MALLIGE COLLEGE OF PHARMACY

#71, SILVEPURA, CHIKKABANAVARA POST, BANGLORE - 560 090

(Recognized by AICTE, PCI, New Delhi, RGUHS Bangalore)

Web: www.mallige.ac.in, E - mail: mcpbangalore@ymail.com, Ph: 080-28446666, 9353729763

Number of research papers published per teacher in the Journals no5fied on UGC website during the last five years

Academic Year	No. of Research Papers
2022	17
2021	10
2020	15
2019	04
2018	04
2017	01

MALLIGE COLLEGE OF PHARMACY IQAC CELL BANGALORE

MALLIGE COLLEGE OF PHARMACY BANGALORE

PRINCIPAL,
Mallige College of Pharmacy
Bangalore-560 090

3.3.1 Number of research papers published per teacher in the Journals notified on UGC website during the last five years

Title of paper	Name of the author/s	Department of the teacher	Name of journal	Year of publication	ISSN number	Link to the recognition in UGC enlistment of the Journal /Digital Object Identifier		
						Link to website of the Journal	Link to article / paper / abstract of the article	Is it listed in UGC Care list
Formulation and Evaluation of Ganciclovir Novel Buccoadhesive Tablets with Different Polymers	Sujatha Muchalambe, Dependra Kumar, Suma, Mamatha	Pharmaceutics	International Research Journal of Pharmacy and Medical Sciences	2019	2581-3277	http://irjpms.com/	http://irjpms.com/wp-content/uploads/2019/07/IRJPMS-V2N5P19-19.pdf	google scholar, IP indexing
Nephrotoxicity, its mechanism and biomarkers: A systemic Review.	Hemalatha S, Shivakumar Swamy, Narayan Sah Sonar, Hariprasad MG, Nandini	pharmacology	Nephrology and Hepatology Science.	2019		https://www.ipinnovative.com/journals/IJUNHS	https://www.ipinnovative.com/journal-article-file/10660	Crossref, doi,
Mechanism of Haematotoxicity induced by Phenylhydarzine: A Review.	Shwetha BR, SiddalingaPrasad HS, Shivakumar Swamy, Nagalakshmi NC, Hariprasad	pharmacology	Journal of applied Pharmaceutical Research.	2019	ISSN No. 2348-0335	https://www.japtronline.com/	https://www.japtronline.com/index.php/joapr/article/view/112	Crossref, CAS, google scholar, indian itation index, Scilit, J-Gate
Hepatotoxicity and Hepatotoxicants: A systemic Review.	Narayan Sah Sonar, Nagalakshmi NC, Hemalatha S, Hariprasad MG	pharmacology	Indian Journal of Pharmacy and Pharmacology.	2019	Print ISSN: 2393-9079, Online ISSN: 2393-9087	https://www.ijpp.org.in/	https://www.ijpp.org.in/article-details/10486	Crossref, Crossmark,Clockss, NISO, ORCID

Review Article

Nephrotoxicity, its mechanism and biomarkers: A systematic review

Hemalatha S[1*], Shivakumar Swamy[2], Narayan Sha Sonar[3], Hariprasad M.G[4], Nandini S[5]

[1,3,5]Student, [2]Principle and Professor, [4]Professor, Dept. of Pharmacology, Mallige College Of Pharmacy, Bengaluru, Karnataka, India

***Corresponding Author: Hemalatha S**
Email: lathahema3396@gmail.com

Abstract
Nephrotoxicity is a common condition responsible for variety of pathologic conditions on kidney due to the destruction of renal function by one or more nephrotic substance. For renal injury to occur, combination of risk factors (drug, kidney and patient related factors) are generally present. The mechanism involved in nephrotoxicity include altered intraglomerular haemodynamic, crystal nephropathy, inflammation, tubular cell necrosis, throbotic microangiopathy, rabdomyolysis. Nephrotoxicity can be assessed by estimating the blood urea nitrogen (BUN), centralization of serum creatinine, glomerular filtration rate and creatinine clearance. Development of new biomarkers are required for specific diagnosis of nephrotoxicity at earlier stages.

Keywords: Nephrotoxicity, Risk factors, Mechanism, Biomarkers.

Introduction
Kidney assumes a significant role in filtration of some noxious substances, which are the fundamental driver for the nephrotoxicity.[1] Medication instigated nephrotoxicity is a very basic condition and is answerable for an assortment of obsessive impacts on the kidneys[2]. The nephrotoxicity of medications is a convoluted procedure that includes a blend of elements. These incorporate the natural nephrotoxic capability of medication, basic patient attributes that improve their hazard for kidney damage, and the digestion and discharge of the potential culpable specialist by the kidney.[3] Acute renal failure (ARF) represented 20% of all ARF case.[2] Finlay et al. characterize nephrotoxic medications (ND) as therapeutic operators that can possibly cause unfriendly impacts on renal capacity because of direct harmfulness or traded off renal perfusion, and this lethality may rely upon the clinical setting included. The sorts of kidney brokenness that are induced by nephrotoxic medications incorporate acute tubular necrosis, glomerular and tubulointerstitial damage, haemodynamically interceded damage and obstructive nephropathy.[4] Exposure to drugs regularly brings about lethality in kidney which speaks to the significant control framework keeping up homeostasis of body and along these lines is particularly powerless to xenobiotics.[5]

Most medications found to cause nephrotoxicity apply harmful impacts by one or more pathogenic components. These incorporate adjusted intraglomerular hemodynamics, tubular cell lethality, inflammation, crystal nephropathy, rhabdomyolysis, and thrombotic microangiopathy[6]. Nephrotoxicity can be analyzed through a basic blood test. Assessment of nephrotoxicity through blood tests incorporates the estimations of blood urea nitrogen (BUN), centralization of serum creatinine, glomerular filtration rate and creatinine clearance.[5] There is an ascent in the advancement of biomarkers for recognizing nephrotoxicity, as standard strategies are not dependable because of absence of particularity and sensitivity.[1] However, these evaluations of nephrotoxicity are just conceivable when a larger part of kidney work is damaged.[5]

In this article we summarize the risk factors (hazard factors), mechanism of nephrotoxicity and biomarkers of nephrotoxicity.

Hazard factors[3]
Renal explicit components

1. High rate of blood conveyance to kidney [approximately 20% of heart output].

Journal of Applied Pharmaceutical Research
Volume 7, Issue 4, Year of Publication 2019, Page 1 – 6
DOI: 10.18231/j.joapr.2019.v.7.i.4.001

Review Article

JOURNAL OF APPLIED PHARMACEUTICAL RESEARCH | JOAPR
www.japtronline.com
ISSN: 2348 – 0335

MECHANISM OF HAEMATOTOXICITY INDUCED BY PHENYLHYDRAZINE: A REVIEW

Shwetha B R[1], Siddalingaprasad H S[2], Shivukumar Swamy[1], Nagalakshmi N C[1], Hariprasad M G[1]*

Article Information
Received: 12th March 2019
Revised: 19th August 2019
Accepted: 23rd September 2019

Keywords
Haemolytic anaemia, Erythropoietin receptors, Phenylhydrazine, Haematotoxicity

ABSTRACT
This work was carried out to show the effects of phenylhydrazine (PHZ) induced anaemic condition. Anaemic condition is defined as reduction in red blood cells (RBC) than normal number of red blood cells. The anti-anaemic activity can be studied using the changes in haematological parameters (PCV, RBC & Haemoglobin) influenced by PHZ [(40mg/kg p.o.)] in rats. PHZ, a potent chemical that causes different effects on different tissues at several levels. Administration of PHZ causes haemolytic anaemia, genotoxic effects and rose in iron absorption in spleen, liver and duodenum & causes change in iron metabolism. PHZ acts by activating immune response which triggers phagocytosis and also interfere with the binding of erythropoietin (EPO) receptors and further JAK-STAT pathway. PHZ also causes genotoxic effect by forming single strand DNA damage. In view of lipid peroxidation along with the formation of Thiobarbituric acid (TBA)-reactive malonyldialdehyde, it is recommend that PHZ induces anaemia as an outcome of peroxidation of RBC membrane lipids and this effect may be a upshot of the autoxidation of the drug and the interaction of membrane lipids and oxygen radicals

INTRODUCTION

In 1895 Hermann Emil Fischer used PHZ for various reactions in sugars. PHZ has some adverse effect on human subjects. PHZ exposure may cause red blood cell damage and in turn leads to anaemia, it may also cause complications on the other tissues like spleen and liver. PHZ is proved to be mutagenic invitro and known to exhibit genotoxicity invivo in rats [1]. PHZ is employed to make phenyl hydrazone of natural mixtures of sugars so as to render the differing sugars easily separable from one another. This Molecule is also found to induce acute haemolytic anaemia in animal models. PHZ is one of the major intermediates used in the various industries for variety of purposes. Due to the toxic effects of Phenyl hydrazine derivatives, use of them as anti-pyretics has been stopped [2].

[1]*Mallige College of Pharmacy, Department of Pharmacology, Silvepura, Bangalore-90*
[2]*Department of Studies and Research in Biochemistry, Tumkur University, Tumakuru- 572103*

***For Correspondence:** hariprasadmg2004@yahoo.com

Review Article http://doi.org/10.18231/j.ijpp.2019.024

Hepatotoxicity and hepatotoxicants: A systematic review

Narayan Sah Sonar[1*], N C Nagalakshmi[2], Hemalatha S[3], Hariprasad MG[4]

[1,3]Student, [2]HOD, [4]Professor, Dept. of Pharmacology, Mallige College of Pharmacy, Bengaluru, Karnataka, India

***Corresponding Author: Narayan Sah Sonar**
Email: sarraf.narayan@gmail.com

Abstract
Liver act as metabolic factory of the body and metabolism of virtually every foreign substance. Its functioning is crucial for health and disease. Chemicals that cause liver injury are called hepatotoxicants. More than 950 drugs have been implicating in causing liver injury and were withdrawn from the market. Mechanism of hepatotoxicity induced by drug, immune based and genetic based toxicity. The drug models which are applied to study hepatotoxicity are Paracetamol, CCl4, Galactosamine, Alcohol, Azathioprine, Rantidine and different classof drugs cause acute and chronic liver injury.

Keywords: Hepatotoxicity, Hepatotoxicants, Mechanism, Liver injury, Drugs.

Introduction
Liver is the fundamental organ for digestion and metabolism of medicines.[1] Hepatotoxicity alludes to liver dysfunction or liver harm that is related with an over-burden of medications or xenobiotics[2] and some of the time in any event, when presented inside therapeutics ranges, may harm the organ,[3] may result not just from direct poisonous quality of the essential compound yet additionally reaction influencing hepatocytes, biliary epithelial cells and additionally liver vasculature.[2]

The synthetics that reason liver damage are called hepatotoxins or hepatotoxicants.[2]

Hepatotoxicants are exogenous mixes of clinical significance and may incorporate overdose of certain therapeutic medications, mechanical synthetic concoctions, regular synthetic concoctions like microcystins, home grown cures and dietary supplements,[2] these operators are convert in synthetically receptive metabolites in liver, which can interconnect with cell macromolecules, for example, protein, lipids and nucleic acids, prompting protein brokenness, lipid per oxidation, DNA harm and oxidative stress.[3]

An awkwardness among forceful and defensive powers brings about harm to the liver and complex system are associated with such hepatotoxicity actuated by an assortment of ecological and synthetic specialists.

Liver-the target organ
Liver performs in excess of 500 fundamental metabolic capacities. It is associated with the union of items like glucose got from glycogenesis, plasma proteins, thickening variables and urea that are discharged into the circulatory system. It directs blood levels of amino acids.[2] Liver parenchyma fills in as a capacity organ for a few items like glycogen, fat and fat solvent nutrients.[2] It is likewise engaged with the creation of a substance considered bile that is discharged to the intestinal tract. Bile helps in the evacuation of poisonous substances and fills in as a channel that isolates out harmful substance.[2] Simultaneously liver is inclined to numerous ailments like hypersensitivity to nourishment and includes safe framework too.[1] Hepatitis is caused due to infections, toxic substances, autoimmunity and can likewise result from non-alcoholic greasy liver malady associated with stoutness and steatosis.[1] Hepatic encephalopathy is brought about by aggregation of poisons in the circulatory system that are typically expelled by the liver.[1] Liver harm can likewise be brought about by drugs, such as tubercular drugs, general soporifics, paracetamol in moderately high doses and some against drugs used in oncology. Harmful hepatitis is the most extreme antagonistic response to antituberculosis drugs,[1] it generally starts in the initial scarcely any long stretches of treatment alongside liver putrefaction, which may advance to encephalopathy and death.[1] Alcoholic liver sicknesses with cirrhosis brought about by inordinate liquor utilization is a typical event.[1] Liver can now and again be harmed by certain synthetic compounds called hepatotoxins, for example, galactosamine and chloroform.[1] Additionally, steroids, immunizations and antiviral medications which are utilized as treatment for liver ailments, may create unfriendly impacts particularly after constant organization.[1] There are in excess of 900 medications that can prompt hepatotoxicity and is one of the significant explanations behind a portion of the medications pulled back from showcase.[1]

Mechanism of hepatotoxicity
Medication related hepatotoxicity can't be seen as a solitary sickness. A wide range of components lead to hepatotoxicity, including disruption of the cell membrane and cell death resulting from covalent binding of the drug to cell proteins, which creates new adducts that serve as immune targets, in this way actuating an immunologic response hindrance of cell pathways of medication digestion, strange bile stream coming about because of interruption of subcellular actin fibers or interference of

2019

International Research Journal of Pharmacy and Medical Sciences
ISSN (Online): 2581-3277

Formulation and Evaluation of Ganciclovir Novel Buccoadhesive Tablets with Different Polymers

Sujatha Muchalambe[1], Geetha Lakshmi[1], Dependra Kumar[1], Suma[2], Mamatha[2]
[1]Department of Pharmaceutics, R.R College of Pharmacy
[2]Department of Pharmaceutics and Pharmacognosy, Mallige College of Pharmacy

Abstract— *The main objective of purpose study is to formulate buccal drug delivery system of Ganciclovir by using different types of mucoadhesive polymers which may increase the intimacy and duration of contact between drug- containing polymer and a mucous surface which will increase the residence time of drug in the body and finally increase the bioavailability of this highly water soluble drug. The direct drug absorption and decrease in excretion rate will also increase the bioavailability. Buccal cavity was found to be the most convenient and easily accessible site for the delivery of therapeutic agents for both local and systemic delivery as retentive dosage form.*

Keywords— *Ganciclovir, Mucoadhesion, HPMC K15M, Ethylcellulose.*

I. INTRODUCTION

The concept of mucoadhesives was introduced in the early 1980s. Mucoadhesion can be defined as the phenomenon of the attachment of natural or synthetic polymers to a mucosal surface[1]

Since from the last 40 years, the concept of mucoadhesion has provided the great application in prolonging the residence time as well as controlled release effect of various bioadhesive dosage forms through different mucosal routes. The formulations based on the mucoadhesive drug delivery system have shown the enhanced bioavailability of many drugs. The use of various mucoadhesive polymers have achieved the significant interest in formulating the sustained release, extended release as well as prolonged release dosage forms. The mucoadhesive drug delivery provides greater absorption and enhanced bioavailability of dosage forms due to the large surface area and higher blood flow in the mucosal cavities[2]

The main advantages of these formulations are: drug targeting, sustained release, increased permanence time in the buccal mucosa, increased bioavailability, and decreased potential adverse effects[3].

Mucoadhesion is known to increase the intimacy and duration of contact between drug- containing polymer and a mucous surface. It is believed that the mucoadhesive nature of the device can increase the residence time of the drug in the body. Increased residence time and adhesion may lead to lower API concentrations and lower administration frequency to achieve the desired therapeutic outcome[4].

Among all dosage form, oral route is more preferred to patient. Transmucosal routes of drug delivery offer distinct advantages over per oral administration for systemic drug delivery.

The buccoadhesive dosage forms along with sub-lingual tablets, oral gels and ointments, lozenges, rapidly dissolving tablets and chewing gums are the formulations targeting drug delivery in the oral cavity[5].

Bio-adhesion can be defined as a state in which two components, of which one is biological in origin, are held together for extended periods of time by the help of interfacial forces. It is denoted (esp. in pharmacy) as mucoadhesion since the main biomaterial involved is mucus present at various sites in the body[6].

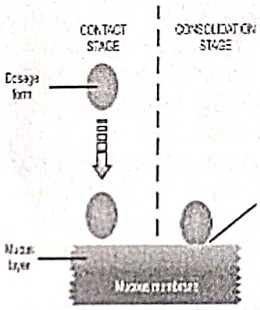

Fig 1: Two steps of mucoadhesion

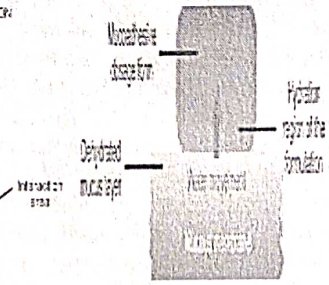

Fig 2: Mucoadhesion theory

Buccoadhesive dosage forms: Over the past few years, different dosage forms intended for buccal drug delivery have been developed. Buccal mucoadhesive dosage forms can be categorized into three types based on their geometry illustrated in the following figure 3.

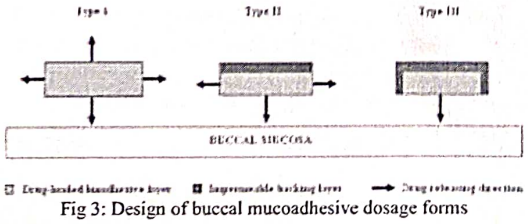

Fig 3: Design of buccal mucoadhesive dosage forms

II. MATERIALS AND METHODS

Ganciclovir was a gift Sample obtained from Natco Pharm ltd., Carbopol was received from Balaji Drugs , HPMC and Guar gum were from Yarrow Chem products Mumbai, Ethyl cellulose,Lactose and Magnesium Stearate from S. d Finechem limited, Mumbai.

Sujatha Muchalambe, Geetha Lakshmi, Dependra Kumar, Suma, and Mamatha, "Formulation and Evaluation of Ganciclovir Novel Buccoadhesive Tablets with Different Polymers," *International Research Journal of Pharmacy and Medical Sciences (IRJPMS)*, Volume 2, Issue 5, pp. 10-15, 2019.