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Inflammation-Driven Carcinogenesis: Mechanisms, Preventive Approaches, and Therapeutic Innovations

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Abstract: Chronic inflammatory diseases as a cause of global cancer trend? Chronic inflammation is a condition where inflammation has persisted for a long time and is not relieved on its own as it promotes tumor growth. This paper aims to outline some of the vital biological processes and signaling pathways involved in the association of cancer with chronic inflammation. One of the functions of this relationship is to create reactive oxygen species (ROS) and nitric oxides (NOs) involved in mediating signal pathways that activate inflammation, the migration of immune cells, and cytokines. Recent studies imply that chronic inflammation may be involved in the pathogenesis of cancer conditions like hepatitis-B virus-induced hepatocellular carcinoma and colon carcinoma arising in the context of inflammatory bowel disease. These processes are necessary for creating therapies for both prevention and diseases. Consequently, the discovery and production of new drugs that can prevent cancer prevalence and increase survival rate within a population depends on correctly identifying the molecular mechanisms that connect disease to inflammation. The study collects the current research and lays out the foundation for future research that may use these conclusions to improve further the diagnosis and treatment of cancer and cancer prevention strategies.

Keywords: Chronic inflammation; Cancer pathogenesis; Reactive oxygen/nitrogen species; Pro-inflammatory signaling pathways; Immune cell infiltration; Therapeutic implications

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1. Introduction

Cancer is still one of the leading causes of morbidity and mortality, and although the causes of the leading kinds of cancer are complex and a culmination of genetic and environmental factors as well as lifestyle. Among these, there is chronic inflammation, which contributes significantly to the progression of many types of cancer [1]. It is a prolonged, frequently chronic state that keeps recurring as opposed to acute inflammation, a reversible response by the body to any injurious stimuli or occurrences. Maintaining the chronic state of inflammation can cause chronic inflammation and initiate the development of cancer [2].

One of the critical roles in the development of carcinogenesis belongs to chronic inflammation, which is performed through many mechanisms. Another critical factor in this process is the formation of ROS/RNS as byproducts, which may contribute to deoxyribose oxidation in DNA and other cellular components. Oxidative stress generates mutations in the DNA, and hence, it will activate the oncogenes and will decontrol the function of the tumor suppressor genes, and this can lead to cancer growth and metastasis [3]. Non-resolving chronic inflammation is associated with the persistent activation of cyclooxygenase-2 (COX-2), nuclear factor kappa-light-chain-enhancer of activated B cells (NF-kappaB), and the signaling mediators and transcription activators 3 (STAT3). These are essential in the development and spread of cancer as they control cell growth (proliferation), cell death (apoptosis), and angiogenesis [4]. They are attracted to the sites of chronic inflammation and release inflammatory chemicals called chemokines and cytokines to promote and perpetuate chronic inflammation. These mediators can create conditions for the growth of metastatic cancer cells in a way that the environment would be ideal for the colonization and proliferation of such cells [5]. The concept of chronic inflammation as a precursor to cancer is a well-known phenomenon in several cancers. Inflammatory bowel disease (IBD), mainly consisting of ulcerative colitis and Crohn's disease (CD), also increases the risk of developing colorectal cancer (CRC) [6]. They lead to chronic inflammation of the gastrointestinal tract lining and develop dysplasia or even cancer growth. Potential people with chronic HBV or HCV infection may develop hepatocellular carcinoma (HCC). Chronic hepatitis can lead to liver cancer because it results in fibrosis, leading to cirrhosis and chronic inflammation [7].

Additionally, there is a high correlation between stomach cancer and persistent *Helicobacter pylori* infections. The bacterium causes stomach mucosal inflammation that can progress to gastric cancer, atrophic gastritis, intestinal dysplasia, and intestinal metaplasia. Chronic inflammation and tissue remodeling in the lungs lead to carcinogenesis. Another example is the link between chronic obstructive pulmonary disease (COPD) and lung cancer [8].

In order to create new methods of cancer prevention and treatment, it is essential to comprehend the complex link underlying chronic inflammation and the disease. Potentially curable cancers and better outcomes for people with inflammation-associated malignancies could be achieved by focusing on particular inflammatory pathways and mediators [9]. Additional research is required to implement these findings in clinical practice to wholly and effectively understand the molecular mechanisms behind this link. By summarizing what is already known and drawing attention to possible future research and treatment intervention paths, this review hopes to delve into how chronic inflammation leads to cancer formation.

2. Molecular pathways involved in chronic inflammation and carcinogenesis

An inflammatory response that lasts for an extended period, known as chronic inflammation, can contribute to the onset and advancement of several diseases, including cancer. Multiple molecular pathways contribute to the change of normal cells into malignant ones in the link between chronic inflammation and carcinogenesis [10]. **Figure 1** also shows the critical molecular pathways that connect chronic inflammation with cancer, emphasizing the functions of cytokines, transcription factors, signaling pathways, and cellular reactions.

2.1. Inflammation-related mediators

Inflammatory mediators and cytokines are essential in the pathogenesis of chronic inflammation, a precursor to the onset of cancer. They also mediate immune responses.

(1) Tumor necrosis factor-alpha (TNF-α)

TNF-α is a pro-inflammatory cytokine that plays a role in inflammation as well as in progression of

cancer. These include events crucial in fine-tuning immune regulation, suppressing inflammation, and promoting cell survival through triggering nuclear factor-kappa B (NF-κB) and mitogen-activated protein kinase (MAPK) pathways. Some genes can encode for the receptors for TNF-α that are close to pro-inflammatory genes, and they get activated when the TNF-α receptor binds to the ligand and thus activates the NF-κB. It can be noted, though, that TNF-α also stimulates the MAPK pathway, including the ERK, JNK, and p38 kinases, which thus influence gene expression, cell proliferation, and cell death. Moreover, cancer development is facilitated by the TNF-α that maintains chronic inflammation through the expression of several pro-inflammatory genes and the overexpression of adhesion molecules. This leaves a conducive condition that favors DNA damage and genomic instability [11].

(2) Interleukins (IL-1 and IL-6)

IL-1, IL-6, IL-10, and IL-17 family of interleukins are significantly involved in regulating inflammation and immune response in cancer. IL-1 causes the production of adhesion molecules and an inflammatory chemokine that leads to more white blood cells sticking to irritated tissues. Moreover, IL-6 stimulates the JAK/STAT pathway that increases the expression of genes that regulate cell proliferation and survival, resulting in promoting tumorigenesis and blocking apoptosis. The angiogenesis associated with IL-6 also supports tumor vascularization and helps to increase tumorigenesis [12].

(3) Chemokines (CCL2 and CXCL8)

Chemokines are small proteins that help in the movement of immune cells, and this part explains how chemokines are involved in the recruitment and activation of immune cells and the growth of tumors. The tumor microenvironment becomes more inflammatory and tumorigenic when CCL2 brings monocytes, memory T cells, and dendritic cells. Neutrophils are drawn to CXCL8 by its ability to release reactive oxygen species and proteases, which then contribute to DNA damage and mutations in tissues. Moreover, CXCL8-mediated attraction of immune and endothelial cells sustains inflammation, supporting tumor angiogenesis and metastasis, thus promoting cancer progression [13].

2.2. Transcription factors and signaling pathways

Chronic inflammation induces the activation of several key transcription factors and signaling pathways that regulate gene expression related to inflammation, cell proliferation, and survival.

(1) NF-κB pathway

Cell proliferation, survival, and the regulation of immunological and inflammatory responses are all regulated by the NF-κB pathway. When not in use, inhibitors of κB (IκB) keep NF-κB dimers (usually p65/RelA and p50) locked up in the cytoplasm. Proteasomal degradation of IκB occurs when the IκB kinase (IKK) complex phosphorylates IκB in response to inflammatory stimuli such as TNF-α, IL-1, and bacterial lipopolysaccharides (LPS). After being released from IκB inhibition, NF-κB moves to the nucleus and attaches to particular DNA sequences to control the transcription of specific genes [14]. Some of the genes that are being targeted include those that code for cytokines and chemokines that promote inflammation (such as IL-1, IL-6, and TNF-α), as well as proteins that prevent cell death (such as Bcl-2 and Bcl-xL). Cell proliferation is regulated by cyclins and cyclin-dependent kinases, which are affected by NF-κB. The genesis and progression of certain malignancies, including colorectal and liver tumors, are influenced by the persistent activation of NF-κB, which is a characteristic of chronic inflammatory disorders such as rheumatoid arthritis as well as inflammatory bowel disease [15].

(2) STAT3 pathway

The STAT3 pathway is crucial for mediating responses to cytokines and growth factors, playing a

significant role in inflammation and cancer. When cytokines like IL-6 bind to their receptors, they activate associated Janus kinases (JAKs). STAT3 undergoes phosphorylation at a tyrosine residue through JAK, leading to dimerization of this protein and nuclear translocation. Both the STAT3 and the DNA are in the nucleus, and dimers facilitate gene expression. STAT3 up-regulates genes required for angiogenesis (e.g., VEFG) and helps cell growth. In the case of cancer, the genes form complexes with cyclin D1 and help to sustain cell survival (e.g., Bcl-2, Mcl-1). The activation of STAT3 can affect cytokine genes at the transcription level and lead to chronic inflammation and inflammatory processes. It is related to cancers like pancreatic, breast, and prostate cancers, as it facilitates tumorigenesis, metastasis, and survival or also provides resistance to apoptosis [16,17].

(3) MAPK pathway

MAPK regulates cell responses to the microenvironment by regulating inflammation, cell proliferation, and differentiation. The ERK, JNK, and p38 MAPKs are noted as three signals to activate each part of this system. ERK is a downstream target of mitogenic and developmental factors for cell proliferation and differentiation. JNKs in the immune system control the regulation and mechanisms of activation. The expression of inflammatory cytokines by stress also activates p38 MAPK to mediate cell cycle arrest and apoptosis and to regulate inflammation. When MAP2Ks are blocked by various extracellular signals, MAP3K activates MAP2K to initiate MAPK phosphorylation. After that, they phosphorylate specific proteins, such as kinases and transcription factors. MAPKs control the expression of genes related to inflammation, cell proliferation, and cell death by regulating transcription factors, including AP-1. Sustained MAPK signaling in chronic inflammation continuously activates pro-inflammatory gene expression, contributing to chronic inflammatory responses. Melanoma, in which ERK pathway hyperactivation results from alterations in the BRAF gene (a MAP3K), and lung cancer, in which tumor growth and inhibition of apoptosis are promoted by mutations as well as overexpression of MAPK components, are two examples of the many cancers that involve dysregulation of MAPK pathways [18,19].

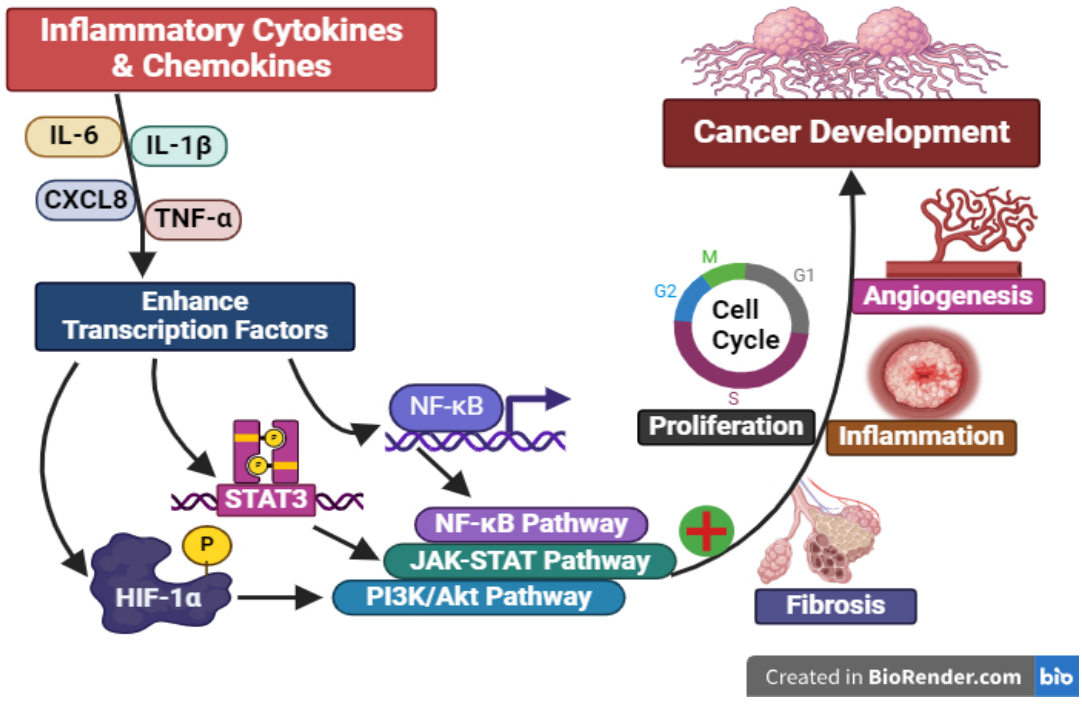

Figure 1. The interplay between inflammation and cancer via different pathways.

3. Cellular responses and microenvironmental changes

Chronic inflammation leads to significant alterations in the cellular microenvironment, contributing to cancer initiation and progression.

3.1. Reactive oxygen species (ROS)

(1) ROS generation and cellular impact

As part of their defense mechanism against infections, inflammatory cells like neutrophils and macrophages produce ROS during the respiratory burst. On the other hand, oxidative stress is caused by chronic inflammation, which leads to an extended generation of ROS.

(2) DNA damage and mutagenesis

Base changes, strand breakage, and cross-linking are only some DNA-damaging mechanisms that reactive oxygen species (ROS) can bring about. Damage can take several forms, including the creation of 8-oxo guanine. This mismatch with adenine throughout replication can result in G-C to T-A transversions, a mutation frequently found in cancer.

(3) Impact on proteins and lipids

ROS can chemically alter proteins by reacting with the amino acids present in the protein molecule. This modification can change the protein's function or make it more susceptible to degradation. The interaction between ROS and lipids leads to lipid peroxidation, which produces secondary mutagenic chemicals such as malondialdehyde and 4-hydroxynonenal aldehydes. These molecules can then bind to DNA, inducing mutations.

(4) Evasion of apoptosis and proliferation

ROS signaling can inhibit apoptotic cell death in cells with ROS-induced DNA damage by reducing p53 levels or through other mechanisms, therefore enabling the accumulation of mutations and promoting cell proliferation. This event triggers the mutation occurrence and accumulation process, which subsequently leads to the clonal evolution of cancer cells [20,21].

3.2. Epigenetic modifications

(1) DNA methylation

Persistent inflammation can lead to alterations in DNA methylation. One example of such a sequence occurs when there is excessive methylation of CpG islands in the promoter regions of tumor suppressor genes, resulting in the silencing of these genes. Conversely, hypomethylation can result in the activation of certain sequences, such as transposable elements and oncogenes.

(2) Histone modifications.

The proliferation of proinflammatory cytokines may enhance or attenuate histone acetylation and methylation status of the DNA, upholding the structure of chromatin and gene transcription. Therewith the oncogenic stimulation is achieved by the STAT3-dependent activation of the acetyltransferase activity of the SETD7 on the promoter region of the genes with oncogenic activity.

(3) Non-coding RNAs

MicroRNAs (miRNAs) and long non-coding RNAs (LncRNAs) are some of the most important gene regulatory elements and are highly dysregulated in chronic inflammatory conditions. Elevated miRNAs might suppress tumor suppressor genes (TSGs) or stimulate the expression of oncogenes, which will promote or reverse tumor development [22].

3.3. Tumor microenvironment (TME)

(1) Cellular composition

There might be several cells present in the tumor microenvironment when there is chronic inflammation. These cells are regulatory T cells (Tregs), myeloid-derived suppressor cells (MDSCs), cancer-associated fibroblasts (CAFs), and tumor-associated macrophages (TAMs). Such cells are capable of giving rise to tumors.

(2) Cytokines and growth factors

The TME also produces other cytokines, e.g., TNF-α, IL-1β, and IL-6, and growth factors, such as TGF-β and EGF, that are responsible for increasing the survival, proliferation, and migration of the tumor cells. For example, TGF-β contributes to cancer metastasis by promoting the process of epithelial-mesenchymal transition (EMT).

(3) Matrix remodeling

There is a protease activity in chronic inflammation, known as the matrix metalloproteinases (MMP), which contributes to chronic extracellular matrix (ECM) remodeling. The reason for this hypothesis is evidence that ECM degradation not only promotes cancer cell invasion and metastasis but also increases the release of greater amounts of bioactive substances that can stimulate tumor growth.

(4) Immune evasion

The following part serves the main purpose of highlighting the proteolytic actions that participate in matrix turnover during chronic inflammation with a special emphasis on the matrix-degrading enzyme group, the MMPs. It should be noted that both carcinogenic and metastasizing processes lead to the mechanical strength reduction of the ECM. However, this decrease is also needed to achieve optimum conditions for the performance of the released chemicals that contribute to tumor growth [23].

3.4. Angiogenesis

(1) VEGF and other angiogenic factors

It may be stated, though, that both angiopoietins and bFGF may also be produced not only by the cancer tissues and cells but also by the pro-inflammatory immune cells, while the VEGF is derived only from cancer cells. These chemicals obviously can dock on the receptors to cause movements and proliferation of cells of the epithelium of the new vessels.

(2) Hypoxia and hypoxia-inducible factor 1-alpha (HIF-1α)

A common feature of rapidly proliferating tumors is a hypoxic tumor microenvironment that stabilizes a transcription factor named HIF-1α that enhances angiogenesis by upregulating genes for the pro-angiogenic protein VEGF and other related proteins.

(3) Vessel abnormalities

Some tumors become well-pronounced areas of hypoxia and acidity because their blood supply is typically characterized by the structural and functional defects of the newly generated vessels. These conditions favor the selection of aggressive cancer phenotypes and lead to increased genetic instability.

(4) Therapeutic resistance

Tumor angiogenesis may promote therapeutic resistance by preventing the flow of immune cells and chemotherapeutic agents to certain areas of brain tumors. Therefore, effective therapeutic modalities targeting angiogenesis, including bevacizumab (anti-VEGF), have become crucial in treating cancer [24].

Table 1. Cancer development pathways, their functions, and potential treatment targets.

Sr.	Molecular pathway	Key players	Implications in cancer	Therapeutic target
1	TNF-α pathway	TNF-α, NF-κB, MAPK	Promotion of oncogenesis, DNA damage, genomic instability	TNF-α inhibitors
2	IL-1 and IL-6 pathway	Interleukins, JAK/STAT pathway	Tumor growth promotion, inhibition of apoptosis, tumor vascularization	IL-6 receptor blockers
3	Chemokine pathway	CCL2, CXCL8	Pro-tumorigenic microenvironment, tumor progression	Chemokine receptor antagonists
4	NF-κB pathway	NF-κB, IKK complex	Implicated in various cancers, including colorectal and liver cancer	IKK inhibitors
5	STAT3 pathway	STAT3, JAKs	Associated with breast, prostate, and pancreatic cancers, tumor growth promotion	STAT3 inhibitors
6	MAPK pathway	ERK, JNK, p38 MAPKs	Implicated in melanoma, lung cancer progression	MAPK pathway inhibitors
7	ROS generation	Macrophages, neutrophils	Clonal evolution of cancer cells, genomic instability	Antioxidants
8	Epigenetic modifications	DNA methylation, histone modifications, non-coding RNAs	Tumor progression, altered gene expression	Epigenetic modifying agents
9	Tumor Microenvironment	TAMs, CAFs, MDSCs, Tregs	Tumor progression, metastasis	Immunotherapies targeting TME
10	Angiogenesis	VEGF, HIF-1α	Tumor growth support, therapeutic resistance	Anti-angiogenic agents

4. The link between chronic inflammatory conditions and cancer risk

Inflammatory-related chronic diseases are associated with a higher incidence of cancer review. One of these can be obtained from the fact that several vital conditions have different mechanisms in carcinogenesis.

4.1. Inflammatory bowel disease (IBD) and colorectal cancer

CRC is a major potential increased risk for IBD, particularly especially ulcerative colitis (UC) and CD. Fulfillment of all three-row criteria increases the risk of colitis: severity of colitis, illness duration, and inflammation level. Chronic inflammation results in never-ending cycles of tissue trauma and healing, which creates a perfect niche for cancer to form. The amount of pro-inflammatory cytokines, such as TNF-α, IL-6, IL-1β, etc., increases, and the pro-tumorigenic microenvironment is formed. Epigenetic modifications, such as DNA methylation and histone modifications, and the elevation of non-coding RNA levels can be provoked by inflammation-induced oxidative stress and cytokines, where these genes have been associated with cancer. The imbalance of gut bacterial lifestyle causing dysbiosis in IBD leads to enhanced inflammation by producing carcinogen metabolites and hence enhances the cancer risk [25,26].

4.2. Chronic hepatitis B and C infections and hepatocellular carcinoma

Epidemiology of HBV and/or HCV infection represents a major risk factor for the development of HCC. HCV individuals have a higher risk of approximately fifteen to twenty times of the general population, and HBV chronic carrier poses about fifteen to twenty percent higher risk. Other ways in which HBV leads to cancer

include insertional mutagenesis where oncogene transcription is activated or where the TSG is inactivated by HBV DNA integrating into the host genome. Chronic infection leads to continuous inflammation, and also constant regeneration of the liver that further increases the risk of mutations in part of the genome of an organism. Chronic infection also leads to consistent release of inflammation-related cytokines such as IL-6 and TGF-β which continuously promotes the development of liver fibrosis and cirrhosis that precede HCC. ROS are generated in the host cell from viral infections and cause DNA damage and genetic instability [7,27].

4.3. Chronic *Helicobacter pylori* infection and gastric cancer

The International Agency for Research on Cancer (IARC) has categorized *Helicobacter pylori* infection as a Group 1 carcinogen. Infected persons have a 2- to 6-fold more significant risk of stomach cancer, making it a substantial risk factor. *H. pylori* causes chronic gastritis, characterized by persistent inflammation and epithelial cell turnover. Certain strains of *H. pylori* express virulence factors such as CagA and VacA, which directly damage gastric epithelial cells and disrupt cellular signaling pathways. The infection can also lead to hypermethylation of promoter regions of tumor suppressor genes, silencing their expression. *H. pylori* manipulate host immune responses to create a chronic inflammatory state while avoiding immune clearance, further contributing to carcinogenesis [28,29].

4.4. Chronic pancreatitis and pancreatic cancer

Chronic pancreatitis significantly increases the risk of pancreatic cancer, with studies showing a 2- to 3-fold increase in risk. Prolonged inflammation leads to fibrosis and genetic mutations in pancreatic cells. High levels of cytokines such as IL-6 and TNF-α promote a pro-tumorigenic microenvironment, while chronic inflammation increases oxidative stress, leading to DNA damage and genomic instability [30].

4.5. Barrett's esophagus and esophageal adenocarcinoma

Barrett's esophagus is a pre-malignant condition caused by metaplastic change that is a result of decades of reflux esophagitis, and it is associated with cancer of the esophagus known as adenocarcinoma. Long-term accumulation of stomach acid and bile enhances chemical toxicity and mutation of esophageal cells. Certain types of chronic inflammation led to the production of more cytokines and growth factors such as IL-6, EGF, and the like. These insults are localized at a micro and possibly a macroscale and are associated with epigenetic changes that may enhance the carcinogenic process [31].

5. Key risk factors for chronic inflammation and cancer development and preventive strategies

5.1. Key risk factors

(1) Genetic predispositions

Some polymorphic gene variants may determine a risk for chronic inflammation and cancers. These genetic variations may induce changes in the expression of mRNAs related to immune function, immune pathways related to inflammation, DNA repair pathways, or inflammatory oncogenesis.

(2) Lifestyle choices

Cigarette smoking and diet have emerged as the major risk factors that cause most of the inflammation and cancers. The direct effect of smoking causes DNA damage and also incites inflammation in the lungs which majorly leads to lung cancer. Nutritional factors that increase the risk for colon cancer in humans. For example, eating unhealthy diets rich in red and processed meat and poor in fruits and

vegetables can exacerbate inflammation and increase a person's probability of developing cancer due to oxidative stress and proinflammatory pathways.

(3) Environmental exposures

It is becoming evident that the toxicants, pathogens, and other factors in the environment can induce chronic inflammation and that this can worsen cancer. The environmental pollutants mentioned, such as air pollution, industrial chemicals as well as pesticides, have the potential to induce inflammatory responses, and the condition might end up leading to prolonged carcinogenesis [32,33].

5.2. Preventive strategies

(1) Diet and nutrition

The anti-inflammatory diet is an evidence-based approach to management with natural products. One method of attaining this goal is to take foods that are anti-inflammatory foods such as fresh vegetables, whole grain products, and omega-3 fatty acids in seafood. It is also possible to suppress the inflammation process simply by reducing the consumption of pro-inflammatory foods such as red meat and processed meat and also the intake of refined carbohydrates and trans-fats.

(2) Physical activity

Both forms of exercise during the training and the rest periods are associated with reduced inflammatory markers, enhanced innate immunity, and lower cancer incidences. Lower body mass index (BMI) and physical activity contribute to the preservation of weight and reduced inflammation, which are risk factors for obesity-related cancers. It also helps in the proper circulation and distribution of oxygen in tissues, which is vital for a tissue inflammatory response.

(3) Smoking cessation

One of the best methods to lessen the likelihood of chronic inflammation and cancer, especially lung cancer, is to stop smoking. Reducing the overall inflammatory load on the body and quitting smoking reduces inflammatory indicators and enables the repair of damaged tissues.

(4) Anti-inflammatory agents

Aspirin and other non-steroidal anti-inflammatory drugs (NSAIDs) have demonstrated encouraging results in cancer prevention trials. Because of its ability to inhibit COX-2 and reduce prostaglandin levels molecules that are important in inflammation and carcinogenesis regular aspirin use is linked to a decreased risk of colorectal cancer [33,34].

5.3. Conventional and emerging treatment strategies

(1) Chemotherapy

Cancer cells and other fast-dividing cells are the targets of cytotoxic medicines used in chemotherapy. These medications inhibit the growth and spread of cancer because they interfere with cell division and DNA replication. Alkylating agents, antimetabolites, anthracyclines, and plant alkaloids are only a few examples of the many chemotherapeutic agents available. These compounds attack cancer cells in unique ways. Chemotherapy can be administered orally, intravenously, or through other routes depending on the specific drug and cancer type. Other common side effects demonstrated are hair loss, nausea, vomiting, fatigue, and susceptibility to infections because chemotherapy targets also damage healthy cells that can reproduce quickly, such as cells in the bone marrow. It is commonly combined with other types of treatments like surgery or radiation to strengthen its positive action and treat cancer from several directions [35].

(2) Radiation therapy

Radiation treatment is very useful in eradicating cancer cells and making them non-functional due to the fact that it damages and hampers their DNA function. This method uses an intense source of radiation, such as X-rays, gamma rays, or protons, among others. There are two types of radiation treatment available: intracavitary and interstitial brachytherapy, or the placement of needles or transrectal radioactive sources into or near the tumor, and external radiation therapy, which applies an external beam of radiation aimed at the tumor. The evolution of technology has made modern radiation treatments very effective with the use of treatments like stereotactic body radiotherapy (SBRT), intensity-modulated radiotherapy (IMRT), and image-guided radiotherapy (IGRT). These particular treatments are designed in such a way that they destroy the affected areas without interfering with the healthy tissues. Radiation therapy may lead to skin peeling, fatigue, organ toxicity, or inflammation when radiation is directed to the lungs. Radiation therapy in cancer treatment has three principal applications: such as primary treatment, which employs the surgical removal of cancerous cells, the use of adjuvant treatment, which is given after an individual undergoes surgery to eliminate all the cancer cells from the body and the palliative treatment which is applied in the most advanced stages of cancer to alleviate symptoms of the disease [36].

(3) Surgery

The primary aim of tumor excision using surgery is to completely remove all the diseased cells within the tumor and also any nearby cells. Therefore, cancer does not have to be life-threatening, when it is localized in one specific area, it can be effectively treated to eradication. The prevention and therapeutic management of cancers in the adults involves least invasive procedures, such as laparoscopy, to the more invasive procedures, such as the excision of the breast for the treatment of breast cancer and excision of the entire colon for the treatment of colon cancer depending on the nature and site of the cancer. Therapy with palliative or radiation candidate chemotherapeutic agents after surgery has become a common adjunctive therapy; diagnostic imaging and biopsy must be performed prior to surgery. Such complications may include infection, bleeding, pain after surgery, and possible organ dysfunction during and after the surgical procedure and also the entire body outcome on the final evaluation. When you cannot completely remove the cancerous mass, then palliative surgery can be done if only it helps the patient in reducing the symptoms and helps in improving their condition [37].

(4) Biologic therapies

For inflammatory response is well known for tumor promotion, biotherapy such as the use of monoclonal antibodies that interfere with a specific proinflammatory cytokine will get necessary research from numerous studies. For instance, existing anti-tumor necrosis factor-alpha formulations like infliximab or adalimumab have been investigated for possible use in cancer treatment. These antibodies have already been used to treat inflammatory diseases. These biologics can reduce chronic tissue inflammation that is often found to correspond to tumor growth and metastasis. In addition, the use of receptor antagonists or monoclonal antibodies to block cytokines such as IL-6 and IL-1β may help in curtailing the pro-tumorigenic signaling inside the tumor environment, which may aid in cancer progression [38,39].

(5) Immunotherapy

The following part of the paper will discuss immunotherapy as a novel cancer treatment that stimulates the immune system to fight cancer. The latter include cancer vaccines, cellular immunotherapy, checkpoint inhibitors, and oncolytic virus therapy. Pembrolizumab and ipilimumab are among the

checkpoint inhibitors that block such proteins to boost T-cell activation, so the immune system gears toward killing cancer cells. Immunotherapies could work against some of the immunosuppressive effects of chronic inflammation at tumor sites. The immunity can lead to the migration of immunosuppressive Tregs and MDSCs into the tumor environment. At the same time, immunotherapy can counter this activity of the Tregs and the MDSCs [40].

(6) Personalized medicine

Because every patient and tumor is different in genetic and molecular aspects, the treatment with personalized medicine is more specific and efficient. This approach identifies potential therapy targets through genome sequencing of tumors. Using erlotinib as a targeted medicine to treat non-small cell lung cancer (NSCLC) is one of the most critical examples proving that personalized medicine is effective. Another example is imatinib, which is used to treat part of chronic myelogenous leukemia (CML). Biomarkers are used to determine the progression of disease as well as the future success of specific targeted therapy tests. Successful treatment for the same kind of cancer on an individual basis depends on the knowledge of the unique genome of this illness in a specific patient. Information from this ensures doctors achieve better symptom control/treatment and better prognoses [41,42].

(7) Integrating anti-inflammatory strategies

Combining these anti-inflammatory approaches in cancer treatment will help to make a shift towards the therapy related to targeting inflammatory pathways and/or modulating tumor protection or the microenvironment. Incorporating these methods with traditional treatments boosts patient wellness and helps inhibit cancer cell proliferation, metastasis, and tumor progression [43].

6. Future directions and research opportunities

Therefore, it is necessary to perform additional studies to explore the link between chronic inflammation and cancer. Moreover, the application of such huge treatments as NSAIDs remains ineffective with side effects. The subsequent research should be dedicated to the creation of anti-inflammatory drugs that act on specific inflammatory pathways such as TNF-alpha, IL-6, and IL-1 beta, to ensure that they are the most beneficial without disrupting immunity. This has the potential to improve cancer evaluations and the suppression of cancer recurrences by inflammation [44].

The third component of the study encompasses the significance of specified biomarkers in the determination and management of carcinomas that are connected to inflammatory conditions. Such biomarkers might be specific genes or proteins or any other molecules that are involved in the development of chronic inflammation leading to cancer. A screening program is the process of predicting the probability of the development of a disease and the illness before any apparent sign of the disease can be observed and the presence of the patient developing bio-molecular markers. This is very important to cancer treatment requirements. The introduction of biomarkers assists in regulating standards and ensuring the efficiency of the treatment procedures by establishing the condition of the diseases and the potency of the provided medication.

The gut microbiome is of great value in showing how pathogens play an essential role in inflammation-related disorders and cancers. Health professionals should also investigate these relationships to modulate microbiota activity to prevent cancer [45]. Moreover, new therapeutic treatments or preventatives, like changes in nutrition (e.g., prebiotics and probiotics), could be manufactured.

7. Conclusion

The interplay between chronic inflammation and cancer elucidates the requirement for interdisciplinary approaches to cancer management that address behavioral and preventive factors as well as novel interventions. This can be avoided by making positive lifestyle changes such as eating healthy, being physically active, and even giving up smoking to tackle chronic inflammation. Prevention strategies are therefore vital in detecting and intervening early. It includes frequent high-risk testing and effective anti-inflammatory drug management. Additionally, there are opportunities for better cancer management and treatments because of the new biologics, immunotherapy, and targeted medications that target the inflammatory pathways responsible for cancer onset and progression. Further scientific investigations into this subject will better understand the mechanisms associated with chronic inflammation and cancer. This would also contribute to the search for new treatment strategies. With the help of more customized and targeted therapies, patients can benefit by alleviating the emotionally and physically challenging effects of inflammation-related malignancies. For this, it is imperative to appreciate that a comprehensive approach is needed to address the issue of chronic inflammation in carcinogenesis.

Disclosure statement

The authors declare no conflict of interest.

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