

Role of Processed Foods on Carcinogenesis

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Almost a 2 trillion industry, processed foods are amongst the most profitable in today's fast paced society¹. Limited research exists on the health effects of these foods due to the prevailing emphasis on reducing costs, increasing shelf lives, and enhancing flavors in food formulation, which has overshadowed the exploration of their potential impacts on health. This research paper will focus on the underlying mechanisms of cancer development related to the consumption of processed foods, specifically by highlighting the production of reactive oxygen species (ROS), which are unstable molecules containing oxygen that can easily damage cells. It will also discuss the role of natural antioxidants, phytochemicals, omega-3-fatty acids, fiber, and vitamins and minerals that have cancer preventive effects. Due to the presence of carcinogens like polycyclic aromatic hydrocarbons, acrylamide, heterocyclic amines, and nitrites and nitrates in processed foods, understanding how processed foods impact human health is necessary.

Introduction

Cancer is a complex disease characterized by uncontrolled cell division and has become a significant public health concern worldwide. With an alarming rise in mortality rates, from approximately 6 million deaths in 2000² to 10 million deaths in 2022 (World Health Organization, 2022), it has emerged as a formidable research challenge in the scientific community. Oxidative stress, which results from an imbalance between reactive oxygen species (ROS) and the body's antioxidant system, is a primary contributor to the development of cancer³. Excessive reactive oxygen species (ROS), which encompass highly reactive molecules such as hydroxyl radicals (OH⁻), superoxide radicals (O₂⁻), and hydrogen peroxide (H₂O₂), act as oxidants. These oxidants are predominantly produced due to dietary intake, particularly from processed foods with high carbohydrate, lipid, and animal-based protein content⁴. This is because processed foods directly or indirectly increase the number of ROS in the cells, while lowering the antioxidant response from the body⁵. Mitochondria, being a major cell organelle, is a significant source of these oxidants; to counterbalance the production of ROS and protect the cells from oxidative damage, cells also possess a robust antioxidant system. However, when the dietary intake results in the production of high levels of ROS, the body's antioxidant system may become overwhelmed, leading to oxidative stress. This representation can be seen in the figure below³.

This oxidative stress can lead to DNA damage, resulting in trans-version mutations that involve the switching of a purine base with a pyrimidine base or vice versa. These mutations primarily affect cancer-suppressing genes and can occur through processes such as base oxidation or strand breaks⁶.

Hydroxyl radicals, in particular, are responsible for causing approximately 60-70% of total cellular DNA damage⁷. Hydroxyl radicals inflict damage to the deoxyribose sugar, a crucial component of DNA structure, by selectively abstracting a hydrogen atom from within the gene. This hydrogen abstraction leads to the formation of mutagenic lesions with potential consequences for gene function and integrity. This is just one of many ways excessive ROS damages the cells and leads to carcinogenic mutations. Other processes may include sugar-base cyclization, DNA-protein cross-links, and intra- and interstrand cross-links; all of which are linked to increased DNA strand breaks⁸. Diets including processed foods directly increase ROS generation while reducing the capability of the body's antioxidant system, thus inducing oxidative stress. It is also important to note that the influence of diet on carcinogenesis ranges from 10-80%⁹, values which are only expected to increase with growing demand of processed foods. Additionally, multiple prospective studies have linked a relation between increased ROS diets and oxidative DNA damage, hinting at an increased risk of carcinogenesis⁹⁻¹¹. The primary goal of this paper is to discuss how processed food intake generates reactive oxygen species that can increase the risk of oncogenesis, with a focus on foods that contain a significant number of nitrites and nitrate, additives, polyaromatic hydrocarbons, heterocyclic amines, trans fats, and sugar. Additionally, the paper also discusses how natural foods can significantly alter the cancer development mechanism.

Nitrates and Nitrites

Nitrites are biomolecules found in green vegetables that are crucial for blood pressure reduction, inhibition of platelet ag-

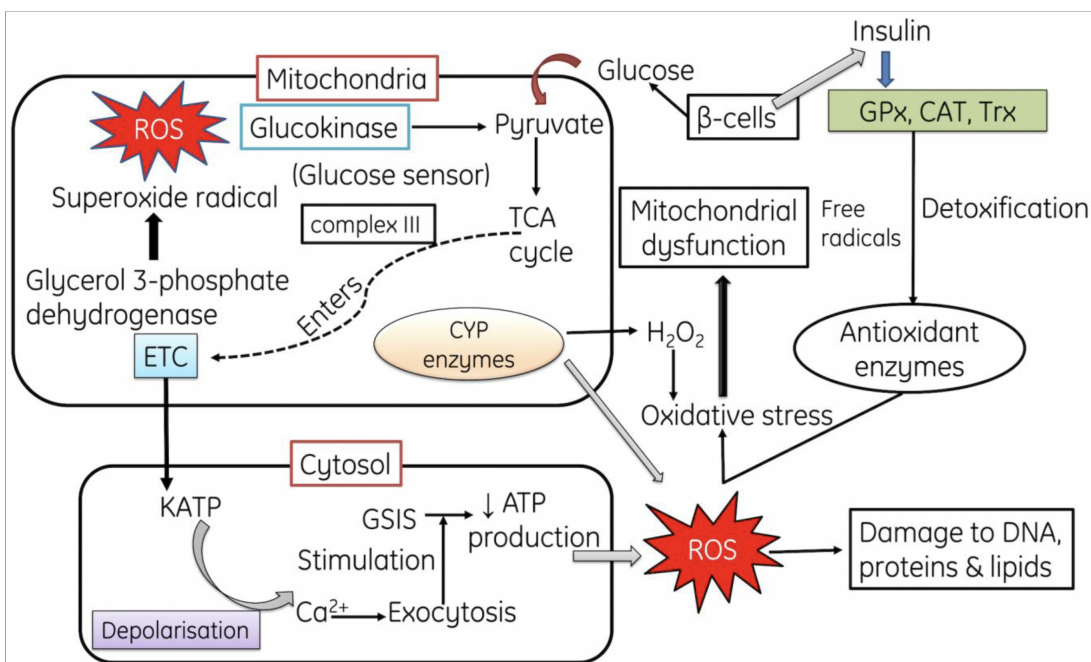


Fig. 1

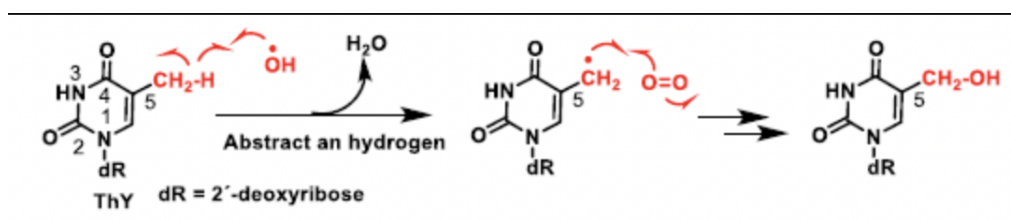


Fig. 2 (Juan et al., 2021)

gregation, vessel protection, antioxidant activity, and bacteria inhibition. In processed meats, nitrites and nitrates are added during the curing process to preserve flavor, smell, and shelf life. This process accounts for immense use of nitrites and nitrates in the processed food industry. The breakdown of nitrates into nitrites forms nitric oxide, which is essential for transporting blood and oxygen. Nitric oxide plays a vital role in the formation of nitroso myoglobin, which is responsible for the characteristic red color of myoglobin and metmyoglobin. This reaction occurs when nitric oxide reacts with the iron present in the heme group. Due to the oxygen supplied by the nitrites and nitrates, the desired red coloring of the meat is preserved for the consumer and the companies. Additionally, nitrites and nitrates bonded to sodium increase the shelf life of meats by inhibiting microbial growth and lipid oxidation¹². Excessive production of nitric oxide inhibits the growth of a pathogenic bacterium, specifically *Clostridium botulinum*, which is known to cause food poisoning¹³. The

nitric oxide inactivates the iron-sulfur enzyme needed for respiration, DNA repair, RNA modification, and gene expression in bacteria¹⁴, thus extending the shelf life of the meat. Nitrites also prevent oxidative breakdown of lipids¹⁵, which allows for greater shelf life and good odor. Since the meat quality is measured by the amount of fats, it is necessary for companies to inhibit lipid oxidation as much as possible. Added nitrites reduce lipid oxidation as nitrites bind to heme and non heme iron, which are responsible for initiating lipid oxidation, and thus the meat quality is maintained.

However, high consumption of processed meats -with high levels of nitrites and nitrates- corresponds to increased cancer risk¹⁶. At high temperatures (approximately 99°C-180°C), these additives react with nitrosating agents, nitric oxide, and secondary amine groups to form nitrosamines, a potent carcinogen. Under these conditions, the elevated concentration of nitrites and nitrates undergo thermal decomposition, forming nitric oxide which is a reactive nitrogen specie (RNS)¹⁷.

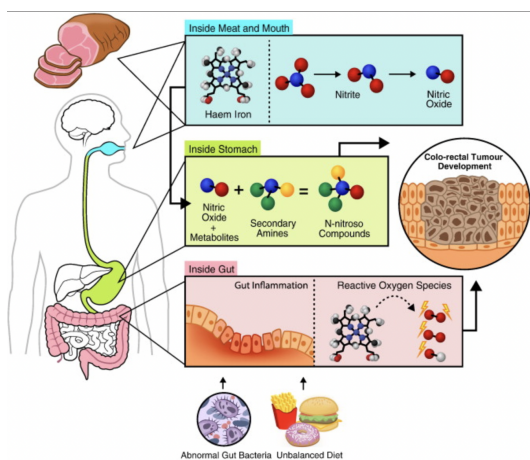


Fig. 3

While nitric oxide is a significant biomolecule that can even inhibit carcinogenesis¹⁸, its presence in greater concentration produces a greater number of nitrosamines which will harm the cell¹⁹ and can promote carcinogenesis by mechanisms like genotoxic effects, antiapoptotic effect, angiogenic effect, and metastatic effect¹⁸. Figure below illustrates this in depth²⁰. Meats are also composed of high-level proteins that facilitate reactions that form N-nitroso compounds with nitric oxides. As high temperatures supply extensive energy to the meat, the necessary activation energy is overcome in a shorter time span, thus accelerating the production of nitrosamines.

Oxidative stress, characterized by an imbalance between reactive oxygen species and antioxidants, is induced by these nitrosamines. Heme iron, which is present in meat, fish, and poultry, can react with polyunsaturated fatty acids in the colorectal and gut area which produces reactive oxygen species and disrupts normal cell division.²⁰

Additionally, a 2015 study illustrated below showcased a correlation between cancer related mortality and consumption of processed meat.²¹ Processed meats also increase the risk of cardiovascular diseases.²¹

Poly Aromatic Hydrocarbons

Poly aromatic hydrocarbons are natural chemicals found in the air created from incomplete combustion during high temperature cooking. PAH chemicals, such as naphthalene, acenaphthene, and fluorene, adhere to the surface of the meat, leading to the generation of O₂- and H₂O₂ through the action of aldoketo reductase enzymes. In this process, PAH metabolites are catalyzed into PAH-o-quinones, which can undergo reduction by electron donors, resulting in a continuous cycle of reactive oxygen species.²² The ROS produced during this process induce oxidative DNA damage that can mutate the p53 genes,

the most important tumor suppressor genes; a common way this mutation occurs is through the transversion of the nucleotide Guanine to Thymine.²³ This showcases the relative risk of PAH-o-quinones, and therefore their formation in red meats should be controlled.

Scientists have also observed PAH chemicals in toasted bread at temperatures of 220-250°C, but the quantity remains unclear due to incomplete carbonization. This undermines the authorities' ability to reach a consensus and impose clear limits on concentration PAH chemicals in food.²⁴ Researchers have also observed the presence of PAH in beverages like roasted coffee. A research study investigated the transformation of trigonelline, stigmasterol, and fatty acids— major components of coffee beans—into carcinogenic PAH chemicals through the complex process of lipid, carbohydrate, and protein pyrolysis at high temperatures.²⁵ They additionally observed other carcinogenic compounds form such as acrylamide and 5-hydroxymethyl-2-furfural. However, the study is based on specific models presented, disregarding other varieties of coffees and processing methods.²⁵ The extent of research conducted in this area is still limited and should be prioritized due to the regular dietary intake of substances like bread and coffee.

Consumption of red and processed meat significantly contributes to CRC carcinogenesis by facilitating the generation of Polycyclic Aromatic Hydrocarbons (PAHs) and Heterocyclic Amines (HCAs) during high-temperature cooking processes. These compounds, upon metabolic activation, induce DNA adduct formation, potentially leading to mutations in proto-oncogenes and p53 genes. Moreover, it is imperative to note that mutations are not solely attributed to the presence of PAH and HCA DNA adducts but are also attributed to the end products of these reactions, which generate Reactive Oxygen Species (ROS). This ROS-mediated mechanism possesses the potential to cause additional DNA damage, thus further exacerbating the risk of CRC development.²⁶

This causative relationship elucidates the direct influence of our dietary intake on the heightened production of ROS.

Acrylamides and HCA

Intense heating of foods containing the amino acid asparagine forms acrylamide as another product. The deamination and decarboxylation of asparagine in the presence of sugars such as fructose and glucose produce acrylamide. The Maillard reaction, a chemical reaction between amino acids and reducing sugars, forms intermediate Schiff bases. These Schiff bases then stimulate the production of acrylamides through various mechanisms, including hydrolysis, activation of pathways, and rearrangement.²⁷ Acrylamides are epoxidated, resulting in a change in their molecular structure, by an enzyme that converts them to glycinamide. These molecules not only in-

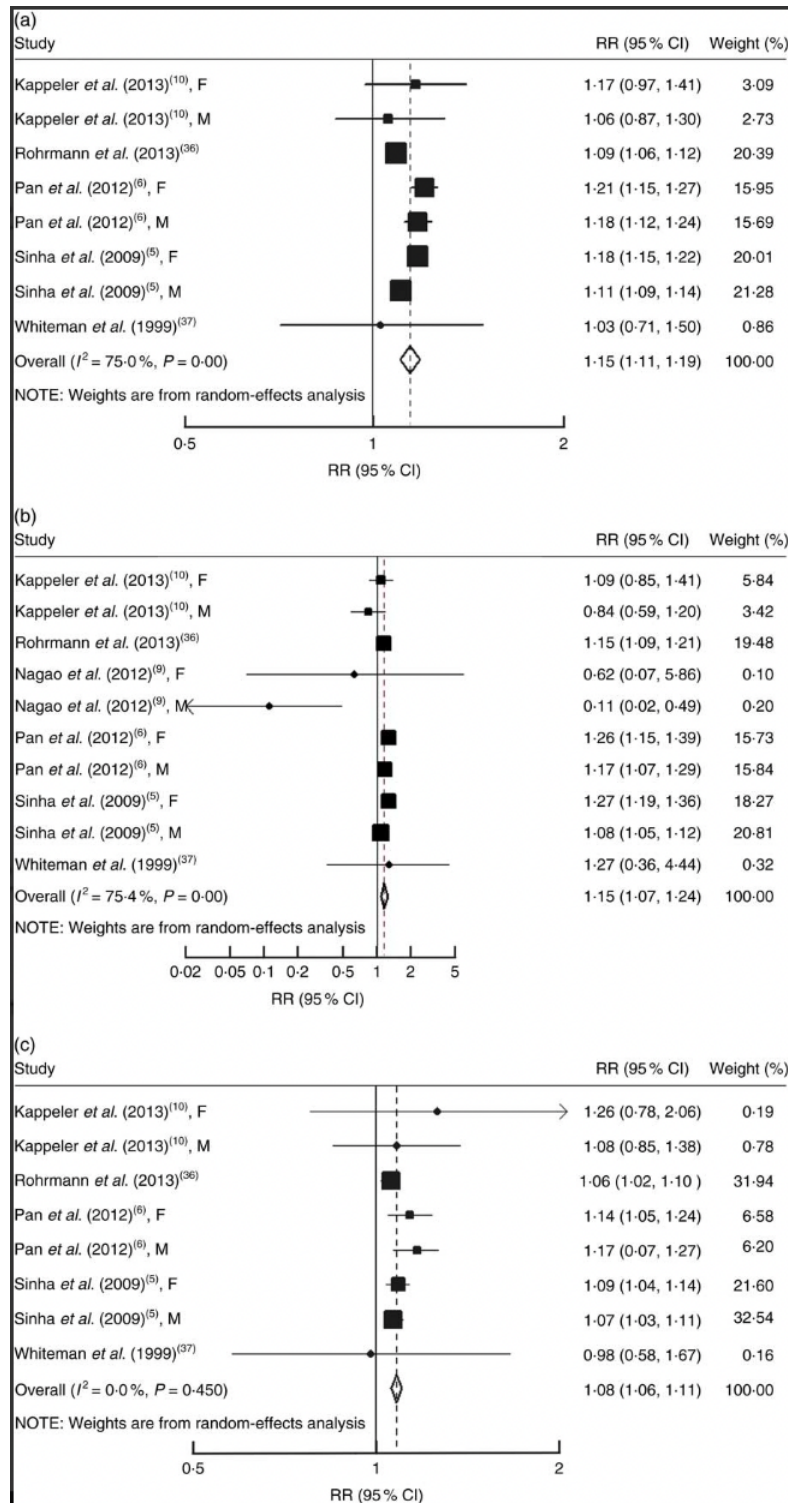


Fig. 4 Risk of (a) all-cause mortality, (b) cardiovascular mortality and (c) cancer mortality associated with each serving per day of processed meat. The study-specific relative risk (RR) and 95 % CI are represented by the black square and horizontal line, respectively; the area of the black square is proportional to the specific-study weight to the overall meta-analysis. The center of the open diamond presents the pooled RR and its width represents the pooled 95 % CI. Weights are from random-effects analysis (F, female, M, male)

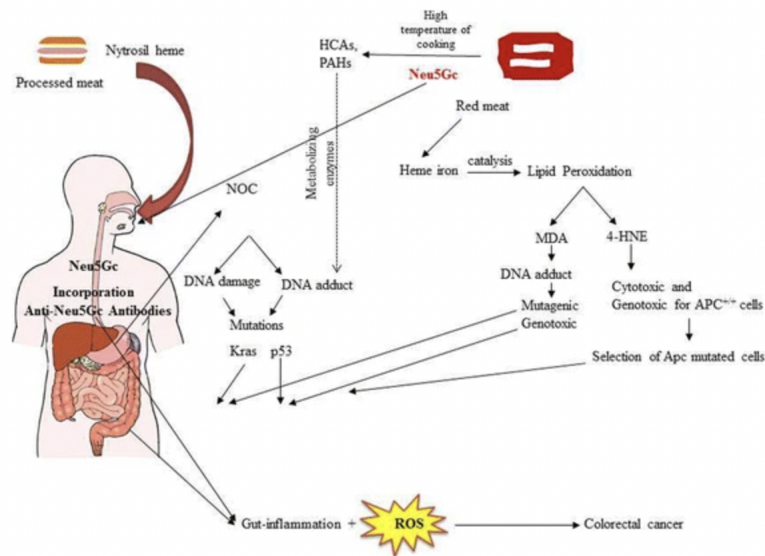


Fig. 5

duce oxidative stress by producing excessive reactive oxygen species (ROS), but they also inhibit glutathione, an antioxidant that plays a crucial role in maintaining cellular homeostasis.²⁸

Cooking meat at high temperatures produces heterocyclic amines (HCA), which serve as another source of carcinogens. Multiple epidemiological studies have shown that increased production of acrylamide and HCA is associated with increased risk of breast, prostate, and colorectal cancer. The exact mechanism is an area of ongoing research, but HCAs induce endoplasmic reticulum stress, disrupt the functioning of the organelle that produce reactive oxygen species²⁹, and contribute to oxidative stress. This is because H₂O₂ is a byproduct of protein folding during endoplasmic reticulum stress.³⁰ These stresses can induce cancer by causing cellular damage and apoptosis and activating pathways that promote tumor growth and survival. Numerous studies have provided strong evidence of a link between HCAs and malondialdehyde concentrations, which are indicators of oxidative stress. This association suggests an elevated risk of cancer development.³¹

Additives

Additives like food coloring and artificial sweeteners are other sources of carcinogens. With the anticipated increase in demand for processed foods by 4.2% by 2024, companies are compelled to develop new food coloring products that can significantly improve or maintain the visual appearance of the food. Food colorings such as red 40, red 3, yellow 5, and yellow 6, which currently constitute 90% of the total food coloring market, are known to have carcinogenic properties.³²

These dyes are non-nutritious and are known to induce cancer due to their genotoxic (Center for Science in the Public Interest, 2010) and cytotoxic properties.³³ For example, yellow 5 dye can induce chromosomal aberrations in cell division.³³ The enzyme mediated reactions of the yellow dye formulate active aromatic amines that attack DNA.³⁴ A 2015 study also concluded the excessive genotoxic effect of yellow 5 dye at all concentrations.³⁵ Dyes such as red 40 and yellow 6 also contain potent carcinogens like benzidine that initiates bladder cancer.³⁶

In addition to coloring, the industry also uses artificial food sweeteners that contain zero sugar but are harmful for consumers. Aspartame, a popular sweetener that is present in 6000 products, is associated with increased cancer risk in living rodents, while also affecting cancer susceptibility in offspring.³⁷ The relationship between oncogenesis in laboratory rodents and humans has resulted in great scientific success as both animals follow a similar series of tumor progression.³⁸ In the study conducted by the Ramazani Institute, 72 out of 76 cases initially diagnosed as hematopoietic and lymphoid tissue tumors (HLT) progressed into malignant tumors, indicating a potential association between aspartame and carcinogenesis in lymphoid tissues.³⁹ However, it is important to note that there is still limited clarity regarding the specific link between aspartame and cancer risk, as other studies have shown no significant effects on cancer initiation. One possible explanation is because of major changes in the study design. The largest human prospective study in contradiction to aspartame as cancerous (study 1),⁴⁰ a 2006 study by the researchers from NCI, explains this effect by examining 285,079 men and 188,905

women ages 50 to 71. This study 1 showcases a larger pool than study 2 that supports aspartame as cancerous⁴¹ which only accounts for 77,218 women and 47,810 men. The primary objective of study 2 was to conduct a prospective evaluation of the consumption of aspartame-containing soda and its potential impact on the initiation of hematopoietic cancer. Additionally, study 1 collected the data using self-administered frequency questionnaires while study 2 assessed diets through repeated assessments. While both methods are scientifically accepted, the repeated assessments are a better method in terms of assessing one's diet. This is because reviewing one's diet in a 24-hour time span increases the level of accuracy as it provides researchers with a comprehensive and detailed understanding of the diet. Additionally, the self-administered questionnaires are recall biased; they are dependent on the individual's memory and ability to recall the amount of food eaten. However, repeated assessments require excessive time and method, and it would have been in-viable for study 1 to employ this methodology as it had a significantly larger pool. The researchers for study 1⁴⁰ could have employed both the study designs; while maintaining their large statistical pool by self-administered questionnaires, they could have also utilized repeated assessments for greater detail and low risks of misreporting. Changes like this in study design limit the ability to reach a scientific consensus, and thus the availability of conclusive data; further research is needed to fully understand the potential relationship between aspartame and cancer development.

Trans Fats

Fats are crucial components of the human body, serving various essential functions. They play a major role in long-term energy storage, participate in signaling pathways, and provide thermal insulation. Unsaturated trans fats and saturated fats have detrimental effects on human health. They elevate the levels of low-density lipoproteins (LDL) while simultaneously reducing the levels of high-density lipoprotein (HDL). This imbalance promotes the development of atherosclerosis and other cardiovascular problems.⁴² Trans fatty acids, specifically elaidic acids, induce oxidative stress by increasing oxidized LDL levels, promoting superoxide production, triggering inflammatory cytokines, and upregulating NADPH oxidase expression. All these factors lead to atherosclerotic lesions, elevated reactive oxygen species, and inflammation in smooth muscle cells. These mechanisms contribute to oxidative DNA damage which in turn increases the risk of cancer development.⁴³ An increased intake of ectopic fat, deposition of triglycerides within cells that normally only contain small amounts of fat, can initiate major problems for the cells; this is associated with the activation of the NF- κ B pathway that when deregulated initiate inflammatory response, leading to

the generation of reactive oxygen species.⁴⁴

Partially hydrogenated cooking oil in processed foods contains the highest levels of trans fats. Manufacturers conduct hydrogenation, a process that converts liquid oil into semi-solid fats, to prolong shelf life, enhance food texture, and modify melting characteristics.⁴⁵ During hydrogenation, the addition of hydrogen molecules saturates the chemical structures of unsaturated fats in the oil. This process not only converts cis fats into trans fats but also disrupts the original cis structure by rearranging the hydrogen atom, facilitated by increased kinetic energy and added catalysts.⁴⁶ These trans fats increase the number of free radicals and induce oxidative stress.

In human diets, trans fats make up less than 1% of the total energy intake and consuming trans fats increases the risk of death by 34% (World Health Organization, 2018). Their presence in one's diet contributes to cardiovascular diseases and cancer. Major sources of intake for trans fats are refined carbohydrates such as cookies, cakes, bread ETC- comprising a significant portion of diet.⁴⁷

Refined Sugar

Another ingredient very prominent in our diets is refined sugar, which is extracted from sugar canes and left with no nutritional value; high glucose content creates an acidic environment due to the production of the lactic acid which is a desirable source of energy for cancer cells, but not for healthy cells.⁴⁸ The need to address the lack of research is significant because the excessive consumption of sugars exceeds the recommendations of the WHO.⁴⁹ Excessive sugar consumption inhibits certain antioxidants and activates NADPH oxidase, leading to increased levels of ROS. The excessive sugar intake leads to the over secretion of insulin in β -cells. The activated NADPH oxidase, through the pentose pathway, converts O₂ to O₂⁻ which is then converted into OH⁻, a highly reactive radical that can severely damage the DNA.⁵⁰ Under normal glucose metabolism, the cytochrome system generates Reactive Oxygen Species (ROS), and consequently, cells naturally express antioxidants to counteract their effects. However, elevated glucose levels resulting from increased consumption of refined sugar lead to an upsurge in ROS production. As a consequence, an excess of ROS molecules leads to DNA damage.⁵¹

Higher glucose intake is also related to the over-expression of antioxidant enzymes, which produce antioxidants, such as CuZn-superoxide-dismutase, catalase, and glutathione peroxidase that also increase the risk of cancer development.⁵² Although antioxidants remove oxidants, an overexpression of antioxidants can be harmful. A study demonstrated that overexpression of catalase drives an aggressive phenotype in glioblastoma, deadliest brain cancer.⁵³ The notion that excessive sugar consumption is highly detrimental has received lim-

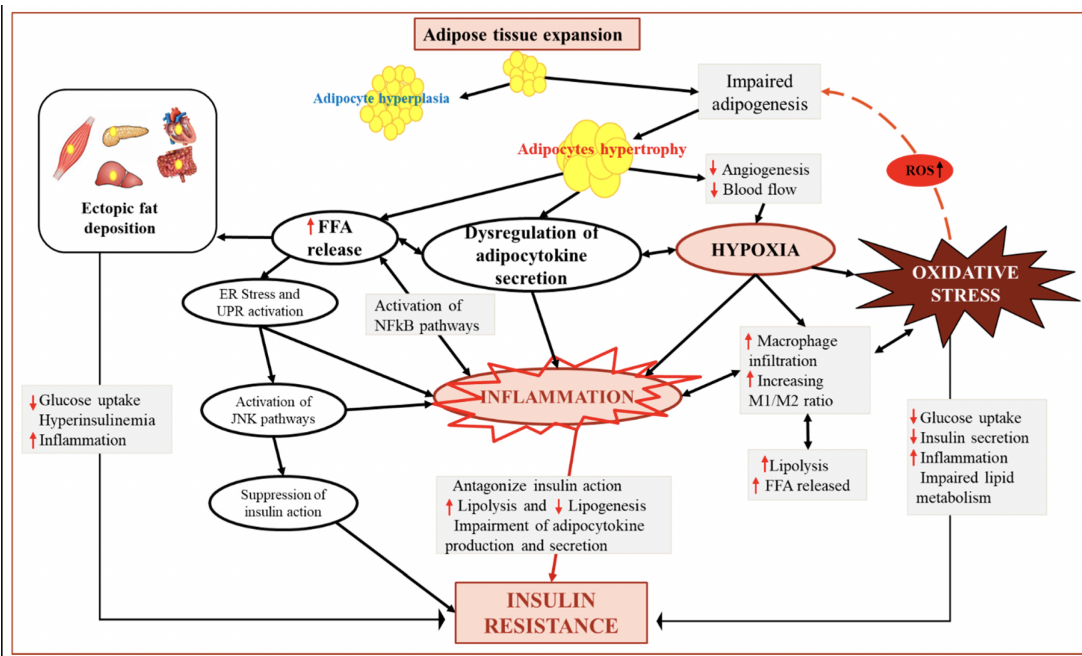


Fig. 6

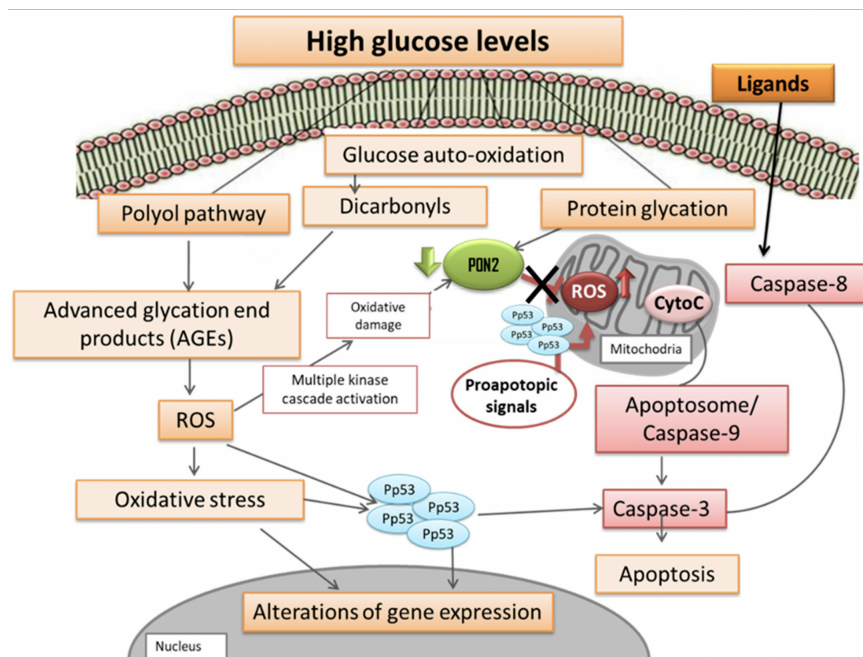


Fig. 7

ited credibility; however, its overuse is associated with various diseases related to brain dysfunction (Harvard University, 2023). Present in 60% of all the foods and beverages, sugar is much loved in the processed foods industry due to its taste, color, bulk, and thickness it provides for the products. However, overuse of sugar induces the production of ROS,⁵⁰ overexpression of antioxidant enzymes,⁵² and weight gain.⁵⁴

The Role of Natural Foods in Cancer Prevention

Dietary modifications, focusing on natural foods without artificial additives, offer a highly promising lifestyle change that can significantly decrease the likelihood of developing cancer by approximately 40%.⁵⁵ Natural foods provide the cells with high nutrition without any artificial preservatives; plants rich in antioxidants, phytochemicals, fiber, and omega-3 fatty acids, vitamins, and minerals enhance cell metabolism, differentiation, and growth. Healthy foods create an environment that hinders the proper functioning of cancer cells, making them an important aspect to reduce cancer risk.⁵⁶

Antioxidants are significant molecules that reduce the number of free radicals generated from aerobic respiration or external sources; this mechanism is necessary to regulate cellular homeostasis. Some antioxidants are produced by the cell in the form of glutathione, ubiquinol, urea acid; while other principle exogenous antioxidants like vitamin E (-tocopherol), vitamin C (ascorbic acid), and B-carotene are required from a diet.⁵⁷ Antioxidants inhibit the ROS by either generating new antioxidants or decreasing ROS producing enzymes.⁵ For instance, low bush blueberries, which are native to central Canada and are high in anthocyanin content, exhibit these properties as strong antioxidants. The radical scavenging system in these blueberries includes enzymes like superoxide dismutase, catalase, glutathione peroxidase, and other antioxidants that convert hydrogen ions and superoxide into water. Anthocyanin, unlike many endogenous antioxidants, reduces the activity of ROS producing metals, iron and copper, by forming a complex bond between the lone pair of the metal; this results in the formation of stable complexes and fewer ROS. The polyhydroxyl structure of anthocyanin enables its strong reducibility, allowing it to inhibit free radicals, regulate antioxidant activity, and chelate with metal ions.⁵⁸

Other natural compounds are phytochemicals that are specifically present in plants for their immune system. Phytochemicals have shown potential for use in human medicine. They can suppress the activity of malignant cells, facilitate antioxidant activity, and prevent angiogenesis.⁵⁹ Capsaicin, a phytochemical found in chili pepper extract, blocks the role of tumor activating pathways such as nuclear factor kappa B (NF-kB), activator protein 1 (AP-1), signal transducer, and activator of transcription (STAT3).⁶⁰ Curcumin, a polyphenol molecule, actively inhibits lymph angiogenesis, the for-

mation of blood vessels that transport oxygen and nutrients to cancer cells, by suppressing HMGB1's (High mobility group box 1 proteins) signaling. This helps prevent the development of chemoresistance in existing cancer cells.⁶¹ Indole, another strong phytochemical, is known to reduce cancer risk as well as protect cells from the side effects of chemotherapy. Glucosinolate, a strong antioxidant in brassica vegetables, inhibits certain carcinogen activating enzymes such as cytochrome P450; it also stimulates the production of antioxidant enzymes that help maintain oxidation level in the cells.⁶²

Furthermore, fiber is also an essential source to fight against cancerous cells. Cellulosic fibers prevent carcinogenesis by increasing stool bulk, diluting fecal carcinogens, and decreasing transit time for digestion.⁶³ Fiber, being susceptible to human digestive enzymes, is able to traverse the gastrointestinal tract without undergoing significant changes, thereby leading to the production of short-chain fatty acids as a predominant metabolite during its fermentation process.⁶⁴ These short chain fatty acids reverse the production of the ROS and increase the level of the antioxidant enzyme superoxide dismutase.⁶⁵

The presence of soluble cellulosic fiber, specifically pectin, in the colon also leads to changes in pH levels. This altered environment facilitates the binding of the major laboratory carcinogen, 1,2-dimethylhydrazine, to the soluble fiber. In fact, studies have shown that the binding efficiency of this carcinogen to pectin is nearly 42%.⁶⁶ By effectively binding to the carcinogen, the fiber prevents it from coming into direct contact with the colon lining, thus reducing the potential harm and lowering the risk of cancer development.⁶⁶

Another significant group of natural compounds is omega-3 fatty acids, which are present in high quantities in fish, seeds, plant oils, and nuts. These fatty acids are necessary to produce hormones involved in immune response, cell signaling, maintaining cell membrane fluidity, and supporting structural integrity. They also play a role in regulating the nervous system, blood pressure, blood clotting, and glucose tolerance.⁶⁷ The primary reason that omega-3-fatty acids prevent carcinogenesis is due to their ability to stimulate the production of SIRT1, a protein coding gene. By reducing inflammation, activating AMP-k pathway,⁶⁸ and promoting lipid metabolism,⁶⁹ the omega fatty acids upregulate the production of SIRT1 enzymes. SIRT1, also known as an anti-aging enzyme, acts as a deacetylase for cell arrest in cell cycle, DNA repair, and major activation sites of p53.^{70,71} SIRT1 is not only stimulated by omega-3-fatty acids, but also by plant derived compounds, strigolactone GR24 and pinosylvin, that are present in grape vine and witchweed.⁷² Despite these beneficial properties, only 20% of the world population's diet includes sufficient omega-3 fatty acids, which can help reduce the risk of cancer.⁷³

Vitamins and minerals, perhaps the most common of all,

are both necessary for the functions of the human body. Vitamins can be divided into 6 major groups, vitamin A, C, D, E, K, and B. Minerals, with the support of vitamins, are composed of five major molecules: calcium, magnesium, phosphorus, potassium, sodium, and sulfur. Additionally, there are 72 minor minerals that are also essential for various bodily functions.⁷⁴ For example, vitamin C also known as ascorbic acid is a direct regulator in the proper functioning of TET1 proteins,⁷⁵ which function to suppress tumors,⁷⁶ by acting as a cofactor in the demethylation process.⁷⁷ Vitamin C is a strong antioxidant as it donates high-energy electrons to neutralize free radicals.⁷⁸ It also upregulates the genes necessary for white blood cell maturation,⁷⁹ and contributes to the development of connective tissues.⁸⁰

The 5 major sources of minerals contribute extensively to the human body. Magnesium is an essential mineral as it acts as a cofactor for purine and pyrimidine nucleotide bases and maintains the appropriate structure of the genetic code by binding to macromolecules' surfaces.⁸¹ Zinc, an important mineral that allows for the fast exchange of ligands and stabilizes different coordination geometries - thereby enhancing the stimulation of signaling pathways.⁸² Zinc promotes an anti-tumor environment primarily because it is a significant part of superoxide dismutase, an antioxidant enzyme. It catalyzes the conversion of highly reactive radicals, like O₂⁻ and OH⁻, into H₂O₂, a comparatively less reactive radical. Zinc does this by preventing the chemical or physical denaturation of the superoxide dismutase and stabilizing the tertiary structure of the enzyme. Interestingly, zinc has also been shown to protect healthy cell from H₂O₂ and intensify its attack in tumor tissue. Not only does zinc act as a catalyst for antioxidant enzymes, but it can also be optimized in treatments related to cancer. And because zinc cannot be stored or produced by the cells, diets should include enough zinc to refrain from zinc deficiency as it increases cancer risk.⁸³ Pomogranate is a major dietary source with almost 0.4 mg zinc per 100g; in this fruit, zinc is necessary to maintain the enzymatic activity of matric mellaprotienase (MMPs) which are responsible for tissue allostasis, achieving stability through change. If not maintained, alteration in this enzyme can lead to tumor progression.⁸⁴ Overall, zinc plays a crucial role in preventing oxidative DNA damage, chromosomal breaks, and catalyzing antioxidant enzymes.⁸⁵

Conclusion

In conclusion, various additives have been known to increase the risk of oncogenesis. Nitrates and nitrites are commonly used additives in processed meats to preserve flavor, smell, and shelf life. Acrylamide is another compound that forms in foods during high-temperature cooking, particularly in starchy foods like bread and fried potatoes. High consumption of such

Type of Food Intake	Major Property
Low Bush Berries	Antioxidant
Capsaicin	Antitumor
Indole	Chemo preventive
Curcumin	Antitumor and Chemopreventive
Grapevine	Anticancer
Pomegranate	Zinc (Antioxidant and Anti-Tumor)

Table 1 Types of Food Intake and Their Major Properties

additives has been associated with an increased risk of cancer. In addition to additives, unhealthy foods contain components such as polyaromatic hydrocarbons (PAHs) and trans fats been shown to have carcinogenic properties. On the other hand, natural foods without artificial additives are generally considered healthier and can help reduce the risk of cancer. They provide high nutritional value, antioxidants, phytochemicals, fiber, and omega-3 fatty acids, which promote cell metabolism, differentiation, and growth. Antioxidants help regulate cellular homeostasis and reduce the number of free radicals, which can damage cells. Phytochemicals, found in plants, have been shown to suppress malignant cell activity, facilitate antioxidant activity, and prevent angiogenesis. Addressing the effects of processed foods and highlighting the dearth of research in this field is crucial, especially considering the ubiquitous consumption of these foods in our daily lives. Government bodies and regulatory agencies should encourage healthy diets, one with phytochemicals, fiber, vitamins, antioxidants, and omega-3-fatty acids. This would not only proliferate a healthier genetic background, but also increase the life expectancy of individuals. However, with the increased demand for fast foods deeming the 21st century, this is likely not to change. That is why it is even more important for authorities to ensure that processed foods are safe for consumption. To create a healthy environment, governments should implement policies that create incentives for food manufacturers to produce healthier alternatives, such as providing subsidiary for fresh produce or implementing stricter labeling requirements for processed foods.

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