Nutritional Strategies in Cancer Management: The Effects of Glutamine-Free Diets

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ABSTRACT

The rapid and extensive growth of malignant cells results in increased need for energy. In response, most cancerous cells alter their metabolic pathways to fulfil these augmented requirements and this has been acknowledged as a novel hallmark of cancer. This is a review study that combines a comprehensive literature review from various platforms, including Google Scholar, Pub Med, Springer, and others, with a survey conducted via Google Forms. The survey involved a sample size of 200 paramedic staff working in diverse healthcare settings. The study did not impose restrictions based on gender or age groups. However, individuals who were unaware of the topic under investigation were excluded from participation. The study included individuals with various types of cancer, and data were collected based on their adherence to a glutamine-free diet. This hypothesis is based on the notion that glutamine serves as a primary energy source and precursor for biosynthesis in highly proliferative cancer cells. As a result, investigating the impact of a glutamine-free diet on tumor cell proliferation is of significant interest in the context of cancer research. On the basis of the results, it is concluded that glutamine is an important amino acid involved in the growth and its deprivation lowers the rate of proliferation in cancer cell lines. Although some studies show that the overall effect of diet-free regulation underlying the glutamine deprivation induced effects over cancer cells has not been completely clarified as dietary changes in glutamine have shown to have a little effect against growth in cancerous cells.

Keywords: Glutamine, cancer cells, Tricarboxylic acid cycle, Go/G1 phase.

INTRODUCTION

The most prevalent amino acid in the body, glutamine is mostly generated in the lungs and stored in the muscles. In healthy conditions, the body can produce enough glutamine to meet its requirements. This amino acid is essential to the body for several processes. One of its most important functions is to eliminate excess ammonia, which is a byproduct of metabolism in the body and is crucial to detoxification. Furthermore, glutamine has a critical role in immune system function, supporting the body's defense against many infections. It also aids the digestive system and helps the brain work normally. Even though many people can get enough glutamine from their diet and their body's production, there can be times when high stress increases the need for glutamine, such as after a hard workout or injury. In these circumstances, the body could need more glutamine than it can generate. Lower glutamine levels may be experienced by people with particular health issues, such as extended stress, infections, operations, and injuries, to meet the greater need consuming a glutamine supplement may be helpful in these situations.

A major component of amino acid nitrogen transport in the plasma is accounted for by glutamine, a five-carbon amino acid with two amino groups. Glutamine plays an essential part in this process. The "nitrogen shuttle" it performs is essential in shielding the organism from the damaging consequences of high blood ammonia levels. Moreover, glutamine performs special metabolic tasks including promoting the transport of carbon between tissues, providing energy for cells that divide quickly, and functioning as a precursor for several biologically active compounds.

Effects of glutamine on the body:

Cellular Energy Production*:** The principal source of energy for quickly proliferating cells, like those in the immune system and gut lining, is glutamine. ***Protein Synthesis: It is essential for the production of proteins and helps build and repair tissues, particularly muscle tissue. ***Immune System Function:*** Since glutamine serves as an energy source for immune cells and promotes the development and activity of white blood cells, the immune system needs to operate properly.

Gut *Health*: This amino acid aids in the preservation of the intestinal lining and serves as an essential source of energy for the cells that line the digestive tract.

Nitrogen Transport: The movement of nitrogen throughout the body's tissues and organs depends on glutamine.

Detoxification and Ammonia Regulation: By transforming ammonia, a waste product of metabolism, into urea in the liver, glutamine assists in the detoxification process.

Antioxidant Properties: Due to its antioxidant qualities, glutamine can shield cells from oxidative stress and harm.

The topic of glutamine's interaction with cancer cells is intricate and complicated. Even though glutamine is necessary for regular cellular processes, cancer cells frequently have altered metabolisms and high glutamine requirements. This is called the ***Warburg effect*** in which cancer cells prefer the metabolic process of glycolysis and use a lot of glucose and glutamine to fuel their fast development and multiplication. Glutamate impacts cancer cells

in someways;

Glutamine as a Source of Energy: Glutamine is an essential energy source for a variety of cell types, including cancer cells. Cancer cells frequently require more energy because of their fast growth and division. The main source of energy for cells, adenosine triphosphate (ATP), can be produced from glutamine via a number of metabolic processes, one of which is the tricarboxylic acid cycle (TCA cycle).

Macromolecule Biosynthesis: Glutamine serves as a precursor to a number of vital biomolecules. It supplies the carbon and nitrogen atoms required for the production of amino acids, lipids, and nucleotides—the fundamental components of DNA and RNA. For cancer cells to support their rapid growth, they need an elevated level of protein synthesis and DNA replication, which makes these metabolic pathways essential.

Protection Against Ammonia Toxicity:

One typical waste product of cellular metabolism, excess ammonia, must be eliminated from the body, and glutamine is essential for this process. Since ammonia is poisonous, the body detoxifies itself by converting glutamine to glutamate during this process. Because of their changed metabolism, cancer cells frequently produce more ammonia; therefore, glutamine utilization plays a crucial role in controlling ammonia levels.

Metabolic Reprogramming: Often called the "Warburg effect," this refers to a change in many malignancies towards modified metabolic pathways, including enhanced glutamine utilization. Cancer cells can adjust to their energy and biosynthetic needs because of this metabolic reprogramming. Glutamine can support anabolic activities by supplying carbon, which helps cancer cells proliferate and endure.

Glutamine Addiction: A glutamine-dependent state occurs in certain cancer cells; this state is sometimes called "glutamine addiction." To maintain their particular metabolic needs, they are dependent on glutamine. Currently, researchers are investigating medicines that impede glutamine utilization as a potential cancer therapeutic approach. This glutamine addiction has led to this exploration.

***Sensitization to Cancer Therapies*:** Certain chemotherapy and radiation treatments can make cancer cells more sensitive if glutamine metabolism is inhibited. Their metabolic systems have been upset, which leaves them more susceptible to conventional cancer treatments. Research on combining glutamine-targeted medicines with conventional cancer treatments is ongoing.

Tumor Microenvironment and Nutrient Competition: Because surrounding normal cells and cancer cells fight for little nutrients, such as glutamine, the tumor microenvironment may become nutrient-depleted. Cancer cells may develop more slowly if glutamine levels are low because it puts them at a disadvantage in the competition.

METHODOLOGY

Research Objectives:

- To assess the knowledge, attitudes, and practices of paramedics and cancer patients regarding the potential benefits of a glutamine-free diet in cancer prevention.
- To gather insights into the dietary habits of these populations.

- To identify potential areas for education and intervention.

Study Design:

Study Type: Cross-Sectional Survey Duration: The survey will be conducted over 2 months.

Participants:

Inclusion Criteria:

Paramedics working in Pakistan. Cancer patients of all ages and genders. Willingness to participate in an online survey.

Exclusion Criteria:

Inability to access and complete an online survey.

Survey Development:

A Google Form survey will be designed, including questions related to knowledge about glutamine, dietary practices, and opinions on the potential role of glutamine- free diet in cancer prevention.

Survey Distribution:

The survey link will be distributed through various channels:
Paramedic associations and forums. Cancer support groups and healthcare facilities. Social mediaplatforms. Local healthcare organizations. Informed consent will be obtained before participants can access the survey.

Data Collection:

Participants will complete the online survey at their convenience. The survey will include questions about dietary habits, awareness of the role of glutamine, and any dietary changes made by cancer patients.
Data collection will be anonymous to protect the participants' privacy.

Data Analysis:

Survey data will be analyzed using appropriate statistical methods, including descriptive statistics and cross-tabulations. Subgroup analyses will be conducted to identify any differences between paramedic staff and cancer patients.

Ethical Considerations:

Informed consent will be included at the beginning of the survey. Participants' confidentiality and privacy will be ensured. The study will adhere to ethical guidelines for online surveys and data collection.

Data Interpretation:

The survey results will be interpreted to assess the knowledge, attitudes, and practices related to glutamine-free diets in the context of cancer prevention.

Conclusion and Dissemination:

A report summarizing the survey findings will be prepared. The results will be disseminated

through local healthcare conferences, and seminars, and published as a report for broader public access.

This methodology provides a framework for conducting a survey-based study on the awareness and practices of paramedic staff and cancer patients in Pakistan regarding a glutamine-free diet and its potential role in cancer prevention. It leverages the accessibility and convenience of online surveys through Google Forms while ensuring the privacy and consent of participants.

RESULT:

GLUTAMINE METABOLISM IN CANCER CELLS:

Oncogenotypes, which rewire glutamine metabolism for energy production and stress suppression, and the tissue of origin have an impact on the expression of glutamine metabolism-related enzymes, which varies widely in malignancies. Of the two glutaminase enzymes. GLS is expressed more widely in normal tissue and is believed to be involved in many malignancies, whereas GLS2 expression is mainly found in the pituitary gland, liver, brain, and pancreas. GLS pre-mRNA is spliced into either kidney- type glutaminase (KGA) isoforms or glutaminase C (GAC), which adds even more complexity. The regulation and activity of GLS2 and the two GLS isoforms vary as well. GLS2 but not GLS is triggered by its product ammonia, whereas glutamate inhibits GLS but not GLS2 in vitro. While inorganic phosphate activates both GLS and GLS2, GLS (and especially GAC) exhibits a significantly greater increase in catalysis when inorganic phosphate is present. In contrast to SIRT3, which may deacetylate GLS2 to encourage its enhanced activity with caloric restriction, Sirtuin 5 (SIRT5), which is overexpressed in lung cancer can desuccinylate GLS to decrease its enzymatic activity. Nutrient absorption and metabolism affect the availability of phosphate, acetyl-CoA, and succinyl-CoA, indicating that GLS and GLS2 activity may be sensitive to the metabolic condition of the cell. Furthermore, translocation, RNA- binding protein control of alternative splicing post-transcriptional regulation by miRNAs and pH stability of the GLS mRNA, and protein degradation via the CDH1 E3 ubiquitin ligase complex and the anaphase-promoting complex (APC).

Numerous cancer types have greater expression of GAC, which is more active than KGA. This suggests that GLS alternative splicing may be a significant factor in the higher glutaminolytic flux that is thought to occur in cancer. The function of GLS2 in cancer, however, appears to be more intricate. Re-expression of GLS2, which is suppressed by promoter methylation in glioblastoma, colorectal cancer, and liver cancer, has been demonstrated to exhibit tumor suppressor properties in colony formation experiments. Indeed, a recent study shown that GLS2 sequesters the small GTPase RAC1 to inhibit metastasis in a non- metabolic role. Nonetheless, GLS2 appears to encourage radiation resistance and assist the growth of some cancer forms. Tumor suppressor p53 and associated proteins p63 and p73 stimulate GLS2, which may indicate that it is involved in radiation resistance, or plays a significant role in tumors that still have wild-type p53. Furthermore, in neuroblastoma, GLS2 is an essential downstream target of the N-MYC oncogene. Further research is definitely warranted given the context-dependent involvement of GLS2 in cancer.glutamine is then produced by glutaminase and further converted to glutamate by one of two mechanisms: GLUD catalyses the reversible degradation of glutamate to produce glutate and release ammonium. This reaction is almost thermodynamically stable in the liver.

GLUD functions both ways in this organ, but in cancer, it is thought to primarily work in the direction of glutarate. Like GLS, GLUD is regulated by post-translational modifications and allosteric regulation. It is activated by adenosine triphosphate (ADP) and inactivated by gated transaminases (GTP), palmitoylnol-CoA and adenosine monophosphate-dependent adenosine ribosyl transferase MTOR (which is also activated by leukine) can also promote GLUD activity by suppressing SIRT4 expression. These observations suggest that low energetic state might promote GLUD allosteric activity via ADP to promote ATP production, while high leucine availability might also promote glutamine alloster.

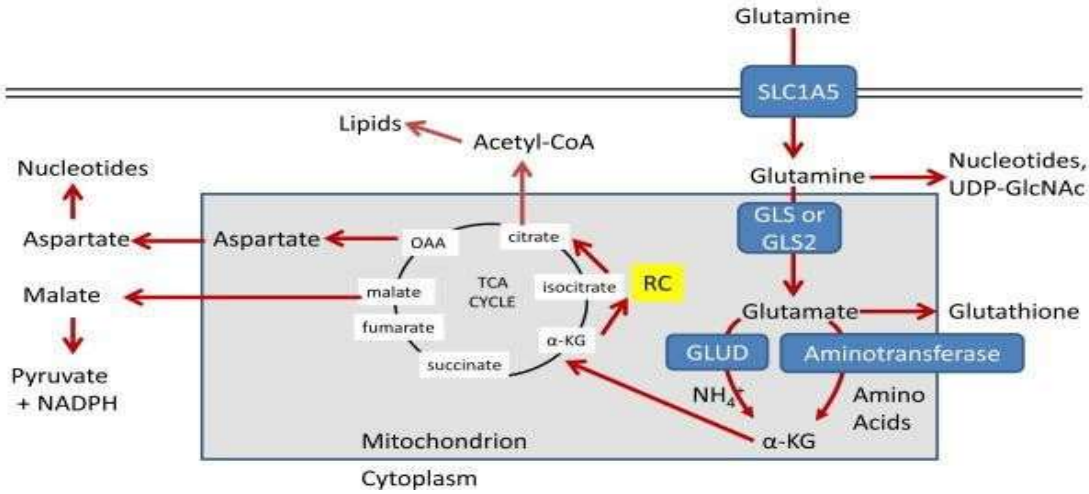

Figure 1 Major metabolic and biosynthetic fates of glutamine.

Aspartate Amino- Amino-Transaminases Amino-transaminases are enzymes that convert glutamate to aspartate without the production of ammonia. Alanine Amino-Transaminase GPT (Glutamic-Pyruvate Transaminase) Glutamic-Oxaloacetic Transaminase GOT (Aspartate Amino-Transaminase) Transferring nitrogen from glutamate Aspartate Producing alanine -Acetylated - Glutamic-Pyrruvate Significance of liver enzymes Liver enzymes. Mitochondrial isoenzymes Types of liver enzymes 1. GPT 2 GPT2 Mitochondrial GLUTAMIC-OXALACETATE TRANSAMINATE TRANASTRASMASECYP1.

Phosphoserine Amino Biosynthesis (PSAT1) PSAT1 is involved in the synthesis of phosphoserine and the formation of phosphatidylinositol in the serine pathway. PSAT1 transfers nitrogen from glutamate into 3-phosphatidyl-thiol pyruvate for the formation of phosphoserine. Tissue Distribution Aspartate Amino-Transferase activity is highly distributed across most tissues. Alanine Amino-transferase activity is concentrated in the liver, but the expression is still relatively universal. Tumors Aminotransferases like PSAT1 may inappropriately express in tumors.

Role of glutamine in cancel cells with respect to amino acid production

Glutamine nitrogen supports the levels of many amino acid groups in cells through aminotransferase activity. Independent of metabolic reactions, the carbon and nitrogen of glutamate can be used to produce proline, which plays a key role in the production of the extracellular matrix protein collagen. Although proline can be degraded to glutamate, the MYC oncoprotein can alter the expression of proline synthesizing and degrading enzymes to promote net synthesis of proline from glutamine-derived glutamate. Overall, labeling experiments determined that at least 50% of the non- essential amino acids used by cancer cells during protein synthesis in vitro can be derived directly from glutamine

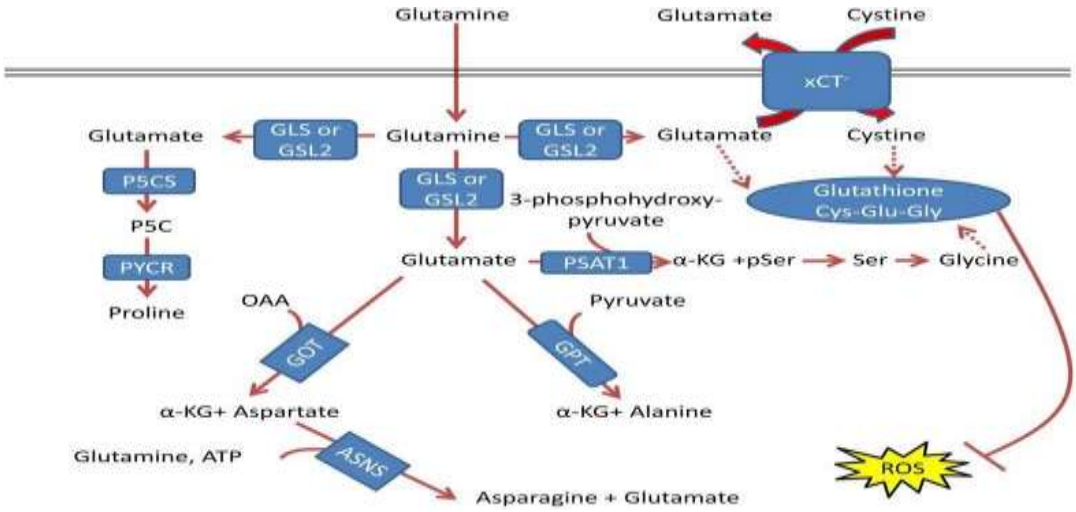

Figure 2: Glutamine regulates amino acid levels and helps manage reactive oxygen species (ROS).

While the Various amino acids derived from glutamine contribute to cancer cell survival. Recent studies have shown that aspartate biosynthesis, may depend on both glutamine flux through the TCA cycle and glutamate metabolism, which is especially important due to its key role in the biosynthesis of purines and pyrimidines to support cell division.

Role of glutamine in cancer cells with respect to control glutathione and reducing equivalents:

Numerous glutamine metabolic fates directly promote protein synthesis and trafficking while inhibiting stress reactions mediated by the endoplasmic reticulum (ER) stress pathway and the integrated stress response (ISR), two related pathways. The cell's total amino acid pools are supported by glutamine intake, which suppresses the ISR. The amino acid-sensing kinase GCN2 (encoded by EIF2αK4) activates the ISR under amino acid deficiency. General cap-dependent protein synthesis via the ISR is inhibited when GCN2 phosphorylates eIF2α. However, cap- independent synthesis of ATF4 is induced, which in turn triggers a cascade that increases transcription of ER-associated chaperones, stops cap-dependent translation, and ultimately leads to cell death. Deprivation of glutamine can either directly result in uncharged tRNAs or lead to a decrease in downstream

products like asparagine, which might subsequently result in uncharged tRNAs and activate GCN2, which in turn triggers the translation of ATF4. It has been demonstrated that glutamine input-induced ISR suppression is essential for the survival of various cancer cell and tumor types, such as neuroblastoma and breast cancer. Additionally, it was noted that asparaginase, which the US Food and Drug Administration (FDA) has licensed for the treatment of acute lymphoblastic leukemia (ALL), activates GCN2 in mice and may deplete serum glutamine and asparagine..As a component of the hexosamine biosynthesis pathway, glutamine also aids in the synthesis of uridine diphosphate N-acetylglucosamine (UDP- GlcNAc), which is necessary for glycosylation, appropriate ER-Golgi trafficking, and regulation of the ER stress pathway, all of which occur upstream of ATF4 induction. It has been demonstrated that abnormal expression and function of O- Linked β-N-acetylglucosamine transferase (OGT), which binds UDP-GlcNAc to

Proteins are essential for the survival and advancement of chronic lymphocytic leukemia, prostate cancer, and breast cancer. Thus, by suppressing the ISR and ER stress pathways, glutamine input directly maintains translation, protein trafficking, and survival.

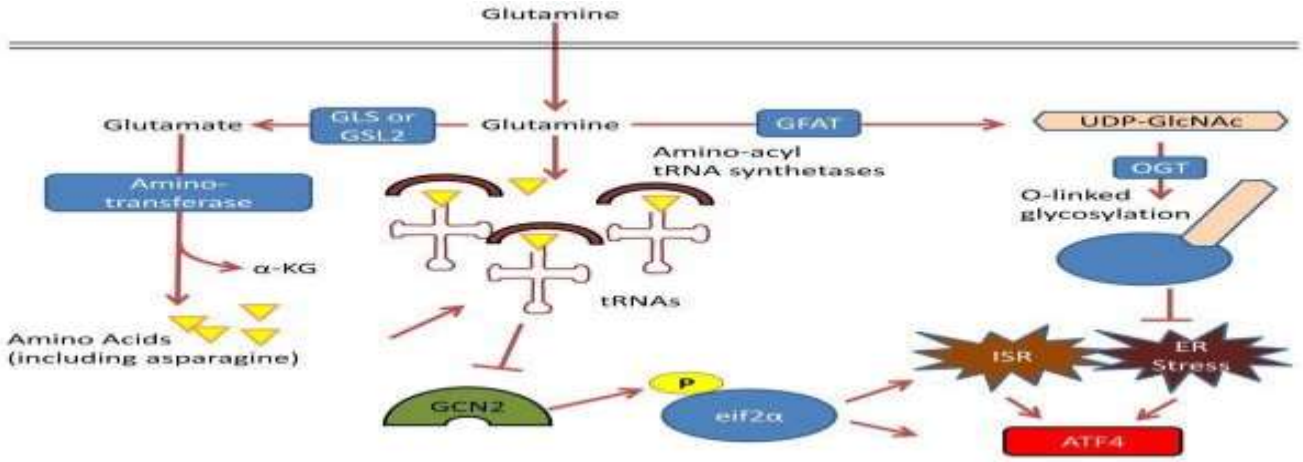

Figure 3 Glutamine helps control the integrated stress response, protein folding and trafficking, and ER stress.

Role of glutamine in cancer cells with respect to control glutathione and reducing equivalents:

When ROS levels are at healthy levels, ROS- mediated cell signaling may be pro- tumorigenic; yet, when levels are elevated, ROS can be extremely harmful to macromolecules. Superoxide (O_2^-) is produced when electrons in the mitochondrial electron transport chain leak to oxygen, one of the many sources of reactive oxygen species (ROS). As a result, higher ROS generation may be correlated with increased glutamine oxidation. Nevertheless, glutamine metabolism is essential for maintaining cellular ROS homeostasis because it produces products through a number of metabolic pathways that directly regulate ROS levels. The most well-known mechanism by which glutamine regulates reactive oxygen species is through glutathione production. The tripeptide glutathione (Glu-Cys-Gly) works to scavenge peroxide free radicals. It has long been known that the rate-limiting step in the synthesis of glutathione is glutamine intake, which is both directly and indirectly responsible for the other two glutathione amino acid constituents. Given that glutathione levels are linked to cancer growth and drug resistance, a deeper comprehension of this process could lead to more effective cancer treatment plans. Indeed, a number of studies have demonstrated that giving cancer patients acute glutamine while they are undergoing chemotherapy or radiation therapy lowers the toxicity of the treatment by promoting glutathione synthesis. Through the production of NADPH via GLUD, glutamine also influences ROS homeostasis. Additionally, glutamine provides reducing

equivalents for glutathione through at least two other related mechanisms, wherein aspartate or malate derived from the TCA cycle is exported to the cytoplasm and subsequently converted to pyruvate to produce NADPH through the malic enzymes.

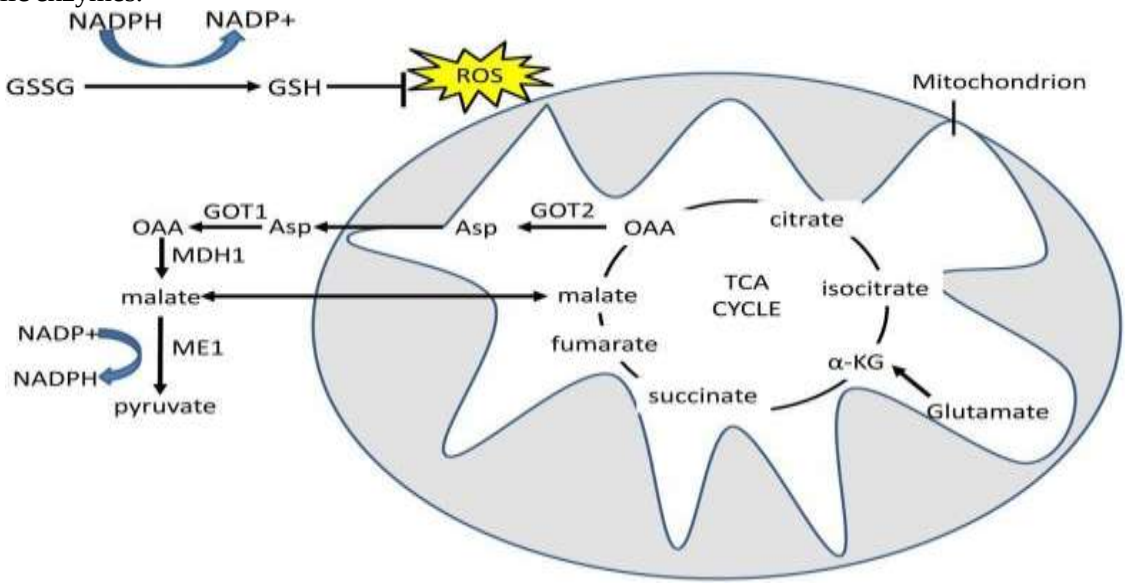

Figure 4 Glutamine can contribute to the TCA cycle and be utilized in two pathways to generate NADPH and neutralize ROS through the malic enzyme.

Role of glutamine in cancer cells with respect to regulation of mTOR.

Sensing amino acids, the TOR pathway inhibits degradative processes like autophagy while broadly promoting biosynthetic pathways like protein translation and fatty acid synthesis. Therefore, to avoid unintended cell growth, mTOR activity needs to be strictly regulated. Glutamate does this through a number of different mechanisms. It is necessary to maintain amino acid availability regardless of the state of mTOR pathway mutations because it stimulates mTOR activity independently of these mutations, which are frequently found in human cancer. Because amino acids derived from macropinocytosis can also support

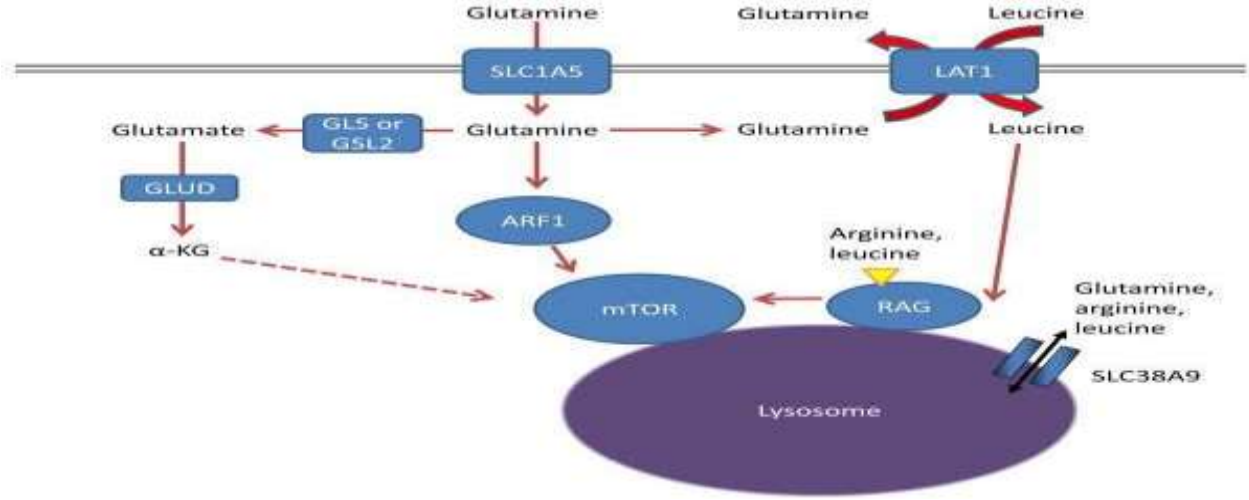

Figure 5 Glutamine controls mTOR activity.

mTOR activation,On the other hand, GLUD repression can be relieved by mTOR itself through cell-type-specific mechanisms that either inhibit GLUD expression while upregulating the expression of aminotransferases, as will be covered in more detail below, or by inhibiting the expression of mitochondrial SIRT4. These results have a significant implication: mTOR activity may be strongly stimulated by increased glutamine uptake and metabolism, which is common in many cancers, even in the absence of direct mutations of negative regulators of the mTOR pathway itself, such as tuberous sclerosis 1 protein (TSC1; also known as hamartin) and TSC2 (also known as tuberin). More research will be required to completely comprehend this fascinatingly complex process of mTOR regulation by amino acid availability, including glutamine. This is a rich and developing field.

Role of glutamine in cancer cells with respect to nucleotide biosynthesis:

Glutamine directly contributes to the biosynthesis requirements for cell division and growth. Nitrogen from glutamine directly contributes to both de novo purine and pyrimidine biosynthesis, while carbon from glutamine is used for the synthesis of amino acids and fatty acids.The fact that glutamine-deprived cancer cells experience cell cycle arrest, which is not reversible by TCA-cycle intermediates like oxaloacetate but is reversible by exogenous nucleotides, highlights the significance of glutamine as a nitrogen reservoir. In fact, ex vivo cultured human primary lung cancer samples have been shown to synthesize nucleotides from exogenous glutamine. Via different mechanisms, glutamine can also aid in the biosynthesis of nucleotides. Purine and pyrimidine require aspartate, which is produced from glutamine by the TCA cycle and transamination, as a vital source of carbon. Aspartate synthesis and supply can prevent cell cycle arrest brought on by glutamine shortage. Furthermore, the enzymes carbamoyl-phosphate synthetase 2, aspartate transcarbamylase and

dihydroorotase (CAD), which catalyze the incorporation of glutamine-derived nitrogen into pyrimidine precursors, may be activated by glutamine-dependent mTOR signaling. Nucleotide synthesis may be further supported by NADPH that is produced as a byproduct of glutamine metabolism and fluxes through the malic enzymes. Overall, by directly supplying carbon and nitrogen, indirectly producing reducing equivalents, and activating the signaling pathways required for their synthesis, glutamine can support the biomass accumulation of fatty acids, amino acids, and nucleotides

Table: 1 Prognosis of different types of cancer concerning Glutamine.

Cancer Type	Prognosis	Key Findings
Colorectal Cancer (CRC)	Overall Survival (OS)	In univariate analysis, low serum glutamine levels are linked to poorer OS. However, no statistical significance is observed in multivariate analysis. Glutamine levels also impact Progression-Free Survival (PFS) and emerge as an independent prognostic factor in multivariate analysis.
Ovarian Cancer	Treatment Response	Glutamine metabolism is closely associated with a less favorable prognosis. It shows potential as a target for addressing drug-resistant ovarian cancer. It plays a critical role in processes like proliferation, invasion, and drug resistance, making it an area of investigation for diagnosis, treatment, and prognosis.
Organ- Specific Metabolism	Inter-Organ Dynamics	Glutamine metabolism reveals intricate dynamics among different organs. Skeletal muscle, lungs, and adipose tissue contribute to the plasma glutamine pool, while the liver's role is influenced by regional variations and systemic factors.

Pharmacology targets glutamine metabolism in cancer

Targeting glutamine metabolism in cancer includes disturbing the role of glutamine, an essential amino acid, in assisting the growth and survival of cancer cells. This method is founded on the understanding that many cancer cells have an amplified demand for glutamine to encounter their metabolic and energy requirements.

Table:2 The pharmacological strategies

DRUG CLASS	DESCRIPTION
01. Glutaminase Inhibitors	➢ Glutaminase is an enzyme that catalyzes the transformatio n of glutamine into glutamate, a significant step in glutamine metabolism. ➢ Pharmacologi cal agents, such as CB- 839, are being produce to prevent glutaminase, decreasing the accessibility of glutamate for cancer cells. ➢ By hindering this conversion, these inhibitors can bound the availability of glutamate and downstream metabolites, which are indispensable for cancer cell

	production and survival
02. Glutamine Transport Inhibitors	➢ Cancer cells frequently depend on definite transporters, such as ASCT2, to import extracellular glutamine. ➢ Pharmacological agents, such as V- 9302, are intended to prevent these transporters, stopping glutamine from entering the cancer cells. ➢ This method can basically famish cancer cells of their glutamine supply, deterring their growth and survival.
03. Targeting Glutamine - Dependent Pathways	➢ Some cancer types display a need for definite metabolic pathways that are glutamine-dependent. For example, the mTOR pathway can be influenced by glutamine levels. ➢ Pharmacological schemes intend to target these pathways directly, disturbing the signaling and metabolic processes that depend on glutamine.
04. Combination Therapies	➢ Combining inhibitors of glutamine metabolism with other Cancer treatment manner is a common method. This combination can improve the efficiency of both methods. ➢ For example, combining glutaminase inhibitors with chemotherapy or immunotherapy may produce a synergistic outcome, leading to enhanced cancer cell destruction.
05. Personalized Medicine	➢ Understanding the degree to which a specific tumor trusts on glutamine metabolism can be vital. ➢ Biomarkers or genetic indicators can help recognize tumors that are more reliant on glutamine for their survival. This evidence allows for altered treatment approaches, confirming that patients receive the maximum operative therapies based on their tumor's metabolic profile.

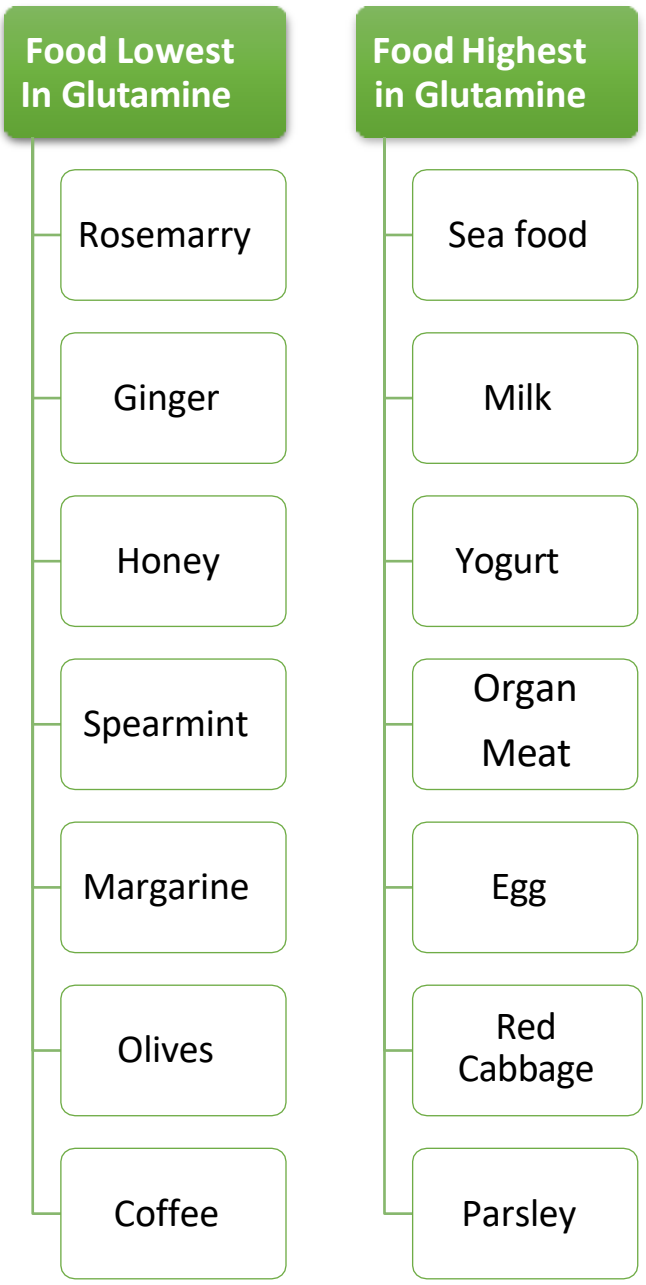

Figure 6 Dietary factors that contribute to the cancer

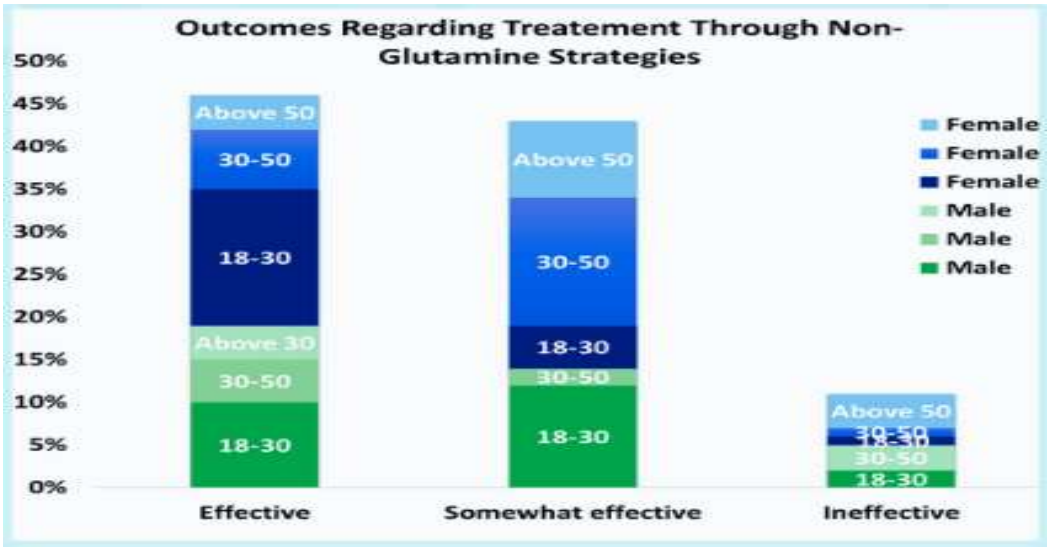

Figure 7 Shows the outcomes regarding treatment through non-glutamine strategies and the bars clearly shows the treatment goal is achieved as the percentage of treatment effectiveness is greater than ineffective.

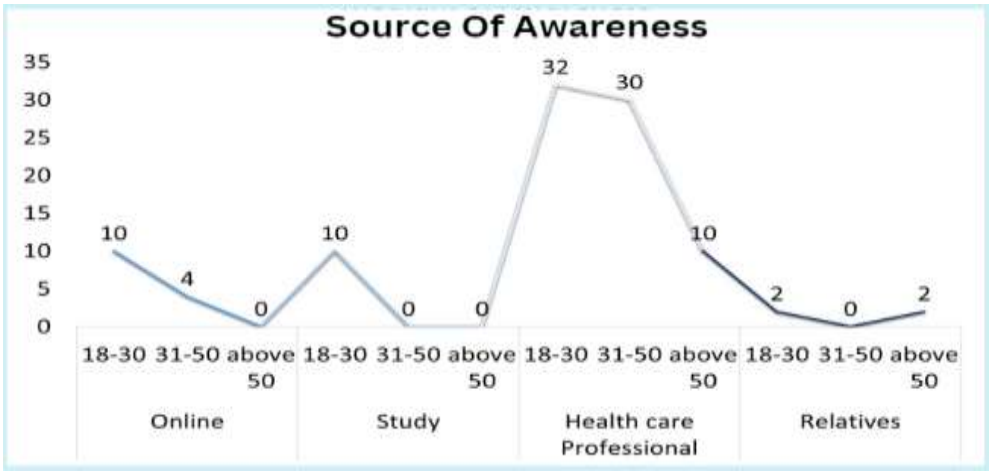

Figure 8 Shows the medium of awareness about the strategy that reducing the progression of cancer by giving glutamine free diet

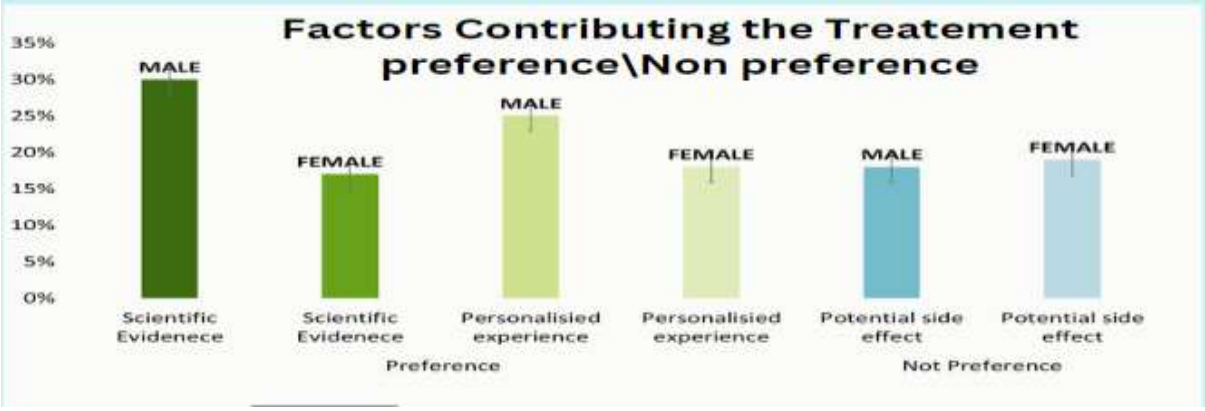

Figure 9 Shows the preference and non-preference factor correlate with the gender responses in percentage.

DISCUSSION:

The interpretation of the results of the study showed that 59% of the participants presented knowledge about the potential impact of a glutamine-free diet on cancer progression. This ensures that a considerable part of the population is aware of the concept. 90% of respondents were aware about glutamine-free diet and its potential implications for cancer progression. The increased level of awareness indicates that majority of the population acknowledged of the relevance of glutamine in context of cancer which could be due to enhanced media coverage, public discussion or dissemination of information by cancer support organizations and other healthcare professionals. 55% of participants had awareness about the role of glutamine in body which shows that more than half of the survey population had a basic understanding of the physiological significance of glutamine. 35% of the respondents had awareness of the scientific studies regarding the impact of glutamine-free diet on cancer progression. These findings indicates that further improvement is required in dissemination of scientific research regarding the concept to the general public. 37% of the population had belief in the effectiveness of glutamine-free diet for slowing oncologic progression. This result demonstrates that while some respondent recognized the potential benefits. lf glutamine-free diet, a major portion remains uncertain or skeptical about its effectiveness. 63% of the population acknowledged the evidential existence of the potentials benefits of the diet in cancer management. In this regard, the higher level of perception is promising as it suggests that a considerable part of the population is informed about the scientific studies and research in this field. 75% of the participants showed willingness to consider the incorporation of glutamine- free diet in cancer management. This high percentage shows an openness among the respondents population to incorporate dietary strategies into their cancer care plan. 42% of respondents considered medical advice as a significant factor influencing their preference for implementing a glutamine-free diet. 39% mentioned scientific evidence as an important factor. 44% mentioned concern about potential side effect as key factor in the decision making process which indicates the individuals depend on combination of the factors i.e. potential side effects, scientific evidence and personalized medical advice when making dietary choices in the process of cancer management.

The above results suggests growing awareness of the dietary choice in field cancer management and willingness to incorporate this dietary approach. Although advancement in the dissemination and education of scientific evidence is still needed to empower and inform the general public to make well informed choices in regards of dietary decisions for cancer management. Moreover, healthcare professionals play a pivotal role in providence of recommendations and guidance to the population seeking to evaluate their exploration of glutamine-free diet into their cancer-care plans.

CONCLUSION:

The comprehensive analysis of the study overwhelmingly supports the effectiveness of a glutamine-free diet in mitigating cancer progression. The results revealed majority of awareness with health care professionals exhibiting the highest

awareness of the potential benefits of the diet, meanwhile; a noteworthy of participants gained awareness through other resources. The findings also underscore the significance of scientific evidence over personalized experience in influencing the dietary interventions. Notable age and gender-based differences in outcome of the effectiveness of non-glutamine strategies for treatment was interpreted. As moving forward, the data consistently indicates a significant positive trend, with a notable percentage of participants giving outcome in the efficacy of this dietary approach. However, gender disparity influences the need for targeted educational interventions in account for significant factors influencing the decision making process in males and females.

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