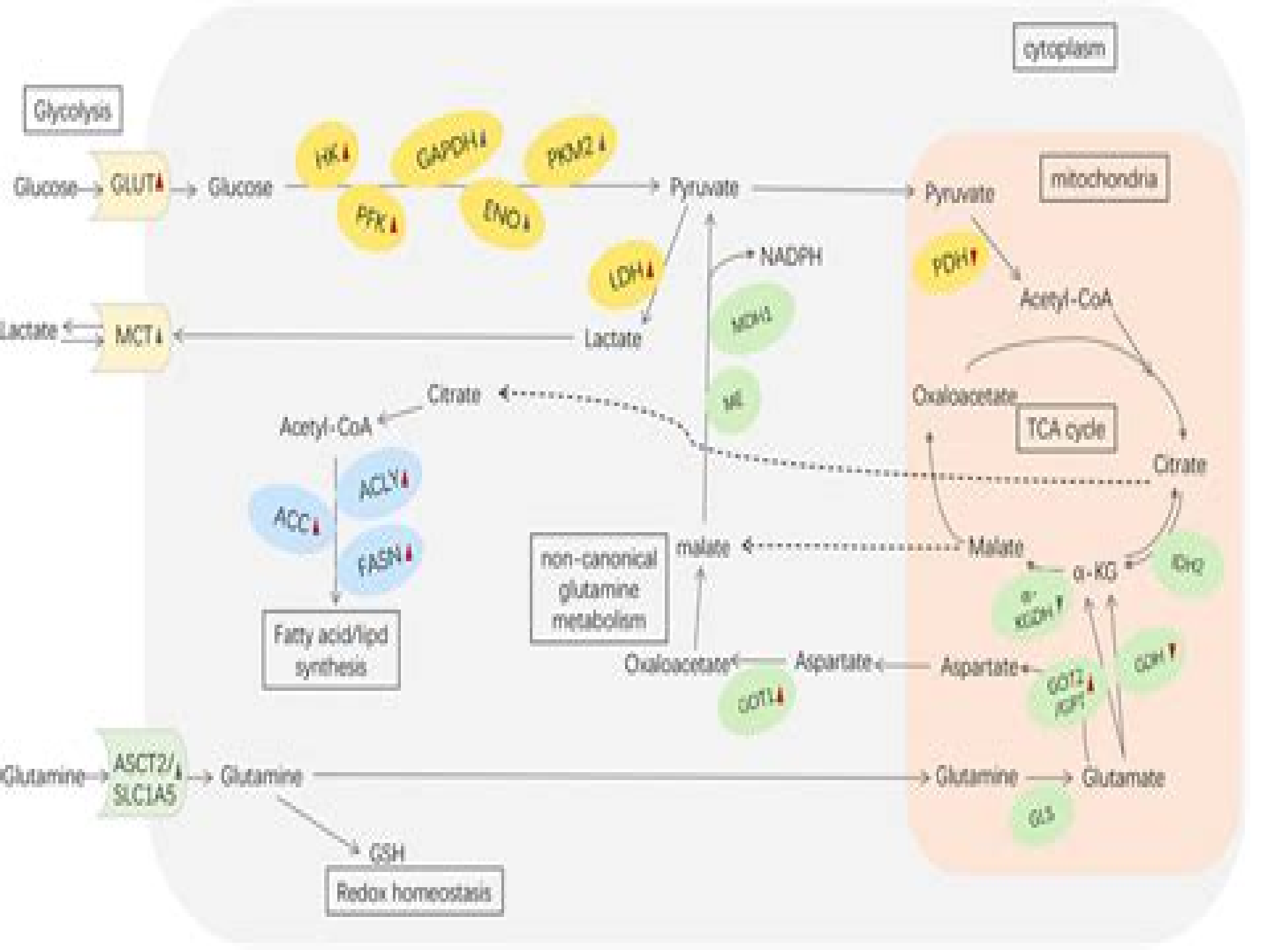
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Acetyl-CoA
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ACC
FASN
Fatty acid/lipd synthesis
non-canonical glutamine metabolism
mitochondria
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TCA cycle
Citrate
Malate
malate
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IDH2
α-KGDH
Oxaloacetate
Aspartate
Aspartate
GOT1
GOT2
GDH
Glutamine
ASCT2/ SLC1A5
Glutamine
Glutamine
Glutamate
GLS
GSH
Redox homeostasis

Metabolic Reprogramming Diminishes Pancreatic Cachexia

Fabrizio Bronte,Kanjoormana Aryan Manu,Emilio Francesco Giunta

Metabolic Reprogramming Diminishes Pancreatic Cachexia:

Metabolic Reprogramming Induced by Ketone Bodies Diminishes Pancreatic Cancer Cachexia Manchester Manchester Press,2015-12-15 This open access article by various authors presents key research on Metabolic reprogramming induced by ketone bodies diminishes pancreatic cancer cachexia Aberrant energy metabolism is a hallmark of cancer To fulfill the increased energy requirements tumor cells secrete cytokines factors inducing muscle and fat degradation in cancer patients a condition known as cancer cachexia It accounts for nearly 20% of all cancer related deaths However the mechanistic basis of cancer cachexia and therapies targeting cancer cachexia thus far remain elusive A ketogenic diet a high fat and low carbohydrate diet that elevates circulating levels of ketone bodies i e acetoacetate beta hydroxybutyrate and acetone serves as an alternative energy source It has also been proposed that a ketogenic diet leads to systemic metabolic changes Keeping in view the significant role of metabolic alterations in cancer we hypothesized that a ketogenic diet may diminish glycolytic flux in tumor cells to alleviate cachexia syndrome and hence may provide an efficient therapeutic strategy **Reviews in Gastrointestinal Cancers** Fabrizio Bronte,Kanjoormana Aryan Manu,Emilio Francesco Giunta,2023-08-02 *The Metabolic Approach to Cancer* Nasha Winters,Jess Higgins Kelley,2017 The Optimal Terrain Ten Protocol to Reboot Cellular Health Since the beginning of the twentieth century cancer rates have increased exponentially now affecting almost 50 percent of the American population Conventional treatment continues to rely on chemotherapy surgery and radiation to attack cancer cells Yet research has repeatedly shown that 95 percent of cancer cases are directly linked to diet and lifestyle The Metabolic Approach to Cancer is the book we have been waiting for it offers an innovative metabolic focused nutrition protocol that actually works Naturopathic integrative oncologist and cancer survivor Dr Nasha Winters and nutrition therapist Jess Higgins Kelley have identified the ten key elements of a person s terrain think of it as a topographical map of our body that are crucial to preventing and managing cancer Each of the terrain ten elements including epigenetics the microbiome the immune system toxin exposures and blood sugar balance is illuminated as it relates to the cancer process then given a heavily researched and tested non toxic and metabolic focused nutrition prescription The metabolic theory of cancer that cancer is fueled by high carbohydrate diets not bad genetics was introduced by Nobel Prize laureate and scientist Otto Warburg in 1931 It has been largely disregarded by conventional oncology ever since But this theory is resurging as a result of research showing incredible clinical outcomes when cancer cells are deprived of their primary fuel source glucose The ketogenic diet which relies on the body s production of ketones as fuel is the centerpiece of The Metabolic Approach to Cancer Further Winters and Kelley explain how to harness the anticancer potential of phytonutrients abundant in low glycemic plant and animal foods to address the 10 hallmarks of cancer an approach Western medicine does with drug based therapies Their optimized genetically tuned diet shuns grains legumes sugar genetically modified foods pesticides and synthetic ingredients while emphasizing whole wild local organic fermented heirloom and low

glycemic foods and herbs Other components of their approach include harm reductive herbal therapies like mistletoe considered the original immunotherapy and common in European cancer care centers and cannabinoids which shrink tumors and increase quality of life yet are illegal in more than half of the United States Through addressing the ten root causes of cancer and approaching the disease from a nutrition focused standpoint we can slow cancer s endemic spread and live optimized lives *Keto for Cancer* Miriam Kalamian,2017 A Comprehensive Guide for Patients and Practitioners Although evidence supporting the benefits of ketogenic diet therapies continues to mount there is little to guide those who wish to adopt this diet as a metabolic therapy for cancer Keto for Cancer fills this need Inspired by the work of Dr Thomas N Seyfried PhD nutritionist Miriam Kalamian has written the first book to lay out comprehensive guidelines that specifically address the many challenges associated with cancer and particularly the deep nutritional overhaul involved with the ketogenic diet Kalamian a leading voice in the keto movement is driven by passion from her own experience in using the ketogenic diet for her young son Her book addresses the nuts and bolts of adopting the diet from deciding whether keto is the right choice to developing a personal plan for smoothly navigating the keto lifestyle It is invaluable for both beginners and seasoned users of the ketogenic diet as well as for health care professionals who need a toolkit to implement this targeted metabolic therapy The book guides readers to a deeper understanding of the therapeutic potential of the ketogenic diet which extends well beyond simply starving cancer emphasizing the powerful impact the diet has on the metabolism of cancer cells Nutritional nuances are explored in sections such as Fasting Protocols and Know What s in the Foods You Eat while meal templates and tracking tools are provided in Preparing Keto Meals Kalamian also discusses important issues such as self advocacy Readers of Keto for Cancer are empowered to get off the bench and get in the game To that end Kalamian offers tips on how to critically examine cancer care options then incorporate what resonates into a truly personalized treatment plan **Insights in Metabolomics: 2021** Wolfram Weckwerth,2023-10-30 We are now entering the third decade of the 21st Century and especially in the last years the achievements made by scientists have been exceptional leading to major advancements in the fast growing field of Metabolomics Frontiers has organized a series of Research Topics to highlight the latest advancements in science in order to be at the forefront of science in different fields of research This editorial initiative of particular relevance led by Dr Wolfram Weckwerth Specialty Chief Editor of the Metabolomics section is focused on new insights novel developments current challenges latest discoveries recent advances and future perspectives in the field of Metabolomics

Cancer Diet Susan Zeppieri ,2022-06-27 The Cancer Diet Book is a cancer diet to help reduce the risk of cancer and boost the immune system with a cancer diet for beginners and a cancer diet for advanced This diet helps restore a healthy body and mind promoting a holistic approach to cancer prevention Written by a nutritionist this book is a must read for anyone who wants to recover from cancer Cancer Diet Book is an easy to follow dietary guide for those struggling with cancer This cookbook helps you to avoid the most common pitfalls and manages to get you on track quickly The book s main

goal is to help people living with cancer to regain their health and to enjoy a long and healthy life The Cancer Diet Book is a comprehensive guide to the fundamentals of a healthy diet and lifestyle Here you will find information on how to nourish your body and restore your health while healing your body from cancer The Cancer Diet Book is a cancer diet for beginners made especially for those who have had cancer or are currently going through cancer treatment HERE S WHAT MAKES THIS BOOK SPECIAL Is Cancer a Genetic or a Metabolic Disorder The Science Behind Cancer Diet The Healthiest Diets for Cancer Patients and Survivors Cancer Nutrition Much much more Interested Then Scroll up Click on Buy now with 1 Click and Get Your Copy Now Animal Models in Cancer Drug Discovery Asfar Azmi,Ramzi M. Mohammad,2019-04-16 Animal Models in Cancer Drug Discovery brings forward the most cutting edge developments in tumor model systems for translational cancer research The reader can find under this one volume virtually all types of existing and emerging tumor models in use by the research community This book provides a deeper insight on how these newer models could de risk modern drug discovery Areas covered include up to date information on latest organoid derived models and newer genetic models Additionally the book discusses humanized animal tumor models for cancer immunotherapy and how they leverage personalized therapies The chapter on larger animal canine models and their use in and their use in pre investigational new drug pre IND development makes the volume unique Unlike before the incorporation of several simplified protocols breeding methodologies handling and assessment procedures to study drug intervention makes this book a must read Animal Models in Cancer Drug Discovery is a valuable resource for basic and translational cancer researchers drug discovery researchers contract research organizations and knowledge seekers at all levels in the biomedical field Encompasses discussions on innovative animal models xenograft genetic models primary models organoid systems humanized and other models in modern biology paradigms that are enhancing research in the field of drug discover Covers the use of these models in personalized medicine immunotherapy toxicology pre IND assessments and related drug development arenas Presents protocols procedures and a comprehensive glossary to help new readers understand technical terms and specialized nomenclature

Experimental Biology and Medicine ,2003 **Nutrition Abstracts and Reviews** ,2003 *Molecular Mechanisms of Metabolic Reprogramming in Pancreatic Cancer* Sonam Kumari,2019 Pancreatic cancer ranks as third deadliest cancer worldwide in both men and women The lack of diagnostic methods chemo resistance and invasiveness of the disease diminishes the overall survival rate in patients Dysregulated glucose metabolism and several molecular determinants including oncogenic signaling pathways play pivotal roles in the development progression and metastasis of pancreatic cancer The aberrant glucose metabolism is the most prevalent key factor that influences pancreatic tumorigenesis The cancer cells of pancreas utilize oxygen for the breakdown of glucose to lactate which is known as Warburg effect This phenotype of pancreatic cancer encourages proliferation growth migration and invasive phenotypes of cancer cells that worsens the disease The upstream regulatory molecules responsible for deviant activity of glucose transporters during

aberrant glucose metabolism was not very well defined Thus identification of new molecular determinants that regulate glucose metabolic pathway would provide promising therapeutic outcomes Our lab has established the role of MUC13 protein in pancreatic cancer development Another interesting molecule Protein Kinase D1 has been reported to play essential role in hypoxic metabolism in squamous cell carcinoma and glucose uptake in cardiac hypertrophy Also it has been extensively studied in our research group and has been suggested by some other groups to have an essential role in pancreatic cancer However its role in regulating glucose metabolism in pancreatic cancer remains elusive In this dissertation we investigated that MUC13 and PKD1 proteins are involved in abrupt glucose metabolism in pancreatic cancer cells We have demonstrated that targeted overexpression of these oncogenes upregulates key oncogenic signaling components involved in aberrant glucose metabolism in pancreatic cancer cells We also investigated a natural agent Steviol that restored glucose metabolism in pancreatic cancer cells via repression of MUC13 and PKD1 expression MUC13 is a transmembrane glycoprotein which has an elevated expression in pancreatic cancer Due to the presence of a cytoplasmic domain it confers kinase activity and regulates many signaling processes Therefore we investigated the effect of MUC13 in metabolic reconditioning of pancreatic cancer As a result it was observed that presence of MUC13 in pancreatic cancer upregulated glucose uptake and lactate secretion This in turn led to more aggressive behavior of the tumor as evidenced by enhanced proliferation migration and invasive characteristics of the cells as compared to MUC13 null cells Interestingly we observed the interaction of MUC13 with Glut 1 protein which influences glucose uptake in cancer cells This was confirmed through various assays such as immunoprecipitation immunofluorescence co capping and proximity ligation assay The interpretation of inhibitor results envisaged the role of NF kappa B pathway during the molecular interactions of MUC13 and Glut 1 To understand the clinical importance of this mechanism we evaluated this interaction in tumor samples from the patients with pancreatic disease in advanced stages Our findings fortified the mechanistic role of MUC13 in rewiring of aberrant glucose metabolism Altogether we believed that MUC13 has clinical implication as a key molecule that might be responsible for dysregulated glucose metabolism As discussed earlier about the paucity of associated proteins related to dysregulated glucose metabolism we further investigated the possible key players with kinase domain for association and activation with other signaling molecules Protein Kinase D1 PKD1 a Ser Thr kinase has been shown to be involved in progression of pancreatic cancer In this study we established PKD1 as a novel molecular target and its involvement in regulating aberrant glucose metabolism in pancreatic cancer We observed that PKD1 was overexpressed in pancreatic cancer tissues compared to normal human tissues We also found constitutive expression of PKD1 in various pancreatic cancer cells as compared to normal pancreatic ductal epithelial cells The overexpression of PKD1 in low PKD1 expressing pancreatic cancer cells enhanced tumorigenic characteristics We observed that specific knockdown of PKD1 inhibited key oncogenic signaling components in pancreatic cancer cells suggesting its oncogenic role in pancreatic cancer Additionally during the PKD1

overexpression cells displayed increased glucose consumption and lactate production This suggested role of PKD1 in metabolic reprogramming in pancreatic cancer Also we observed that PKD1 stimulates the glucose uptake through mTORC1 a component of mTOR signaling pathway Additionally the knockdown of PKD1 promoted the chemosensitivity of cells towards gemcitabine and 2DG 2 deoxyglucose Overall these results indicated a significant function of PKD1 in rewiring of glucose metabolism in pancreatic cancer It was mandating to target and counteract the aberrant glucose metabolism along with the proteins identified in our previous results In this perspective herein we proposed steviol a natural sweetener from plant origin to assess its inhibitory action towards dysregulated glucose metabolism and its associated proteins MUC13 and PKD1 The reason behind steviol selection for this approach includes that it mimics the glucose molecule that facilitates enhanced uptake of drug steviol within the cells and on the other side it provides glucose homeostasis to adjacent normal cells These features made steviol as dual functional drug by overtaking other natural drugs for promising results The exposure of steviol inhibited the proliferative IC50 10mM and clonogenic abilities of pancreatic cancer cells It also suppressed the cell migration and cell invasion capabilities of pancreatic cancer cells Further steviol caused the selective inhibition of intracellular glucose intake and lactate accumulation in a concentration dependent manner The selective inhibition of MUC13 and PKD1 upstream key proteins of glucose metabolism that caused the disruption of glucose intake in cancer cells was observed during steviol treatment Secondly steviol interfered with translation initiation machinery causing destabilization of the cellular functionality The impairment of cellular translation process had promoted G1 cell cycle arrest in cells due to the lack of G1 S transition proteins during drug exposure Collectively all these events engendered the cellular integrity and directed towards activation of apoptosis In conclusion we evaluated the role of MUC13 and PKD1 in aberrant glucose metabolism leading to growth and aggressive metastatic phenotypes of pancreatic cancer We also demonstrated efficacy of steviol to repress aberrant glucose metabolism via suppression of MUC13 and PKD1 proteins Chemical Abstracts ,2002 **Abridged Index Medicus** ,1984 Conference Papers Index ,1987 **Exploring Pancreatic Metabolism and Malignancy** Ganji Purnachandra Nagaraju,Aramati BM Reddy,2019-11-23 This book comprehensively describes the association between metabolic syndrome and pancreatic cancer progression and the mechanism of action and target definition with a view to drug discovery Metabolic syndrome which includes adnominal obesity hypertension dyslipidemia and hyperglycemia has recently been shown to play an important role in the etiology and progression of various cancers Further obesity and diabetes have been associated with an increased incidence of gastric cancers The book reviews the key biological mechanisms underlying the association between metabolic dysregulation including obesity associated enhancement of growth factor signaling inflammation and perturbation in pancreatic cancer cell growth and metastasis It also illustrates the role of the inflammatory signaling pathway in metabolic diseases as well as tumor growth and explores the potential of these pathways as the rational targets for pancreatic cancer therapy Lastly the book offers a comprehensive description of the challenges associated with diabetes

and pancreatic cancer therapy **Metabolic Reprogramming of Pancreatic Ductal Adenocarcinoma Cells in Response to Chronic Low PH Stress** Jaime Abrego,2017 Pancreatic ductal adenocarcinoma PDAC is among the most lethal of all cancers with a 5 year survival rate of only 8 2% This is because PDAC is diagnosed in its advanced stages and is characterized by radio and chemotherapy resistance Aggressiveness of PDAC tumors is attributed to its high metabolic phenotype which is characterized by increased glycolysis rate and lactate secretion while oxidative metabolism is reduced These metabolic features are required to fulfill the biosynthetic demands of proliferating PDAC cells However this increase in metabolic activity results in acidification of the extracellular space because the dense fibrotic stroma of PDAC tumors limits venting of protons into the vasculature thereby creating a chronic low pH microenvironment Little is known regarding the physiology and metabolism of cancer cells enduring chronic low pH exposure To demonstrate effects of low pH PDAC cells were cultured in low pH 6 9 7 0 to establish chronic low pH as it occurs in tumors These cells were compared to cells in physiological pH of 7 4 which is also the pH of cell culture in order to evaluate physiological differences between these pH values In these experiments it was observed that cells in low pH have reduced clonogenic capacity and undergo a metabolic shift to oxidative metabolism that is supported by an increase in glutamine uptake These observations exhibit a robust contrast to PDAC cells in control pH conditions that are highly glycolytic Furthermore in low pH there is increased transcription of the GOT1 enzyme which mediates metabolic flux through the non canonical glutamine metabolic pathway that allows synthesis of other metabolic substrates from glutamine Upon shRNA mediated depletion of GOT1 survival of PDAC cells in low pH was significantly impaired due to increase in ROS to cytotoxic levels However supplementing transfected clones with GOT1 metabolic product oxaloacetate resulted in growth rescue and reduction in ROS levels Thus in chronic low pH stress PDAC cells up regulate non canonical glutamine metabolism through increased transcription of GOT1 which allows PDAC cells to generate energy and metabolic co factors to suppress cytotoxic ROS levels Low pH is a universal feature of the PDAC tumor microenvironment and further dissection of metabolic adaptations to microenvironment conditions will result in more effective therapy for PDAC Exploring Pancreatic Metabolism and Malignancy ,2019 This book comprehensively describes the association between metabolic syndrome and pancreatic cancer progression and the mechanism of action and target definition with a view to drug discovery Metabolic syndrome which includes adnominal obesity hypertension dyslipidemia and hyperglycemia has recently been shown to play an important role in the etiology and progression of various cancers Further obesity and diabetes have been associated with an increased incidence of gastric cancers The book reviews the key biological mechanisms underlying the association between metabolic dysregulation including obesity associated enhancement of growth factor signaling inflammation and perturbation in pancreatic cancer cell growth and metastasis It also illustrates the role of the inflammatory signaling pathway in metabolic diseases as well as tumor growth and explores the potential of these pathways as the rational targets for pancreatic cancer therapy Lastly the

book offers a comprehensive description of the challenges associated with diabetes and pancreatic cancer therapy

Molecular Mechanisms and Therapies of Pancreatic Cancer Donatella Delle Cave,Claudio Luchini,2024-11-18 Pancreatic cancer PC is one of the most aggressive solid malignancies with an overall 5 year survival rate of 8% and it is predicted to become the second leading cause of cancer related death by 2030 PC progression and metastasis are strongly influenced by metabolic stress imposed by the tumor microenvironment TME due to limited oxygen and nutrient supply and unfavorable pH In this context deregulation and the reprogramming of energy metabolism are hallmarks of PC which leads tumor cells to rewire their glucose amino acid and lipid metabolism on the basis of the bioenergetic and biosynthetic demands needed to survive and escape immunosurveillance For this reason exploiting cellular plasticity through the targeted reprogramming of metabolic features in PC could lead to the generation of promising and novel selective therapeutic approaches for patients treatment **Metabolic Derangements in Pancreatic Cancer** Franziska Romrig,2013

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