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Leveraging Cancer Mutations and Metabolism for Regenerative Medicine Applications

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ABSTRACT

Cellular replication rates can vary between different cell types and the genes encoded within them. However, there are ways to manipulate cells in order to increase their rate of cell turnover. Cancer cells are an important point of study, as one of their characteristic traits is their rapid reproduction. The mutations that cause cancer cells to grow faster than normal either alter genes or change metabolic pathways. This can be manipulated in normal cells through upregulation or downregulation to artificially induce rapid rates of reproduction in normal cells. Mitochondrial function also impacts the rate of cellular processes and turnover, and can be made more efficient by providing specific nutrients to cells that stimulate mitochondrial function. These technologies can be used to induce higher rates of turnover in normal cells from different parts of the body for the purpose of regenerative medicine and medicinal research.

Introduction

Regenerative medicine has come far in the past couple of years, but still has limitations. Often, cells don't grow fast or well together, despite being similar in origin or cultured from the same original sample. Organoids, grown using stem cells, can take months or even years to grow despite being the size of a fingernail. At this point, growing organs big enough to perform all the needed functions in the human body may take years or even decades to grow. As of now, regenerative technology is focused on using stem cells to grow tissues and organs. Examples of the types of stem cells used for this include ESCs, which are isolated from a blastocyst, iPSCs, which are reprogrammed from adult tissues, or ASCs, which are isolated from nature tissues. Stem cells themselves are defined as undifferentiated cells that are capable of self-renewal and differentiation into diverse, mature progenies. This allows them to play a central role in homeostasis, regeneration, and tissue genesis through their ability to provide new elements to increase tissue mass during growth and replace cell loss due to senescence or damage. Different stem cells have different capabilities in their potency to grow different types of tissues, with zygote SCs having totipotency, embryonic SCs having pluripotency, and adult SCs having multi/unipotency. ESCs can generate any differentiated phenotype of the three primary germ layers through the differentiation process, while ASCs have a limited differentiation potential and are mainly responsible for turnover and repair within the tissue of origin.

One of the most significant factors currently limiting the technology is the rate of cell growth. This paper explores the use of cancer mutations in specific genes and metabolic pathways and the enhancement of mitochondrial function to accelerate cell growth, opening the door to further applications in regenerative medicine. Cancerous mutations are cancer-specific genetic variants that allow cancer cells to have an increased reproduction rate, and often upregulate or downregulate key genes. Cancer cells also have specific metabolic profiles due to the difference in their metabolic pathways compared to normal cells, most involve changes to pathways that allow cells to reproduce faster by increasing the rate at which cellular processes can occur. Additionally, mitochondrial function is one of the determining factors for the rate of cellular process, including

reproduction, because the efficiency of mitochondria correlates directly with the amount of ATP available for use in the cell.

Cancer Mutations

What Are They?

Cancer mutations are forms of gene mutations, or abnormal changes in a gene's DNA, that help cancer grow, form, and spread throughout the body. Cancer mutations come in two main forms: an oncogene, which is a mutated proto-oncogene whose main function is to regulate cell growth, and in mutated tumor suppressor genes, such as *BRCA1*. Both types of mutations provide grounds for cancerous cells to grow, in the case of oncogenes, by no longer suppressing cancerous growth or, in the case of tumor-suppressor genes, by not inhibiting the growth of cancerous tumors. In addition, both types of mutations are associated with a high cellular growth rate, which is currently the main point of exploration for these genes.

How Can They Be Leveraged?

Many different cancer mutations can be leveraged to increase the rate of cellular reproduction, and different mutations can be either upregulated or downregulated to do this (Figure 1).

Insulin-Like Growth Factor-1

Insulin-like Growth Factor-1 (*IGF-1*) is a gene that encodes a peptide growth hormone and is associated with an increased risk for breast cancer, making it a cancer gene (Sachdev & Yee, 2001). The protein this gene encodes is a crucial regulator of cell proliferation, differentiation, and apoptosis, and also has both mitogenic and anti-apoptotic activity in normal cells and cancer cells. It acts cooperatively with the hormone estrogen to increase neoplastic cell proliferation (Costa-Silva et. al, 2016). *IGF-1* is also found in the majority of human tissues, though it is mainly expressed in stromal and rarely in epithelial cells. This is ideal for use in reproductive medicine because it can be used to grow and regenerate a variety of tissues if implanted and upregulated in different types of tissues. The peptide growth hormone can be artificially activated in tissues where present but not currently active and can be upregulated in tissues where present. Activation and upregulation should be done with GH, insulin, and sex hormones to increase the amount of mitosis and decrease the amount of cell apoptosis occurring in a tissue at any given time. The activities of mitosis and cell apoptosis themselves are mediated by the transmembrane tyrosine-kinase of the IGF-1 receptor (IGF-1R). In partnership with the *IGF-1* gene, this can increase the amount of cell proliferation possible at one time for multiple tissues (Costa-Silva et. al, 2016). This gene is fascinating because of its versatility, since it is found in most human tissues, which offers the potential for it to be manipulated in any of the body's tissues. However, since it is mainly expressed in stromal cells and occasionally in epithelial cells, it would be best manipulated in those types of cells through upregulation and an increase in GH, insulin, and sex hormones. Given that IGF-1 is found mainly in the pancreas, it could be upregulated in pancreatic cells for diabetic research, such as a restoration of the production of insulin in type-II diabetics.

Acetylcholinesterase

Acetylcholinesterase (AChE) is a serine hydrolase enzyme whose primary function involves degrading the neurotransmitter acetylcholine (ACh) and terminating neurotransmission. However, AChE is involved in cellular growth, apoptosis, and drug resistance pathways in non-neuronal cells. The AChE pathway functions by inhibiting MAPK, PI3K, and AKt enzymes, all of which cause cellular growth to occur, and by promoting the enzyme

JNK , which results in apoptosis. Additionally, in the normal AChE pathway, phosphorylated GSK-3β is the product of the activation of the AKt enzyme, and promotes cell apoptosis. In human tumors, AChE is altered to become a vital regulator of oncogenic signaling pathways that involve the proliferation, differentiation, adhesion, migration, invasion, and metastasis of cells in tumors. The AChE signaling pathway itself is altered and a significant contributor to cancer progression, with its isoforms, AChE-T, AChE-R, and AChE-S expressed in cancer cell lines and tumors.

In cancerous tissues, AChE is downregulated in comparison to normal tissue levels, with the cancer cells elevating the functional activity of the proteins involved in ACh synthesis and transport while breaking down Ach-degrading enzymes like AChE to subsequently increase the amount of ACh neurotransmitter in cells (Richbart et al., 2021). The ACh neurotransmitter functions as an autocrine and paracrine growth factor in cells by acting as a signaling molecule in non-neuronal tissues (Friedman et al., 2019). As a result, both the AChE signaling pathway and ACh signaling loop can be manipulated when regenerating or creating different types of tissues throughout the body. For example, it could downregulate the production of the degrading enzyme AChE, while increasing the production of ACh to serve as a signal for cell growth in tissues to increase the rate of cellular growth. This signaling loop has exciting potential for manipulation because it is found in all body cells and can be manipulated for its intended goal of increasing growth in all non-neuronal cells as it regulates cell growth and apoptosis in all body cells. The AChE pathway and ACh signaling loop can be useful for regenerative medicine given that it is a cell-signaling checkpoint that either stops or allows for division to occur, so altering its function can result in an altered cell-growth cycle.

miR-506

MicroRNAs, also known as miRNAs, function as small, non-coding RNAs that regulate gene expression post-transcription. They are involved in almost all biological processes and have been identified as potential oncogenes or tumor suppressor genes depending on the context of the cancerous tissues itself. miR-506 specifically plays a crucial role in the regulation of cell proliferation, differentiation, migration, and invasion. Dysregulation of this microRNA has been demonstrated in multiple types of cancers and is caused by promoter methylation and changes in upstream transcription factors (Li et al., 2016). The down- or up-regulation of miR-506 affects diverse biological behaviors by working to suppress the translational output of several other target genes within the genome, with several miR-506 targets having known roles in various types of cancers and related to different biological processes. The targeted genes are downregulated in the specific context of the disease. Thus, they are associated with several physiological events, including increased cell proliferation and differentiation (genes CDK 4/6, PIM3, N-Ras, and SPHK1), inhibition of cell apoptosis and angiogenesis (genes SPHK1, ROCK1, and Gli3), increased cell migration and invasion (genes ETS-1, DNMT3B, SNAI2, and VIM), and increased chemoresistance (genes RAD51 and DNMT3B). The miR-506 gene can be found with reduced expression in pancreatic cancer and suppresses cell proliferation, enhances apoptosis rates, and induces cell cycle arrest at the G1/S transition stage (Li et al., 2016). This essentially makes miR-506 a tumor suppressor in pancreatic cancer as it supports PC cell proliferation (Li et al., 2016). In gastric cancer, however, miR-506 was upregulated and acted to inhibit endothelial cell angiogenesis and metastasis invasion by suppressing the proto-oncogene transcription factor ETS-1 (Li et. al, 2015), though miR-506 still functioned as a tumor suppressor in this form of cancer (Li et al., 2016). In the context of cell proliferation, miR-506 can be either up or down-regulated depending on the context of the tissue being regenerated to increase the rate of cell growth possible in tissue formation. Because of its versatility, miR-506 can be used in various tissues to alter their reproduction rates, making it the ideal type of miRNA to be used in such a context.

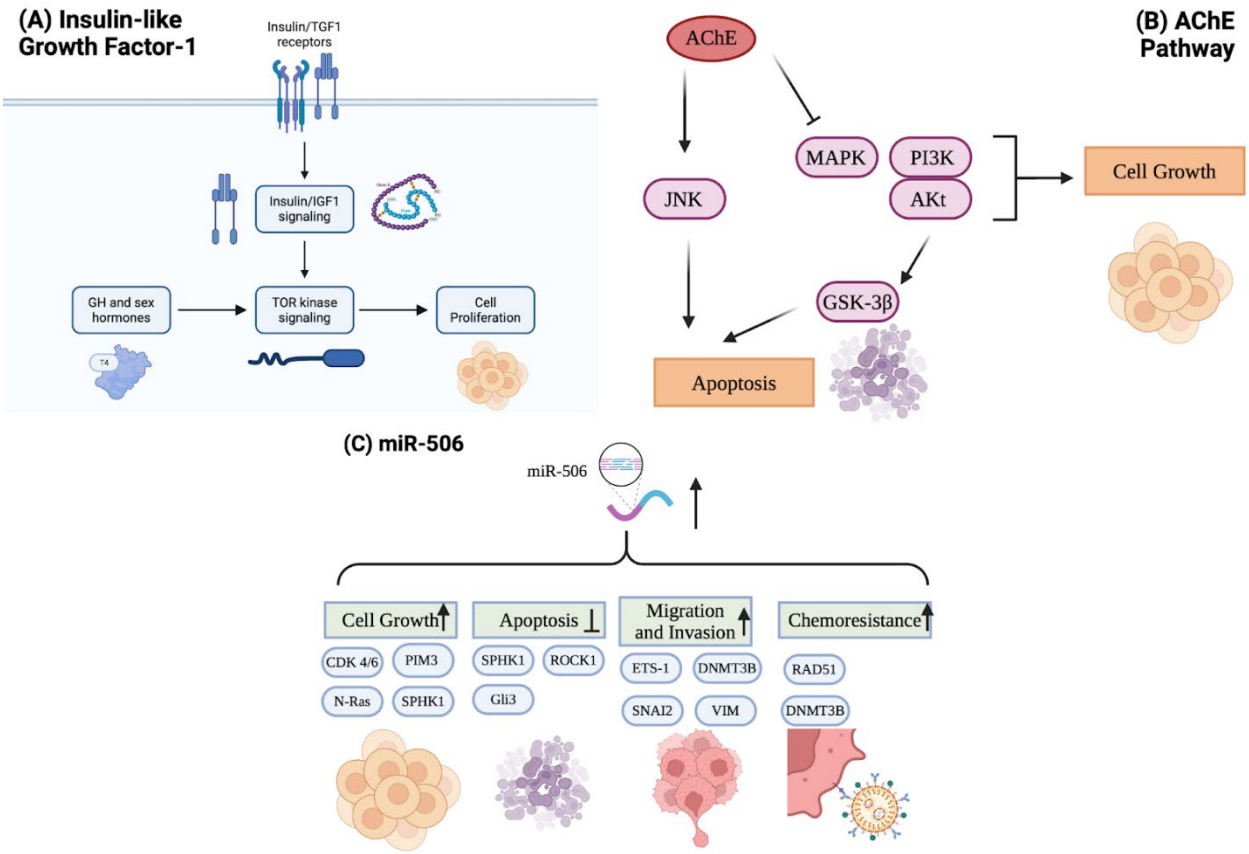

Figure 1. Potential methods to leverage cancer mutation pathways. Three pathways are depicted. (A) The Insulin-like growth factor signaling pathway results in cell proliferation. (B) The Acetylcholinesterase pathway results in the promotion of apoptosis and the inhibition of cellular growth. (C) miR-506 is shown to act as an oncogene, which, when upregulated, will cause an increase in cell growth, migration/invasion, and chemoresistance, while inhibiting apoptosis. Created with BioRender.com.

Transforming Growth Factor Beta Type I

Transforming growth factor beta type I (TGF-beta) is a cytokine known for its ability to inhibit epithelial cell proliferation as a tumor suppressor, with mutations abrogating the TGF-beta signal transduction pathway found in many different gastrointestinal cancers involving epithelial tissue. In cancers outside the gastrointestinal zone, a resistance to the growth-inhibitory effects of TGF-beta is acquired, known as part of a signaling switch where the original function of TGF-beta, growth inhibition, is reversed. This leads to the use of TGF-beta as a growth-promoting factor in epithelial cells. Studies also suggest that the ability of a cell to use TGF-beta as a growth-promoting/invasive cytokine is a result of different nuclear and cellular factors, one of which includes the absence or disruption of cyclin-dependent kinase inhibitors. This imbalance in cell cycle regulators is the key element determining whether a cell's response to TGF-beta is growth-stimulatory or growth-inhibitory and allowing for TGF-beta signaling to possess a dual nature (Bachman & Park, 2005). In this way, the absence or disruption of cyclin-dependent kinase inhibitors can be induced to allow a cell to respond to TGF-beta as growth-stimulatory to encourage cell proliferation in epithelial cells outside the gastrointestinal region or TGF-beta can be either downregulated in epithelial cells inside the gastrointestinal region to prevent it from functioning as growth inhibitory, both of which promote cell proliferation in regenerative tissues. This cytokine is particularly interesting in that it can be manipulated in different ways in epithelial cells depending on what part

of the body is being focused on, as upregulation for regenerative purposes outside the gastrointestinal region will result in growth stimulation. In contrast, downregulation inside the gastrointestinal region will result in a lack of growth inhibition. Manipulation of TGF-beta can be tested both inside and outside the gastrointestinal region and is also used for research on the effect of the cell-signaling pathways on the cell growth cycle in cases outside the gastrointestinal region.

Metabolic Pathways

What are they?

Metabolic pathways are a linked series of chemical reactions occurring within the cell. In cancer cells, metabolic pathways are often changed to alter the activity of nutrient uptake in cancer cells, allowing them to grow and divide faster than normal. These metabolic pathways can be reprogrammed in normal cells to resemble those of cancer cells, allowing cells to take in nutrients faster and be more readily available for cellular processes, causing these processes, such as reproduction, to occur faster.

How Can They Be Leveraged?

Glucose Metabolic Reprogramming

The PI3K/AKT/mTOR pathway is a prominent signaling pathway that imports nutrients into the cell and is commonly involved in the regulation of cancer cell growth, metabolism, survival, and proliferation. It is antagonized by the tumor suppressor PTEN, leading to the inhibition of downstream proteins PDK1, AKT1, and mTOR (Yang et al., 2019). Typically, the activation of this pathway induces glycolytic flux upon the stimulation by growth factors such as insulin. However, in cancer cells, this pathway is frequently altered by mutations in components of the PI3K complex, or by the hyperactivation of RTKS in combination with the observed loss of PTEN. This can lead to a dysregulation of glucose metabolism, and thus an expression of glycolytic enzymes and glucose transporters. The dysregulation of glycolytic enzymes results in the activation of c-MYC through single nucleotide polymorphisms and chromosomal translocations in oncogenes found in cancer cells. This ultimately leads to energy production and anabolic processes, such as lactic acid production and NADPH production, even in the absence of growth factor stimulation by the expression of key glycolytic enzymes (Pal et al., 2022). This pathway is exciting because of its ability to allow for energetic processes to occur in the absence of the growth factor, thereby allowing for increased cell survival and proliferation, necessary when considering the conditions of regenerative medicine processes–likely those where normal cells are not able to survive due to vulnerability to malfunction in growth factors properly, a risk eliminated by the manipulation of the PI3K/AKT/mTOR pathway. This pathway can be manipulated in normal cells by reducing the amount of PTEN present and by using CRISPR to alter components of the PI3K complex or activation of the RTKS by single nucleotide polymorphismsIt can then be returned to normal by bringing up levels of PTEN and by undoing the single nucleotide polymorphisms brought about by CRISPR. Additionally, mutp53, found in cancerous cells, can be added to cells to alter the PI3K/AKT/mTOR metabolic pathway by inhibiting AMPK and upregulating glucose transporters GLUT1, GLUT3, and GLUT4. This would allow for proliferation despite conditions lacking energy, allowing cells to transport more glucose in and out of the cell to help with proliferation and reproduction.

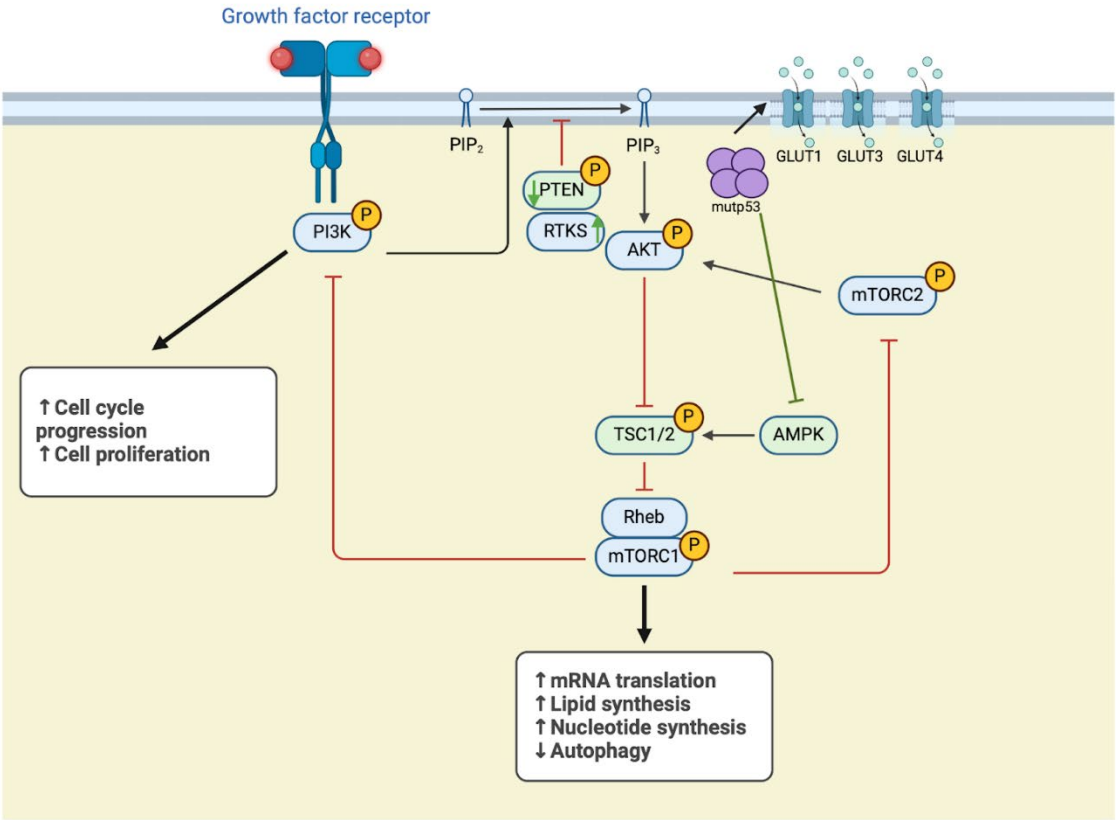

Figure 2. The PI3K/AKT/mTOR pathway can be leveraged for cell proliferation. This pathway can result in increased mRNA translation, lipid synthesis, nucleotide synthesis, and autophagy. The inhibition of the Rheb/mTORC1 also inhibits the production of mTORC2 and PI3K, which inhibits cell cycle progression and cell proliferation. However, in cancer, mutp53 downregulates PTEN, upregulates RTKS, and inhibits AMPK, which results in the lack of formation of TSC1/2 and thus leads to decreased mRNA translation, lipid synthesis, and nucleotide synthesis, while increasing autophagy and increasing cell cycle progression and cell proliferation. mutp53 also upregulates glucose transporters 1, 3, and 4. Created with BioRender.com.

Dysregulations in Amino Acid Metabolism

Amino acid metabolism is closely connected with the glycolytic pathway as amino acid pools can generate various components of the TCA cycle used in glycolysis via anaplerotic pathways, and metabolites such as lipids, glucose, and precursors of purines and pyrimidines. In cancer cells, amino acid pools are utilized during points for glucose deprivation to fulfill the energy requirements needed for glycolysis. The primary amino acid crucial for cancer proliferation is glutamine, as it provides both carbon and nitrogen that help support homeostasis and biosynthesis in cancer cells (Wei et al., 2021). To maintain high glutamine pools, cancer cells upregulate expression of glutamine transporters such as Alanine/Serine/Cysteine/Threonine Transporter 2 (ASCT2; SLC1A5). Additionally, glutamine synthetase (GS) is overexpressed to convert glutamate to glutamine. At the same time, GS produced by glial cells also converts ammonia to glutamine in glioblastoma tissues, allowing for an alternate source of glutamine to be accessed (Marin-Valencia et al., 2012). The process by which glutamine is used to produce glutamate is called glutaminolysis, which is upregulated in cancer cells due to the increased activity of the glutaminase 1 (GLS1) enzyme, which has been linked to the overexpression of TGF-beta (Pal et al., 2022). Thus, TGF-beta expression can be increased in cells to increase the activity of GLS1. This allows for increased conversion of glutamate to glutamine, providing more sources of glutamine to support

both homeostasis and biosynthesis in cells. To gather additional sources of glutamine, the expression of glutamine transporters can be increased along with the upregulation of GS. In addition, the TCA cycle can be altered to resort to using amino acid pools in times of low glucose. This will allow cells to become more resilient when grown, as cells would have easy access to another form of energy that would enable them to both maintain homeostasis while proliferating during deprivation of glucose, leading them to become more resistant to change and thus, better able to be used when regenerating tissues.

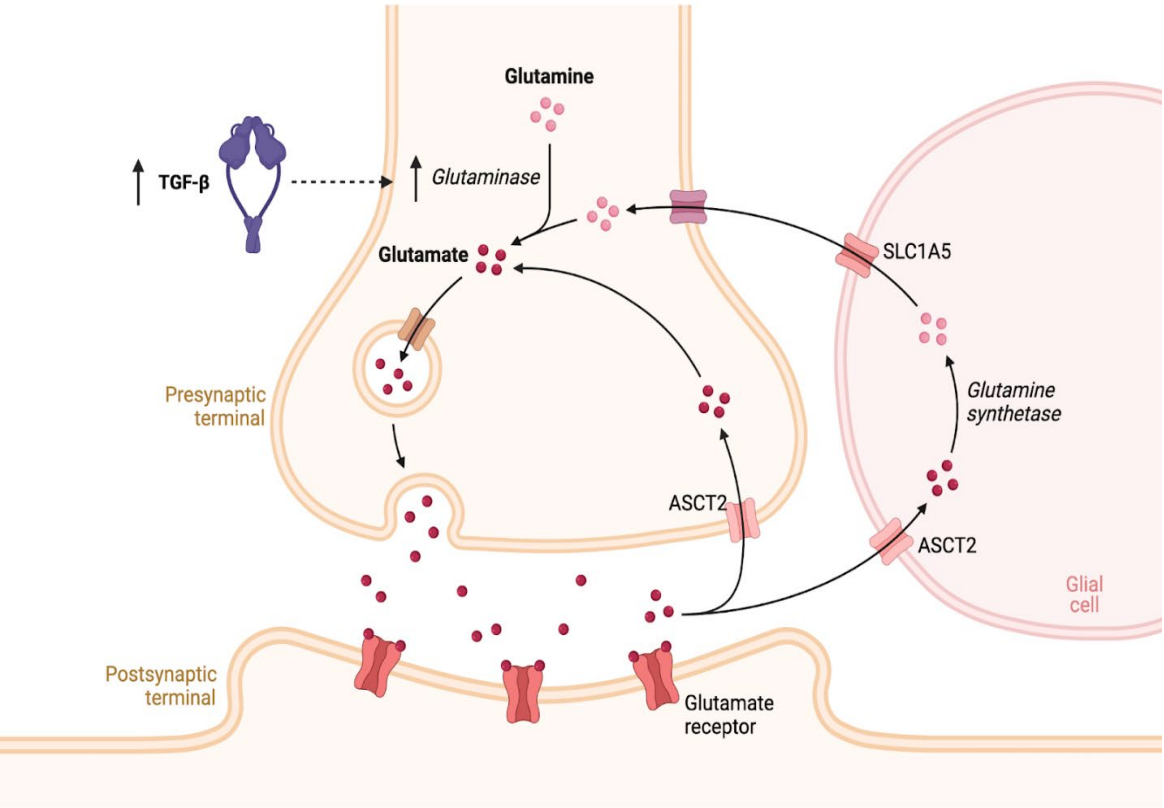

Figure 3. Dysregulation in amino acid metabolism through the glutamine/glutamate pathway can result in increased TGF-beta expression. In the glutamine/glutamate pathway, glutamate is transported to and from cells by glutamate transporters such as ASCT2 and SLC1A5. In glial cells, glutamate is converted to glutamine by glutamine synthetase, where it is then transported to neuronal cells and converted back into glutamate by glutaminase, the presence of which can be increased by increasing TGF-beta and released out of the cell for other glial cells to transport. Created with BioRender.com.

Mitochondrial Function

What Is It?

The mitochondria are present in all eukaryotic cells and are where cells get most of their energy and power. Energy is produced in the form of ATP in the mitochondrial membranes through the process of oxidative phosphorylation. By increasing the rate at which mitochondria can function, the rate of cellular processes, including reproduction, can be increased because there will be more available energy for the cell to use at any one given time. There are many different ways to improve mitochondrial function, including through optimizing mtDNA

and providing optimal nutrients that limit oxygen and high energy electron leakage in the ETC while protecting from oxidative stress and facilitating mitochondrial ATP production.

How Can They Be Leveraged?

mtDNA

Mitochondrial DNA, also known as mtDNA, is composed of the following genes: 2 rRNAs, 22 tRNAs and 13 of 83 genes for respiratory chain subunits (MT-ATP6, MT-ATP8, MT-CO1, MT-CO2, MT-CO3, MT-CYB, MT-ND1, MT-ND2, MT-ND3, MT-ND4, MT-ND4L, MT-ND5, MT-ND6). This DNA is required to produce key catalytic subunits of the mitochondrial respiratory chain complexes, causing it to be essential to the production of ATP by oxidative phosphorylation. mtDNA also encodes three short peptides, gau proteins, humanin, and MOTS-c, all of which have important biological functions (Rusecka et al., 2018). In order to optimize mitochondrial function, however, the genes in the mtDNA itself can be optimized by altering the genes specific to the regulation of oxidative phosphorylation, cellular metabolism, and apoptosis (Atig et al., 2009). In modifying these genes, cells will be able to increase the amount of oxidative phosphorylation occurring at one time, while also preventing apoptosis of mitochondria and decreasing the amount of oxidative stress by increasing the amount of stress control possible through the optimization of the mtDNA coding for a reduction in stress control.

Nutrients

Another way to optimize mitochondrial function is to increase mitochondrial metabolism. One way to do this is by increasing nutrient availability. Cells can be provided with nutrients that stimulate mitochondrial function by preventing oxidative stress and facilitating ATP production. These nutrients include CoQ10, α-Lipoic Acid + Acetyl-l-Carnitine, Reseveratol, N-Acetyl Cysteine (NAC), and Vitamin E. CoQ10 carries high-energy electrons through the electron transport channel found in the mitochondria. A deficiency of this nutrient would result in decreased ATP production and increased electron loss, which would cause oxidative damage since there would be fewer high-energy electrons carried through the ETC with more being lost to the environment, and thus, a lack of proton gradient allowing for ATP production (Pizzorno, 2014). α-Lipoic Acid + Acetyl-l-Carnitine has been found to reduce oxidant production and, thus, helps to reduce oxidative stress created when mitochondria produce reactive oxygen species, leading to improved mitochondrial function (McMackin et al., 2007). Resveratrol can increase mitochondrial ATP production, protect from ROS, and up-regulate sirtuin 1 by inducing genes for oxidative phosphorylation and mitochondrial biogenesis through an RS-mediated decrease in PGC-1 alpha acetylation and an increase in the activity of PGC-1alpha, given that RSV is a known activator of SIRT1 and the protein deacetylase (Lagouge et al., 2006). NAC increases intracellular glutathione, which is pumped into the mitochondria, protecting the mitochondria from oxidative damage as it prevents the mitochondrial toxicity if C26:0 by replenishing mtGSH and protecting cells from additional oxidative stress and death (Zhou et al., 2021). Vitamin E is an antioxidant that protects mitochondria from oxidative stress by preventing lipid peroxidation chain reactions in cellular membranes through interference with the propagation of lipid radicals (Ryan et al., 2010). Increasing the availability of these nutrients in cells and their growth plates can result in increased mitochondrial function through a decrease in oxidative stress, thus increasing ATP production by reducing the amount of stress incurred on the mitochondria. An increased mitochondrial function through an increased production of ATP will result in increased availability of free energy that can be used to perform cellular processes such as cellular reproduction, meaning that cells can divide faster and in more significant quantities as they have the means to do so.

Future Perspectives and Challenges

When considering broader adoption, many challenges and limitations arise when considering the implications of leveraging cancerous properties for tissue regeneration. The most prevalent among these limitations is mutating the cell from its baseline to have cancerous properties and returning it to its normal state. To have this process occur, gene editing technology such as CRISPR must be used to change the genetic makeup of cells to allow them to have cancer-like properties, which are only gained in natural cells through mutated genes. It must then return cells to their baseline after tissue regeneration. The biggest concern with editing the cells is the decreased stability of the cells. However, given that cancer cells tend to be more resilient than other types of cells, there likely will be a higher rate of success with these cells, as the two rounds of opposite genetic editing will not take as much of a toll on the cells as it would normal cells who don't have the properties that give cancer the ability to reproduce and spread the way it does.

Despite the risks, this technology holds immense potential to impact the medical field positively. Leveraging biological pathways to enhance the rate of cellular regeneration could be used to create artificial tissues for individual tissue transplant surgeries so the tissue can be similar in molecular makeup to the area it is being transplanted to instead of the molecular differences that come with transplanting tissues from a different location on the body, such as the thigh. Additionally, increased rates of cellular reproduction can be used to create organs for a person from the existing tissues in their body, as the tissue will grow rapidly enough to make an entire organ in a shorter amount of time than is already possible. Doing this will decrease the amount of deaths caused by a lack of functioning organs every year and will reduce the need for organ transplants.

There are multiple different ways that cancer pathways can be inserted into normal cells. Gene editing technology, like CRISPR, can be used to edit specific parts of genes through up or downregulating certain genes that promote faster reproduction rates. This would be one of the quickest and easiest ways to edit the different cellular genomes to increase the reproductive success rate, and this process would likely have a generally high success rate due to the genetic makeup of cancer cells. Mutating cells in this way would allow cells to reproduce at a faster rate, but could also be done on a variety of different cells with no restriction to only one type of cell tissue. Faster rates of reproduction would allow for tissues to rebuild themselves faster, which would be extremely useful when healing more extensive wounds, or could also be used to generate and grow new tissue from small amounts of previously existing tissue, which would be particularly helpful in tissue grafting and organ transplantation as well. This technology could be used in various ways, and application would depend on the means of regenerative technology. Cells themselves could be modified to grow 2D layers of tissues, which could then be layered upon each other to form 3D structures that would be useful inside the body for both tissue and organ transplantation. This process could also be applied to individual cells, which, in tandem with layers of tissue, could be used to test out different types of medications for faster, more ethical testing purposes. Additionally, if the cells in question were blood cells, the cells could be cultivated outside the body and injected directly into a patient's bloodstream.

Conclusion

Cancer cells have long been thought to be only harmful, but in reality, they have the immense potential to be used in medicinal technology to the benefit of society. Their processes have evolved to have faster rates of reproduction, something that is key when thinking about tissue regeneration and medicinal testing of drugs or medications. Cancer cells achieve enhanced reproduction in two specific ways: first, they have genes linked to reproductive processes that they ether upregulate or downregulate based on function, and second, they have rewired metabolic pathways that allow them to get nutrients to the cell faster, which enables them to conduct more cellular processes efficiently. Besides cancer mutations, cellular replication rates can be increased by

increasing mitochondrial function to increase the production of ATP in the cell. By implementing these edited processes in normal cells, the reproduction and replication rates of the cells can be used, which will be beneficial in medicine hinging upon cellular reproduction, such as tissue regeneration, organ restoration, and medicinal testing.

Cells can be changed using cancerous properties in three different ways. First, genes can be upregulated or downregulated to speed up the reproduction rate. Second, metabolic pathways of different cells can be rewired to increase the amount of nutrients a cell has access to at any given time, allowing it to reproduce faster. Lastly, the mitochondrial DNA of cells can be altered, in combination with an increase of nutrients that stimulate mitochondrial function, to allow the cell to be more efficient in terms of mitochondrial function. These cells can be used in different parts of the body to optimize function and can also be used to replace failing organs or tissues in the body. They can also be transported to places wherein the body lacks the right amount of tissue. They can be used in medicinal testing to observe the effects of different drugs before testing occurs.

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Biorender.com was used in the creation of figures and schematics.

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