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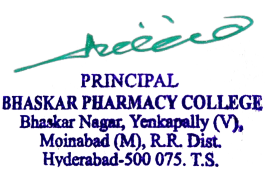

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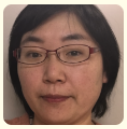

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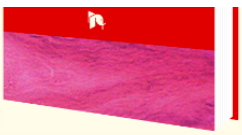

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The Gout

WRITTEN BY

Narottam Pal

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ABOUT THIS SPECIAL ISSUE

This special issue focuses on those aspects with much implication for the healthcare. The articles published in this special issue will certainly bring a positive effect for thedeveloping health care and to make use of available resources and to remove certain obsoletefactors and process which may delay or harm the existing health care system. It enhances maximum utilization of scientific knowledge to potentiate therapy and diagnosis in the healthcare system. Authors presented short review articles as well as research articles in which they focused recent developments in their subject, emphasising the aspects that, in their opinion, are most important. In addition, they provide short annotations to the papers that they consider to be most interesting from all those published in their topic over the previous year. The conference intends to focus on pre-clinical trials. The conference will be offering a unique gathering in respect to scenario of global challenges. It aims to bring together students, teachers, and researchers working in sciences. The conference is expected to be a platform for the gathering of the ideas for the development of clinical research.

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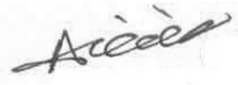
Dr.A.SRINIVASA RAO	M.PHARM,PH.D PRINCIPAL, BHASKAR PHARMACY COLLEGE	
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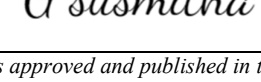
Dr.G.SUSMITHA	PHARM-D ASSISTANT PROFESSOR	

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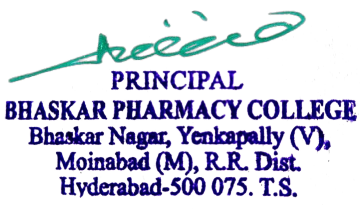

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SP-04

DEVELOPMENT AND CHARACTERIZATION OF ELETRIPTAN HYDROBROMIDE FASTDISSOLVING FILMS

MANNAM MOUNIKA[1] AND V LOKESWARA BABU[2]

Department of Pharmaceutics, Bhaskar Pharmacy College, Bhaskar nagar, Yenkapally (V), Moinabad (M), RR Dist., Hyderabad 500075, Telangana, India.

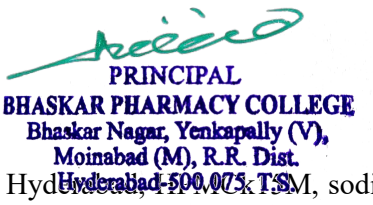
ABSTRACT

The aim of present study was to prepare fast dissolving films of eletriptan hydrobromide quick disintegration and faster dissolution with satisfactory taste in oral cavity. Film formulation can be taken within the pocket and patient can take it without need of water by simply putting it on the tongue without any grittiness that is frequently found during the disintegration of fast dissolving tablets. The developed formulation will disintegrate within a minute and ultimately provides good bioavailability and quick onset. The films were prepared by solvent casting method using various polymers as film former. The IR studies confirmed complete complexation of eletriptan hydrobromide with taste masking resin. The prepared formulations were evaluated for in vitro dissolution, disintegration time and physical properties. The optimized eletriptan hydrobromide film containing HPMC K15 and eudragit RS100, propylene glycol, glycerine showed better drug dissolution (more than 90% within 30 min) with satisfactory taste masking and other physicochemical properties. The development of fast dissolving film one of the alternative routes to provide quick onset of action.

Keywords: Eletriptan hydrobromide, FTIR Studies, Polymers, Solvent casting method, Fast dissolving film, Drug release studies.

INTRODUCTION

Oral route is the most preferred route of administration for systemic effect. About 60% of all the formulations are solid dosage form. Tablet is the most preferred dosage form due to ease of transportation, specially designed for the drugs which have extensive manufacturing and more patient compliance[1, 2.] Fast dissolving drug delivery system is a new generation delivery system also known as fast dissolving disintegrating film for the oral delivery of the drugs. The delivery system consists of a very thin oral strip which is simply placed on the patient's tongue or any other oral mucosal tissue and instantly gets wetted by saliva. The film rapidly hydrates onto the site of application. It then rapidly dissolves and disintegrates to release the medication for oro-mucosal absorption. It improves the efficacy of APIs by getting dissolved within few minutes in the oral cavity after coming in contact with saliva, without chewing and no need of water for administration[3.] Eletriptan hydrobromide is a selective 5-hydroxytryptamine 1B/1D (5-HT1B/1D) receptor agonist, used in the treatment of migraine attacks. The terminal elimination half-life of Eletriptan is approximately 4 hours, and is primarily metabolized by cytochrome P-450 enzyme CYP3A4 after oral administration[4, 5].



MATERIALS AND METHOD

MATERIALS

Eletriptan hydrobromide was collected as a gift sample from Hetero labs, Hyderabad, HPMC K15M, sodium alginate, eudragit RS100 and other excipients were purchased from AR chemicals.

METHODODOLOGY

FTIR Studies [6]

IR study was performed for polymer incompatibility studies with that of Eletriptan hydrobromide using Fourier transformed infrared spectrophotometry. The KBr disk technique was employed using 1:1 ratio of Eletriptan hydrobromide and various polymers. The study was repeated separately for each polymer blend with Eletriptan hydrobromide.

SP-05

HEPATOPROTECTIVE ACTIVITY OF TABEBUIA ARGENTEA LEAF EXTRACTS AGAINST PARACETAMOL INDUCED LIVER DAMAGE INALBINO WISTAR RATS.

A SRINIVASA RAO[1], S SHOBHA RANI[2], M SRI RAMACHANDRA*[3,]

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[2] Institute of Science & Technology, JNTUH, Hyderabad, India.

ABSTRACT

The study was designed to evaluate *in vitro* antioxidant and hepatoprotective activity of *Tabebuia argentea* leaf extracts against Paracetamol induced liver damage in albino Wistar rats.The *in vitro* antioxidant activity was evaluated by measuring superoxide radical, hydrogen peroxide radical, hydroxyl radical, nitric oxide radical reducing power and estimation of phenolic content of petroleum ether, methanolic and aqueous extracts of *Tabebuia argentea*. Hepatoprotective activity of the extracts was screened against Paracetamol (3gm/kg b.w) at doses of 200 and 400 mg/kg by estimating biochemical parameters (SGPT, SGOT, SALP, TB and TP), physical parameters (liver weight, liver volume) and histopathological changes in liver with silymarin (50mg/kg, b.w.) as standard.Methanolic, aqueous and Pet.ether extracts of plant showed good antioxidant activity. Administration of plant extracts resulted in significant reduction in SGPT, SGOT, SALP and TB and increase in TP as compared to disease control group. They also reduced the liver weight and liver volume. In plant extract treated groups histological aberrations were reduced as compared to disease control. Among all the extracts the META showed a significant effect at high dose (400 mg/kg) ($p< 0.001$) compared to lower dose. This evidence showed that the plant has antioxidant and hepatoprotective activity. From the results it can be concluded thatthe*Tabebuia argentea* possesses antioxidant and hepatoprotective effect against Paracetamolinduced liver damage in rats.

Key words: ***Tabebuia argentea*, Paracetamol, Silymarin and Hepatotoxicity**

1. INTRODUCTION:

Liver is a vital organ which maintains homeostasis by performing various activities like production of bilirubin, plasma proteins, metabolism of carbohydrates, lipids, proteins and detoxification of chemicals and drugs etc[1]. *Tabebuia argentea* (*Bignoniaceae)* is a flowering trees and commonly called as 'silver trumpet tree with Silvery gray leaves, bark is corky, Leaves are palmate, opposite, 11 inches long and 4 inches wide.[3] They are rich source of many organic compounds, especially, of phenolic and polyphenolic substances. It was intended to investigate the hepatoprotective activity of *T.argentea* using paracetamol model.

2. MATERIALS AND METHODS:

2.1 Preparation of plant extracts: The leaves of *Tabebuia argentea* were collected and authenticated by Dr.*Madhava Setty, d*epartment of Botany, S.V University, tirupati. The leaves were shade dried and powdered. The powder was subjected to successive solvent extraction by using petroleum ether, methanol and water. Then the dried extract was obtained by evaporation of the solvent using a rotatory vacuum evaporator at 50 ºC and kept in dessicator and studied for phytochemical, *in vitro* antioxidant activity and examined for their hepatoprotective activity in rats.

2.2 *In Vitro* antioxidant studies: Petroleum ether, methanol and aqueous extracts were subjected for *in vitro* antioxidant studies namely viz., superoxide[4], hydrogen peroxide[5] , nitric oxide [6] hydroxyl radical scavenging activity [5] and reducing power[7]. Its total phenol content [8]were also studied.

2.3. Experimental animals:

Healthy Wistar albino rats weighing 170-180g were acclimatized for 7 days and selected for study. Standard

SP-15

REVIEW ARTICLE ON HALF CORD PARALYSIS: TRANSVERSE MYELITIS

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ABSTRACT:
Acute transverse myelitis (ATM) is an etiologically heterogenous syndrome with acute onset, in which inflammation of spinal cord results in neurologic deficits, manifesting as weakness, sensory loss and autonomic dysfunction. The chance of occurrence is 1-4 people in 1 million[1], it can be associated with infection of bacteria, virus, fungi and auto immune disorders which constitute as 40% of etiology, rest 60% is idiopathic (unknown). It can affect persons from age 10 to 70 years. By treatment it can be cured if diagnosed very soon if not it is nearly complete treatable and can maintain it with medications and physiotherapy.

INTRODUCTION:
Transverse Myelitis is defined as inflammation of the myelin sheath of neurons[4] present in the spinal cord. This inflammation may be through the length i.e. longitudinal (or) it can be perpendicular i.e. transversely, so based on the inflammation the name is given.In either way of inflammation, it causes some of the sensory and motor signal obstruction due to inflammation of myelin sheath which results in non-functioning of some of the body parts because of improper signaling through it.

ETIOLOGY AND OCCURENCE:
There are many idiopathic ways by which Transverse myelitis can occur and among those known are infections through various kind of microorganism and autoimmune disorder[2].Transverse myelitis is rare condition occurring to an individual in which a individual looses control over posture of body and voluntary actions.

SYMPTOMS:
Rapid onset of weakness, bladder dysfunction and alteration of sensory and motor function.[5]

PATHOPHYSIOLOGY:
The progressive loss of protective fatty myelin sheath around nerves in the affected or infected spinal cord occurs for unclear reasons following infections and autoimmune response of the body. Infection may cause inflammation of spinal cord where as auto immune response may demyelinate the neurons of spinal cord which causes non-functioning or improper functioning of motor and sensory functions of body[1].

TREATMENT:
The recovery from the Transverse myelitis differs from person to person depending on the underlying cause. Few populations recover within 2-12 weeks of treatment some take longer than that and some patient may not show any sign of improvement.
If it is treated in early, patient felt completely or nearly complete recovery from the condition[3].
Treatment include medications depending upon the cause and other suggestions like physiotherapy, plasmapheresis.

CASE:
CLINCAL PRESENTAION:
A 13 years old female patient came to a tertiary hospital with chief complaints of following cough associated with sputum, fever (low grade) with intermitted chills and rigors, and blebs on gluteal mass.
When she came her posture was not good, she was malnourished, and had anemia, she lost control for walking and was unable to all kind of activity from lower region of body.

DIFFRENTIAL DIAGNOSIS:

SP-16

MUSHROOM THERAPY-PREVENTING THE ADR'S OF ANTI NEOPLASTIC AGENTS- A REVIEW

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Department of Pharmacy Practice, Bhaskar Pharmacy College, Hyderabad- 500075, India.
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ABSTRACT:
Cancer is perilous and is reason for quietus all over the creation, espousing long term repercussion for a life time. Hence many drugs have been hatched in anticipation of cancer nevertheless engendering reactions. So an auxiliary remedy for the carryover of anticancer drugs is to avail oneself of mushrooms. As mushrooms bear active compounds alike β-glycans, β-proteoglycans,lectins, triterpenes, ergosterols, glutamine, arginine, they are urging compromising effect in chemotherapy. Widely utilized mushrooms are of genus *Cordyceps, Fomes, Laricifomes, Ganoderma, Grifola, Lentinula, Piptoporus, Inonotus, Agaricus.* Yet an investigation stipulate that the organic food along with periodic ingestion of mushrooms declines the threat of emerging cancer.

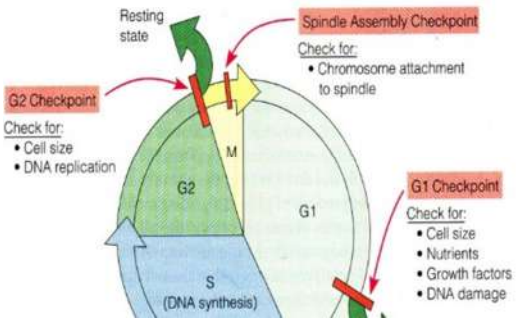
KEYWORDS: Cancer, Quietus, Auxiliary remedy, Mushrooms, Clinical trials, Potency, Anti tumor effects.

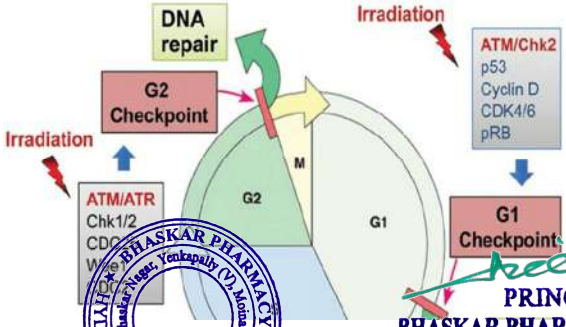
INTRODUCTION:

The gauge of attack of cancer in 2019 is about 1 million and fatal rate is estimated to be 6 lakh. Unfortunately highest fatal rates is due to uterine cancers and next place occupies the lung and skin cancer separately(U.S Department of Health and Human services, NIH,NCI). As there is huge increments in fatal rates steadily, it is important to decrease mortality rates to some extend by proper medical adherence. But the majority of consequences rising from the therapy mainly radiotherapy and chemotherapy, results in impairing and depleting the patient's immune system and triggering several unwanted effects[1].Keeping all these in mind, certain alternative remedy like *biotherapy*have been approached. Biotherapy includes monoclonal antibodies, cancer vaccines, interferons, interleukins, colony-stimulating factors, gene therapy, non-specific immune modulating agents[1]. Apart from all these, use of medicinal mushrooms dug a path in fields of therapeutics. These mushrooms not only have anti-cancer properties, but also can act against cardiovascular diseases, diabetes, act as anti-viral, anti-bacterial, anti-parasitic, anti-inflammatory, nephroprotective, neuroprotective and hepatoprotective properties[1].But medicinal mushrooms gained attentiveness due to its immune modulatory and immunostimulatory properties. Hence these are regarded as *immunomodulators or biological response modifiers.*

Cell cycle and regulation:

1) *Cell cycle checkpoints*: It is a stage at which the cell examines internal and external conditions and decides whether to cause cell division or not. There are many checkpoints but the most important are G1, G2, M check points.
2) *Cell cycle regulators:* The proteins that regulate the cell cycle are called as CCR's. They are cyclins, CDK's, APC/C complex, retinoblastoma protein, p53 protein and p21 protein.





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AQUAGENIC URTICARIA

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Abstract
The other term used for Aquagenic Urticaria is water allergy or water urticaria . It is rarely diagnosed form of physical urticaria. This usually occurs when the skin corner in contact with water (in some patients even tears, sweat) and causes wheals. They are pin head-small pea sized wheals surrounded by variable sized erythematousflares following skin contact with water on face, neck and trunk, irrespective of temperature or source, pathogenesis of aquagenic urticaria is unknown.

KEYWORDS:Aquagenic urticaria, water, physical urticaria, diagnosis

INTRODUCTION
Water is found everywhere in our daily lives and it is harmless, but for some patients water is the main problem. Such person is unable to bear a few minutes of moisture on his body and by chance if he comes in contact with water he develops severe symptoms of allergy like itching, redness on skin. This type of allergy is regarded as Aquagenic urticaria.

DEFINITION:
Aquagenic urticaria is rare form of physical urticaria caused when skin comes in contact with water, irrespective of temperature and source, causes wheals[1,2]. Wheals are small punctate lesions are located mainly on upper body(neck,trunk,shoulder,arm,back)[3].

EPIDEMIOLOGY:
Aquagenic urticaria is first seen in shelly and rawnsley in 1964[4]. Nearly 100 cases reported in literature, most commonly seen in females during puberty or post puberty and some of them reported itching too. There have been reports of childhood onset disease, some are sporadic in nature, while several cases of familial disease[6].

CAUSES
Some of the researches are still working to know the causes of aquagenic urticaria. some of them are presence of Chemical additive in water, like chlorine causes development of wheels. Allergy like symptoms are experienced from the rash is due to release of histamine. Due to allergic reaction, immune system release histamine in response to fight against the harmful substance[5]. Histamine causes allergy like symptoms and affects parts of the body.

Pathogenesis
The exact mechanism of aquagenic urticaria is not known. The mechanism of aquagenic urticaria of first reported case Shelly and rawnsely in 1964 is hypothesized that when water comes in contact with sebum or sebaceous glands of skin it produce a toxic component, leads to degranulation of mast cells and release of histamine leads to formation of urticarial lesions[4]. Some of them proposed ,that it is due to change in osmatic pressure cause indirect action of urticaria[8]. Another mechanism is presence of water-soluble antigens in the epidermis which diffuse and dissolve into dermis and causes release of histamine from mast cells[7]. Recent studies proposed that they are completely independent of histamine release ,based on reports of several patients with aquagenic urticaria[5].

Clinicalpresentations
Symptoms develops within 30minutes after contact with water irrespective of temperature and source. The pruritic wheals of(1-3mm diameter) surrounded by (1-3cm) erythematous flares which usually last less than 1hour[4] and located on the neck, upper trunk and arms. Other symptoms include pruritus, burning sensation,

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PSITTACOSIS (PARROT FEVER)

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ABSTRACT:
Psittacosis, otherwise parrot fever and ornithosis, is an infection which is caused by bacteria namely known as chlamydia psittaci. It leads to severe pneumonia and other health problems in humans. Chlamydia psittaci can infect different kinds of birds and is referred to as avian chlamydiosis. Most often human cases are seen after the exposure to infected parrot type of birds which are used as pets particularly cockatiels, budgerigars, macaws and also from non-psittadian species such as pigeons, sparrows, ducks, hens, gulls. The disease is contracted to the humans through the exposure bacteria present in feces and nasal discharges, shaded by infected birds.

INTRODUCTION:

Figure -1 Bird with psittacosis[12]

Definition: parrot fever and ornithosis are the other terms of psittacosis. Psittacosis is a respiratory tract infection which is clinically reported with influenza like symptoms (fever, headache, malaise, productive cough for 50% but delayed) and perhaps commonly known as simple community-acquired pneumonia (CAP) or influenza.[5]

- The term psittacosis is arrived because the major cause
- for the transmission is through the infected birds mainly identified to be psittacine (parrot fever) birds, especially cockatiels and budgerigars.[2]
- Among non-psittacine birds (pigeons, doves, poultry
- species such as hens, ducks, turkeys) are likely to be affected, hence ornithosis is the term for psittacosis.[2]

Etiology: Psittacosis (respiratory tract infection), chlamydial psittaci which is an obligate intracellular bacterium is the causative agent of infection and was 1st described in 1879.

- Chlamydial psittaci is a bacterial species, belongs to the family chlamydiaceae.[1]
- Although there are other sources that person may get affected, contact with the infected birds plays an important role and major risk factor for illness.[2]
- Genotype-A is indigenous among cockatoos, parakeets and Lories(psittaci forms) which is well known as zoonotic agent.[3]
- Genotype-B is indigenous for pigeons and doves.[3]
- Genotype C and D are mostly detected in non-psittacine birds.[3]
- But early studies says that Genotype E/B strain was identified in both parrots and humans but so far psittaci forms are most frequently affected by Genotype-A strain.[3]

SP-20

MECONIUM ASPIRATION SYNDROME

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ABSTRACT:
Meconium aspiration syndrome is common condition that is see in 8-25 % of all deliveries. Meconium is the first intestinal discharge of the newborn, which is composed of intestinal secretions such as bile, mucus and intestinal epithelial cells; this is formed during 3rd trimester of intrauterine life. In post-dated babies due the increased motilin, there is a Release of meconium into the amniotic fluid, making it contaminated and thereby it is aspired into the lungs of baby causing respiratory distress and so called meconium aspiration syndrome

INTRODUCTION:

It is a common cause of morbidity and mortality[9,1,4]seen in 8-25% of all deliveries, where the baby experiences respiratory distress due to aspiration of meconium stained amniotic fluid[5.] Usually the meconium is the first stool passed just after the baby is delivery i.e. within 24hrs[3]. It is composed of bile acids, desquamated skin, proteins, intestinal epithelial cells, lanugo, cholesterol and its precursors, lipids, enzymes. Enzymes include pancreatic phosholipaseA2[7.] 80-90 %[3,8] of water is also present it and it appears to thick, black greenish colored. The MAS is observed in both preterm and term babies but most commonly seen in term babies[2], where the gastric maturation of the baby results in the passage of meconium, due to increased gestational age. Passage of meconium was highly associated with birth asphyxia and resulted in a bad prognosis. Fetal distress is directly correlated with the APGAR SCORE, therefore meconium stained amniotic fluid along with low APGAR score[3], will ultimately lead to poor fetal outcome309.

ETIOLOGY:
The causes include prolonged labor, increased levels of motilin, maternal hypertension, [1]low APGAR score[,3] utero-placental insufficiency[3],oligohydramnios[1], ante partum hemorrhage.

PATHOPHYSIOLOGY:
Normally meconium passage from the fetus into amniotic fluid is prevented by lack of intestinal peristalsis, caused due to the low levels of motilin, but in case of postdated babies[8], due to transient parasympathetic stimulation due to the head compression, increase in motilin levels and cholinergic innervations. Apart from fetal maturation, meconium is also passed due to relaxation of anal sphincter which is caused by inutero-stress with hypoxia and acidosis.[3]

1.BALL-VALVE OBSTRUCTION:
When meconium enters the lungs, it causes air trapping thereby, results in mechanical obstruction to airways. Meconium migrates from proximal to distal airways during breathing movements and cause complete atelectasis[7], ventilation perfusion mismatch and air leak[2].

2. PNEUMONITIS:
Within few hours of meconium aspiration into the airways, there is an accumulation of macrophages and neutrophils, as the meconium acts as a chemoattractant. This further results in release of pro inflammatory mediators such as TNF-alpha, IL-8,RESULTING IN PARENCHYMAL INJURY AND CAUSES TOXIC PNEUMONITIS[2,8].

3. SURFACTANT INACTIVATION:
s bile acids and cholesterol which are the components of meconium will interfere with the lung surfactant changing its viscosity and ultrastructure thereby; results in surfactant in inactivation .meconium present

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A CAUSATIVE RISK OF COLORECTAL CANCER BY RED MEAT

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ABSTRACT:
Colorectal Cancer is the 2ND Most Leading cause of death .It is 2nd most common in Women & 3rd Most Common in Men. It occurs due to changes in the Genes that lies in Colon & Rectum due to which there is no control on Cell Growth that may result in escape of Growth (or)Fast Growth which becomes Tumor leading to COLORECTAL CANCER. These genetic changes builds up over the years,& by the age of 50-60 people starts with development of POLYPS , which are small growths on the lining of the intestinal tract that develops into cancer,and these few of polyps becomes causative agents for Cancer.Major Risk of Colorectal Cancer is due to RED MEAT or PROCESSED MEAT, since it contains Large amount of Iron ,Creatine ,Minerals such as Zinc & Phosphorus,and vit B (niacin,Vit B12,thiamin & riboflavin, Also red meat is a source of Lipoic Acid, other related risk factors are:Age above 45yrs, Inflammatory Bowel Disease(IBD), Family history ,Diet-high Fat diet, low fiber, Smoking ,Sedentary lifestyle, History of Polyps, Obesity .The Signs & Symptoms of Colorectal Cancer Includes : A Persistent change in bowel habbits including diarrhea or constipation or change in consistency of your Stool. Rectal bleeding or blood in your stool, persistent abdominal discomfort, such as gas ,Cramps, or Pain.Weakness or fatigue , Unexplained weight loss.Mostly the Diagnosis of Colorectal Cancer is done by Colonoscopy, Tissue Biopsy during Sigmoidscopy .However Preventive measures when taken will reduce the risk of colorectal Cancer by reducing the intake of Red Meat or Processed Meat in diet,changing the sedentary lifestyle , doing physical activity, Avoiding Smoking &Alcohol consumption, Diary Foods, Fried Foods ,including dietary Fiber.

KEYWORDS: **Red Meat, Processed Meat,Colorectal Cancer, Polyps Formation,Sedentary Lifestyle.**

INTRODUCTION:

Colorectal Cancer , also known as Bowel Cancer.It is a is a disease originating from epithelial cells lining the colon or rectum of the GIT,most frequently as a result of Mutations that are inherited or acquired and most probably occur in the Intestinal crypt stem cell. Current research shows there are certain chemicals in red &Processed meats-both added & naturally occurring-that cause these foods to be carcinogenic.For example, When a chemical in red meat called Haem is broken down in gut ,N-nitroso chemicals are formed &these have been found to damage the cells that lines the bowel,which can be called as bowel cancer. [9] These chemicals also form when processed meat is digested. In addition,the nitrite & nitrate preservatives used to preserve processed meat produce these N- nitroso chemicals &can lead to bowel cancer.The Major cause of Colorectal Cancer is due to productive intake of Red Meat over time which develops polyps in the intestine which and further becomes tumor in colon firstly and due to lack of appropriate measures further growths in rectum. A Subset of colorectal Cancer is characterized with deficient DNA mismatch repair. This phenotype has been linked to mutations of Genes such as MSH2, MLH1, &PMS2,these mutations results in so called high frequency microsatellite instability (H-MSI)which can be detected with an immunocytochemistry Assay.H-MSI is a hallmark of hereditary non polyposis Colon cancer syndrome(HNPCC,LYNCH SYNDROME)which accounts for about 6%of all colon cancer.H-MSI is also found in about 20% of sporadic colon cancer.[10]

Other important genes in colon carcinogenesis include the KRAS oncogene,Chromosome 18 loss of heterozygosity (LOH) leading to inactivation of SMAD4(DPC4)& DCC Tumor suppression genes. Chromosome arm 17p deletion &mutations affecting the p53 tumor suppression gene confer resistance to Programmed Cell Death(Apoptosis)& are thought to be late in colon carcinogenesis.[3]

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SP-23

A REVIEW ON SODIUM GLUCOSE CO-TRANSPORTER(SGLT-2) INHIBITORS

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ABSTRACT:
All over the world, incidence of diabetes is increasing day by day. Even after using proper medications, the target to stop this is not reached. Sadly, only half of the population is able to get control over it. Type- 2 diabetes adds the risk of cardiovascular and macro and micro vascular complications. SGLT-2 inhibitors are the latest group of drugs for treating Type - 2 diabetes having insulin independent action. The appreciable worth of using these drugs is that they show increment in urinary glucose excretion without causing hypoglycaemia and causes weight loss. This review highlights the physiology and pharmacology of SGLT-2 inhibitors.

INTRODUCTION:

Currently available anti-hyperglycaemic drugs work either by improving β-cell secretion of insulin(e.g.: sulfonylureas, and the new incretin agents) or by improving peripheral and hepatic insulin sensitivity (e.g.: metformin and pioglitazones). Even though, current anti-hyperglycaemic drugs have proven efficacy, they cause weight gain on long term use.[5]Recently a new class of anti-hyperglycaemic agents have been approved, which have better glycaemic control and improve insulin resistance in Type 2 DM patients. This new class of drugs are renal sodium linked glucose transporter-2 (SGLT-2) inhibitors which reduce the blood glucose levels by blocking renal glucose reabsorption system. SGLT-2 inhibitors acts by excretion of glucose in urine. They are indicated for glycaemic control and reduction of already ingested calories.

ROLE OF KIDNEYS IN GLUCOSE HOMEOSTASIS:

In normal individuals, glucose homeostasis is maintained by regulating production, reabsorption and utilisation of glucose. In spite of tight glucose regulation some individuals develop diabetes or hypoglycaemia[6]. Kidneys play a crucial role in filtration and reabsorption of glucose. Kidneys filter approximately 180gm of plasma glucose each day, of which 99% filtered plasma glucose is reabsorbed in proximal convoluted tubule. In normal individuals the reabsorption of glucose is 100% i.e., 180gm of filtered glucose is reabsorbed.[7] When there are hyperglycaemic conditions the amount of filtered glucose reabsorbed increase in proportion to the plasma glucose concentration. When the glucose levels exceeds the maximum capacity of the carrier proteins, which is usually 200mg/dl of plasma glucose concentration, glucose appears in the urine. In diabetes patients, the hyperglycaemia causes hyperfiltration of glucose by the kidneys and increases the luminal glucose levels, which exceeds the maximum reabsorption rate resulting in glucosuria.[8]

MECHANISM OF GLUCOSE TRANSPORT BY THE CARRIER PROTEINS ACROSS MEMBRANE:

Cell membrane is made up of lipids, which is impermeable to glucose, as glucose is a polar compound. So the glucose reabsorption in PCT and its transport across cell membrane needs carrier proteins which are located in cell membrane. Glucose enters the cell via two types of cell membrane associated carrier proteins: they are the facilitated glucose co-transporters(GULTs) and the active sodium coupled co-transporters (SGLTs). Secondary active sodium and glucose symporters – SGLT 1 and SGLT 2 helps in glucose transport through the apical membrane of intestinal and kidney epithelial cells. The active glucose transport is coupled with the downhill transport of sodium across the basolateral cells into the intracellular fluid. The energy produced by this transport is used in the concentration of glucose inside the cells. GLUTs catalyse the facilitated diffusion of glucose across the basolateral membrane. This process is independent of energy. GLUT-4 is primarily involved in insulin mediated glucose uptake in muscles and adipose tissue

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A REVIEW ON RECIPROCATION OF CANCER AND CIRCADIAN RHYTHMS: EMERGING THERAPY FOR CANCER.

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ABSTRACT:
Circadian rhythm is the natural cycle of physical, mental and behavior changes that the body goes through in a 24-hour cycle. Disruption of circadian rhythm is associated with a variety of human pathologies, including cancer. The Circadian clock controls the "On" and "Off" cycling of many functions that are important for cancer development. REV-ERB proteins are the key components of the clock machinery that repress biological functions that cancer cells depend on, such as cell division and cell metabolism. This suggests that pharmacological modulation of clock-related proteins may be suitable as an effective Anti-cancer strategy. The integration of circadian biology into cancer research and a better appreciation of this underlying fundamental biological phenomenon and its potential for chronotherapy could benefit many cancer patients worldwide.

Keywords:Circadian rhythm, REV-ERB Proteins, Cancer therapy, Chronotherapy.

INTRODUCTION:

Cancer is a condition in which abnormal cells divide uncontrollably and can invade the nearby tissues. It can also spread to other parts of the body through the blood and lymph systems. The treatment options include Surgery, Chemotherapy and Radiation therapy. Targeted therapies are also available for some types as of now. Cell physiology is regulated by 24-hour Circadian clock that are coordinated by the Suprachiasmatic nucleus, a hypothalamic pacemaker that will help the organism in adjusting to the environmental cycles.[1] Altered Circadian rhythms predict poor survival in cancer patients. The concept of treating cancer patients according to the biological clock is termed as Chronotherapy. It aims for administering the drugs at an appropriate time of the day to show maximum efficacy and minimum side effects. Several experiments have been conducted which reported positive associations between circadian clock and drug response in cancer patients.[2]The nuclear hormone receptors REV-ERB alpha and REV-ERB beta are found to be very crucial. Two REV-ERB receptor agonists i.e. SR9009 AND SR9011 have been developed with in vivo activity. They induced apoptosis of almost all the cells that had different mutations that drive cancer growth without causing any harm to the surrounding normal cells. In cells that had undergone oncogene induced senescence REV-ERB agonism blocked autophagy and induced apoptosis.[3,4]Thus, we summarize the link between the cancer and disruption of circadian rhythms.

CENTRAL AND PERIPHERAL CLOCK:

The circadian rhythms are hierarchical, and the central clock in the human body is composed of a biological pacemaker that is located in the brain's suprachiasmatic nucleus (SCN).[2]Peripheral clocks are synchronized by signals from the central clock are present in nearly all other tissues of the body. For synchronization, a major signal is the daily light–dark cycle that is detected by photoreceptors present in the mammalian retina, which relay signals to the SCN. These electronic and endocrine signalsgenerated are sent to organs and tissues in the body (peripheral clocks) via the endocrine and autonomic nervous systems, thus leading to synchronization. Peripheral cellular clocks also respond to other stimuli including temperature shock and glucocorticoids.[1,5]However, without external indicationor from a central clock, they lose synchronization with one another.

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CADD IN ANTICANCER DRUG WITH RECEPTOR PDB ID (3RE2 PROTEIN)

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ABSTRACT:
Cancer can be described as the uncontrolled growth of abnormal cells. Menin is a tumor suppressor protein that is encoded by theMEN1(multiple endocrine neoplasia 1) gene and controls cell growth in endocrine tissuesare present in cancers cells. 3RE2 proteins inhibitors play a vital role in deactivating cancer disease.[12]Some of the commonly used cancerdrugs are Imatinib, Sunitinib, Tandutinib etc.These drugs mainly work against the effects of kinase .Methods: The Protein- Ligand interaction plays a role in structural baseddrug designing. In our research work I have taken the Protein available in protein data bank PDB ID 3RE2& commercially available drugsSunitinib against cancer. The 3RE2was docked to the above said drug Sunitiniband the energy value obtained is -242.94 using the Argus Lab & Hex docking software. Results: Depending on the energy values we have chosen the marketed drug. We tried to improve the binding efficiency and steric compatibility of the drugs namely Several modifications were made to the groups which were interacting with the receptor molecule. This drug molecule was prepared using ACD ChemSketch and docked using Argus Lab &Hex version 5.1dockingsoftware.Discussion: Taking as a reference drug,several analog structure can be prepared to get a potent lead molecules.From this work we can improve the steric[3] compatibility and ADMET of the drugs.Further research is under process.

Keywords: **Cancer,Sunitinib,MEN1,Argus lab,Hexversion5.1**

INTRODUCTION:

Anticancer[1] chemotherapy ,targeted drug therapy are now a days a very active field of research, so it clearly indicate that there is a need of an updated treatment from the point of view of medicinal chemistry & drug designing.[1]In silico drug designing now a days is very updated technology to discover drug molecules with less consumption of materials &manpower.The basic target of our work is to develop some novel antineoplastic [10]compound & forward for further evaluation.Antitumour[2] chemotherapy,Menin is a tumor suppressor proteinnow a days a random target for cancer[7] chemotherapy.The effect of drug structure on MEN1activity will be seen in the docking studies.

OBJECTIVES OF WORK:

- Identification of cancer drug target.
- In silico analysis of compound library against drug.
- Selection of scaffold based in silico result.
- ADMET of chemical compound to check their suitability of drug.
- Docking of drug with a particular protein.

APPROACH:

The basic focus of current study is to elucidate the docking of antineoplastic[2] drug from the atomistic point of view of chemistry on the relationship between chemical structure & chemical & biochemical reactivity of antineoplastic agent aiming at rationalization of the action of drug in order to allow the design of new active molecules.

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A REVIEW ON CURRENT TRENDS IN INSULIN THERAPY: SCOPE AND FUTURE

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ABSTRACT:
Diabetes Mellitus is a metabolic disorder characterized by elevated blood glucose levels.All patients with T1DM and Many patients with advanced type 2 diabetes mellitus (T2DM) require insulin to maintain blood glucose levels in the normal range. insulin injections via subcutaneous route are most commonly used and it is the standard route of administration but it isassociated with injection pain, needle phobia, lipodystrophy, noncompliance and peripheral hyperinsulinemia.To overcome thenoncompliance, various alternative and noninvasive insulin delivery methods have been developed

KEY WORDS: Diabetes Mellitus, Hyperinsulinemia, noninvasive insulin delivery.

INTRODUCTION:

Diabetes mellitus prevalence is increasing worldwide and insulin is necessary to maintain the blood glucose levels in the normal range in diabetes patients by various insulin delivery methods [1,2]. The most commonly used method of insulin administration is via subcutaneous routes such as vials, syringes, insulin pens and pumps. Alternative insulin delivery systems that are non invasive are currently available for the administration of insulinwhich includesoral, nasal,buccal, mucosal, transdermal, nasal, peritoneal. All these delivery systems are developed to provide predictable and effective lowering of glucose levels in the blood. [3,4]This review focuses on various insulin delivery techniques with its advantages and disadvantages in the management of Diabetes.

INSULIN DELIVERY TECHNIQUES: Insulin is administered through subcutaneous route by using vials, syringes and pens.[5]

CURRENT TRENDS IN INSULIN THERAPY: Vials, syringes, and insulin pens are currently used for delivering insulin among which insulin pens are user friendly and are less painful. Insulin pumps are also availablein current generation which is more user friendly with calculated doses and alarms.[6]

Sensor–augmented pump therapy:sensor-augmented pump therapy includesCGM (continuous Glucose Monitors) readings toadjust insulin delivery through insulin pump. SAP requires patient involvement for using CGM readings to adjust insulin pump delivery and patient to wake up to manage nocturnal hypoglycemia. It is designed to reduce the severity and duration of hypoglycemia.[7,8,9]

Advancements in insulin therapies:
Injectable Insulins:

Insulin degludec and VIAject: Insulindegludec is an ultra-long acting insulin used to reduce the blood glucose levels in diabetic patients.it has single amino acid[threonine] deleted from B-chain in the position 30 in comparison to human insulin, and is conjugated to hexadecanedioic acid via glutamic acid linker to lysine in the position 29 of beta chain[10] .It was developed by NovoNordisk under the brand name Tresiba.VIAject is an ultra fast acting insulin that absorbs more rapidly and it is used to reduce the postprandial oxidative stress and improves the endothelial functions. [11,12]

NEW TECHNIQUES TO DELIVER INSULIN:
1.INHALED INSULIN DELIVERY:

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A REVIEW OF APPROACHES IN DEVELOPMENT OF ECO RBC

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ABSTRACT: The entire system of blood collection, distribution and transfusion revolves around ABO Blood groups. Elimination of this need of classification can be done by enzymatic conversion of A, B, AB to O group which can revolutionise blood banking. Current transfusion problems such as blood supply shortage, blood borne diseases, and emergencytransfusions can be solved by this technique.The antigens of A,B,AB differ only in the terminal monosaccharide N-acetyl galactosamine for group A, galactose for group B and no additional saccharide for group O. A possibility was observed that clipping these saccharides would convert them to H antigen.α-galactosidase reacted with RBC survived normally *in vivo*. Transfusions carried out in clinical trials showed no rejection or haemolysis. A slight rise in anti-b titrewas observed with no explanation.30 times more effective enzymes are available which can directly be injected into blood bag. This technique holds tremendous promise and further research can ensure development of this technology into reality.

KEY WORDS: ECO RBC, Transfusions, Enzymes, Blood, Galactosidas

INTRODUCTION

According to WHO, India suffers from an annual deficit of two million blood units, as only 1% of Indian population donate blood each year. In 2016, the Ministry of Health and Family Welfare reported a donation of 10.9 million units against a requirement of 12 million units. There is a shortage of 30,00,000 blood units every year. one road accident victim can require up to 100 units of blood. Blood group O being universal donor is most requested by hospitals. It can be transfused to A, B and AB recipients. By seeing the great demand of O blood group, scientists discovered different methods of blood conversion and making all blood type universal donor. Enzyme conversion of A,B,AB group was proposed more than 30 years ago as it was a promising approach to achieve the goal of producing universal donor RBC to improve blood supply and enhance safety of clinical transfusion.

VARIOUS APPROACHES TO UPGRADE ENZYMATIC CONVERSION OF RBC

Previous approaches involved masking the antigen on RBC with PEG but survival rate *in vitro*was not considerable as it was immunogenic and induced production of antibodies in rabbits (Garratty, 2004)[1]. an alternate approach was enzymatic conversion of A & B antigen to universally accepted O blood group which was done by Goldstein *et al* (1982).This would eliminate shortage of blood supply. The major distinction between the A, B, AB&O lies with additional α1-3- linked sugar. A cells have α1-3- linked N acetyl galactosamine and B has α1-3- galactose, O cells have no sugar and AB has a mix of both chains. Study was carried out at American Society of Hematology where Goldstein used glycolytic enzyme to alter sugars present on RBC surface.

α galactosidase from Sanos coffee beans(*coffee canephora*)was extracted and purified .RBC treated with α galactosidase maintained structural integrity and functionality *in vitro.* Cells did not haemagglutination. Treatment of RBC with α galactosidase resulted in conversion of B cells to O cells .clinical trials were done in which patients were given 2 units of ECORBC. No antibody responses post transfusion of ECORBC were recorded. Anti B titers monitored in all O group recipients remained unchanged post transfusionand no evidence of overt hemoglobinuria was observed. However this enzyme suffered low turnover rates and required copious amount of enzyme (1-2 gm/unit B cell).another challenge was maintaining pH 5.5 while carrying out conversion reactions which rendered the approach impractical[2,8].

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CLINICAL EVALUATION OF ADVERSE DRUG REACTIONS IN PROTRACTED ILLNESS PATIENTS IN A TERTIARY CARE TEACHING HOSPITAL

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ABSTRACT

The study mainly evaluates the causality, severity, preventability of adverse drug reactions and associated factors with the development of ADRs in chronic disease patients from various departments of tertiary care teaching hospital.A Prospective observational study was conducted in a tertiary care teaching hospital at Hyderabad, India, for a period two years. All the patients were distributed according to their gender, age, number medications used, disease condition, and socioeconomic state. The reported ADRs were analyzed by WHO-UMC causality assessment, Hartwig's Siegel's scale and modified Shumock and Thornton criteria respectively. Descriptive statistics were used for data analysis. A total of 691 patients enrolled in the study, in that 391 patients reported with 510 ADRs. Of these 37.0% are in-patients and 62.9% are out-patients. Majority of the patients are from female category (58.0%) and 45.8% of ADRs reported from adult age group (41-60 years). 65.8% patients are non-adherent to their prescription medication. Life style habits, economic status and education are also found to be predictors in experiencing ADRs. WHO's ADR probability scale showed that 42.9% of ADRs were probable.Hartwig's and Siegel's the severity assessment scales shown that 13.1 % ADRs are severe followed by 33.7% moderate ADRs and 40% of ADRs were preventable.This study provides a database of ADRs due to commonly used drugs. Hence our study advises that there is a need of improvement in ADR reporting from health care professionals. This study also suggests further research in India for the improvement of possible intervention strategies to reduce burden and cost of ADRs.

Key words: Diabetes mellitus, Adverse drug reactions, Spontaneous reporting, Naranjo's and Hartwig's Siegel's Severity assessment.

INTRODUCTION

According to WHO Pharmacovigilance (PV) is defined as the science and activities relating to the detection assessment, understanding and prevention of adverse effects or any other drug-related problem. WHO established its Programme for International Drug Monitoring in response to the thalidomide disaster detected in 1962.[1] The objective of PvPI is, to monitor ADRs in Indian population, to create awareness amongst health care professionals about the importance of ADR reporting in India, to monitor benefit-risk profile of medicines, generate independent, evidence based recommendations on the safety of medicines, support the CDSCO for formulating safety related regulatory decisions for medicines, communicate findings with all key stake holders and create a national centre of excellence as par with global drug safety monitoring standards.[2]

It is generally recommended to treat each chronic condition in accordance with disease-specific guidelines. However, most clinical practice guidelines do not modify or discuss the applicability of their recommendations for older patients with multiple diseases and following all guidelines for each and every drug a patient is taking will inevitably lead to polypharmacy.[3]According to estimates, India has the highest number of adults with diabetes reported at 50.8 millions in 2010 which is expected to rise to 87 million by 2030.[4] The prevalence of diabetes has been reported to be rapidly increasing in both rural and urban India.[5] Prescriber's knowledge about pharmacokinetics and pharmacodynamic aspects of medicines and their interaction with normal aging physiology is critical in the management of diabetes mellitus. The knowledge needed to minimize and even avoid the potentially adverse effects of hypoglycemia and side effects associated with the anti-diabetic drugs.[6]

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A REVIEW ON ROLE OF IGF-1 IN OSTEOPOROSIS.

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ABSTRACT:
Osteoporosis is a progressive metabolic bone disease that decreases bone density with deterioration of bone structure, skeletal weakness leading to fractures with minor or inapparent trauma. A new treatment is introduced to treat osteoporosis i.e. Insulin like growth factor(IGF-1)is a primary mediator for effects of growth hormone.Growth hormone is secreted in the anterior pituitary gland is released into the blood streams and then stimulates the liver to produce IGF-1.IGF-1 then stimulate systemic body growth on every cell of body especially skeletal muscles cartilage bone.IGF-1 can also regulate cellular DNA synthesis.Both GH and IGF-1 administration significantly increase bone resorption and bone formation.It is also stipulated but highly debated that IGF-1 can increase the size of a tumour in cancer patients. It can be taken in the form of tablets or injections.

KEY WORDS: Osteoporosis, GH, IGF-1, Bone resorption, Osteoclasts, Osteoblasts, Bone metabolism, Tumor, Side effects, Dosing.

INTRODUCTION:

One of the vitaltissues of our body is bone, which undergoes continuous resorption by osteoclasts and new bone is formed by osteoblasts[(1)].The growth hormone/insulin-like growth factor I (GH/IGF-I) axis is pivotal for the management of bone formation. GH and IGF-I play a major part in the formation of bone mass in adolescence as well as maintain the bone mass in the adult stage. Osteoporosis and bone-loss disorders are caused due to the lack of GH/IG-1[2,3,4,5].Aging results in a rapid decline in the number of osteoblasts and less osteoblast activity, but osteoclast activity is not changed. Secondary osteoporosis is associated with bone loss which may result from different causes, like Cushing's syndrome, or overuse of glucocorticoids[6,7].

The IGF-1 plays a crucial part in cellular growth, survival, and cell cycle progression, differentiation[8] and IGF-1 level is necessary for the proper building of high bone mass; furthermore, GH protects against ovariectomy-induced bone loss. IGF-1 helps to reduce osteoblast apoptosis and promotes osteoblastogenesis through the phosphoinositide 3-kinase (PI3K) pathway[9].Pre-clinical studies showed that IGF-1-deficient mice developed smallerskeletonswithasignificantdelayinmineralization.In agreementwiththefindingsfrompre-clinical studies, clinical studies showed a positive association between serum IGF-1 levelsandBone-Mass-Densityindifferentracial groups[10,11].Osteoporoticfractures are caused due to low serum levels of IGF[12,13]. This explains us about the importance of IGF-1in bone formation and mineralization.

GH/IGF-1 AXIS:

GH is secreted by the anterior pituitary gland which is a polypeptide hormone. The GH primarily acts in the liver where it stimulates IGF-1 production[14]. The action of GH is mediated by the binding of GH to the trans membrane GH receptor (GHR) which is present on the surface of most cells[15]. The GH has two different actions i.e., dependent and independent mechanism of action, one directly through the GHR and the other inducing IGF-1 secretion by the liver. Circulating IGF-1 is mostly synthetized in the liver, but IGF-1 is expressed in all tissues, suggesting that paracrine/autocrine effects of local IGF-1 may be a major mechanism controlling tissue growth [16].

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