Open Access Mini-Review

Journal of Renal Endocrinology

J Ren Endocrinol 2025;11:e25196.
https://www.jrenendo.com
doi: 10.34172/jre.2025.25196

Nickan Research Institute

Obesity, diabetes, and cancer; a triad of interconnected risks and emerging evidence

Hafez Mohammadhassanzadeh[1], **Zeinab Aliyari Serej**[2*]

Abstract
Obesity has become a major risk factor for different types of cancer, and highlights research emphasizes the impact of inflammation related to obesity in this connection. Chronic low-grade inflammation associated with excess adipose tissue can disrupt metabolic signaling and immune responses, which may facilitate the tumorigenesis.
Keywords: Obesity, Inflammation, Type 2 diabetes, Cancer, Cytokines
Citation: Mohammadhassanzadeh H, Aliyari Serej Z. Obesity, diabetes, and cancer; a triad of interconnected risks and emerging evidence. J Ren Endocrinol. 2025;11:e25196. doi: 10.34172/jre.2025.25196.

Introduction

Obesity is associated with a state of chronic low-grade inflammation primarily due to dietary factors and excessive fat accumulation (1). Adipose tissue, especially visceral fat, secretes various pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), which contribute to systemic inflammation (2). This chronic inflammatory environment is implicated in several malignancies, containing breast tumor, colorectal cancer, and endometrial cancer (3). The inflammatory mediators released in obesity can promote cell proliferation, inhibit apoptosis, and lead to insulin resistance, further augmenting cancer risk (4). Numerous studies showed that, insulin is a potent growth factor that promotes cell proliferation and survival by activating several carcinogenic signaling pathways linked to cancer development, like the phosphoinositide 3-kinase (PI3K)/AKT and the mitogen-activated protein kinase (MAPK) pathways (5,6). Excess insulin in obesity or diabetes can bind to the insulin receptor (INSR) as well as insulin-like growth factor 1 receptor (IGF1R), which not only enhances cellular proliferation but also reduces apoptosis in various cell types, including cancer cells (7). Moreover, the interaction between insulin and insulin-like growth factors (IGFs) plays a significant role in cancer development (8). The IGF signaling pathway, particularly IGF-1, is known to stimulate cell growth and inhibit apoptotic pathways, contributing to both tumor initiation and progression (9). The overexpression of the IGF-1 receptor has been detected in various malignancies and is associated with a poor prognosis, underscoring the importance of insulin and IGF dysregulation in the context of diabetes-induced carcinogenesis (6). Meanwhile, insulin resistance contributes to a cascade of biochemical changes, including hyperglycemia and dyslipidemia, which further exacerbate cancer risk (10). Elevated blood glucose levels can provide a readily available energy source for rapidly proliferating cancer cells, a phenomenon often referred to as the Warburg effect, where cancer cells preferentially utilize glycolysis for energy production even in the presence of oxygen (11). Moreover, the metabolic disturbances associated with diabetes, such as dyslipidemia, can lead to the accumulation of lipid peroxides and free fatty acids, which have been interacted in promoting tumor cell proliferation and metastasis (12). This metabolic dysregulation underlines the importance of managing diabetes not only for the prevention of macrovascular and microvascular complications but also in mitigating cancer risk (13). This study sought into the specific mechanisms through which obesity-related inflammation (ORI) increases cancer risk, providing insights into the biological interplay between obesity and cancer development (14).

Search strategy

For this review, we conducted a search across several databases, including PubMed, Web of Science, EBSCO, Scopus, Google Scholar, the Directory of Open Access

Received: 11 Mar. 2025, Revised: 10 May 2025, Accepted: 31 May 2025, ePublished: 8 Jun. 2025

[1]Tabriz University of Medical Sciences, Tabriz, Iran. [2]Department of Applied Cell Sciences, Faculty of Advanced Sciences, Tabriz University of Medical Sciences, Tabriz, Iran.
***Corresponding Author:** Zeinab Aliyari Serej, Email: z_aliyari@yahoo.com

Implication for health policy/practice/research/ medical education

Obesity is recognized as a major public health concern, leading to various metabolic disorders and chronic diseases, comprising type 2 diabetes and cardiovascular disorders. In this condition the state of chronic low-grade inflammation is driven by various key cytokines, counting TNF-α, IL-6, leptin, IL-1β, and IL-10, which is triggered by excess adipose tissue

Journals (DOAJ), and Embase. We utilized various keywords such as "obesity," "inflammation," "type 2 diabetes," "cancer," and "cytokines."

Chronic inflammation in obesity

Prior studies showed that, obesity induces a state of chronic inflammation characterized by the infiltration of immune cells into adipose tissue, leading to the release of various pro-inflammatory cytokines, such as TNF-α, IL-6, and leptin, a hormone produced by adipocytes (15). These inflammatory mediators significantly modify the tumor microenvironment, creating conditions that favor cancer cell proliferation and survival (16). For instance, studies show that inflammation in adipose tissue alters the local extracellular matrix environment, promoting cancer progression through enhanced cell migration and invasion mechanisms (17). The inflammatory cytokines produced in obese individuals can also activate signaling pathways that are crucial for tumor development (18). For example, the activation of nuclear factor κB (NF-κB) and signal transducer and activator of transcription 3 due to persistent inflammation can lead to increased genes expression associated with cell growth, angiogenesis, and evasion of apoptosis, fostering a conducive environment for tumor initiation and progression (19). Additionally, the ORI contributes to genetic instability, which is a critical factor in cancer development (20). Immune infiltrates, particularly macrophages in the adipose tissue, produce ROS (reactive oxygen species) as part of the inflammatory response (21). Elevated levels of ROS cause oxidative stress, leading to DNA damage, mutations in oncogenes and tumor suppressor genes, and ultimately promoting the transformation of normal cells into cancerous ones (22). In individuals with obesity, the continuous proliferation of adipose tissue coupled with cellular stress from hypoxia results in an environment rife with genomic changes (23). Various studies have identified a correlation between obesity-related oxidative stress and mutations in the TP53 tumor suppressor gene as a crucial regulatory point in the cell cycle and apoptosis pathways (24). The mutations result in the loss of critical tumor suppression functions, thus leading to uncontrolled cell division and malignancy (25). Moreover, a significant aspect of obesity-related inflammation is its impact on immune system function (26). Chronic inflammation alters the balance between pro-inflammatory and anti-inflammatory responses, which can impair the immune system's ability to detect and eradicate emerging malignant cells (27). Meanwhile, inflammatory conditions promote the expansion of regulatory T cells (Tregs) and myeloid-derived suppressor cells (MDSCs), which dampen the anti-tumor immune responses by inhibiting the functions of cytotoxic T lymphocytes and natural killer (NK) cells (28). Furthermore, the increased levels of leptin can directly affect immune cell function (29). Leptin signaling not only enhances the proliferation of pro-inflammatory macrophages but also skews T cell populations toward an inflammatory phenotype that can exacerbate tumor growth while simultaneously reducing effective anti-tumor immunity (30,31). As a result, the altered immune environment in obesity decreases the efficacy of immune surveillance, allowing for the survival and expansion of malignant cells (30). Finally, obesity is closely linked to insulin resistance, a condition that can further promote cancer risk through multiple mechanisms (32). Insulin resistance leads to elevated circulating insulin levels, which can drive tumor cell proliferation due to insulin's mitogenic effects (33). Elevated insulin levels may enhance the bioavailability of IGF-1, which is implicated in promoting cell growth and inhibiting apoptosis and has been associated with breast, prostate, and colorectal cancers (8). Likewise, the inflammatory cytokines characteristic of obesity can independently contribute to insulin resistance, which complicates the metabolic landscape further (1). As an example, TNF-α and IL-6 released from inflamed adipose tissue can interfere with insulin signaling pathways, exacerbating insulin resistance and establishing a feedback loop that facilitates further weight gain and higher cancer risk (34).

Role of cytokines in ORC

Cytokines, are small proteins released by different cell types to communicate and orchestrate immune responses, play a noteworthy role in the inflammatory processes associated with obesity (35). TNF-α is one of the most critical pro-inflammatory cytokines related to obesity. It is primarily secreted by adipocytes and macrophages present in adipose tissue (18). In obese individuals, elevated levels of TNF-α have been linked to insulin resistance, a hallmark of type 2 diabetes (36). TNF-α mediates inflammation and promotes apoptosis in adipocytes, contributing to the dysregulation of metabolic processes (37). Furthermore, its persistent expression can impair insulin signaling by interfering with the action of insulin receptor substrates, exacerbating insulin resistance (38,39). The role of TNF-α in promoting inflammation reflects how adipose tissue behaves as an active endocrine organ, where increased levels of this cytokine contribute to the overall inflammatory milieu (40). This impact is particularly apparent in visceral fat, which is more metabolically active than subcutaneous fat and is associated with greater health risks. IL-6 is another cytokine significantly involved in the

inflammation associated with obesity (41). It is produced by some cells, involving adipocytes, macrophages, and T cells (42). Elevated levels of IL-6 are commonly found in obese cases which is accompanying by metabolic syndrome and insulin resistance (43). IL-6 plays a dual role: while it can promote inflammatory responses, it also has some anti-inflammatory properties under specific conditions (44). The cytokine is released in response to infection and injury and stimulates the production of acute-phase proteins from the liver (45). Chronic elevation of IL-6 due to obesity can lead to persistent inflammatory states that contribute to various obesity-related diseases, including cardiovascular disease and cancer (2,46). Notably, research suggests that IL-6 can induce pathways of energy utilization that impact fat metabolism, indicating its role in both inflammation and metabolism (47). Additionally, leptin is an adipokine primarily produced by adipocytes (48). It has a fundamental role in regulating energy balance and appetite (49). However, in the context of obesity, leptin levels are often elevated due to increased adipose tissue mass (50). This hyperleptinemia can lead to leptin resistance, where the normal physiological effects of leptin, such as appetite suppression, become diminished, leading to further weight gain (51). It should remember that, leptin also has pro-inflammatory effects, promoting the recruitment and activation of immune cells, and enhancing the secretion of other inflammatory cytokines (52,53). It serves as a mediator linking obesity with inflammation and metabolic dysregulation, creating a vicious cycle that exacerbates chronic inflammatory states within adipose tissue (53). Likewise, interleukin-1 beta (IL-1β), is a potent pro-inflammatory cytokine produced by activated macrophages and adipocytes (54). It plays an essential role in mediating inflammation and has been connected with insulin resistance in obesity (55). Besides, IL-1β induces the expression of other inflammatory mediators and can initiate an inflammatory cascade leading to the recruitment of additional immune cells to sites of chronic inflammation (56,57). In obesity, increased levels of IL-1β are associated with adipose tissue disorders and contribute to the development of insulin resistance (1). Clinical studies have indicated that targeting IL-1β could lead to improvements in glycemic control and body weight regulation in individuals with obesity-related diseases (58). In contrast to other cytokines that promote inflammation, IL-10 acts as an anti-inflammatory cytokine, playing a crucial role in limiting inflammatory responses and maintaining homeostasis (59). Produced by various immune cells, including Tregs and macrophages, IL-10 inhibits the synthesis of pro-inflammatory cytokines and promotes the survival of adipocyte progenitor cells (60,61). In the background of obesity, the levels of IL-10 can be negatively affected by the obesity-induced inflammatory state, diminishing its protective effects (62). A previous study suggests that bolstering IL-10 activity or levels could provide therapeutic benefits in managing obesity-related inflammation and metabolic dysfunction (63).

Cancer risks in diabetes and obese individuals

Type 2 diabetes, is accompanying by an increased risk of malignancy due to several mechanisms such as hyperinsulinemia and insulin resistance, since elevated insulin levels can activate insulin and IGF receptors on tumor cells, promoting their growth and survival (64). Meanwhile, diabetes and obesity create a pro-inflammatory environment through cytokines and adipokines, which can enhance tumor progression (65). Similarly, high glucose levels contribute to the oxidative stress and the formation of advanced glycation end-products (AGEs), which can damage cells and promote carcinogenesis (66). On the other hand, diabetes alters sex hormone levels, which may strengthen the potential of hormone-sensitive malignancies like breast and endometrial cancer (67). Finally, diabetes-associated processes, such as epithelial-to-mesenchymal transition (EMT), facilitate tumor invasion and metastasis (68,69). Several investigation found that, diabetes is strongly linked to an increased risk of various cancers (69). These studies showed that, hepatocellular carcinoma, likely due to chronic inflammation, insulin resistance, and fatty liver disease (70). Moreover, hyperglycemia and hyperinsulinemia are key contributors to the elevated risk of pancreatic cancer in diabetic patients (71). In addition, hormonal dysregulation and obesity, are correlated with enhanced endometrial malignancy risk (72). Similarly, postmenopausal women with diabetes have a greater risk of breast malignancy (73). Diabetic patients also prone to colorectal cancer, possibly through hyperinsulinemia and altered gut microbiota and lastly, kidney and bladder cancers have an increased risks, potentially due to chronic hyperglycemia and oxidative stress (4). Interestingly, diabetes is associated with a reduced risk of prostate cancer, though the reasons remain unclear (74).

Conclusion

Obesity-related inflammation significantly contributes to increased cancer risk through multiple intricate biological mechanisms. The chronic inflammatory environment alters the tumor microenvironment, induces genomic instability, impairs immune surveillance, and establishes insulin resistance, all of which play critical roles in cancer development. As obesity raise globally, understanding these underlying mechanisms can provide valuable insights for developing interventions aimed at mitigating obesity-associated cancer risks.

Authors' contribution

Conceptualization: Zeinab Aliyari Serej.
Investigation: Hafez Mohammadhassanzadeh.
Writing–original draft: Hafez Mohammadhassanzadeh.
Writing–review and editing: Zeinab Aliyari Serej.

Conflicts of interest

The authors declare that they have no competing interests.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the authors utilized Perplexity to refine grammar points and language style in writing. Subsequently, the authors thoroughly reviewed and edited the content as necessary, assuming full responsibility for the publication's content.

Ethical issues

The authors have fully adhered to ethical standards, including those related to plagiarism, data fabrication, and duplicate publication.

Funding/Support

None.

References

1. Rohm TV, Meier DT, Olefsky JM, Donath MY. Inflammation in obesity, diabetes, and related disorders. Immunity. 2022;55:31–55.
2. Divella R, De Luca R, Abbate I, Naglieri E, Daniele A. Obesity and cancer: the role of adipose tissue and adipo-cytokines-induced chronic inflammation. J Cancer. 2016;7:2346–59.
3. Hirano T. IL-6 in inflammation, autoimmunity and cancer. Int Immunol. 2021;33:127–48.
4. Gallagher EJ, LeRoith D. Obesity and Diabetes: The Increased Risk of Cancer and Cancer-Related Mortality. Physiol Rev. 2015;95:727–48.
5. Liu P, Cheng H, Roberts TM, Zhao JJ. Targeting the phosphoinositide 3-kinase pathway in cancer. Nat Rev Drug Discov. 2009;8:627–44.
6. Khan MZ, Zugaza JL, Torres Aleman I. The signaling landscape of insulin-like growth factor 1. J Biol Chem. 2025;301:108047.
7. Bowers LW, Rossi EL, O'Flanagan CH, deGraffenried LA, Hursting SD. The Role of the Insulin/IGF System in Cancer: Lessons Learned from Clinical Trials and the Energy Balance-Cancer Link. Front Endocrinol (Lausanne). 2015;6:77.
8. Brahmkhatri VP, Prasanna C, Atreya HS. Insulin-like growth factor system in cancer: novel targeted therapies. Biomed Res Int. 2015;2015:538019.
9. Werner H. The IGF1 Signaling Pathway: From Basic Concepts to Therapeutic Opportunities. International Journal of Molecular Sciences. 2023;24:14882.
10. Biadgo B, Tamir W, Ambachew S. Insulin-like Growth Factor and its Therapeutic Potential for Diabetes Complications - Mechanisms and Metabolic Links: A Review. Rev Diabet Stud. 2020;16:24–34.
11. Liberti MV, Locasale JW. The Warburg Effect: How Does it Benefit Cancer Cells? Trends Biochem Sci. 2016;41:211–8.
12. Broadfield LA, Pane AA, Talebi A, Swinnen JV, Fendt SM. Lipid metabolism in cancer: New perspectives and emerging mechanisms. Dev Cell. 2021;56:1363–93.
13. Yapislar H, Gurler EB. Management of Microcomplications of Diabetes Mellitus: Challenges, Current Trends, and Future Perspectives in Treatment. Biomedicines. 2024;12:1958.
14. Rakib A, Al Mamun MA, Mandal M, Sinha P, Singh UP. Obesity–cancer axis crosstalk: Molecular insights and therapeutic approaches. Acta Pharmaceutica Sinica B. 2025.
15. Engin A. The pathogenesis of obesity-associated adipose tissue inflammation. Obesity and lipotoxicity. 2017:221–45.
16. Divella R, De Luca R, Abbate I, Naglieri E, Daniele A. Obesity and cancer: the role of adipose tissue and adipo-cytokines-induced chronic inflammation. Journal of Cancer. 2016;7:2346.
17. Himbert C, Delphan M, Scherer D, Bowers LW, Hursting S, Ulrich CM. Signals from the Adipose Microenvironment and the Obesity-Cancer Link-A Systematic Review. Cancer Prev Res (Phila). 2017;10:494–506.
18. Uti DE, Atangwho IJ, Omang WA, Alum EU, Obeten UN, Udeozor PA, et al. Cytokines as Key Players in Obesity Low Grade Inflammation and Related Complications. Obesity Medicine. 2025;54:100585.
19. Yu H, Lin L, Zhang Z, Zhang H, Hu H. Targeting NF-κB pathway for the therapy of diseases: mechanism and clinical study. Signal Transduction and Targeted Therapy. 2020;5:209.
20. Ramos-Nino ME. The role of chronic inflammation in obesity-associated cancers. ISRN Oncol. 2013;2013:697521.
21. Vieira-Potter VJ. Inflammation and macrophage modulation in adipose tissues. Cell Microbiol. 2014;16:1484–92.
22. Srinivas US, Tan BWQ, Vellayappan BA, Jeyasekharan AD. ROS and the DNA damage response in cancer. Redox Biol. 2019;25:101084.
23. Trayhurn P. Hypoxia and adipose tissue function and dysfunction in obesity. Physiol Rev. 2013;93:1–21.
24. Jovanović M, Kovačević S, Brkljačić J, Djordjevic A. Oxidative Stress Linking Obesity and Cancer: Is Obesity a 'Radical Trigger' to Cancer? Int J Mol Sci. 2023;24:8452.
25. Sherr CJ. Principles of tumor suppression. Cell. 2004;116:235–46.
26. de Heredia FP, Gómez-Martínez S, Marcos A. Obesity, inflammation and the immune system. Proc Nutr Soc. 2012;71:332–8.
27. Tripathi S, Sharma Y, Kumar D. Unveiling the link between chronic inflammation and cancer. Metabolism Open. 2025;25:100347.
28. Lindau D, Gielen P, Kroesen M, Wesseling P, Adema GJ. The immunosuppressive tumour network: myeloid-derived suppressor cells, regulatory T cells and natural killer T cells. Immunology. 2013;138:105-15.
29. Kiernan K, MacIver NJ. The Role of the Adipokine Leptin in Immune Cell Function in Health and Disease. Front Immunol. 2020;11:622468.
30. Dudzinski SO, Bader JE, Beckermann KE, Young KL, Hongo R, Madden MZ, et al. Leptin Augments Antitumor Immunity in Obesity by Repolarizing Tumor-Associated Macrophages. J Immunol. 2021;207:3122-30.
31. de Candia P, Prattichizzo F, Garavelli S, Alviggi C, La Cava A, Matarese G. The pleiotropic roles of leptin in metabolism, immunity, and cancer. J Exp Med. 2021;218:e20191593.
32. Jee SH, Kim HJ, Lee J. Obesity, insulin resistance and cancer risk. Yonsei Med J. 2005;46:449–55.
33. Godsland IF. Insulin resistance and hyperinsulinaemia in the development and progression of cancer. Clin Sci (Lond). 2009;118:315–32.
34. Kern L, Mittenbühler MJ, Vesting AJ, Ostermann AL, Wunderlich CM, Wunderlich FT. Obesity-Induced TNFα and IL-6 Signaling: The Missing Link between Obesity and Inflammation-Driven Liver and Colorectal Cancers. Cancers (Basel). 2018;11:24.
35. De Heredia FP, Gómez-Martínez S, Marcos A. Obesity, inflammation and the immune system. Proceedings of the Nutrition Society. 2012;71:332–8.
36. Akash MSH, Rehman K, Liaqat A. Tumor Necrosis Factor-Alpha: Role in Development of Insulin Resistance and Pathogenesis of Type 2 Diabetes Mellitus. J Cell Biochem. 2018;119:105–10.
37. Cawthorn WP, Sethi JK. TNF-alpha and adipocyte biology. FEBS Lett. 2008;582:117–31.
38. Berbudi A, Khairani S, Tjahjadi AI. Interplay Between Insulin Resistance and Immune Dysregulation in Type 2 Diabetes Mellitus: Implications for Therapeutic Interventions.

Immunotargets Ther. 2025;14:359–82.
39. Skolnik EY, Marcusohn J. Inhibition of insulin receptor signaling by TNF: potential role in obesity and non-insulin-dependent diabetes mellitus. Cytokine Growth Factor Rev. 1996;7:161–73.
40. Smitka K, Marešová D. Adipose tissue as an endocrine organ: an update on pro-inflammatory and anti-inflammatory microenvironment. Prague medical report. 2015;116:87–111.
41. Popko K, Gorska E, Stelmaszczyk-Emmel A, Plywaczewski R, Stoklosa A, Gorecka D, et al. Proinflammatory cytokines Il-6 and TNF-α and the development of inflammation in obese subjects. European journal of medical research. 2010;15:1–3.
42. Han MS, White A, Perry RJ, Camporez JP, Hidalgo J, Shulman GI, et al. Regulation of adipose tissue inflammation by interleukin 6. Proc Natl Acad Sci U S A. 2020;117:2751–60.
43. Almuraikhy S, Kafienah W, Bashah M, Diboun I, Jaganjac M, Al-Khelaifi F, et al. Interleukin-6 induces impairment in human subcutaneous adipogenesis in obesity-associated insulin resistance. Diabetologia. 2016;59:2406–16.
44. Tanaka T, Narazaki M, Kishimoto T. IL-6 in inflammation, immunity, and disease. Cold Spring Harb Perspect Biol. 2014;6:a016295.
45. Ramadori G, Christ B. Cytokines and the hepatic acute-phase response. Semin Liver Dis. 1999;19:141-155. doi:10.1055/s-2007-1007106
46. Battineni G, Sagaro GG, Chintalapudi N, Amenta F, Tomassoni D, Tayebati SK. Impact of Obesity-Induced Inflammation on Cardiovascular Diseases (CVD). Int J Mol Sci2021;22:4798.
47. Covarrubias AJ, Horng T. IL-6 strikes a balance in metabolic inflammation. Cell Metab. 2014;19(6):898–9.
48. Picó C, Palou M, Pomar CA, Rodríguez AM, Palou A. Leptin as a key regulator of the adipose organ. Rev Endocr Metab Disord. 2022;23:13–30.
49. Van Dijk G. The role of leptin in the regulation of energy balance and adiposity. J Neuroendocrinol. 2001;13:913–21.
50. Stefanakis K, Upadhyay J, Ramirez-Cisneros A, Patel N, Sahai A, Mantzoros CS. Leptin physiology and pathophysiology in energy homeostasis, immune function, neuroendocrine regulation and bone health. Metabolism. 2024;161:156056.
51. Obradovic M, Sudar-Milovanovic E, Soskic S, Essack M, Arya S, Stewart AJ, et al. Leptin and Obesity: Role and Clinical Implication. Front Endocrinol (Lausanne). 2021;12:585887.
52. La Cava A. Leptin in inflammation and autoimmunity. Cytokine. 2017;98:51–8.
53. Pérez-Pérez A, Sánchez-Jiménez F, Vilariño-García T, Sánchez-Margalet V. Role of Leptin in Inflammation and Vice Versa. Int J Mol Sci. 2020;21:5887.
54. Bing C. Is interleukin-1β a culprit in macrophage-adipocyte crosstalk in obesity? Adipocyte. 2015;4:149–52.
55. Gao D, Madi M, Ding C, Fok M, Steele T, Ford C, et al. Interleukin-1β mediates macrophage-induced impairment of insulin signaling in human primary adipocytes. Am J Physiol Endocrinol Metab. 2014;307(3):E289–304.
56. Pyrillou K, Burzynski LC, Clarke MC. Alternative pathways of IL-1 activation, and its role in health and disease. Frontiers in immunology. 2020;11:613170.
57. Lopez-Castejon G, Brough D. Understanding the mechanism of IL-1β secretion. Cytokine Growth Factor Rev. 2011;22:189–95.
58. Osborn O, Brownell SE, Sanchez-Alavez M, Salomon D, Gram H, Bartfai T. Treatment with an Interleukin 1 beta antibody improves glycemic control in diet-induced obesity. Cytokine. 2008;44:141–8.
59. Iyer SS, Cheng G. Role of interleukin 10 transcriptional regulation in inflammation and autoimmune disease. Crit Rev Immunol. 2012;32:23–63.
60. Park J, Sohn JH, Han SM, Park YJ, Huh JY, Choe SS, et al. Adipocytes Are the Control Tower That Manages Adipose Tissue Immunity by Regulating Lipid Metabolism. Front Immunol. 2020;11:598566.
61. Liu R, Nikolajczyk BS. Tissue immune cells fuel obesity-associated inflammation in adipose tissue and beyond. Frontiers in immunology. 2019;10:1587.
62. Lai HC, Chen PH, Tang CH, Chen LW. IL-10 Enhances the Inhibitory Effect of Adipose-Derived Stromal Cells on Insulin Resistance/Liver Gluconeogenesis by Treg Cell Induction. Int J Mol Sci. 2024;25:8088.
63. Gkrinia EMM, Belančić A. The Mechanisms of Chronic Inflammation in Obesity and Potential Therapeutic Strategies: A Narrative Review. Current Issues in Molecular Biology. 2025;47:357.
64. Cohen DH, LeRoith D. Obesity, type 2 diabetes, and cancer: the insulin and IGF connection. Endocr Relat Cancer. 2012;19:F27-F45. doi:10.1530/ERC-11-0374
65. Kim DS, Scherer PE. Obesity, Diabetes, and Increased Cancer Progression. Diabetes Metab J. 2021;45:799–812.
66. Schröter D, Höhn A. Role of Advanced Glycation End Products in Carcinogenesis and their Therapeutic Implications. Curr Pharm Des. 2018;24:5245–51.
67. Joung KH, Jeong JW, Ku BJ. The association between type 2 diabetes mellitus and women cancer: the epidemiological evidences and putative mechanisms. Biomed Res Int. 2015;2015:920618.
68. Wang L, Bai Y-Y, Yang Y, Hu F, Wang Y, Yu Z, et al. Diabetes mellitus stimulates pancreatic cancer growth and epithelial-mesenchymal transition-mediated metastasis via a p38 MAPK pathway. Oncotarget. 2016;7:38539.
69. Suh S, Kim KW. Diabetes and cancer: is diabetes causally related to cancer? Diabetes Metab J. 2011;35:193–8.
70. Yu J, Shen J, Sun TT, Zhang X, Wong N. Obesity, insulin resistance, NASH and hepatocellular carcinoma. Semin Cancer Biol. 2013;23:483-491. doi:10.1016/j.semcancer.2013.07.003.
71. Li D. Diabetes and pancreatic cancer. Mol Carcinog. 2012;51:64–74.
72. Schmandt RE, Iglesias DA, Co NN, Lu KH. Understanding obesity and endometrial cancer risk: opportunities for prevention. Am J Obstet Gynecol. 2011;205:518–25.
73. Wang CY, Shih SR, Huang KC. Increasing risk of diabetes mellitus in postmenopausal women with newly diagnosed primary breast cancer. J Diabetes Investig. 2020;11:490–8.
74. Drab A, Wdowiak K, Kanadys W, Zajączkowski K, Koczkodaj P, et al. Diabetes Mellitus and Prostate Cancer Risk-A Systematic Review and Meta-Analysis. Cancers (Basel). 2024;16:4010. doi: 10.3390/cancers16234010.