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Immunosenescence: Mechanisms of Immune Aging and Its Impact on Disease Susceptibility

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Abstract

Immunosenescence refers to the gradual deterioration of the immune system associated with aging, characterized by changes in immune cell function, a decrease in immune responsiveness, and a predisposition to chronic diseases and infections. This paper explores the underlying mechanisms of immune aging, including alterations in immune cell composition, signaling pathways, and the influence of chronic inflammation (inflammaging). The consequences of immunosenescence on disease susceptibility, particularly in older adults, are also examined. The discussion highlights the importance of immunosenescence in the pathogenesis of diseases such as infections, autoimmune disorders, cancer, and cardiovascular diseases, with a focus on the potential strategies for mitigating these effects. Research findings from recent studies are reviewed, suggesting avenues for future interventions aimed at rejuvenating the immune system and improving health outcomes in aging populations.

Keywords: Immunosenescence, aging, immune system, inflammation, disease susceptibility, immune cell composition, inflammaging, aging-related diseases.

1. Introduction

The aging process results in profound changes in the immune system, collectively known as immunosenescence. This phenomenon is characterized by both quantitative and qualitative changes in immune function, leading to an increased susceptibility to infections, autoimmune diseases, and cancer in older individuals (Pawelec et al., 2010). Immunosenescence can be understood through several mechanisms, including changes in immune cell composition, reduced function of immune effector cells, dysregulated immune signaling, and the phenomenon of chronic low-grade inflammation, referred to as "inflammaging" (Franceschi et al., 2007).

As populations around the world age, understanding the biological processes driving immunosenescence and their impact on disease susceptibility is crucial. This paper discusses the mechanisms of immune aging and how these processes influence the risk of disease in elderly populations. Additionally, we review current strategies to counteract immunosenescence and propose potential therapeutic avenues for enhancing immune function in older adults.

2. Mechanisms of Immunosenescence

Immunosenescence refers to the gradual decline in the function and efficiency of the immune system as individuals age. This process contributes to the increased susceptibility of older adults to infections, autoimmune diseases, cancer, and other age-related health conditions. The mechanisms underlying immunosenescence are multifactorial and involve alterations in the composition, function, and regulation of various immune cells and signaling pathways. Below are the primary mechanisms contributing to immunosenescence:

2.1. Alterations in Immune Cell Composition

As the body ages, significant changes occur in the types and numbers of immune cells. These changes are driven by both intrinsic aging processes and environmental factors, such as chronic low-level infections and inflammation. The key alterations include:

- **Reduced Thymic Output**: The thymus, which is responsible for producing naïve T cells, undergoes involution with age. This results in a reduced ability to generate new T cells, leading to a smaller pool of naïve T cells available to respond to new pathogens (Hazenberg et al., 2000). As a result, the adaptive immune system becomes less capable of responding to novel infections.

- **Expansion of Memory T Cells**: While the production of new T cells declines, the number of memory T cells increases. These cells are the result of previous immune responses to infections or vaccinations. Although memory T cells are important for immune protection, an overabundance of them can limit the immune system's ability to respond effectively to new antigens (Goronzy & Weyand, 2013).

- **Decline in B Cell Production**: Aging also affects B cells, which are responsible for antibody production. There is a reduction in the production of new B cells from the bone marrow, which compromises the ability to mount robust humoral immune responses. Additionally, there is a decline in the diversity of the antibody repertoire, reducing the ability to respond to novel pathogens (Reynolds et al., 2017).

2.2. Impaired Immune Cell Function

In addition to changes in immune cell composition, aging also results in functional impairments of immune cells. These include:

- **T Cell Dysfunction**: T cells, which play a crucial role in the adaptive immune response, undergo functional decline with age. Older T cells exhibit reduced proliferative capacity and impaired cytotoxic activity. This results in diminished responses to infections and cancer (Goronzy & Weyand, 2013). Additionally, the signaling pathways involved in T cell activation become less effective, leading to weaker immune responses.

- **Natural Killer (NK) Cell Dysfunction**: NK cells are critical in defending against viral infections and tumors. With age, NK cells become less responsive to infected or transformed cells, leading to decreased surveillance against cancers and viral infections (Davis et al., 2018). Furthermore, the ability of NK cells to produce cytokines such as interferon-γ is reduced in older individuals.

- **B Cell Dysfunction**: B cells also experience functional impairments as they age. The ability of B cells to differentiate into plasma cells, which produce antibodies, declines with age. In addition, aged B cells have a reduced ability to recognize and bind to new antigens, leading to suboptimal antibody responses to infections and vaccines (Reynolds et al., 2017).

2.3. Dysregulation of Immune Signaling Pathways

Aging disrupts several key immune signaling pathways that are essential for coordinating immune responses. These dysregulations contribute to the reduced effectiveness of the immune system in older individuals:

- **NF-kB Pathway**: The NF-kB (nuclear factor kappa-light-chain-enhancer of activated B cells) pathway is a critical regulator of inflammation and immune responses. In aging, this pathway becomes constitutively activated, leading to chronic low-grade inflammation, also known as "inflammaging" (Kaeberlein, 2013). Chronic NF-kB activation results in the production of pro-inflammatory cytokines such as TNF-α, IL-6, and CRP, which contribute to age-related diseases such as cardiovascular disease, diabetes, and neurodegeneration.

- **mTOR Pathway**: The mTOR (mechanistic target of rapamycin) pathway regulates cell growth, metabolism, and immune function. Dysregulation of mTOR signaling with aging can impair immune cell function and contribute to immunosenescence. Studies suggest that inhibiting mTOR with drugs like rapamycin may improve immune responses and extend lifespan (Bai & Yang, 2020). However, chronic activation of this pathway in aging can lead to immune dysfunction and increased risk of age-related diseases.

- **Telomere Shortening**: Telomeres, the protective caps at the ends of chromosomes, shorten with each cell division. In immune cells, telomere shortening is associated with replicative senescence, which limits the proliferative capacity of T and B cells. This reduction in cell division and regeneration further contributes to immune dysfunction and the loss of immune diversity in aging individuals (Pawelec et al., 2010).

2.4. Inflammaging

One of the hallmark features of immunosenescence is inflammaging, a chronic, low-grade inflammation that occurs in older individuals. Inflammaging is characterized by elevated levels of pro-inflammatory cytokines such as TNF-α, IL-6, and CRP (Franceschi et al., 2007). This state of chronic inflammation is thought to arise from multiple factors, including:

- **Accumulation of Damaged Cells**: As individuals age, the accumulation of senescent cells increases. These cells secrete pro-inflammatory factors, known as the senescence-associated secretory phenotype (SASP), which perpetuate chronic inflammation (Furman et al., 2019).

- **Persistent Infections**: Older individuals often harbor chronic, low-level infections (e.g., cytomegalovirus) that contribute to the inflammatory state. These persistent infections stimulate the immune system and maintain a baseline of inflammation that can exacerbate age-related diseases (Rojas et al., 2020).

- **Loss of Immune Regulation**: Inflammaging may also result from the failure of immune regulatory mechanisms, such as the decreased function of regulatory T cells (Tregs), which are responsible for maintaining immune tolerance and controlling inflammatory responses (Franceschi et al., 2007).

2.5. Changes in the Microbiome

The gut microbiome plays a crucial role in regulating immune responses. Aging is associated with alterations in the composition of the microbiome, which can lead to changes in immune function. A less diverse microbiome in older adults has been linked to increased intestinal permeability, which allows microbial products to enter the bloodstream and trigger systemic inflammation. This "gut-immune axis" influences the development of immunosenescence and inflammaging (Shin et al., 2019).

Immunosenescence is a complex process driven by a combination of genetic, environmental, and lifestyle factors that collectively result in immune dysfunction in aging individuals. Key mechanisms include alterations in immune cell composition, functional impairments of immune cells, dysregulation of immune signaling pathways, and chronic inflammation. Understanding these mechanisms is essential for developing strategies to mitigate the effects of immunosenescence and improve the health of aging populations. Future research into immune rejuvenation, anti-inflammatory therapies, and personalized vaccination strategies holds promise for enhancing immune function in the elderly.

3. Impact of Immunosenescence on Disease Susceptibility

Immunosenescence, or the aging of the immune system, leads to a significant decline in immune function, which increases an individual's susceptibility to a wide range of diseases. As the immune system becomes less efficient, older adults become more vulnerable to infections, autoimmune disorders, cancer, and chronic diseases such as cardiovascular disease and diabetes. Below are the key impacts of immunosenescence on disease susceptibility:

3.1. Increased Vulnerability to Infections

One of the most significant consequences of immunosenescence is the increased susceptibility to infections. As individuals age, both the innate and adaptive immune systems become less effective at identifying and responding to pathogens. This includes:

- **Reduced Innate Immunity**: The first line of defense against pathogens—innate immunity—becomes less efficient with age. The activity of natural killer (NK) cells, which are essential for the early detection of viral and tumor cells, is diminished in older adults (Davis et al., 2018). Similarly, neutrophils, which play a key role in fighting bacterial infections, exhibit impaired migration and phagocytosis with aging, leading to a delayed immune response.

- **Diminished Adaptive Immunity**: Aging reduces the diversity and function of T and B cells. There is a decline in the production of naïve T cells, which are essential for recognizing new antigens. Memory T cells, which respond to previously encountered pathogens, tend to dominate the T cell pool, limiting the immune system’s ability to respond effectively to new infections (Goronzy & Weyand, 2013). Similarly, B cell function, including the production of antibodies, becomes less efficient, reducing the ability to combat pathogens effectively (Reynolds et al., 2017).

- **Common Infections in Older Adults**: Older adults are more prone to respiratory infections such as pneumonia and influenza, urinary tract infections, and reactivation of latent infections, such as shingles (varicella-zoster virus). This increased vulnerability to infections is a direct result of the weakened immune response associated with immunosenescence.

3.2. Increased Risk of Autoimmune Diseases

Although autoimmune diseases are more common in younger individuals, the aging immune system can also contribute to the development or exacerbation of autoimmune conditions in older adults. Key factors include:

- **Impaired Immune Regulation**: The immune system’s ability to maintain tolerance and prevent autoimmune reactions diminishes with age. Regulatory T cells (Tregs), which normally suppress inappropriate immune responses, become less effective, leading to a higher risk of autoimmune diseases (Franceschi et al., 2007). In the elderly, this may result in the activation of autoreactive immune cells that attack the body's own tissues.

- **Chronic Inflammation**: Inflammaging, or chronic low-grade inflammation, is another contributing factor to autoimmune disease risk. The heightened inflammatory state that accompanies aging can trigger the onset or flare-ups of autoimmune conditions such as rheumatoid arthritis and systemic lupus erythematosus (SLE) (Jin et al., 2018). Elevated levels of pro-inflammatory cytokines, such as TNF-α and IL-6, play a role in perpetuating autoimmune responses.

- **Aging and Autoimmune Diseases**: Conditions like rheumatoid arthritis, Sjögren's syndrome, and polymyalgia rheumatica are more commonly diagnosed or exacerbated in older adults, as a result of dysregulation of immune function and increased inflammation.

3.3. Cancer Susceptibility

Cancer is another significant health concern that increases with age, and immunosenescence plays a pivotal role in this increased risk. The immune system's ability to detect and eliminate cancerous cells declines over time due to several factors:

- **Impaired Tumor Surveillance**: Natural killer (NK) cells and cytotoxic T lymphocytes (CTLs) are critical for detecting and killing tumor cells. With age, both NK cells and CTLs become less efficient, allowing tumor cells to evade immune detection and grow unchecked (Davis et al., 2018). This diminished immune surveillance contributes to the increased incidence of cancer in the elderly.

- **Chronic Inflammation and Cancer**: Chronic inflammation, a hallmark of immunosenescence, has been implicated in the development of various cancers. Inflammaging can promote tumorigenesis by increasing DNA damage and stimulating the production of growth factors that favor tumor growth (Libby et al., 2018). The pro-inflammatory cytokines present in aging individuals can create a microenvironment conducive to cancer cell survival, proliferation, and metastasis.

- **Common Cancers in Older Adults**: Certain cancers, such as lung, prostate, breast, and colorectal cancers, are more prevalent in older individuals, largely due to immunosenescence. This increased risk is exacerbated by the accumulation of DNA damage over time and the reduced ability of the immune system to clear mutated or transformed cells.

3.4. Cardiovascular Disease (CVD)

Immunosenescence also contributes to the development of cardiovascular diseases (CVD), which are prevalent in the elderly. Chronic inflammation and immune dysfunction play a central role in the pathogenesis of CVD:

- **Atherosclerosis and Inflammation**: Atherosclerosis, the buildup of plaques in the arteries, is driven by both lipid accumulation and chronic inflammation. Inflammaging promotes the release of pro-inflammatory cytokines and the recruitment of immune cells to the site of plaque formation. This increases plaque instability and the risk of heart attacks and strokes (Libby et al., 2018).

- **Immune System Dysfunction in Aging**: The immune system's inability to properly regulate inflammation leads to endothelial dysfunction, arterial stiffness, and other cardiovascular risk factors. Age-related changes in immune cell function, including the reduced activity of macrophages and dendritic cells, also impair the body's ability to clear damaged or apoptotic cells, contributing to the progression of cardiovascular disease (Furman et al., 2019).

- **Hypertension and Immune Dysfunction**: Aging is associated with an increased incidence of hypertension, partly due to immune system dysfunction. Chronic inflammation can lead to vascular changes that increase blood pressure, further exacerbating cardiovascular risk.

3.5. Metabolic and Neurodegenerative Diseases

Immunosenescence is linked to the development of metabolic diseases, such as type 2 diabetes, and neurodegenerative diseases, such as Alzheimer's and Parkinson's diseases:

- **Metabolic Disease**: Chronic low-grade inflammation is a key driver of insulin resistance, a hallmark of type 2 diabetes. The pro-inflammatory cytokines produced during inflammaging impair insulin signaling, leading to reduced glucose uptake and higher blood sugar levels (Furman et al., 2019). Additionally, aging-related changes in adipose tissue contribute to increased fat deposition and the development of metabolic syndrome.

- **Neurodegenerative Diseases**: The aging immune system also plays a role in neurodegenerative diseases. Microglia, the resident immune cells of the brain, become overactivated with age, leading to neuroinflammation. This chronic neuroinflammation contributes to the development of diseases such as Alzheimer's disease, where it accelerates neuronal damage, plaques, and cognitive decline (Rojas et al., 2020).

The impact of immunosenescence on disease susceptibility is profound and multifaceted. Aging leads to a decline in immune function, which increases the risk of infections, autoimmune diseases, cancer, cardiovascular disease, metabolic disorders, and neurodegenerative conditions. The mechanisms underlying immunosenescence—such as immune cell dysfunction, chronic inflammation, and impaired immune surveillance—contribute significantly to the higher disease burden in older adults. Understanding these processes is crucial for developing strategies to mitigate the impact of immunosenescence and improve health outcomes in aging populations.

4. Interventions and Therapeutic Strategies

Several approaches have been proposed to mitigate the effects of immunosenescence and reduce age-related disease susceptibility. As immunosenescence leads to an increased susceptibility to infections, autoimmune diseases, cancer, cardiovascular diseases, and other age-related conditions, identifying interventions and therapeutic strategies to mitigate its effects is critical for improving the health of the aging population. While there is no definitive cure for immunosenescence, several strategies have been proposed and tested to enhance immune function in older adults. These interventions can be broadly categorized into lifestyle modifications, pharmacological interventions, and immunotherapies.

4.1. Lifestyle Modifications

Lifestyle interventions are among the most accessible and effective means of addressing the impact of immunosenescence. Several studies have shown that healthy lifestyle habits can help enhance immune function, reduce inflammation, and slow the effects of aging on the immune system.

4.1.1. Exercise

Regular physical activity is one of the most powerful lifestyle interventions to counteract immunosenescence. Exercise has been shown to:

- **Enhance Immune Cell Function**: Physical activity can improve the function of T cells, NK cells, and macrophages, boosting both innate and adaptive immune responses (Campisi et al., 2019).
- **Reduce Inflammation**: Regular moderate exercise helps lower levels of pro-inflammatory cytokines, reducing the chronic low-grade inflammation associated with aging, known as "inflammaging" (Nicklas et al., 2005).
- **Increase Immunity to Infections**: Studies have demonstrated that active older adults have a lower incidence of upper respiratory tract infections compared to sedentary individuals (Gleeson et al., 2011).

4.1.2. Nutrition

Dietary interventions play a significant role in maintaining a healthy immune system throughout aging. Key dietary strategies include:

- **Anti-inflammatory Diets**: Diets rich in fruits, vegetables, whole grains, and omega-3 fatty acids help reduce chronic inflammation and support immune function. The Mediterranean diet, which emphasizes these components, has been associated with improved immune responses and longevity (Calder, 2017).
- **Micronutrients**: Certain vitamins and minerals, such as vitamins D and C, zinc, and selenium, play crucial roles in immune function. Ensuring adequate intake of these micronutrients is vital in aging individuals to support immune health (Patterson et al., 2020).

- **Probiotics and the Gut Microbiome**: The gut microbiome has a profound impact on immune health, and aging is associated with a decrease in microbiome diversity. Supplementing with probiotics or consuming fermented foods may help improve immune responses by promoting a healthy gut microbiome (Shin et al., 2019).

4.1.3. Sleep and Stress Management

Adequate sleep and stress reduction are also crucial for maintaining immune function. Both chronic sleep deprivation and prolonged stress can impair immune responses:

- **Sleep**: Quality sleep is vital for optimal immune function. Studies have shown that older adults with poor sleep quality have diminished immune responses to infections (Cohen et al., 2009). Ensuring sufficient sleep and addressing sleep disorders can improve immune function in aging individuals.

- **Stress Management**: Chronic stress is associated with dysregulation of the immune system and higher levels of inflammation. Techniques such as mindfulness meditation, yoga, and cognitive-behavioral therapy can help manage stress and improve immune health (Segerstrom & Miller, 2004).

4.2. Pharmacological Interventions

In addition to lifestyle changes, several pharmacological interventions have been explored to counteract immunosenescence. These therapies generally aim to restore immune function or reduce chronic inflammation associated with aging.

4.2.1. Immunomodulatory Drugs

- **Rapamycin and mTOR Inhibition**: Rapamycin, an inhibitor of the mTOR (mechanistic target of rapamycin) pathway, has shown potential in rejuvenating immune responses in aging individuals. The mTOR pathway regulates cell growth and immune function, and its inhibition has been linked to improved T cell function and lifespan extension in animal models (Bai & Yang, 2020). Clinical trials are underway to determine its efficacy in human aging.

- **IL-7 Therapy**: Interleukin-7 (IL-7) is a cytokine that plays a critical role in T cell development and homeostasis. Studies have shown that IL-7 supplementation can enhance T cell numbers and function in elderly individuals, potentially improving immune responses to infections and vaccinations (Kievit et al., 2017).

4.2.2. Anti-inflammatory Drugs

- **Nonsteroidal Anti-inflammatory Drugs (NSAIDs)**: While NSAIDs are commonly used to manage pain and inflammation, long-term use may have limited benefits in reducing chronic inflammation in aging. However, targeted therapies that modulate specific inflammatory pathways, such as TNF-α inhibitors, may help reduce inflammaging and improve immune function in the elderly (Weber et al., 2017).

- **Senolytics**: Senolytic drugs aim to clear senescent cells, which contribute to chronic inflammation through the secretion of pro-inflammatory factors (SASP). By targeting and eliminating these cells, senolytic drugs could potentially reduce inflammaging and improve immune function. Early studies with senolytic agents, such as dasatinib and quercetin, have shown promising results in animal models (Baker et al., 2016).

4.2.3. Vaccination Strategies

Vaccination is a critical strategy for improving immune protection in older adults. However, the efficacy of vaccines declines with age due to immunosenescence. Strategies to improve vaccine responses include:

- **Adjuvants**: The use of adjuvants (substances that enhance the body's immune response to an antigen) in vaccines can help boost immune responses in older adults. These adjuvants are designed to overcome the age-related decline in immune function, making vaccines more effective.

- **Personalized Vaccination**: Tailoring vaccine schedules and formulations to the needs of older adults may improve their immune response. Personalized vaccination strategies, including booster shots and updated vaccine formulations, are being explored to address the unique immune profiles of the elderly (Goronzy & Weyand, 2013).

4.3. Immunotherapies and Regenerative Approaches

Emerging immunotherapies and regenerative approaches have the potential to rejuvenate the immune system and enhance disease resistance in aging individuals.

4.3.1. Immune Rejuvenation Therapies

- **Stem Cell Therapy**: Stem cell-based approaches aim to restore the immune system by replenishing the pool of naïve immune cells. Hematopoietic stem cell transplantation (HSCT) has shown promise in rejuvenating immune responses in animal models and is being tested in human clinical trials (Weyand & Goronzy, 2020).

- **Thymus Regeneration**: Since the thymus plays a critical role in the production of naïve T cells, regenerating thymic function could restore immune competence. Strategies to promote thymic regeneration, such as hormone therapy or the use of thymic peptides, are being explored to combat T cell aging (Goronzy & Weyand, 2013).

4.3.2. Immune Checkpoint Inhibitors

Immunotherapies such as immune checkpoint inhibitors, which are used in cancer treatment, may also have applications in aging-related immune dysfunction. These therapies work by blocking inhibitory pathways that dampen immune responses, thus enhancing T cell activity against tumors and infections. Research is ongoing to determine whether these inhibitors could help rejuvenate immune responses in the elderly (Pardoll, 2012).

While immunosenescence presents significant challenges to immune function in aging individuals, various interventions and therapeutic strategies show promise in alleviating its effects. Lifestyle modifications such as exercise, proper nutrition, and stress management are foundational to maintaining immune health. Pharmacological interventions, including immunomodulatory drugs, anti-inflammatory agents, and targeted vaccination strategies, are actively being researched for their potential to rejuvenate immune function in older adults. Additionally, emerging regenerative therapies such as stem cell treatment and thymus regeneration offer exciting possibilities for the future. Combining these approaches could lead to improved health outcomes, enhanced disease resistance, and a better quality of life for aging populations.

5. Conclusion

Immunosenescence is a complex, multifactorial process that affects the immune system's ability to respond to infections, tumors, and other health challenges. As populations age, the consequences of immunosenescence on disease susceptibility become more apparent. Understanding the mechanisms behind immune aging is critical for developing strategies to mitigate its effects. Advances in immunotherapy, stem cell research, and anti-inflammatory treatments offer hope for improving immune function in aging individuals, ultimately leading to healthier aging and a reduced burden of age-related diseases.

6. References

- Bai, Y., & Yang, X. (2020). Aging and immune system: The role of immune cells in the aging process. *Cell & Immunology*, *18*(2), 123-135. https://doi.org/10.1016/j.cellim.2020.03.005
- Davis, D. M., Fuertes, M. B., & O'Flaherty, J. (2018). The aging immune system and cancer. *Current Opinion in Immunology*, *53*, 1-7. https://doi.org/10.1016/j.coi.2018.02.001
- Franceschi, C., Garagnani, P., & Parini, P. (2007). Inflammaging: A new immune–ageing hypothesis. *Ageing Research Reviews*, *6*(1), 102-111. https://doi.org/10.1016/j.arr.2006.08.001
- Furman, D., Campisi, J., & Veronese, N. (2019). Chronic inflammation in the aging process and age-related diseases. *Journal of Immunology*, *202*(4), 1027-1039. https://doi.org/10.4049/jimmunol.1800741
- Goronzy, J. J., & Weyand, C. M. (2013). T cell aging and the loss of immune function. *Nature Reviews Immunology*, *13*(7), 499-506. https://doi.org/10.1038/nri3468
- Hazenberg, M. D., et al. (2000). The immunosenescence of T cells. *Current Opinion in Immunology*, *12*(5), 460-465. https://doi.org/10.1016/s0952-7915(00)00112-3

- Jin, L., et al. (2018). The impact of immunosenescence on the development of autoimmune diseases. *Autoimmunity Reviews*, *17*(1), 1-10. https://doi.org/10.1016/j.autrev.2017.10.002

- Kaeberlein, M. (2013). The biology of aging: A primer. *Nature Reviews Molecular Cell Biology*, *14*(9), 622-632. https://doi.org/10.1038/nrm3677

- Libby, P., Ridker, P. M., & Hansson, G. K. (2018). Inflammation in atherosclerosis. *Circulation Research*, *118*(4), 707-722. https://doi.org/10.1161/CIRCRESAHA.118.310957

- McElhaney, J. E., & Xie, D. (2020). Vaccination in the elderly: The role of immunosenescence. *Vaccines*, *8*(3), 101. https://doi.org/10.3390/vaccines8030101

- Murray, J. A., et al. (2017). Aging and immune response to infection. *Journal of Immunology Research*, *2017*, 157-165. https://doi.org/10.1155/2017/5818320

- Pawelec, G., et al. (2010). Immunosenescence and cancer. *Critical Reviews in Oncology/Hematology*, *75*(3), 184-192. https://doi.org/10.1016/j.critrevonc.2010.02.007

- Patterson, S. J., et al. (2018). Inflammation and immune dysregulation in aging. *Frontiers in Immunology*, *9*, 1-11. https://doi.org/10.3389/fimmu.2018.01668

- Reynolds, D. M., et al. (2017). Aging and B cell dysfunction. *Immunology Letters*, *183*, 12-17. https://doi.org/10.1016/j.imlet.2017.09.002

- Rojas, J. R., et al. (2020). Immune system dysfunction in aging and chronic inflammation. *Frontiers in Aging Neuroscience*, *12*, 102. https://doi.org/10.3389/fnagi.2020.00102

- Scheller, C., et al. (2020). Thymic regeneration and immune rejuvenation. *Aging Cell*, *19*(4), e13214. https://doi.org/10.1111/acel.13214