EUROPEAN JOURNAL OF BIOMEDICAL AND PHARMACEUTICAL SCIENCES
http://www.ejbps.com

ISSN 2349-8870
Coden USA: EJBPBO
Volume: 13
Issue: 11
Year: 2026

PROLONGED STRESS FROM TRANSITION FOR HIGHER STUDY AMONG YOUNG ADULTS AND RISK OF PANCREATITIS PURUSHOTTAM PRAMANIK

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DOI: https://doi.org/10.5281/zenodo.23803204
How to cite this Article: Purushottam Pramanik*. (2026) Prolonged Stress From Transition For Higher Study Among Young Adults And Risk Of Pancreatitis Purushottam Pramanik. European Journal of Biomedical and Pharmaceutical Sciences, 13(11), 18–27.
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Article Received on 22/09/2026 **Article Revised on 13/10/2026** **Article Published on 01/11/2026**

ABSTRACT
Many young adults suffer in chronic stress for transition to college or university from home or school. Chronic stress significantly affects various physiological systems of our body. The aims of this review article to analyze the impact of chronic stress on pancreatic health among young adults engaged in higher study in abord. This review paper critically analyzes the existing literature to explore the complex relationship between chronic stress and risk of pancreatitis. The hypothalamic-pituitary-adrenal (HPA) axis is a neuroendocrine pathway involved body's response to stress by enhancing secretion of glucocorticoids (GC) from adrenal cortex. In acute stress GC level is maintained by negative feedback mechanism. In chronic stress GC level is increased in plasma due to development of GC insensitivity. Prolonged exposure to high GC level from chronic stress may induce upregulation of proinflammatory gene transcription and down regulation of anti-inflammatory gene transcription and the subsequent release of more proinflammatory cytokines to enhance pancreatitis. Activation of HPA axis and inflammation are linked to oxidative stress. Oxidative stress produces necrosis of various cells including acinar cells. The oxidative stress and its constant companion inflammation play critical roles in the pathogenesis of pancreatitis. Chronic stress augments the development of obesity from changed lifestyle. Obesity may result in the increase plasma triglyceride and increase risk of cholelithiasis, both are well-known contributors of pancreatitis. Obesity as persistent low-grade inflammation induces hypoalbuminemia and severity of hypertriglyceridemia-induced acute pancreatitis. Thus, this review article suggested that chronic stress augments the risk of pancreatis by inducing glucocorticoids resistance and lifestyle modification. Social support is the most important tool to overcome prolonged stress induced by transition to abord and to minimize risk of pancreatitis.

KEYWORDS: Chronic stress, oxidative stress, pancreatitis, inflammation, lifestyle, obesity.

INTRODUCTION
Stress is threatened homeostasis from physical or mental forces or both.[1] It is the effect of anything that threatens the constancy of the internal environment. A stressor is a stimulus that disrupts the homeostasis and activates variety of chemical, physiological processes and behavioral coping mechanisms to restore homeostasis. Stressors are physical (physiological) and psychological. Stressor-induce physiological and behavioral changes is known as stress response. On the basis of duration, stress is categorized into acute and chronic. Acute stress is short term stress results from immediate stressors. This leads to fight-or-flight temporary physiological response. Chronic stress occurs due to persistent of stressor over an extended period. Prolong exposure to chronic stress led to cumulative physiological and psychological effects and increasing the risk of health problems like cardiovascular disease, anxiety, depression, inflammation etc.[2]

Acute pancreatitis is an acute inflammation of the pancreas characterized by severe epigastric pain with elevated serum pancreatic enzyme.[3] The main events occurring in the exocrine pancreas that initiate AP include inhibition of secretion, intracellular activation of pancreatic proteases and generation of inflammatory mediators.[4] Patients with more severe conditions can develop systemic inflammatory response syndrome which may be accompanied by multiorgan failure.

Stress is threatened homeostasis from physical or mental forces or both. Acute -short-term stress decreases the severity of experimentally induced pancreatitis. The worsening effects of chronic stress on the exocrine pancreas have received relatively little attention. This review will focus on the influence of chronic stress for transition to higher study in acute pancreatitis.

What is Stress?

Stress can be described as an adversive event (i.e. physical. Mental or emotional) that threatens the wellbeing of an individual. It is the effect of anything that threatens the constancy of the internal environment. A stressor is a stimulus that disrupts the homeostasis and activates variety of chemical, physiological processes and behavioral coping mechanisms to restore homeostasis and promote survival. Stressors are physical (physiological) and psychological. Physical stressors refer to biological agents (virus, bacteria, parasites etc.) or external forces (e.g. radiation, noise etc.) that can modulate and/or elicit physiological response.[5] Psychological stressors are social and physical environmental circumstances that challenge the adaptive capabilities and resources of an organism.[6] Stressor-induce physiological and behavioral changes is known as stress response. The stress response is complex interplay of neural endocrine and immune mechanisms. On the basis of duration of stress is categorized into acute and chronic. Acute stress is short term stress results from immediate stressors. This leads to fight-or-flight temporary physiological response like increase heart rate, release of more adrenaline etc. Chronic stress occurs due to persistent of stressor over an extended period. prolonged exposure to chronic stress led to cumulative physiological and psychological effects and increases the risk of health problems like cardiovascular disease, anxiety, depression, inflammation etc.

What is acute pancreatitis?

Acute pancreatitis is an inflammatory disorder of the pancreas that may cause life-threatening complications. The main events occurring in the exocrine pancreas that initiate it include inhibition of secretion, intracellular activation of pancreatic proteases and generation of inflammatory mediators.[7] These events are correlated with morphological changes in pancreatic acini viz. retention of enzymes, formation of large vacuoles containing both digestive enzymes and lysosomal hydrolases and necrosis in human acute pancreatitis.[8] Patients with more severe conditions can develop systemic inflammatory response syndrome which may be accompanied by multiorgan failure.[9]

Epidemiological evidence suggests that the most common causes of AP are (i). gallstone disease, (ii) long term alcohol consumption and (iii) hypertriglyceridemia,[10,11] However, in 5% to 25% cases remain unclear. According to the Atlanta Criteria, 2 of the following 3 criteria need to be present for the diagnosis of AP: (i) Abdominal pain (epigastric) (ii). Serum amylase and / or lipase more than 3 times the upper normal limit (iii). Abnormal abdominal cross-sectional imaging findings suggestive of AP.[12] AP is divided into three grades: Mild (no organ failure or local complications), moderate (transient organ failure or local complications) and severe (persistent organ failure).[13] Organ failure in AP is categorized into two: early primary organ failure which occurs within 72 hours due to systemic inflammation and late secondary organ failure which typically develop after one week and is linked with infectious pancreatic necrosis. In respect to pathological morphology, AP is divided in to two categories: necrotizing and interstitial edematous.[13] Mild Ap is under interstitial edematous category whereas moderate to severe are under necrotizing category.

Chronic Stress for Transition to College or University From Home or School

Mental health of many young adults are significantly affected by their transition to college or university from home or school.[14] The demands of academic performance, the challenge of independent living and learning and need to establish social connection contribute to the psychological wellbeing.[15] Social isolation, loneliness and poor social integration are potent risk factors for psychological stress.[16] Lifestyle factors such as physical activity, sleep quality, nutrition, substance use and social relationship are closely linked to the mental health outcomes. Physical activity is associated with lower levels of stress, anxiety and depression.[17] Sleep disturbance like irregular sleep pattern, insufficient sleep duration and poor sleep quality are common among students of higher education. Sleep disruption increases susceptibility to mental health disorders.[18] The relationship between sleep and mental health is bidirectional, poor mental health induces sleep problem which further detoriates mental health by creating a vicious cycle.[19] Substance use including alcohol, tobacco and recreational drugs can exacerbate mental health problem.[20]

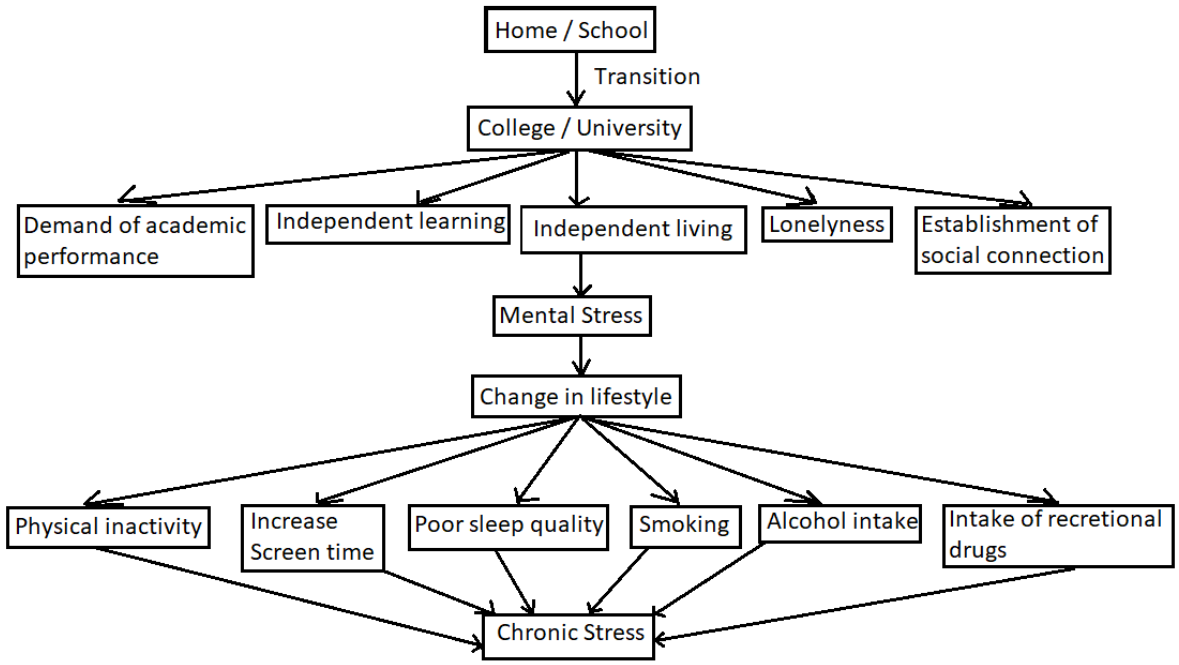


Fig. 1: Hypothesis of Development of Chronic Stress Among Young Adults From Transition to Higher Study.

Psychological Stress, oxidative stress and pancreatitis

Biological response to external stresses e.g., psychological stress involves a complex interaction among the nervous, endocrine and immune system.[21] Stress activates hypothalamic-pituitary-adrenal axis in the neuroendocrine system leads to secretion of glucocorticoids that interconnect central nervous system and immune system.[22] Literature survey indicates that activation of HPA axis and inflammation are linked to oxidative stress.[23] Many studies suggest that the chronic psychological stress is linked oxidative stress.[24]

The disbalance between free radical (ROS) formation and antioxidant capacity to neutralize them as oxidative stress. Thus, oxidative stress is caused by a combination of increased production of ROS and impaired antioxidant capacity. ROS are free radicals containing oxygen atoms viz super oxide anions, hydroxyl radicals, hydrogen peroxide, singlet oxygen etc. ROS produced by oxidative stress can cause cell necrosis or apoptosis by various ways: i). Mitochondrial dysfunction and disruption of lipid and lysosomal membranes (ii). Intracellular calcium overload,[25] (iii). Enhances cell death by inducing membrane peroxidation,[26] (iv). Activation of transcription nuclear factor NFkB,[27] (v). production of inflammatory mediators.

ROS are considered as important causes of pancreatic cell death. Free radicals react with polyunsaturated fatty acids in the cell membrane to induce damage of cell membrane leading to cell death. Hydrogen peroxide promotes apoptosis of acinar cells at low concentration and rapid necrosis at higher concentration.[28] Under normal conditions, ROS are safely neutralized by antioxidant defense system. The body's antioxidant defense network consists of endogenous and exogenous system. The endogenous enzymic antioxidants are the primary protection against oxidative stress. These include super oxide dismutase (SOD), Catalase and Glutathione peroxidase. These enzymes facilitate detoxification of ROS. Nonenzymic antioxidant include glutathione, uric acid, bilirubin and coenzyme Q10. These are free radical scavengers, metal chelators and modulators. These nonenzymic antioxidants operate synergy with enzymatic antioxidants. Levels of nonenzymic antioxidants are tightly regulated through biosynthesis, recycling and dietary intake. An imbalance increases the susceptibility to oxidative damage and alters redox sensitive gene expression that are critical for inflammation and immunity. Oxidative stress is caused by a combination of increase production of ROS and impaired antioxidant capacity.

Proinflammatory cytokines and oxidative stress trigger each other by activating mitogen-activated protein kinases and NFkB and amplify inflammatory cascade.[29] Accumulation of TNF-alfa promotes production of IL-1beta and IL-6 resulting in multiorgan damage. IL-1beta and IL-6 determine the severity of acute pancreatitis.[30] Oxidative stress amplifies the inflammatory process through the recruitment and release of inflammatory mediators. Thus, oxidative stress and its constant companion inflammation play critical roles in the pathogenesis of pancreatitis.

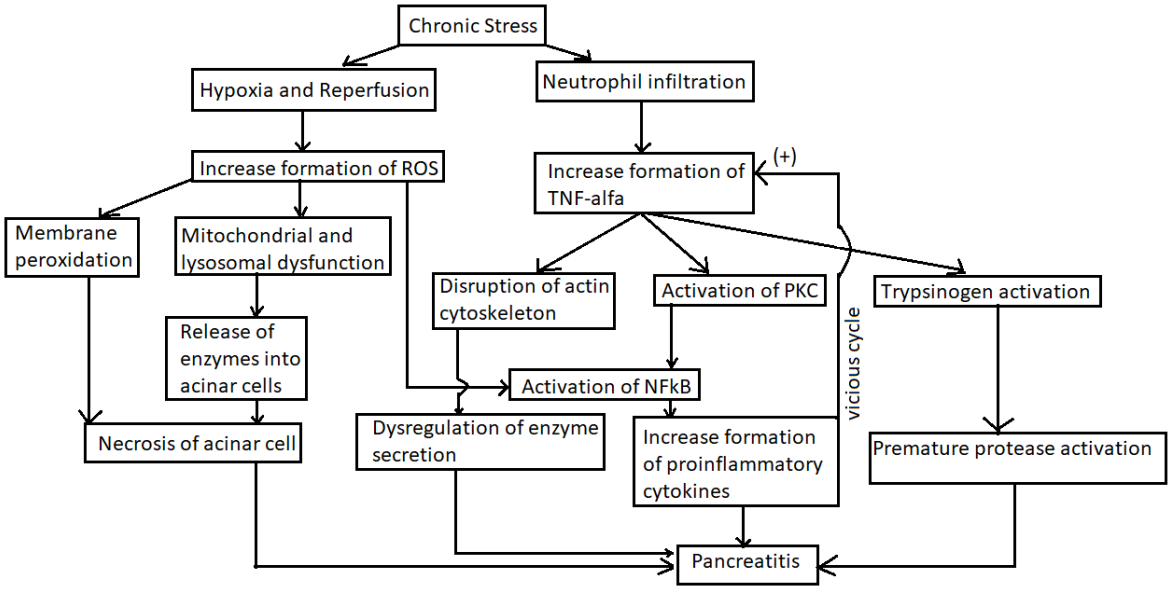


Fig-2: Hypothesis of Pathophysiological Impact of Chronic Stress on Pancreatic Acinar Cells.

Chronic Stress, Obesity and Inflammation

A growing body of research supports a bidirectional link between obesity and mental health. On one hand chronic stress and depression can leads to behaviors and physiological changes that promote weight gain. On the other hand, obesity itself can exacerbate stress due to social stigma and biological factors. A meta-analysis suggested that obesity is associated with 55% increased risk of developing depression, while having depression was associated with 58% higher risk of becoming obese.[31]

Stress-induced eating can contribute stress related obesity. Stress can impair self-control in eating, leading to higher caloric intake by consuming comfort foods that are high in sugar and fat.[32] Previous studies indicate that stressed people tend to gravitate toward caloric rich foods and may eat larger quantities than usual.[33] Chronic stress elevates glucocorticoids level via activation of hypothalamic-pituitary-adrenal axis which promotes the development of visceral or central obesity.[34] Thus, repeated food intake to blunt stress, creating a cycle where stress leads to overeating, which can promote weight gain and subsequently more stress.

Stress is associated with insufficient as well as disturbed sleep. Low quality or insufficient sleep increases secretion of ghrelin and decreases leptin secretion. Disturbs the balance of appetite-regulating hormones induces to intake more foods.[35] On the other hand, sleep deprivation elevates stress and impaired cognitive control which leads to enhance emotional eating.[36] Stressed individuals often experience fatigue that can result in lower exercise frequency and tendency toward sedentary activities like prolonged screen time. Thus, stress creates a cascade of behavioral changes-overeating, unhealthy food choice, sedentary life style and poor sleep quality that synergistically increase the risk of obesity.

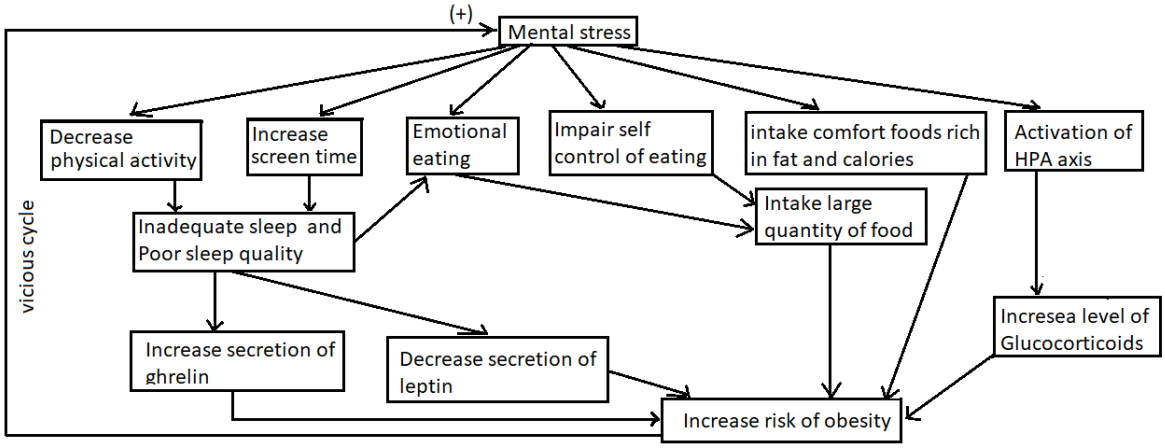


Fig.3: Hypothesis of Pathophysiological Mechanisms of Development of Chronic Stress-Induced Obesity.

BMI is positively correlated with oxidative stress markers (e.g. malondialdehyde) in serum.[37] Activities of the antioxidant enzymes including super oxide dismutase, catalase and glutathione peroxidase levels were significantly lower in obese in compare to nonobese counterpart.[38] Obesity induces oxidative stress by inducing multiple biochemical mechanisms like: i). hyperleptinemia[39] ii). Hyperglycemia,[40] iii). Deficiencies of antioxidant vitamins and minerals,[41] iv). Chronic inflammation.[42]

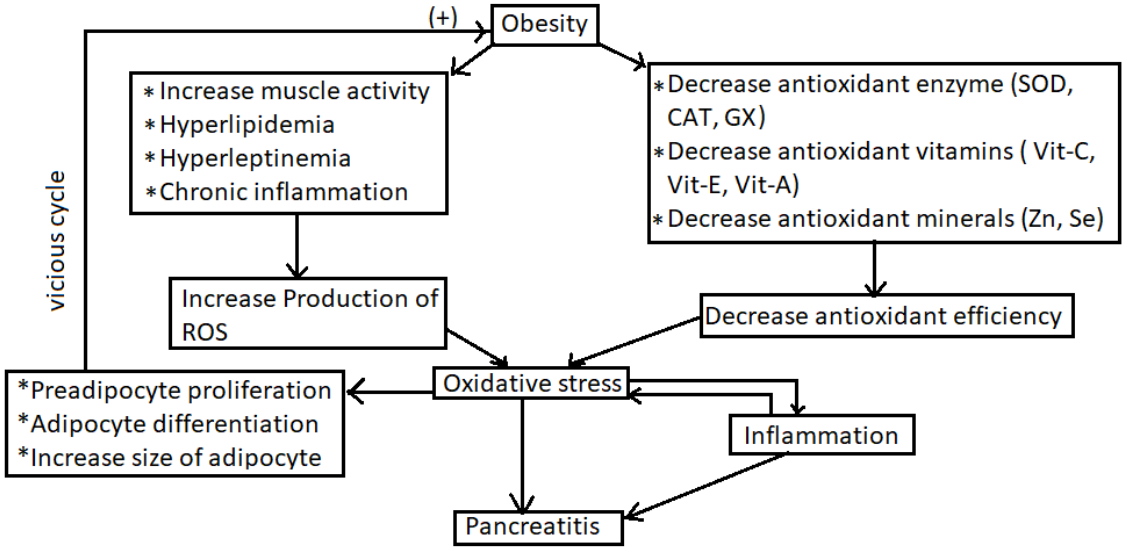


Fig. 4: Hypothesis of Pathophysiological Mechanisms of Risk of Pancreatitis Among Chronic Stress-Induced Obese.

Chronic Stress, Inflammation and Pancreatitis

The hypothalamic-pituitary-adrenal (HPA) axis is a neuroendocrine pathway involved body's response to stress. Magnocellular neurons in the paraventricular nuclei (PVN) hypothalamus synthesize and secrete corticotropin releasing factor (CRF), the main regulator of HPA axis. Acute stress triggers the activation of Agouti-related protein (AgRP) neurons related with glucocorticoids (GC) production.[43] Acute stress triggers the activation of AgRP neurons. Neuropeptide-Y (NPY) secreted from AgRP terminal inhibits GABAergic neurons. As a result, inhibitory effect of GABA on CRF neuron is withdrawn and leading to increase secretion of CRF from PVN of hypothalamus. Signal from limbic areas and prefrontal cortex stimulate secretion of arginine vasopressin (AVP) from PVN. Both CRF and AVP are released into hypophyseal portal system in the median eminence and circulate to anterior pituitary. CRF act on corticotrope cell of anterior pituitary and stimulate secretion of Adreno corticotropic hormone (ACTH) into systemic circulation. AVP facilitates the release of ACTH by binding to its vasopressin V1b receptor on the corticotrope cells. The released ACTH binds to the type-2 melanocortin receptor (MCR-2) of on Zona fasciculata of adrenal cortex and stimulates synthesis and secretion of glucocorticoids (GC) into general circulation. GC secreted from adrenal cortex in response to stress and following activation of HPA axis is responsible for appropriate behavioral responses to stress along with adaptive responses in metabolism, feeding and immunity. GC is released for several hours and at a certain blood concentration it exerts negative feedback on the hypothalamic release of CRF and pituitary release of ACTH. Thus stress-induced cortisol secretion is normally curtailed through a negative feedback mechanism to maintained overall homeostasis.[44]

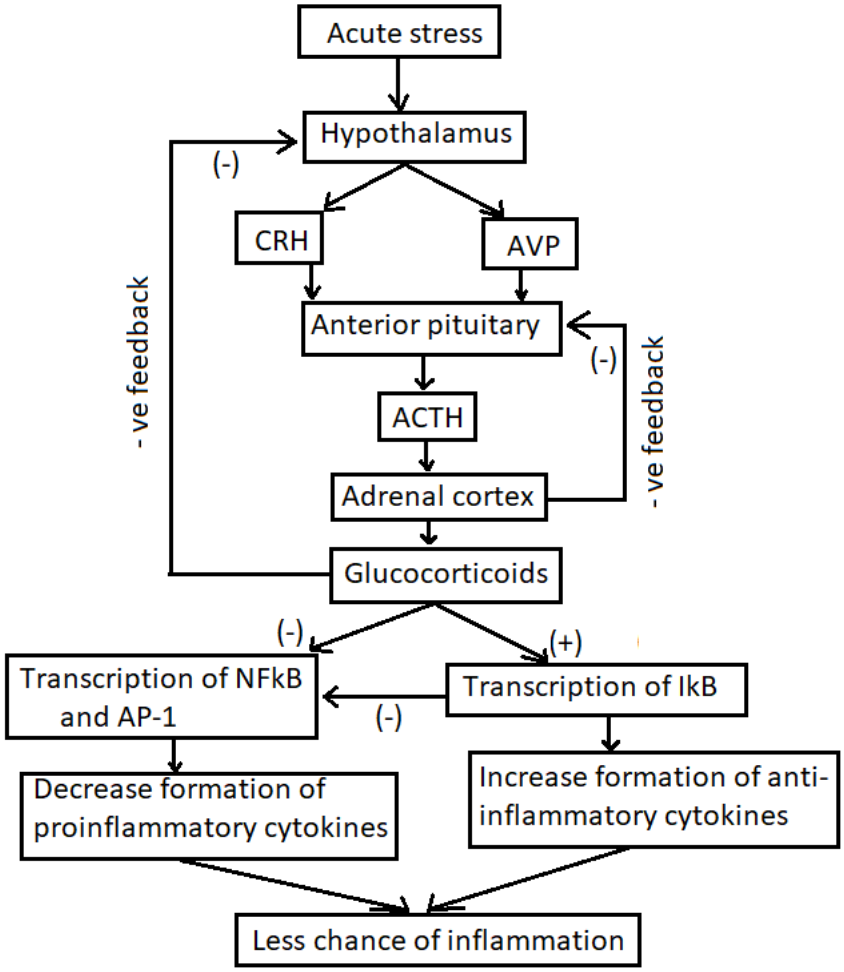


Fig.5: Schematic Representation of Regulation of Secretion of Glucocorticoids and its Impact on Immunity in Acute Stress Exposure.

GC binds with cytosolic glucocorticoid receptor (GR) which translocate to nucleus and regulate transcription of genes. In absence of GC binding GRs are sequestered into cytoplasm by a complex protein. Such proteins are dissociated from GR after binding with GC. Following binding GR-GC complex translocate to nucleus where it can inhibit proinflammatory transcription factors such as NFkB and AP-1 those initiate transcription of genes encoding proinflammatory cytokines (like IL-1. IL-6, TNF-alfa).[45] GC-GR complex also promotes transcription of anti-inflammatory proteins (e.g. IkB) via GC response element. As an anti-inflammatory protein, IkB sequestered proinflammatory transcription factor NFkB in cytoplasm.[46] Thus GC-GR complex down regulates inflammation at molecular level via inhibition of NFkB and AP-1, as well as initiation of anti-inflammatory process via GRE and IkB.

However, under prolog stress exposure, cortisol levels may become dysregulated leading to either hypersecretion or development of GC insensitivity.[47] Long term stress is accompanied by failure of HPA axis negative feedback leads to gradually elevates basal GC levels which cannot be effectively reduced. At this stage there is aggravation of inflammation.[48] It is suggested that chronic elevated cortisol levels in blood decreases the sensitivity of the hypothalamus to cortisol thereby inhibiting negative feedback mechanism.[49] Studies have shown that chronic stress can induce glucocorticoid resistance (GCR). The evidence for GCR in response to chronic stress has been found in parents of children with cancer and persons reporting loneliness.[50] Prolonged exposure to GC from chronic stress may induce upregulation of proinflammatory gene transcription and the subsequent release of proinflammatory proteins.[51]

GCR could be conferred at the level of receptor or in the nucleus at the level of gene transcription. In the nucleus, decrease transcription anti-inflammatory factors (e.g., IkB) and /or reduced inhibition of pro-inflammatory transcription factors (e.g., NFkB, AP-1) could result in upregulation of pro-inflammatory gene transcription rather than down regulation. Wang et al showed that chronic stress-induced GCR could cause significant and persistently high levels of inflammatory cytokines such as TNF-alfa; IL-1beta, IFN-gamma etc.[52] Thus, GCR may play a permissive role in the upregulation pro-inflammatory processes engendered by chronic mental stress.

TNF-alfa is involved in stress related. Secretion of TNF-alfa by several stress stimuli has been demonstrated in various cells including pancreatic acinar cells.[53] Chronic stress induced rat exhibits higher concentration of TNF-alfa and neutrophil infiltration in pancreatic tissue suggesting neutrophil as a source of TNF-alfa.[20] Neutrophil directly induces intracellular trypsinogen activation and cell death and indirectly neutrophil-induced TNF-alfa is responsible for protease activation and necrosis.[54] Hypoxia-reoxygenation conditions increases secretion of TNF-alfa by acinar cells from oxidative stress.[55] TNF-alfa activates pancreatic nuclear factor kB (NFkB), main transcriptional regulator of inflammatory molecules. Literature suggested that treatment of rat pancreatic acinar cell with high doses of TNF-alfa increase production of IL-1beta, IL-4, IL-6, IL-10 as well as TNF-alfa.[56] It was reported that TNF-alfa activates PKC-delta and PKC- epsilon in pancreatic acini of rats[57] which activates NFkB and trypsinogen in rat pancreatic acini and increase synthesis of CCK.

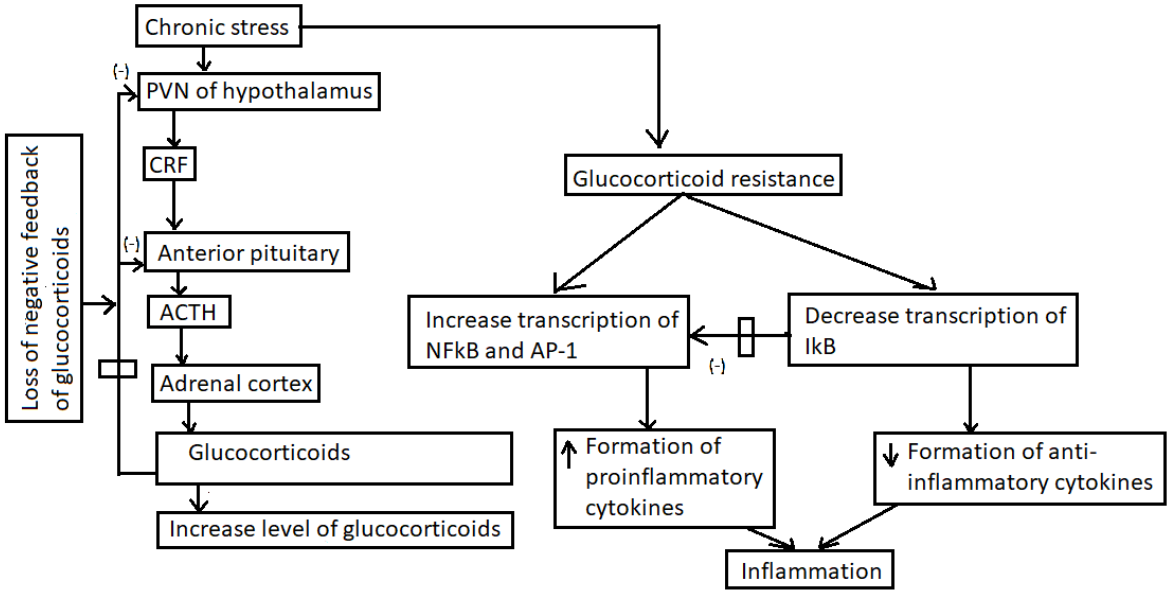


Fig.6: Schematic Representation of Regulation of Secretion of Glucocorticoids and its Impact on Immunity in Chronic Stress Condition.

Chronic Stress Lifestyle and Pancreatitis

The most reported behaviors used to cope with stress included TV watching, surfing the internet, napping, eating, drinking alcohol and smoking.[58] Cigarette smoking is significant risk factor for pancreatitis. The primary metabolite of cigarette smoke has detrimental effect on the acinar cells and zymogen secretion in the pancreas. These effects leads to functional and histological changes characteristic of acute pancreatitis.[59] A Danish population-based cohort study reported that smoking is an independent risk factor of pancreatitis.[60] Recent studies suggested that cigarette smoke extract significantly increase proinflammatory cytokines including IL-1beta, IL-6 and TNF-alfa in vascular endothelial cells.[61]

Long screen time (TV watching, surfing the internet) increases the risk of acute pancreatitis due to: firstly; people with long screen time have abnormal eating behavior without breakfast, low intake of vitamin and fibers and high intake of fat.[62] Such eating habits can induce gall stone. Gallstone is the most common triggers of acute pancreatitis.[63] Secondly more screen time increases the risk of type-2 diabetes, which can potentiate the development of acute pancreatitis.[64]

Chronic excessive alcohol intake is recognized as the second major cause of acute pancreatitis.[65] Ethanol metabolism has been associated with increased oxidative stress.[66] At the cellular level primary source of ROS is mitochondria. Alcohol-induced oxidative stress was related to the impairment of antioxidants including SOD, CAT and glutathione per oxidase activity.[67]

CONCLUSION

Mental Health of many young adults are significantly affected by their transition to college or university from home or school and suffer in chronic stress from educational demand, social isolation, loneliness and poor social integration. P*sychological stress involves a* complex interaction among the nervous, endocrine and immune system. The hypothalamic-pituitary-adrenal (HPA) axis is a neuroendocrine pathway involved body's response to stress by enhancing secretion of GC from adrenal cortex. In acute stress GC level is maintained by negative feedback mechanism. In chronic stress GC level is increased in plasma due to development of GC insensitivity in PVN and anterior pituitary. Chronic stress-induced GCR can disrupt negative feedback regulation of HPA axis and interfere the ability of immune system to control inflammation. Prolonged exposure to GC from chronic stress may induce

upregulation of proinflammatory gene transcription and down regulation of anti-inflammatory gene transcription and the subsequent release of more proinflammatory cytokines like TNF-alfa, IL-6 and IL-1Beta. TNF-alfa activates PKC which in turn activates NFkB to enhance pancreatitis.

Activation of HPA axis and inflammation are linked to oxidative stress. The oxidative stress is caused by a combination of increased production of ROS and impaired antioxidant capacity. ROS produced by oxidative stress can cause necrosis of various cells including acinar cells. Oxidative stress amplifies the inflammatory process through the recruitment and release of inflammatory mediators. The oxidative stress and its constant companion inflammation play critical roles in the pathogenesis of pancreatitis.

Chronic stress is associated with the development of obesity from changed lifestyle. Obesity-induced hypertriglyceridemia and cholelithiasis are well known contributors of pancreatitis. Beside this, obesity as persistent low-grade inflammation induces hypoalbuminemia which potentiates the severity of hypertriglyceridemia-induced acute pancreatitis. Social support in abord is first line defense to overcome prolonged stress and its impact on pancreatic health. Healthy eating, adequate and good quality sleep and regular physical activity may be considered to minimize risk of pancreatitis among young adult living away from home for higher study.

ACKNOWLEDGMENT

The author wishes to dedicate this review article to Dr. Parthiba Pramanik, MD in Respiratory Medicine and coauthor of my various articles, who was died in severe acute pancreatitis just after completion his MD course. His memory inspires me to write this review article.

Conflicts of Interest

The author declares no conflicts of interest.

Ethics Approval

This review work did not involve any clinical or animal experiments and was analyzed only using published open-source studies, therefore, did not involve the approval of the Ethics Committee.

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