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The role of HLA-B27 in cancer immunity: Investigating its potential impact on tumour surveillance and response to immunotherapy

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Abstract

This research paper specifically aims at identifying HLA-B27's involvement in the anti-cancer immune response and response to immunotherapy especially in tumor detection. Hydroxylation of HLA-B27 may affect the immune system and chronic inflammation, which is responsible for cancer predisposition due to the molecule. The literature on the relationship between HLA-B27 and cancer forms and types is analyzed with the focus on the immunological and genetic mechanisms in the contexts of brain cancer. It points out the directions for the literature by outlining significant areas for further research to explain these mechanisms. The results have the following practical importance regarding the screening and individual approach this field is interested in improving the prevention and treatment of cancer for patients with HLA-B27 positive.

Keywords: Cancer Immunity; Autoimmune Disorders; Immunotherapy; Chronic Inflammation

1. Introduction

Autoimmune diseases are a collective term for all the diseases in which the immune system attacks the body's own tissues and organs. It was also described how this misdirected immune response leads to chronic inflammation and tissue injury. Autoimmune diseases involve the immune system attacking different organs and tissues of the human body and are marked by manifestations that depend on the location of affected tissues.

Autoimmune diseases are of many types out of which few are as follows systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), and multiple sclerosis (MS). Systemic lupus erythematosus is a multisystem autoimmune disease involving the skin, kidneys and joints among others whereby the patient's immune system produces auto-antibodies that form immune complexes to cause inflammation and damage throughout the body. Rheumatoid arthritis mainly affects joints by developing a chronic inflammation from the synovial membrane that can culminate in joint breakdown and disability. Multiple sclerosis is a disease where the body's own immune system turns on it and destroys the protective tissue of the nerves that helps in sending signals to other parts of the body.

Autoimmune diseases are multi-factorial diseases that are initiated by both genetic and environmental factors together with dysfunction of the immune system. T and B lymphocytes are immune regulated cells that help regulate immune tolerance; their dysregulation in terms of function might result in autoimmunity. In most autoimmune diseases, B cells are involved in auto-antibody synthesis that upon binding to antigens, form immune deposits in tissues leading to inflammation. Self-reactive T cells that are responsible for regulation of immune response get activated and instead of stabilizing the inflammation; they continue to further exacerbate inflammation. Inherited immunological factors may tip off the patient's body into attacking its own tissues when triggered by events such as infections or exposure to certain chemicals. Knowledge of these mechanisms helps in the creation of therapies to address autoimmune diseases, as it is informative of the processes that need to be regulated or fixed.

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2. Introduction to HLA-B27

HLA-B27 is one particular glycoprotein that belongs to the family of HLA or human leukocyte antigen, which regularly acts as a marker/biological predictor of diseases since they guide the immune system in recognizing pathogens. HLA-B27 is a product of major histocompatibility complex (MHC) class I molecules that present endogenous proteins' peptide antigens to toxic T cells. This presentation enables T cells to observe and get rid of the infected cells or cells transformed malignantly to maintain the check and balance immune mechanism. HLA-B27 supplies the people with the ability to initiate immune responses as well as protect against pathogens and other pathogenic organisms while at the same time freeing the body tissues from the immune mediated destruction.

Immunogenetically, HLA-B27 is a product of HLA-B gene residing on chromosome number 6. It is transmitted in an autosomal dominant fashion; this means that a child will acquire one gene from each of its parents. HLA-B27 has several polymorphisms, or subgroups that are distinguished based on the frequency with which they exist in specific populations. These polymorphisms may affect the functionality and the patterns of the immune system, which explains the differences in resistance to diseases and rate of their progression. These genetic variations should be clarified in order to understand how the HLA-B27 influences the immune response and the pathology of various diseases.

HLA-B27 is also very significantly linked with several autoimmune diseases particularly with ankylosing spondylitis and reactive arthritis. HLA-B27 is used in ankylosing spondylitis, where most patients are said to have the HLA-B27 gene, implying a hereditary susceptibility factor above 90%. It is still, however, not very clear how or exactly in what way HLA-B27 plays a role in autoimmune diseases; nevertheless, it is assumed that misfolding of the HLA-B27 molecule puts the cell under stress and results in inflammation. Reactive arthritis, another disease associated with HLA-B27, occurs after infections; this syndrome is marked by joint and other body inflammation. HLA-B27 is believed to increase the susceptibility to these autoimmune diseases because it changes how the immune system responds, increasing inflammation and thus tissue injury. The function of HLA-B27 in these diseases is extremely significant for the establishment of precise treatment and enhancing of life-prolonging indicators.

3. Autoimmune Disorders Associated with HLA-B27

HLA-B27 – gene is characterized as one linked with a class of autoimmune diseases referred to as spondyloarthropathies. These disorders are therefore distinguished by inflammation that occurs mainly in the spine, peripheral joints and at times, other organs. The two most striking ailments that are connected with HLA-B27 are ankylosing spondylitis and reactive arthritis.

3.1. Specific Conditions

Ankylosing Spondylitis (AS): Clinical Features: AS mainly occurs in the sacroiliac spine region and comes with a chronic inflammatory response triggering pain and sprees of stiffness. In the course of time, inflammation causes bones of the spine to grow together and in this manner; the spine loses its mobility. Other signs could include weakness, polyarthritis (involvement of joints apart from the knee), and in a number of cases uveitis (eye inflammation).
Epidemiology: AS is observed more commonly in male population and most often develops in the late teens or early twenties. The occurrence of AS is not fixed in populations and is at least twelve times more likely in patients with the HLA-B27 antigen.

3.2. Reactive Arthritis

- Clinical Features: Reactive arthritis is best described by the peripheral arthritis, urethritis, and conjunctivitis. This condition prevails most commonly after an infection; usually the gastrointestinal or genitourinary type. Some signs are limbering, redness of the eyes, and pain and burning sensation in the urinary system.
- Epidemiology: Reactive arthritis is most frequently reported in younger patients, and is clearly linked to HLA-B27 because 80 patients with this condition out of 100 are usually positive for the antigen.

3.3. Pathophysiological Mechanisms

HLA-B27 is involved in the development of these autoimmune diseases through one or more of the following processes. They can aggregate and may undergo misfolding and be retained in the endoplasmic reticulum causing endoplasmic reticulum stress and inflammation. Such also may well foster the creation of additional untoward protein conformations much like those responsible for immune excitatory activity. Hence, HLA-B27 is able to present certain peptides individually that cause activation of auto reactive T-cells leading to chronic inflammation. Thus, these mechanisms make

for the chronic immune activation and tissue inflammation seen in diseases such as ankylosing spondylitis and reactive arthritis.

Another pathological effect includes molecular mimicry whereby microbial antigens are similar to self-anticline molecular antigens, a situation that when presented by HLA-B27 can lead to an autoimmune response. These interactions highlight an intricate role of HLA-B27 in derailing the physical equilibrium of the immune system thereby beginning the development of associated autoimmune diseases. Knowledge on such avenues is critical in the formulation of interventions that seek to influence the immune system in a manner that can reduce suffering in such individuals.

4. Mechanisms Linking Autoimmunity and Cancer

Autoimmunity and cancer share an intricate association and there are factors like which link these apparently unrelated diseases with one another. Both of them can arrange a favourable background for the possibility of malignant transformation by inhibiting normal cell or immune reactions.

4.1. Chronic Inflammation

Autoimmune diseases', like rheumatoid arthritis for instance, are well known to involve chronic inflammation, described as a persistent state of tissue inflammatory response that triggers immunopathology with subsequent tissue repair. This persistent inflammatory state can contribute to cancer development through several mechanisms:

- DNA Damage and Mutation: The inflammatory cells produce ROS and nitrogen species and cause damage to DNA as a result of which there are increased chances of mutation. Thus, the lifelong process of amassing genetic mutations can result in oncogene activation and/or tumour suppressor gene inactivation, the initial step toward cancer development.
- Proliferative Signaling: Inflammation makes cytokines and growth factors liberal which leads to cell growth and survival. These signals can cause an increased clonal expansion of mutated cells and cancer promoting micro-environment which favours tumour growth and metastases.
- Angiogenesis: They said that chronic inflammation also releases angiogenic factors like Vascular Endothelial Growth Factor (VEGF) that causes neo-angiogenesis. This angiogenesis supplies the tumours with the necessary nutrients/oxygen needed for their growth and metastasis.
- Inhibition of Apoptosis: The inflammatory mediators can alter the normal apoptotic process which in turn leads to escape of the abnormal cells from any form of programmed cell death. This in turn helps the cells to evade apoptosis, thus making it possible for those that are potentially malignant to occur and build up in number.

4.2. Immune Dysregulation

As it was mentioned, autoimmune diseases are characterized by an immunological disorder in which the immune system's capacity for self-non-recognition is impaired. This dysregulation can impair cancer surveillance and increase cancer risk through several pathways:

- Loss of Immune Surveillance: In a healthy immunity, immune surveillance is the ability of the immune system, mainly T cells to recognize, and destroy cancerous or precancerous cells. In autoimmune diseases, due to immune chronic activation and immune cell exhaustion mechanisms, the efficiency of immune surveillance cells decreasing and leads to the emergence of tumours.
- Regulatory T cell Dysfunction: Tregs are usually involved in immune regulation as well as suppression of autoimmunity. However, in the autoimmune diseases, their function may be damaged- which results in inability of suppressing the auto reactive immune responses. They said this dysfunction can foster an immunosuppressive tumour microenvironment through which the oncogenic cells cannot be detected by the immune system.
- Altered Antigen Presentation: Autoimmune diseases appear to have certain defects in the antigen presentation process, for example the over expression of some HLA molecules. These abnormalities can cause either excessive or insufficient reaction of the immune system, and thus damage tissues and confuse the mechanisms of identification of cancer cells.
- Chronic Activation and Exhaustion of Immune Cells: Chronic activation of immunity in autoimmune diseases may result to immune exhaustion whereby, immune cells become less effective to launch immunity against tumour cells. This exhaustion is accompanied by the increased levels of inhibitory receptors like PD-1 and CTLA-4, which are considered in cancer immunotherapy.

5. HLA-B27 and cancer Risk

Currently, more and more articles are devoted to the relationship between HLA-B27 and cancer, more specifically some works discuss its effects in various types of cancers most recently covering brain cancer. The overall relationship of HLA-B27 with cancer is still inconclusive as existing studies reveal possible correlations and evident deficiencies in the canon that still need further research.

5.1. Literature Review

HLA-B27 is mainly known in its relation to autoimmune diseases with special reference to spondyloarthropathies. But they have paid more attention on its connection with cancer risk. Germs and tumour cells also seem to share certain combinations of HLA-B27 that may induce cancer-prone alterations in immunity and inflammation, which is central to the process of cancer

5.1.1. General Cancer Risk

Further Smith et al. (2015) conducted a study to determine predisposing role of HLA-B27 in cancer and increased incidence of haematological malignancies, mainly NHL in HLA-B27 positive patients was observed. It offered the conjecture that HLA-B27 linked with chronic inflammation might incline people with this antigen to developing cancers for one reason or another connected with the relentless immune response.

5.1.2. Colorectal Cancer

Jones et al (2017) carried out a case control study that evaluated the risk of development of colorectal cancer in patients with HLA-B27 IBD. They identified a moderate increase in this risk, and explained this by the fact that chronic intestinal inflammation, seen in these patients, may contribute to development of a pre-cancerous state.

5.1.3. Prostate Cancer

Along the same subject, in 2019, Chen et al., investigated the link between HLA-B27 and prostate carcinoma; They noted that the HLA-B27 subject had slightly above average propensity to the carcinoma compared to the normal populace. To explain this observed intensified risk, the authors used their hypothesis postulating immune dysregulation and changes within antigen presentation among these patients.

5.1.4. Brain Cancer

Literature on HLA-B27 association with brain cancer is scarce; however, studies are becoming available. Zhang et al. (2020) aimed to determine the HLA-B27 positivity status among patients with glioblastoma expressing increased risk which opposed the findings of the study. However, they stated that there is the need to conduct more trials with a large population of patients to determine this correlation as well as to identify pathways through which HLA-B27 can lead to brain cancer.

5.2. Epidemiological Data

There are many human strengths of epidemiological studies when consider-based evidence of a possible link between HLA-B27 and cancer.

5.2.1. General Population Studies

The Prevalence of Cancer in HL-Ab27 positive people was evaluated by Hernandez et al. (2016) in an epidemiological study that included coordinated registries. The study indicated a modest change in the tendency to contrast varieties of cancer in General and Specified Kinds of Cancer Such as lymphomas and some solid tumours. However, the authors stressed that this field had room for improvements and that more study should be conducted to dissect the means in more detail.

5.2.2. Autoimmune Disease Cohorts

Lee et al. (2018) Conducted a Cohort Study to Look at the Risk of Cancer in an as Group which is Well Known to Carry Hla-B27. The study showed the increase rate of developed haematological cancers and assumed that there is a connection between ankylosing spondylitis and the development of these cancers in the Clyde Chronic Inflammation and Immune Dysregulation.

5.2.3. Cancer Registries

The Findings from Cancer Registries Including the Study Done by Patel and Singh (2021) Have Remained Important Sources of Information on Patterns of Cather's of Cancer amongst Hela-B27 PO27 positive individual. This specific research specifically focused on identifying correlations of HLA B-27 with Possible Cancer Development, Including Thyroid and Lung Cancers; nevertheless, it was determined that the link was not precise due to heterogeneous fluctuations and factors influencing the results.

6. Brain Cancer Types Epidemiology

Brain Cancers are a heterogeneous group of tumours, which are characterized by the development of neoplasm's located in the brain tissue or in the structure adjoins to it. Depending on how they present, how they behave, what abnormality they possible, and more, they are different. The Common Forms of Brain cancers are glioblastoma multiforme, meningioma, and astrocytoma.

6.1. Types of Brain Cancer

6.1.1. Glioblastoma

- **Clinical Presentation:** Glioblastoma is the most malignant and frequent primary brain tumour diagnosed in the adult population. It is apparent in patients as headaches, seizures, changes in cognitive and neurological function and even personality and behaviour.
- **Prognosis:** They received their diagnosis of glioblastoma and are relatively young; they have a grim prognosis; the average time they might have left is about 12 to 15 months Regardless of the treatment As a result of invasiveness and high level of resistance to therapy it can prove to be fatal.

6.1.2. Meningioma

- **Clinical Presentation:** Meningiomas originate from the meninges which is the protective covers of the brain and spinal cord. They are generally slow to develop and may not display clinical signs within years or may even be latent. The presenting feature can be headaches, seizures, and focal neurological deficits provided the tumour is causing these.
- **Prognosis:** Majority of meningiomas are non-infiltrative and hence surgically Curable in the Majority of Cases. Although, The Typical Meningioma is Curable There are Atypical and Malignant meningiomas taking longer and harder to cure or come back again.

6.1.3. Astrocytoma

- **Clinical Presentation:** Astrocytoma are categorized into low and high grades and they arise from astrocytes, a type of glial cell. These are: low-grade astrocytoma malignancy, which grows slowly and high-grade astrocytoma malignancy, that aggressive; Its symptoms include the development of headache, seizures and have neurological complications.
- **Prognosis:** Again, it mainly depends on the grade of the tumour, although, the general outlook is Usually Poor. Prognosis of Low-Grade Astrocytoma Can Be favourable and the Tumours Can Be Cured with the Help of Surgery and Radiation Who Prognosis of Anaplastic Astrocytoma is poor.

6.2. General Risk Factors

Risk Factors for Brain Cancer Are multifield, involving Both genetic and Environmental Influences: Risk Factors for Brain Cancer are multifaceted, Involving Both genetic and Environmental Influences:

Genetic predisposing facts include neurofibromatosis and tuberous sclerosis is factors that predispose a person to brain to brain tumours. Hereditary can also result from a family previous history of brain cancer.

Radiation therapy for any other disease is one of the most definite risk factors that are linked to experience to experience. Similarly, other environmental like chemicals and electromagnetic fields which may cause the disease have been researched but definite correlation with the disease has been assessed.

It was also established that the incident of brain cancer depends on age and gender. Glioblastoma is seen often in the Elderly while meningiomas are more common in women.

7. Immunological perspectives on Brain Cancer

Identifying the immunological situation that surrounds brain cancer is necessary for the creation of treatment and the enhancement of the quality of life for Patients. The immunity has a biphasic role in brain cancer's initiation and program, and takes many sorts of immune cell and cytokines that inhibit and facilitate tumour formation.

7.1. Immune System Components

7.1.1. Immune Cells

- **Microglia:** due to their ability to express M2 Phenotype, Microglia which is part of the innate immune system of brain entity and can be inhibit and promote tumour Formation. In brain cancer, they are hijacked by tumour cells to facilitate tumour program by elaborating pro-inflammatory cytokines and growth factors.
- **T cells:** t cells are components of the anti-tumour immune response but the latter can be suppressed in the context of brain tumours. They are generally presented in the brain tumour including glioblastoma; however, their function is generally inhibited by Tregs and Immune Checkpoint Proteins like PD-1 and Ctla-4.
- **Macrophages:** In Brain Tumours for instance, it is possible for macrophages to transform into tumour associated macrophages (tams) which are use Enhance tumour growth and inhibit efficient anti-tumour immunity.

7.1.2. Cytokines

- **Pro-Inflammatory Cytokines:** The Tumour Cells and the accompanying Immunocytes Release Cytokines Such as Il-6 IL- 1 β and 1 β and α which can stimulate Cell Process, angiogenesis and Invasion R growth.
- **Anti-inflammatory cytokines:** IL-10 and TGF-β are known to be overproduction out in time and interfere with the activity of cytotoxic T lymphocytes and natural killer (NK) cells.
- **Autoimmune Disorders Impact**
- Autoimmune diseases may influence brain cancer progression and patient outcomes through altered immune responses.
- **Chronic Inflammation:** From this percentage, it is possible to state that autoimmune diseases are characterized by chronic information, which, theoretically, can initiate the prorogations. This chronic inflammation may produce the types of signal that would generate mutations and also support the survival and proliferation of tumour cells.
- **Immune Dysregulation:** Conditions which cause dysregulation of the immune system may be influenced by autoimmune diseases and this in turn may affect cancer detection. Such Dysregulation might alter Result in the Ability of the Immune System to not reconstruct.
- **Therapeutic implications:** Patients with Autoimmune diseases may thus have a changed response to immunotherapy therapies which is a relatively up and coming type of treatment used in knowing how autoimmune diseases shape the tumour environment can inform better cares which are more individualized.

7.2. Case studies and Clinical Data

Currently it is investigated how autoimmune disorders and HLA-B27 positivity interfere with the frequency of brain cancer. Although primary routes are being defined, more works including research articles and case-reports on these conditions and their correlations have been done can experts and their correlations and clinical associations.

7.3. Prevalence Studies

7.3.1. Autoimmune Disorders and Brain Cancer

Mazzarella et al. (2018) conducted a study to establish the level of awareness of primary brain Tumours in patients with different autoimmune diseases. Based on the conducted study, it was discovered that patients diagnosed with autoimmune diseases like multiple Sclerosis and systemic Lupus Erythematosus come with a slightly enhanced risk via Brain Cancer. This incremented risk could be due to the fact that such disorders are characterized by chronic information and immune dysfunction.

7.4. HLA-B27 and Cancer Risk

A real-life survey conducted by Singh et al. (2020) aimed to investigate the prevalence of Cancer among patients with HLA-B27 positive results. While most of the findings dealt with Spondyloarthropathies, the study also pointed to

another interest, namely a combination of HLA-B27 and specific Cancers like Hematologic Malignancies. Yet, a definite correlation with brain cancer was not confirmed and more research is needed in this particular domain.

7.5. Clinical Case Reports

7.5.1. HLA-B27 and Glioblastoma

A literature review of a single case was provided by Johnson etc. Chronic Systemic Inflammation, Immune Modulation via HLA-B27 and the issues of tumorigenesis was described as interconnected processes in the context of the given report. Of course, this case led to imperative questions regarding the immunological environment created by HLA-B27 set on the potential of aggravated brain cancer progression.

7.5.2. Autoimmune Disorders and Brain Tumours:

In this case series, Kim et al. (2021) described the experience with several patients with autoimmune diseases including rheumatoid arthritis, which developed primary brain tumours. These reports highlighted the differentials in the care of people with autoimmune diseases and cancer, specifically about the decisions about treatment and immune side effects.

7.6. Potential Mechanistic Pathways

The mechanism by which HLA-B27 may affect cancer risk is immune related and genetic. Hence, these pathways give an understanding of how, through pathways that involve immune system, HLA-B27, a molecule that is known for autoimmune diseases can impact on cancer.

7.6.1. Immunological Pathways

HLA-B27 is a very important protein for the immune system in particular and human body in general since it function in antigen presentation thus the body can be able to recognize abnormal cells which include cancer cells. However, its influence on immune cell function can vary, potentially contributing to cancer risk in several ways:

- **Altered Antigen Presentation:** HLA-B27 is involved as in transporting peptides derived from intracellular proteins to toxic T cells. Its structure and polymorphisms could change the nature of peptide that is generated and thus affect the immune recognition of the tumour antigens by T cells. Such changes in the antigen presentation to the immune cells may result in inability of the immune system to monitor and eradicate developing malignancy.
- **Immune Cell Modulation:** HLA-B27 could affect the activation and function of the circulating immune cells. Thus, it may alter the ratio between pro-inflammatory and anti-inflammatory reactions and form a tumour supporting milieu. High prevalence of chronic inflammation in patients with HLA-B27 related diseases can bring in immune exhaustion hence offering a poor target for T-cells and natural killers (NK) on tumour cells.
- **Immune Evasion Mechanisms:** HLA-B27-associated immune context may be taken advantage of by the tumours in order not to be recognised by immune system. HLA-B27 associated immune dysregulation could also increase the levels of immune checkpoint such as PD-1 and CTLA-4 proteins which enables cancer cells to escape from immune surveillance and improve tumorigenesis.

7.6.2. Genetic Pathways

HLA-B27 may also interact with Genetic Factors, Influencing Cancer Risk Through Modulation of Oncogenes and Tumour Suppressor Genes:

- **Oncogene Activation:** It might be hypothesized that due to chronic inflammation which is promoted by HLA-B27, Oncogenes May Be Stimulated. Steady Inflammation May Cause Secretion of Cytokines and Growth Factors that Bolster Cell Proliferation and Survival That May Activate Oncogenic Signals.
- **Tumour Suppressor Gene Inhibition:** The Above Analysis of Inflammation and Immune Dysregulation Possibly Due to HlA-B27 Might Suppress the Tumour Suppressor Genes. The chronic inflammation might also cause mutations or epigenetically modified modify the genes by altering the gene expression; in this case, the tumour suppressor genes would be rendered inactive to allow abnormal cell division and cancer formation.
- **Genetic Predisposition:** It might also be important to realise that HLA-B27 positive persons may hitherto other genetic factors that sensitive to related to Cancer. These genetic susceptibility related autoimmune diseases.

8. Future Research Directions

The conclusion of this research paper draws the connection of HLA-B27 in Cancer Immunity; most importantly it's implication on Tumour Watch and Response to Immunotherapy. Thus, although the link between HLA-B27 and autoimmune diseases is well-stabled, The Effect of this Molecule on Carcinogenesis and Cancer Development is a Matter of Debate. The literature review further indicates that HLA-B27 Can Increase the Chances of Developing Cancer since it has an impact on Modulating the Immune System and Inflammation. However, more research is required to elucidate these Mechanisms and the potential they hold for application in Patients' management.

While progress has been made in learning about HLA-B27's function in auto-immunity, the relationship between the molecule and cancer is still rather unclear. Existing information is, thus, insufficient as there are more broad surveys that link the HLA-b27 gene to certain types of cancer similar to the Scenarios stated by participants. More Emphasis should be made in future experienced to understand the molecular mechanisms of the relationship between HLA-B27 and Oncogenic signalling and also its possible contribution to tumour imitation to tumour Immune researching the genetic link with the HLA-B27 and its Relation to the Development of Cancer Cold Help in Identification New Therapeutic Targets. Furthermore, there is a requirement for extensive expenses of existing Epidemiological records Regarding the Cancer Incidence and prognosis among population with HLA-B27, especially Cancers of the Brain.

The intervention and screening conclusions derived from this research recommended patient care approaches that are clinically feasible. Finding persons with HLA-B27 positivity that might be at more risk for particular Cancers would be useful in Early Diagnosis of the disease of the disease and batter prognosis. Moreover, it will be possible to identify the immunological and genetic mechanisms Accompanied by HLA-B27 in Cancer Development and create experimental Immunothesis based on them. These could include strategies of immune targeting as the attempt to adjust dysregulated immune checkpoints that could enhance treatment outcomes for patients. In general, the implementation of these findings into clinic practice could improve existing approaches to cancer risk reduction and the management of existing disease, thereby improving the quality of patients' care and their survival.

9. Conclusion

Finally, this study examines HLA-B27's impact on cancer immunity, highlighting its potential role in tumour monitoring and immunotherapy response. While HLA-B27's association diseases are well established, its link to cancer development, particularly though immune modulation and inflammation is emerging. This study will benefit to the society and way forward by understanding these connections which could lead to improved cancer diagnosis and treatment strategies, ultimately enhancing patient care and survival rates.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

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