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A Mini Review on Pancreatin: Prodigious Focus on Hepatic Ischemia-Reperfusion Injury

Anurag[1], Kuldeep Singh[2*], Soumyadip Mukherjee[3], Shivendra Kumar[4], Akash Garg[5], Sunam Saha[6], Brajnandan Dubey[7]

[1,2,3,4,5,6,7]Rajiv Academy for Pharmacy, NH-2, Mathura, Uttar Pradesh, India

Corresponding Author: Kuldeep Singh

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ABSTRACT

Pancreatin is a natural mixture of digestive enzymes derived from the pancreas of vertebrates. Recent studies have demonstrated that pancreatin has protective effects against various types of hepatic ischemia-reperfusion (I/R) injury. In this review, we summarize the existing evidence regarding the protective effects of pancreatin on hepatic I/R injury. We discuss the potential mechanisms behind the protective effects of pancreatin, including its anti-inflammatory, antioxidative, anti-apoptotic, and anti-fibrotic effects. Moreover, we discuss the potential application of pancreatin as a therapeutic agent in the treatment of I/R injury in the liver. Numerous studies have shown that pancreatin can protect against hepatic ischemia-reperfusion (I/R) injury by targeting multiple pathways and processes involved in I/R injury, including reduction in oxidative stress, suppressing inflammatory response, promoting cell proliferation as well as improvement in apoptosis process. In addition, pancreatin has a protective effect on liver injury by promoting the antioxidant defence system, inhibiting the accumulation of intracellular lipid peroxides, and restoring cellular antioxidant status. Thus, pancreatin may be a promising and effective treatment for hepatic I/R injury, and further research is needed to explore its clinical efficacy. In conclusion, pancreatin is a potential natural remedy that has been studied to potentially reduce hepatic I/R injury. Although further research is needed to confirm the efficacy of pancreatin in clinical settings, it has been suggested that it can act as a natural remedy for hepatic I/R injury due to its multiple mechanisms of action and its ability to restore the balance of oxidative stress. Pancreatin has been studied as a potential natural remedy for hepatic I/R injury due to its ability to inhibit lipid peroxidation, promote antioxidant defence system activity, and restore cellular antioxidant status.

Keywords: Pancreatin, hepatic ischemia-reperfusion, antioxidant activity, PI3K-AKT pathway, hepatoprotective strategies.

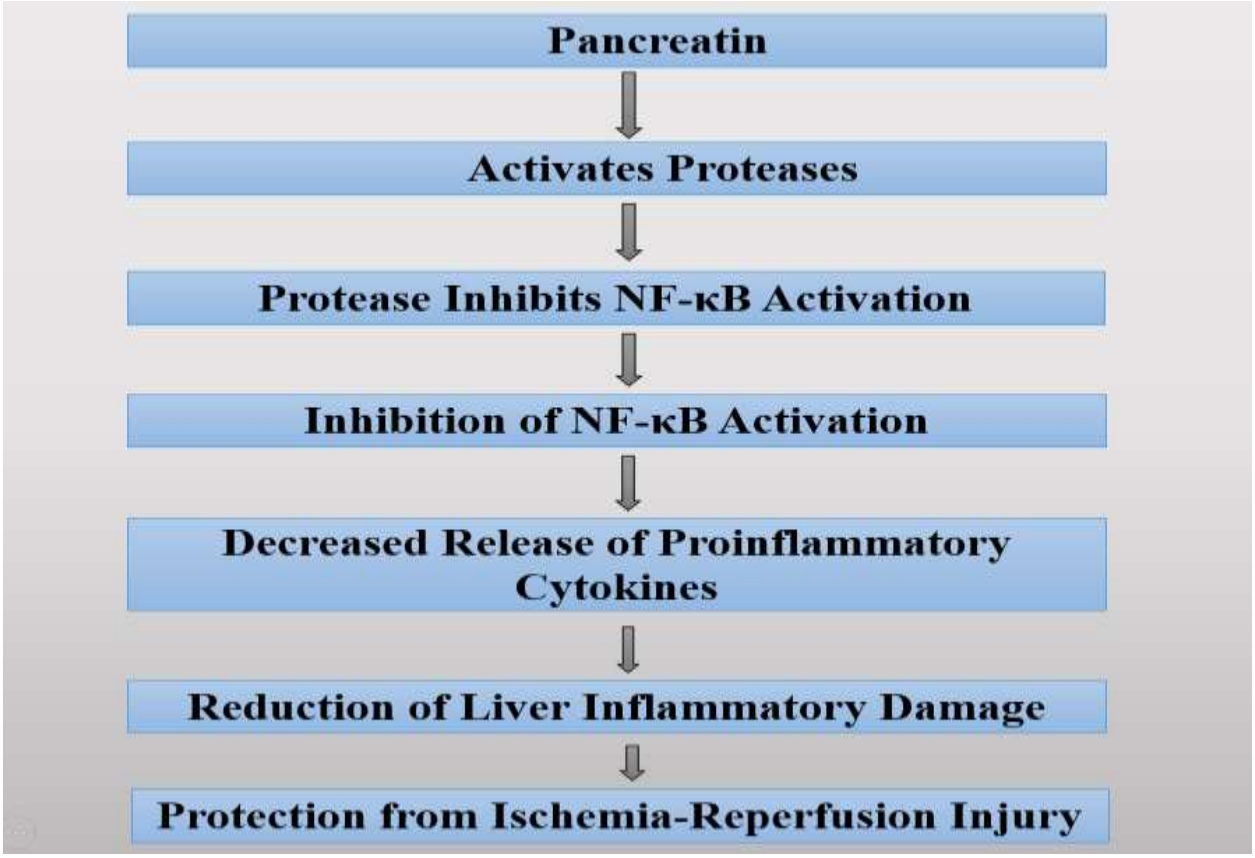
INTRODUCTION

Pancreatin is a combination of enzymes that are naturally produced by the pancreas to help digest food. It consists of several digestive enzymes, including proteases (which break down proteins), amylases (which break down carbohydrates), and lipases (which break down fats)(1). Hepatic is a form of pancreatin derived from the pancreas of hogs, which is then processed to ensure it remains sterile and safe for human consumption. It is used to aid in digestion by breaking down food into smaller pieces that can be more easily absorbed by the body(2). It is commonly used to treat digestive disorders such as irritable bowel syndrome, pancreatic exocrine insufficiency, and malabsorption syndrome. Hepatic is a popular choice of supplement for those who suffer from digestive disorders, as it provides the body with the digestive enzymes it needs to break down food properly(3). Hepatic is an effective and safe choice for treating digestive issues, as it provides the body with the necessary

enzymes to break down food and absorb nutrients efficiently. While the use of hepatic enzymes is not a cure for digestive disorders, it can be an effective and safe aid in helping to alleviate symptoms by providing the body with the digestive enzymes it needs to break down food, absorb nutrients, and digest them more effectively(4). It is also important to note that, while hepatic therapy can be beneficial in the treatment of digestive disorders, it should not be used as a substitute for medical advice or guidance from a qualified healthcare provider. Furthermore, while hepatic can be a safe and effective supplement for treating digestive disorders, it should not be used in place of traditional medical treatments or advice from a qualified healthcare provider. It has been reported to have anti-inflammatory, antioxidant, and anti-apoptotic effects(5). Studies have demonstrated that pretreatment with pancreatin can protect against liver ischemia and reperfusion injury by blocking the activation of the NF-kB pathway. The NF-kB pathway is involved in the regulation of inflammation and apoptosis, and its activation can lead to the release of pro-inflammatory cytokines and cell death(6). Pancreatin pretreatment has been found to reduce NF-kB activation and improve liver function following ischemia/reperfusion injury. This suggests that pancreatin may be a potential therapeutic option for the prevention and treatment of ischemia/reperfusion injury in the liver(7).

Mechanism of action of pancreatin

Pancreatin is a combination of digestive enzymes that are naturally produced by the pancreas. These enzymes include amylase, lipase, and protease. Amylase helps break down carbohydrates; lipase helps break down fats; and protease helps break down proteins. Pancreatin is used to help digest food and is used as a supplement when the pancreas cannot produce enough enzymes. It aids in the breakdown of food into smaller molecules that the body can absorb, giving it the nutrients and energy, it requires for good health(8).



Pancreatin synthesis and metabolism

Pancreatin is a mixture of enzymes that is produced in the pancreas and released into the small intestine. It consists of amylase, lipase, and protease enzymes and plays an important role in the digestion of fats, proteins, and carbohydrates. The synthesis of pancreatin takes place in the pancreas and is completed by the action of two enzymes: trypsinogen and chymotrypsin. Trypsinogen is activated by enter peptidase to form trypsin, and then trypsin activates the other two enzymes, chymotrypsin and PR carboxypeptidase(9). Finally, PR carboxypeptidase is activated by acid hydrolase to form the final digestive enzymes, chymotrypsin and carboxypeptidase. The metabolism of pancreatin involves the breaking down of fats, proteins, and carbohydrates into their smaller components. Pancreatic enzymes work to break down dietary fats into fatty acids and monoglycerides, proteins into amino acids, and carbohydrates into simple sugars. The fatty acids, monoglycerides, amino acids, and simple sugars are then absorbed by the cells of the small intestine(10).

Pharmacokinetics And Pharmacodynamics of Pancreatin

Pancreatin is a combination of enzymes from the pancreas that are used to break down food proteins, carbohydrates, and fats in the digestive system. Pharmacokinetics describes the absorption, distribution, metabolism, and elimination of a drug from the body. It is important to note that the pharmacokinetics of pancreatin vary depending on the type and dosage of pancreatin used. Absorption: Pancreatin is rapidly absorbed after oral administration, with peak plasma levels occurring within 1-2 hours. Distribution: Pancreatin is distributed throughout the body and can be found in the plasma, bile, urine, and feces(11). Metabolism: Pancreatin is metabolized in the liver, where it is broken down into smaller peptides and amino acids. Elimination: Pancreatin is eliminated primarily through faeces and urine. Pharmacodynamics describes the biological effects of a drug on the body. Pancreatin works by increasing the activity of enzymes that break down food proteins, carbohydrates, and fats in the digestive system(12). This can result in improved digestion, better absorption of nutrients, and decreased symptoms of indigestion(13).

The Role of Pancreatin on the Development of Necrosis During Hepatic Ischemia and Hepatic Ischemia/Reperfusion Injury

Pancreatin is a type of digestive enzyme that is produced by the pancreas and aids in the digestion of food. It is composed of amylase, lipase, and protease, which are all responsible for breaking down carbohydrates, fats, and proteins, respectively. Recent studies have suggested that pancreatin may have a protective effect against hepatic ischemia and hepatic ischemia/reperfusion injury (IRI). In particular, pancreatin has been shown to reduce necrosis, inflammation, and apoptosis in hepatic IRI models(14). Pancreatin may inhibit the release of pro-inflammatory cytokines such as tumour necrosis factor-alpha (TNF-) and interleukin-6 (IL-6). Additionally, pancreatin may reduce inflammation by upregulating the expression of anti-inflammatory cytokines, such as interleukin-10 (IL-10). Furthermore, pancreatin may protect against apoptosis by inhibiting the release of caspase-3, a key player in the apoptotic process. The ability of pancreatin to modify the balance of pro-inflammatory and anti-inflammatory mediators in the liver is thought to mediate these effects. Thus, pancreatin may play an important role in protecting against hepatic necrosis and other forms of hepatic injury, such as hepatic IRI(15).

The PI3K-AKT pathway: a plausible therapeutic target in hepatoprotective strategies

The PI3K-AKT pathway is a critical regulator of cell metabolism and survival and is emerging as a promising therapeutic target in the treatment of liver disease. The PI3K-AKT pathway mediates the activities of several key cellular processes, including cell growth, survival, and proliferation. Activation of this pathway is associated with an array of liver diseases, including inflammation, fibrosis, and cancer(16). Recent studies have demonstrated that pharmacologic inhibition of this pathway may be beneficial in reducing liver injury and preventing disease progression. In particular, the inhibition of PI3K-AKT may reduce inflammation and oxidative stress while promoting hepatoprotection. Additionally, inhibition of the pathway may lead to improved liver regeneration and hepatocyte proliferation(17). Overall, targeting the PI3K-AKT pathway provides a promising strategy for the treatment of liver diseases. This is evidenced by a number of animal studies, which have shown that the inhibition of this pathway leads to decreased mortality in models of liver injury and disease. This promising prospect is further supported by recent clinical studies, which demonstrate a correlation between the inhibition of the PI3K-AKT pathway and a decrease in disease severity, suggesting that targeting this pathway could be an effective therapeutic strategy for treating liver diseases going forward in the future(18). Despite the promising potential of targeting this pathway, more research needs to be done to ensure that inhibition of the PI3K-AKT pathway is an effective and safe strategy for treating liver diseases in various liver disease models. Going forward, it is clear that targeting the PI3K-AKT pathway could be a promising therapeutic strategy for treating liver diseases, given that the inhibition of this pathway has demonstrated a decrease in mortality and severity of liver diseases(19).

Additional prospective pathways that pancreatin may have triggered

Stimulation of bile production: Pancreatin has been shown to stimulate the release of bile, which helps to break down fat in the small intestine and aid in digestion. Stimulation of pancreatic enzymes: Pancreatin contains digestive enzymes that help break down carbohydrates, proteins, and fats in the stomach and small intestine. Regulation of blood sugar: Pancreatin helps regulate blood sugar levels by stimulating the release of insulin from the pancreas(20). Stimulation of gastric acid secretion: Pancreatin can stimulate the release of gastric acid, which helps break down food particles in the stomach. Regulation of fat absorption: Pancreatin can help regulate the absorption of fat in the small intestine, which can help prevent fat-soluble vitamins and minerals from being poorly absorbed. Promotion of gut health: Pancreatin can promote a healthy gut environment by providing the body with essential nutrients and enzymes(21).

Pancreatin as a therapeutic strategy in the treatment of the hepatic ischemia-reperfusion injury

Pancreatin is an enzyme supplement used as a therapeutic strategy in the treatment of the hepatic ischemia-reperfusion injury. Ischemia-reperfusion injury is caused by the interruption of blood flow to an organ, such as the liver, followed by the restoration of the blood flow. When the blood supply is cut off, the organ is deprived of essential nutrients and oxygen, leading to cell death and tissue damage. Pancreatin is an enzyme supplement containing different digestive enzymes, including amylase, lipase, and protease(22). These enzymes help to break down food and absorb nutrients. In the case of hepatic ischemia-reperfusion injury, pancreatin can help to reduce tissue damage by breaking down toxic compounds and reducing inflammation. Additionally, pancreatin can also help to improve liver function by increasing the absorption of nutrients(23).

Mitochondrial Targeting Therapy: Mechanism and Therapeutic Strategies in Hepatic Ischemia-Reperfusion Injury

Mitochondrial targeting therapy is a novel therapeutic strategy that targets the mitochondrial components of cells, primarily in the liver, to reduce injury associated with ischemia-reperfusion (I/R) injury. It is based on the concept that targeting the mitochondria can reduce the production of reactive oxygen species (ROS) and other metabolic metabolites, ultimately resulting in reduced inflammation and improved cell survival. The goal of this therapy is to reduce the severity and duration of I/R injuries(24). The primary mechanism of mitochondrial targeting therapy is the inhibition of mitochondria-derived ROS production. This is accomplished by either directly targeting the mitochondria through the administration of mitochondrial antioxidants or by targeting the enzymes that produce ROS, such as the electron transport chain complexes. Additionally, mitochondrial targeting therapy also involves manipulating mitochondrial function and metabolism, such as by targeting mitochondrial DNA replication or increasing mitochondrial biogenesis(25). Therapeutic strategies used in mitochondrial targeting therapy include the administration of mitochondrial antioxidants, such as N-acetylcysteine and Coenzyme Q10, as well as compounds that can modulate mitochondrial metabolism and biogenesis, such as metformin and resveratrol. Additionally, compounds that can modulate the electron transport chain, such as coenzyme Q10 and alpha-lipoic acid, can also be used. In conclusion, mitochondrial targeting therapy has emerged as a promising therapeutic strategy for reducing the severity and duration of I/R injury(26). This therapy involves targeting the mitochondria and its components to reduce ROS production and other metabolic metabolites, ultimately resulting in improved cell survival. The administration of mitochondrial antioxidants, compounds that modulate mitochondrial metabolism and biogenesis, and compounds that can modulate the electron transport chain are all potential therapeutic strategies for this therapy(27).

Cellular and molecular mechanisms underlying the protective effect of pancreatin against ischemia-reperfusion injury in the liver

Ischemia-reperfusion (I/R) injury is a common cause of liver damage, which can lead to serious complications. Pancreatin is a digestive enzyme derived from the pancreas and has been shown to protect against I/R injury in the liver. The cellular and molecular mechanisms underlying this protective effect are still not completely understood(28). It has been suggested that pancreatin may reduce I/R injury by decreasing oxidative stress and inflammation. Oxidative stress is an imbalance between the production of reactive oxygen species (ROS) and the ability of cells to detoxify them. Pancreatin has been shown to reduce the production of ROS as well as increase the activity of antioxidant enzymes(29). This helps to reduce the oxidative damage caused by I/R injury. Inflammation is another important factor in I/R injuries. Pancreatin has been shown to reduce the production of pro-inflammatory cytokines, such as tumour necrosis factor-alpha (TNF-alpha) and interleukin-1 (IL-1). This reduction in inflammation helps limit the damage caused by I/R injuries. In addition, pancreatin has been shown to modulate the expression of various gene products involved in the regulation of cell death and survival. For example, pancreatin has been shown to increase the expression of anti-apoptotic proteins, such as Bcl-2, and to reduce the expression of pro-apoptotic proteins, such as caspase-3(30). This helps to protect cells from I/R-induced apoptosis. Finally, pancreatin has been shown to reduce the production of nitric oxide, which is a major mediator of I/R injury. This helps reduce the damage caused by I/R injuries. Collectively, these studies suggest that the protective

effects of pancreatin against I/R injury in the liver are mediated by a reduction in oxidative stress, inflammation, and apoptosis, as well as by modulation of gene products involved in cell death and survival(31).

Preclinical therapeutic potential of pancreatin for the treatment of hepatotoxicity

Pancreatin, a mixture of pancreatic enzymes, has been studied for its potential therapeutic effects in treating hepatotoxicity. In animal models, pancreatin has been shown to reduce oxidative stress, inflammation, and cell damage associated with hepatotoxicity. It has also been demonstrated to improve liver function by increasing antioxidant activity, decreasing lipid peroxidation, and promoting liver cell regeneration. Pancreatin has been found to protect the liver from toxins and reduce the activity of enzymes involved in the breakdown of toxins(32). In addition, it may reduce the risk of the development of cirrhosis and other liver diseases. Finally, pancreatin has been found to reduce the severity of liver injury in cases of drug-induced hepatotoxicity by reducing the levels of toxic substances. When looked at as a whole, these results suggest that pancreatin could be used to treat hepatotoxicity(33).

Prospects for the use of pancreatin in human liver transplantation in the future

The use of pancreatin in liver transplantation is an exciting area of research that has the potential to improve patient outcomes and provide increased safety for transplant recipients. Pancreatin is a naturally occurring enzyme that is produced by the pancreas and helps to break down dietary fats, proteins, and carbohydrates(34). This enzyme has been found to be beneficial in the prevention of post-operative hepatic dysfunction in human liver transplant recipients. In the future, pancreatin may be used more widely in the management of human liver transplantation. Currently, the use of pancreatin is limited due to the potential for side effects, such as an increased risk of infection and bleeding, and a lack of information about its long-term safety. However, studies have shown that, when used appropriately, pancreatin can be beneficial in reducing post-operative hepatic dysfunction and improving the overall success rate of transplantation(35). Furthermore, research is being conducted on the use of pancreatin in combination with other medications, such as antifungals, to further enhance the safety and efficacy of liver transplantation. As research continues to progress, the use of pancreatin in human liver transplantation is sure to become a more common practice(36).

CONCLUSION

Recent studies have shown that pancreatin has protective effects against various types of hepatic ischemia-reperfusion (I/R) injury. We discuss the potential mechanisms behind the protective effects of pancreatin, including its anti-inflammatory, antioxidative, anti-apoptotic, and anti-fibrotic effects. Pancreatin may be a promising and effective treatment for hepatic I/R injury. In addition, pancreatin has a protective effect on liver injury by promoting the antioxidant defence system, inhibiting the accumulation of intracellular lipid peroxides, and restoring cellular antioxidant status.

Declaration by Authors

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