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Chapter- 5

Epigenomic and other important functions of diet and nutrition in Mesenchymal Stem Cells: A brief review

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Abstract:

Adult stem cells stand for the regenerative ability of organisms during their lifespan. One characteristic feature of healthy aging is the sustainment of healthy SC populations capable of replenishing organs and physiological systems. The native environment of stem cells is known as the niche. It comprises the nutritional surroundings and is crucial to sustain the quality and quantity of stem cells available for renewal and regeneration. It is considered mainly that stem cells have unique metabolism and restricted nutrient requirements compared to completely differentiated cells. Nutrients play a significant role in stem cell physiology because many metabolites derived from nutrients discharged during the catabolic process can affect chromatin remodelling, epigenetic changes, and modulation of gene expression. Nutrient requirements differ throughout the lifespan and are altered by factors like individual health, physiological states including pregnancy, disease, sex, age, and during healing from injury. Even if present nutrition guidance mainly focuses on healthy populations and averting nutritional insufficiency diseases, there are growing efforts to demonstrate food-based and nutrient-based suggestions depending on decreasing chronic disease. Understanding the dynamics of stem cell nutritional needs throughout the life span, including the role of nutrition in extending biological age by blunting biological systems decay, is fundamental to establishing food and nutrient guidance for chronic disease reduction and health maintenance.

Introduction:

Nutrients, which include carbohydrates, proteins, lipids, minerals, and vitamins, are substances in food and are necessary for biological activity in organisms. During metabolism, the nutrients, after conversion into smaller molecules inside the body, are exploited in several life-sustaining chemical reactions. Metabolism constitutes catabolism and anabolism. In catabolism, the breakdown of food or fuel to obtain energy occurs, whereas the reactions in which larger molecules are produced from smaller ones are known as anabolism. Anabolic reactions utilize the energy generated in catabolic reactions. Hence, the cooperative control of both processes is essential to sustain life (Tadokoro and Hirao, 2022).

Stem cells (SCs) are undifferentiated cells with the ability by cell division to generate various cell types in an organism. They possess unique metabolic features in contrast to differentiated cells (Cerletti et al., 2012; Moussaieff et al., 2015; Baksh et al., 2020), and they can exclusively

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sustain their undifferentiated state throughout their entire life while making offspring cells devoted to differentiation in response to specific requirements to maintain tissue homeostasis. Several evidences support the idea that stem cells (SCs) are important in coordinating our body's response to nutrients, mainly because of their key role in tissue homeostasis. To accomplish this, tissue SCs, besides utilizing nutrients for their metabolic requirements, also adjust their functions, such as self-renewal, autophagy, or differentiation, to the metabolic environment and availability of nutrients (Cerletti et al., 2012; Yilmaz et al., 2012; Rafalski et al., 2012). Conversely, their relatively long lifespan, which is crucial to carry out their function in tissue turnover, holds the back of the coin of continually being exposed to important environmental factors like diet and gradually accumulating cell damage at the genetic and epigenetic level, with considerable effects on gene and protein expression as well as on molecular pathways (Blokzijl et al., 2016; Novak et al., 2021; Mondal et al., 2024).

Nutrients are usually essential in SC physiology because of the ability of various nutrient-derived metabolites, produced during the catabolic process, to trigger chromatin reshaping, epigenetic modifications, and modulation of gene expression (Lu et al., 2018). Nutrients also act as donors for moieties engaged in post-translational modifications. For example, in the hexosamine biosynthetic pathway (HBP), uridine diphosphate GlcNAc (UDP-GlcNAc) results in the O-GlcNAcylation of serine and threonine residues embedded in cytoplasmic, mitochondrial and nuclear proteins. In SCs, these post-translational changes have been found to cause linkages between the availability of glucose and nutrients with the epigenetic control of cell fate determination and differentiation (Sun et al., 2016).

Both embryonic and adult SCs have the potential to provide tissues with new lineage of cells throughout their entire life. This new lineage of cells may divide symmetrically or asymmetrically, resulting in either SC self-renewal or differentiation. Besides several other factors, nutrients play a vital role in SC specification, differentiation, and performance, thus extending their effects on aging and disease. Nutrients act directly on SCs or indirectly by controlling the SC niche (non-autonomously). In addition, nutrients can regulate the production of hormones, which can manipulate the nature of SCs and their niche. These direct and indirect stimuli result in the activation of signalling pathways, modification in metabolism, and changes in gene expression in SCs. In this way, the dietary input is converted into fate decisions in SCs (Puca et al., 2022).

For SCs, the primary molecular mechanism linking diet and function is mediated by the AMPK-mTOR-SIRT1 pathway. Fasting or exercise-induced low cellular ATP levels trigger the phosphorylation of AMP-activated protein kinase (AMPK) by the serine-threonine kinase liver kinase B1 (LKB1). This results in direct or indirect modulation of enzymes involved in glucose (Theret et al., 2017) and lipid metabolism (Wang et al., 2018). It also modulates the mTOR pathway, which regulates proteostasis and cell growth (Shackelford et al., 2009). The target proteins of AMPK include the proteins controlling cell polarity, apoptosis (through direct phosphorylation of p53), cell proliferation (cyclin D1) (Shackelford et al., 2009), differentiation (Sarikhani et al., 2020), response to hypoxia (HIF1) and autophagy (Mihaylova et al., 2011).

These proteins have also been found to modify SC fate (Shackelford et al., 2009; Chung et al., 2019). In addition, AMPK enhances cellular NAD^+, which triggers the activation of the NAD-dependent histone deacetylase SIRT1, influencing gene expression (Dai et al., 2020), protein synthesis, and SC self-renewal (Igarashi and Guarente, 2016).

Nutrition has appeared as a chief regulator of the epigenome and gene expression. As a result, nutrition and diet can affect cell metabolism and health (Hahn et al., 2017). This capacity of nutrition and diet is explained by the fact that many metabolites either directly bind to chromatin or indirectly modulate chromatin-modifying enzymes. In SCs, epigenetic changes of DNA and DNA-associated histones determine their function and fate decisions. Hence, inputs from the diet cause modification in chromatin structure and expression of genes (Van Winkle and Ryznar, 2019; Bar-El Dadon and Reifen, 2017; Afarideh et al., 2021; Shyamasundar et al., 2013) in embryonic and adult SCs. This modification, in turn, affects several processes in humans, including embryonic development, cell differentiation, determination of cell fate, aging, immune function, and oncogenic transformation (Chen, 2019; Hernández-Saavedra et al., 2017). These play important roles in closely correlating SC functions, nutrition, metabolism, and epigenetics to each other (Reid et al., 2017).

Nutrients derived from diet, after digestion, produce simple metabolites and can be uptaken by SCs. These biomolecules can act as precursors of substrates or cofactors required by chromatin-modifying enzymes. Sometimes, these enzymes can move to the nucleus and, in association with specific cofactors, bring about chromatin modifications (Boukouriset al., 2016). Epigenetic modifications induced by nutrients result in modifications of both histones (acylation, acetylation, ADP-ribosylation, glycosylation, glycation, methylation, phosphorylation, hydroxylation, and ubiquitylation) and DNA (glycation and methylation). These modifications may be achieved through enzymatic or non-enzymatic reactions (Dai et al., 2020).

Mesenchymal Stem Cells (MSCs) and their Characteristics:

MSCs undergo mitotic divisions. One of its daughter cells remains as a stem cell while the other one differentiates into a mature cell, and only small numbers of these mature cells can be seen in mature organs and tissues in the stem cell niche (Aliborzi et al., 2016). In 1966, MSCs were discovered as fibroblast-like cells within the bone marrow (Aliborzi et al., 2015). Since then, the presence of MSCs has been confirmed in various adult tissues like endometrium (Ghobadi et al., 2018), adipose tissue (Kamali-Sarvestani et al., 2018), intestine (Mani et al., 2023), dental pulp (Zare et al., 2019), and Wharton's jelly (Nazempour et al., 2020). Among various types of MSCs, intestinal stem cells (ISCs) possess a crucial role in the nutritional milieu. They are present in the crypts and do not come in direct contact with intestinal content. On the other hand, differentiated gut cells are present at the villi and come in direct contact with the intestinal lumen. They provide mature cell types of the intestinal epithelium throughout adult life (Barker et al., 2007). Inside intestine, adjacent to ISCs reside a collection of functionally differentiated cells including enterocytes, Paneth and goblet cells. These cells inhabit the intestinal epithelium and play a critical role in the nutritional environment.

The principal function of ISCs is to act as gut regenerative machinery. They undergo continuous division to reinstate their own population and create subtypes of differentiated epithelial cells. The nutritional conditions can modulate the production of secretory lineages such as Paneth cells, enterocytes, and ISCs (Alonso et al., 2018). Specific dietary exposure and fasting have been established to reduce the population of ISCs and their function (Alonso et al., 2018). Two populations of ISCs have been identified, namely Lgr5+ and Lgr4+. Lgr5+ is associated with regular cell renewal, while Lgr4+ is responsible for tissue regeneration. The quiescent Lgr4+ ISCs have the potential to be stimulated in response to injury (Wang et al., 2021). Optimal food intake can control and trigger symmetric divisions of ISCs (O'Brien et al., 2011).

The MSCs can differentiate into one or more types of full-grown cells. This property of stem cells is called "developmental plasticity," and different stem cells have distinct potency levels (Mehrabani et al., 2019). Under both pathologic and physiologic conditions, MSCs can sustain tissue regeneration. In a specialized and dynamic microenvironment along with a separate design as stem cells niche SCs play a major role in tissue homeostasis. These cells have immune-modulating properties due to the low expression of class I MHC, CD40, CD80, and CD86 and the absence of class II MHC expression (Hashemi et al., 2019).

The immune modulating activity of MSCs is attributed to their interaction with immune cells like neutrophils, T and B cells, natural killer cells (NKs), dendritic cells (DCs), and macrophages (Mohammadzadeh et al., 2022). They can be used as drug carriers and can be tracked by MRI (Mehrabani et al., 2022). Their application has also been traced to tissue engineering (Fard et al., 2018). Exosomes or extracellular vesicles (EVs) are the active constituents of paracrine secretion of MSCs. These exosomes are utilized in the management of various diseases (Khajehahmadi et al., 2016). Exosomes are used in the treatment of brain diseases as they can cross the blood-brain barrier (BBB) and enter the CNS (Payehdar et al., 2017). MicroRNAs (miRNAs) are naturally packaged into exosomes in MSCs. This feature of MSCs is successfully applied in the packaging of exogenous therapeutic miRNAs (Jahromi et al., 2017).

Mesenchymal Stem Cells and Nutrition:

Lifestyle and diet are key factors that influence health and vulnerability to diseases. In the stem cell niche, these two factors affect the quality and quantity of stem cells available for renewal, regeneration, and physiological reinstatement as a trademark of health (Stover et al., 2022). Deficiencies in the nutritional environment can modify the niche of stem cells and/or interaction between stem cells and niche, leading to age-associated modulations of the proliferation of stem cells and their functions. Stem cells have distinctive metabolism. Hence, their nutrient requirements are of immense importance. So, consideration of the nutritional requirements of stem cells throughout the life span, together with the involvement of nutrition in expanding biological age by minimizing biological systems degeneration, is key to determining food and nutrient guidance to reduce the occurrence of diseases and to retain the general health (Stover et al., 2022).

MSCs as the tissue precursor were demonstrated to be incredibly relevant for obesity during childhood and metabolic disease risk of skeletal muscle and adipose tissues (Gyllenhammer et al., 2023). In this circumstance, by controlling the stem cell niche, nutrients may directly or indirectly impact stem cells. Nutrients also control hormone production, which can modify the behaviour of the stem cells and their niche. These stimuli trigger the activation of signalling pathways in stem cells, modify their metabolism and gene expression, and transform the dietary input into fate decisions. Many stem cell features are controlled by nutrients, including balanced asymmetric/symmetric divisions, genome and epigenome integrity, gene expression, metabolism, autophagy, oxidative status, differentiation, self-renewal, and exhaustion. When there are adequate nutrients and growth factors, stem cells undergo proliferation. This tight regulation is achieved by “master regulators" like mTORC1, which can monitor nutrients and control stem cells' metabolism and fate (Rafalski et al., 2012).

Alternatively, intracellular metabolites like acetyl-CoA regulate epigenetic processes and metabolic pathways and link stem cell functions with diet and metabolism (Ghosh-Choudhary et al., 2020). For the fate determination of different stem cells, this link is very crucial, and self-renewal of stem cells can be brought about by modification of nutrients or calories (Novak et al., 2021). So, stem cells play a significant role in coordinating the body's response to nutrients because of their essential role in tissue homeostasis and health maintenance (Alvina et al., 2021). This feature of stem cells is achieved by using nutrients for their metabolic requirements and accomplishing many functions, like self-renewal, differentiation, or autophagy. Nutrient availability, metabolic environment, and diet-induced metabolic changes affect the fate of stem cells, lineage specification, and differentiation (Puca et al., 2022).

In this scenario, nutrients are vital in stem cell physiology because of the ability of many metabolites derived from nutrients released during catabolic processes to trigger chromatin reorganization, epigenetic alterations, and modulation of gene expression (Lu et al., 2021). At the same time, molecular mechanisms that sense nutrient availability regulate important self-renewal functions, protein synthesis, autophagy, and differentiation (Bjerkvig et al., 2005). The effect of diet on stem cells becomes more spectacular as stem cells have exceptional metabolic requirements which are changed depending on their developmental stages (Baksh et al., 2020). For precise activities of stem cells, stimulation of metabolic pathways is essential, making stem cells more explicitly dependent on nutrients compared to differentiated cells (Yilmaz et al., 2012). Stem cells have been reported to possess fewer reactive oxygen species (ROS) than differentiated cells. The total intracellular oxidation state and accumulation of ROS have been reported to be primarily influenced by nutrients and diet. They are believed to be key monitors of balance between differentiation and self-renewal (Smith et al., 2000).

Mesenchymal Stem Cells and Amino Acids:

Amino acids (AAs) are engaged in self-renewal, preservation of pluripotency, and differentiation capability of stem cells (Liu et al., 2019). Several essential AAs (EAAs) have been revealed to be crucial for the maintenance of MSCs (Taya et al., 2016), and their affluence was

demonstrated to enhance proliferation without disturbing the stemness (Nikolits et al., 2021). In the intestine, The Mammalian Target of Rapamycin Complex 1 (mTORC1) was reported to be a principal nutrient sensor, functioning as an essential controller of protein synthesis and growth, influencing the proliferation of stem cells and autophagy (Wang et al., 2021).

Restriction of amino acids and proteins in the diet has been described to alter stem cell fate. For instance, methionine deficiency has been shown to reduce the proliferation of ISCs (Saito et al., 2017). In *Drosophila*, in response to a reduction in methionine and the methionine-derived S-adenosyl methionine, the midgut mitosis in ISCs was shown to diminish. This inhibition of mitosis in ISCs was accomplished by regulating the protein synthesis and stimulating the Jak/STAT ligand Unpaired 3 (Upd3) (Obata et al., 2018). Stimulation of the JNK pathway enhances ISC differentiation, while ISC proliferation remains unaffected despite the attenuation of the Jak/STAT pathway (Zhang et al., 2017). Hence, methionine was revealed to regulate cell proliferation (Walvekar et al., 2018).

The function of leucine in carrying out the proliferation and differentiation of myoblasts through an mTORC1-MyoD cascade was reported (Dai et al., 2015). The mTOR has a vital function in various cellular processes, including cell growth, differentiation, and protein synthesis, via its role in regulating specific gene expression (Zhang et al., 2015). Arginine was shown to have a crucial function in the proliferation and renewal of ISCs and tissue regeneration (Hou et al., 2020). During the proliferation stage of myoblasts, glutamine has been revealed to be the second most used nutrient after glucose (Hosios et al., 2016), establishing their significant role in cell proliferation (Gaglio et al., 2009). The conditional EAA glutamine in diet supplementation was found to cause activation of ISCs, which includes an increase in total intestinal cell numbers (Viitanen, 2019). Dietary glutamate activates ISC proliferation and growth via calcium signaling (Deng et al., 2015).

Mesenchymal Stem Cells and Fatty Acids:

Fatty acids (FAs) are another class of molecules derived from nutrients and are crucial for stem cell physiology. It is confirmed by the presence of a particular lipidome signature in MSCs, performing a significant function in self-renewal and quiescence, asymmetric-symmetric division, differentiation, determination of cell fate of MSCs, and cell-to-niche interaction (Clémot et al., 2020). A high-fat diet (HFD) can trigger modifications in intestinal structure and function (Obniski et al., 2018) by modifying the regulation of ISC activity. It was described that some particular fatty acids, including oleic acid and palmitic acid, interact directly with the ISCs and stimulate peroxisome proliferator-activated receptor delta (PPAR-δ) exclusively in ISCs and progenitor cells to increase their stemness (Beyaz et al., 2016).

In stem cells, the presence of excellent coordination between the synthesis of fatty acids and oxidation of fatty acids is necessary, and damage or removal of one or the other can lead to stem cell retardation (Clémot et al., 2020). High-fat diets have been shown to enhance ISC proliferation and self-renewal while reducing Paneth cell number and resulting in an increased risk of intestinal hyperplasia (Wang et al., 2021). It was found that a high-fat western-style diet

in mice resulted in transcriptional reprogramming in both Lgr4+ and Lgr5+ ISCs populations, mutations in stem cells, and nutrient-triggered modifications in stem cell populations, which are in line with a carcinogenesis event (Li et al., 2019). HFD-induced stress causes activation of the JNK pathway, and this pathway leads to Upd3 ligand secretion and activation of ISC proliferation (Richards et al., 2016).

In *Drosophila*, short-chain fatty acids derived from microbiota were reported to control carbohydrate and lipid metabolism to maintain ISCs (Koh et al., 2016). In *Drosophila*, it was demonstrated that high-cholesterol diets, by changing the δ-ligand and Notch stability in the endoplasmic reticulum, can alter ISC cell differentiation (Obniski et al., 2018).

Mesenchymal Stem Cells and Minerals:

Impaired dietary intake of calcium at early stages of life might alter the adipogenic differentiation capability of MSCs from male offspring, with considerable expressions on the Wnt/β-catenin signalling pathway to exacerbate high-fat diet-induced obesity in adulthood (Li et al., 2022). This adipogenic differentiation is controlled by coordinating a complex network of several signalling pathways, which include SIRT1/SIRT2, JAK2/STAT3, TGF-β/BMP, Wnt/β-catenin, ERK1/ERK2, and RHO family GTPase (Porro et al., 2021). Stimulation of Wnt/β-catenin signaling can additionally prevent adipogenic differentiation and trigger osteogenic differentiation with the help of endogenous regulatory genes including Wnt1, Wnt10a, Wnt10b, Wnt5a, CTNNB1, Axin2, Gsk3β, and TGF7L2 (Matsushita., and Dzau, 2017). This differentiation capability was considerably decreased with age (Matsushita and Dzau, 2017). Hence, the nutritional posture and exposure to unfavorable factors during pregnancy and during lactation have a significant function in the differentiation ability of MSCs to influence later metabolic troubles in adulthood (Zhang et al., 2020). The Ca^{2+} produced in the culture medium was reported to have osteo-inductive features to support the osteogenic differentiation of MSCs (Chen et al., 2015).

Mesenchymal Stem Cells and Energy:

It was demonstrated that changes in energy sources can affect stem cell differentiation through glycolysis, the TCA cycle, as well as alterations in the generation of ROS (Burgess et al., 2014). ISCs were reported to have vigorous responses to intake of energy, including caloric constraint, fasting, and a variety of energy sources resulting from ketogenic, high carbohydrate, or high-fat diets (Wang et al., 2021). Under this circumstance, energy has been illustrated as the Lkb1/AMPK triggered kinase pathway to operate as a metabolic checkpoint and principal regulator of stem cell proliferation and fate. This pathway is activated when mTORC1 signalling is suppressed in response to reduced level of ATP and ceased cell growth. So, it can be said that the complex relationship between LKB1-AMPK activity and mTORC1 can affect stem cell proliferation, self-renewal, and apoptosis (Wang et al., 2021; Das et al., 2023) since LKB1-AMPK signalling has an impact on Sirt1 and is triggered by fasting, caloric restriction and exercise which can influence the development of the ISCs and enhance the ability for tissue repair and regeneration (Igarashi., and Guarente, 2016).

Sirt1 functions as a NAD-dependent histone and nonhistone protein deacetylase and controls gene expression, metabolism, cell proliferation and differentiation. As level of Sirt1 declines with age and restored by dietary NAD, it can control the stem cell quantity (Igarashi et al., 2019). Ketogenic diets mimic low caloric states by increasing stem cell self renewal and tissue regeneration and minimizing the gradual loss of tissue functions during aging. However, diets with high fat and carbohydrate levels have opposing effects (Cheng et al., 2019). The intracrine ketone bodies can delineate the fate of ISCs and act as moderators of the pro-regenerative results of fasting. Diets containing high carbohydrates were reported to inhibit the formation of ketone bodies and diminish function, stemness, self-renewal, regenerative power, and epithelial homeostasis of ISCs by activating the formation of goblet cells and Paneth cells at the expense of enterocytes formation (Cheng et al., 2019).

ISCs were shown to monitor and respond differently to macronutrients and dietary energy sources. Ketogenic diets can enrich intestinal health since the increased production of ketone bodies influences the functions of Lgr5+ stem cells and intestinal epithelial homeostasis. Hindrance in the production of ketone bodies in Lgr5+ cells can hamper stemness by increasing the formation of Paneth and goblet cells. It is now established that the release of stem cell growth factors and Wnt ligands by Paneth cells can protect epithelial homeostasis (Cheng et al., 2019). Dietary supplementation with N-acetyl-Dglucosamine (GlcNAc) was sufficient to sustain ISC proliferation amid caloric prohibition independent of food intake (Igarashi et al., 2019). Diets with high sugar can cause modifications in intestinal structure and function and ISCs (Kapinova et al., 2018) through alterations in the control of ISC activity.

Conclusion:

Lifestyle and diet have significant effects on health and vulnerability to diseases. The nutritional requirements of stem cells and their function in quality and quantity are of immense significance for the replenishment of cells and the curative process in wounded tissues, as nutrients play a vital role in stem cell physiology because many nutrient-derived metabolites have genetic and epigenetic roles. Preserving stem cell populations for tissue renewal, regeneration, and restoration is one of the features of health posture. Depending on the participation of stem cells in tissue renewal and regeneration, demonstrating the nutritional needs in diseases, during recovery from trauma, and in the aging process must come into discussion for determining nutrient endorsements to reduce the occurrence of diseases and to progress the interpreting of the biological pathways and mechanisms that link nutritional requirements of stem cells with diseases and aging.

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