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Alcoholic Pancreatitis is One of the Major Forms of Acute and Chronic Pancreatitis

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Introduction

In a new study, employed proteomic techniques to characterise how ethanol alters the redox status of the pancreatic ER proteome. Their findings were published in this edition of Cellular and Molecular Gastroenterology and Hepatology. The ethanol-induced pathophysiology linked to pancreatitis may be influenced by these structural alterations and ER malfunction. One of the most common types of acute and chronic pancreatitis is alcoholic pancreatitis. Although long-term heavy alcohol consumption is a significant risk factor for pancreatitis, not everyone who uses alcohol long-term experiences pancreatic injury and illness. Instead, it's thought that alcohol makes the pancreas more susceptible to other stresses that cause disease aetiology.

Description

There is ongoing research on just how alcohol makes the pancreas more vulnerable to harm. Exocrine acinar cell abnormalities are major causes of pancreatitis. Highly specialised secretory cells called acinar cells make a significant amount of digesting enzymes. The secretory route is used to direct these endoplasmic reticulum (ER)-produced proteins for secretion to the cell surface. When oxidative damage, increasing secretory demand, or genetic abnormalities cause errors in protein folding, unfolded proteins build up and cause ER stress. Using the unfolded protein response, mammalian cells have developed a coordinated set of signalling channels to control ER stress (UPR). Translation is slowed by the UPR, which also helps with protein folding and targets misfolded proteins for destruction.

Proteostasis restoration is the ultimate objective. Unchecked ER stress, on the other hand, causes cells to skip the adaptive UPR and engage cell death mechanisms. Alcohol-induced pancreatitis is thought to be primarily caused by ER malfunction in acinar cells. For instance, persistent ethanol feeding in mice causes a UPR. In this instance, the UPR is an adaptive reaction that enables the cell to handle damage brought on by ethanol. Acinar cells are unable to deal with ER stress brought on by ethanol feeding in the absence of the transcription factor X-box binding protein 1 (XBP1), a crucial mediator of the UPR, which results in acinar cell death. These findings imply that ER dysfunction and acinar cell pathology are caused by a compromised UPR, which may have an impact on the pathogenesis of alcoholic pancreatitis. Although ethanol-induced pancreas pathology is driven by poor UPR signalling and ER dysfunction, it is uncertain what structural alterations or folding errors cause ER stress. To solve this issue, Scientists defined the structural alterations that occur in the ER following ethanol feeding in mice with normal or compromised UPR signalling. In this investigation, fed either control diets or ethanol diets to wild-type or XBP1-deficient (xbp1/-) mice before analysing ER proteins from pancreatic tissue. In order to create a pancreatic redox ER proteome, they used an oxidation state sensitive isotope coded affinity tag technique (dubbed OxICAT) to enrich for cysteine-reactive proteins in the ER fraction.

Aside from that they assayed for changes in activity and ER localization of serine hydrolases produced by acinar cells. In their model, feeding wild-type mice ethanol results in ER oxidation, modest ER stress, and up-regulation of XBP1, but no disease of the acinar cells. However, ethanol feeding increases ER stress and impairs secretory capacity in XBP1-deficient animals, which are linked to the pathophysiology of acinar cells. One of the main conclusions of the present investigation is that ethanol feeding increases the oxidation of several proteins in the ER in wild-type mice. Wild-type mice have the ability to activate an adaptive response and return normal proteostasis when their UPR is intact. In \sxbp1 On the other hand, compared to wild-type mice, control-fed mice have lower quantities of oxidised proteins in the ER, but ethanol feeding causes a more pronounced shift in oxidation. This most likely reflects non-native disulfide-containing proteins that cause ER stress in ethanol-fed xbp1/- animals. Numerous pancreatic ER proteins experience redox status alterations in response to ethanol intake or UPR state. Many of them belonged to the digestive enzyme family of serine hydrolases, which is widely distributed in acinar cells. The vast majority of serine hydrolases are created from inactive precursors [1-5].

Conclusion

To determine whether changes in redox state cause improper activation. ER proteins with a probe that only reacts with active serine hydrolases' catalytic serine. The unique serine hydrolase carboxyl ester lipase (Cel), which was tagged in pancreatic ER fractions, was discovered by this proteomic study. Treatment with ethanol changed the percentage distribution of dimeric Cel that was kept in the ER in XBP1-deficient animals. A configuration of an active dimer was stabilised by oxidation, according to modelling of the Cel structure.

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Conflict of Interest

The Author declares there is no conflict of interest associated with this manuscript.

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