) are substances that can harm your body. They can be swallowed, inhaled, absorbed or injected. Accidental poisoning may be caused by household or environmental accidents, exposure to harmful substances, or accidentally overdosing on a harmful substance, such as paracetamol. Different poisons affect your body in different ways and can take effect quickly or over time. As such, the range of symptoms can be broad and varied. If someone suddenly becomes ill for no apparent reason and has unexplained symptoms, you should consider poisoning a possibility. It's particularly likely if you find him or her near a potentially poisonous substance. Symptoms of poisoning can include feeling sick or vomiting, sometimes vomiting blood, abdominal (tummy) pain, diarrhoea, dizziness, weakness or drowsiness, fever or chills (shivering), fast or irregular pulse, headache, confusion or irritability, pain or burning around or on the affected area, double or blurred vision, seizures (fits), stupor (being sleepy or unresponsive) or unconsciousness. The purpose of this paper is to describe the variety of chemical agents and medicines which can produce oral manifestations following poisoning. Rapid diagnosis is essential in covert cases to ensure early treatment. Furthermore, oral care is an important part of the general management of many poisoned patients.



Abstract - Pharma - 5

Teratogenicity

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Teratogenicity is the ability to cause developmental anomalies in a fetus. Things that can cause developmental abnormalities are known as teratogens, and they include things like viruses, chemicals, and radiation. Their study is known as teratology; all of these words share a Greek root meaning “monster,” a reference to the fact that some developmental abnormalities were viewed as monstrosities or marvels historically. Substances with teratogenic effects can damage the DNA of a developing fetus. They may cause anything from abnormal development of a limb to malformation of an organ, and the effects for the developing fetus can vary depending on the teratogen, the gestational age of the fetus, and other factors. Sometimes, prenatal exposure to teratogenic substances causes the death of the fetus, while in other instances, someone may be born with relatively mild anomalies like extra fingers or toes. Substances with known teratogenicity must be handled carefully. Pregnant women are encouraged to avoid exposure to such substances, and they are tightly controlled to minimize the risk of inadvertent release. As researchers have learned, however, sometimes the danger of a substance is not known until it is too late. Thalidomide, for example, was widely used in pregnant women until medical experts realized that it was causing developmental abnormalities. The absolute risk of 7-10% is about 3-5% higher than that in the general population. Barbiturates and phenytoin are particularly associated with congenital heart malformations, facial clefts, and some other malformations. Valproate and carbamazepine are associated predominantly with spina bifida aperta and hypospadias. Genetic predisposition to the teratogenic side effects of AEDs plays a role, codetermining the recurrence risk if the woman has previously given birth to a child with a major malformation.