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Peripheral Neural Regulation of Antitumor Immunity: Pro-tumor and Anti-tumor Mechanisms Across Nerve Subtypes and Cancer Types

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Abstract. With the research on the interaction between nerves and tumor cells, immune responses, etc. in the tumor microenvironment (TME), the three major neural substars (sympathetic nerve, parasympathetic nerve and sensory nerve) have all been confirmed to have both tumor-promoting and tumor-suppressing effects. The specific effects depend on the type of neurotransmitter, receptor subtype, target immune cell population and tumor background. Critically, tumors are not passive recipients of neural input: they actively remodel local innervation by secreting neurotrophic factors, thereby shaping the receptor landscape of the neural signals they subsequently receive. This article integrates the research on eight types of cancer, consolidates the findings into two major modules: tumor-promoting and tumor-suppressing neural mechanisms.

Keywords: cancer neuroscience, sympathetic nervous system, sensory nociceptors, parasympathetic innervation, neuro-immune crosstalk

1. Introduction

Peripheral nervous system has become an important functional component of tumor microenvironment [1]. The peripheral nervous system is mainly divided into three categories: sympathetic, parasympathetic and sensory nerves, all of which can infiltrate and innervating solid tumors and secrete neurotransmitters to interact with receptors expressed on cancer cells, immune cells, endothelial cells and cancer-associated fibroblasts [1]. Sympathetic nerves release norepinephrine, which acts on α-and β-adrenergic receptors; Parasympathetic nerve fibers mainly transmit signals through acetylcholine and muscarinic receptors. Sensory nociceptors release neuropeptides, mainly calcitonin gene-related peptide (CGRP) and substance P, which act on receptors such as RAMP1 [1].

Typically, the sympathetic nerve can inhibit the function of cytotoxic T cells and promote the infiltration of metastatic macrophages in breast cancer by releasing norepinephrine [2]. However, in melanoma, local sympathetic axons can restrict pro-tumor myeloid cells [3]. Cholinergic signals released by the parasympathetic nerve promote the invasion of prostate cancer and gastric cancer through muscarinic receptors [4], while vagus nerve input inhibits the tumorigenesis of pancreatic

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ductal adenocarcinoma [5]. Sensory nociceptors release calcitonin gene-related peptide (CGRP), which drives $CD8^+$ T cell exhaustion and blocks the assembly of tertiary lymphoid structures in melanoma, lung adenocarcinoma and gastric cancer [6-8]. However, there are also literature reports that in breast cancer, the destruction of sensory neurons in breast cancer accelerates metastasis [9].

This article summarizes previous studies on the neuro-tumor relationship, classifies the research findings into two major mechanism modules: tumor-promoting and tumor-suppressing, and explains the conditions that determine the direction of promotion or inhibition.

2. Pro-tumor neuro-immune mechanisms

2.1. Sympathetic nervous system: adrenergic promotion of metastasis and immune suppression

The sympathetic nervous system (SNS) is a branch of the autonomic nervous system, which releases catecholamines in response to physiological and psychological stress, mainly norepinephrine released from postganglionic nerve terminals and epinephrine released from adrenal medulla, acting on α and β adrenergic receptors expressed on the surface of immune cells, stromal cells and tumor cells in the tumor microenvironment.

Breast cancer studies have long revealed a potential mechanistic link between sympathetic nerve activity and tumor progression. Sloan et al. 's animal experiments found that stress-induced neuroendocrine activation in an orthotopic mouse model of breast cancer did not change the volume of the primary tumor, but increased the number of lymph nodes and lung metastases by about 30 times [2]. Such biologic effects are most likely regulated by β-adrenergic signaling. This pathway promoted the infiltration of $CD11b^+F4/80^+$ macrophages into the primary tumor parenchyma and induced the expression of multiple pro-metastasis genes with M2 polarization. If propranolol is used for drug blocking treatment, the above series of malignant changes can often be effectively reversed [2]. Existing reviews further confirm that this axis is widely involved in the progression of a variety of tumors. Norepinephrine can activate ADRB1 downstream signals on the surface of $CD8^+$T cells to initiate the exhaustion program, and catecholamines can also expand the population size of myeloid-derived suppressor cells to a certain extent [1].

Studies related to prostate cancer have also verified the role of sympathetic nerves in the regulation of early tumorigenesis. Data from Magnon et al. showed that sympathetic nerve fibers are able to drive the early initiation process of prostate tumors. Researchers can effectively inhibit early tumor proliferation in mouse models by chemical or surgical excision of sympathetic nerves or gene knockout of β2 and β3 adrenergic receptors on the surface of stromal cells [4]. The analysis results of clinical prostate adenocarcinoma specimens also confirm that the density of sympathetic nerve fibers in the tumor tissue may be related to the poor prognosis of patients [4]. These findings highlight the role of adrenergic signaling as an important driver of tumor cell survival and early growth. This regulatory mechanism has also been partially verified in the related studies of advanced melanoma and head and neck squamous cell carcinoma [1].

2.2. Parasympathetic nervous system: cholinergic promotion of invasion and metastasis

The parasympathetic nervous system (PNS) is another major branch of the autonomic nervous system, and acetylcholine (ACh) is mainly released by postganglionic fibers. The transmitter can act on nicotinic receptors and muscarinic receptors, the latter contains five subtypes of M1 - M5. The expression levels of the two types of receptors are significantly different in different cells and

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organs, which may partly explain the environment-dependent characteristics of cholinergic signaling in tumor biology.

The subsequent study by Magnon et al. added that prostate cancer tissues can be infiltrated by parasympathetic cholinergic fibers, which may be involved in promoting advanced malignant processes such as tumor invasion and distant metastasis [4]. This phenomenon is significantly different from the tumor-suppressing effect of vagus nerve denervation in pancreatic cancer, which is sufficient to indicate that acetylcholine, this single neurotransmitter, may produce opposite tumor regulatory effects by relying on different receptor subtypes and organ-specific biological characteristics [10]. Pharmacologic intervention or genetic knockout of the stromal cell muscarinic receptor type 1 (CHRM1) partially reduces metastatic disease in the pelvic lymph nodes, gut, and bone. The results suggest that the two major branches of the autonomic nervous system have complementary features and significant differences in the time sequence of action. Sympathetic nerves are mostly involved in the regulation of early tumor colonization and establishment, while parasympathetic nerves tend to mediate late tumor spread and metastasis [4].

Studies on gastric cancer have shown different regulatory mechanisms. Parasympathetic innervation mediated by vagus nerve can promote the occurrence and development of tumors with the help of specific muscarinic receptors. The study by Wang et al. confirmed that the vagus nerve is able to drive gastric cancer progression through Wnt signaling regulated by M3 muscarinic receptors in gastric stem cells. In mouse model experiments, M3 receptor knockout can significantly inhibit tumor proliferation and development [10]. Under this research system, gastric vagotomy can effectively slow down tumor growth. This phenomenon is obviously different from the tumor-suppressing effect of vagus nerve denervation in pancreatic cancer, which indicates that acetylcholine, a single neurotransmitter, may exert opposite tumor regulatory effects based on different receptor subtypes and organ-specific biological characteristics [10].

2.3. Sensory nociceptors: CGRP-mediated immunosuppression and TLS disruption

Sensory nociceptors are assigned to primary afferent neurons, and the population is dominated by fine-diameter C-fibers and Aδ fibers. Calcitonin gene-related peptide (CGRP) and substance P (SP) are highly expressed in these neurons, which can recognize a variety of noxious stimuli and release neuropeptides through peripheral and central synaptic terminals. After entering the tumor microenvironment, these neuropeptides can bind to specific receptors on the surface of immune cells and stromal cells, and this process does not require the initiation of traditional pain signaling pathways.

The tumor-promoting role of sensory nociceptor has been the most intensively studied area of tumor neuroscience in recent years. Based on the results of multiple studies on melanoma, gastric cancer, and lung adenocarcinoma, the CGRP-RAMP1 pathway can be regarded as a conserved core axis mediated by nociceptive receptors for tumor immune escape. This pathway may rely on various cell mediators such as $CD8^+$T cells, natural killer cells, and tumor-associated macrophages to exert different tumor-promoting effects in different tumors [6-8, 11]. A number of studies on different tumors have pointed to calcitonin gene-related peptide (CGRP) as the core immunosuppressive mediator in this neuroregulation process.

Related experiments in melanoma have revealed a complete neuroimmune regulatory pathway. Balood et al. found that melanoma cells can interact with nociception neurons, and such cell interactions can promote neurite proliferation and increase the release level of neuropeptides [6]. CGRP secreted by sensory nerve fibers can bind to RAMP1, a receptor component on the surface of $CD8^+$T cells, and further aggravate the exhaustion state of T cells. This process can be accompanied

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by the up-regulation of exhaustion markers such as PD-1, Lag-3 and Tim-3, and at the same time cause the loss of cellular interferon γ secretion function [6]. In the experiment, gene knockout of TRPV1 lineage neurons, or drug inhibition of nociceptor activity and blockade of RAMP1 function can alleviate the depletion phenotype of tumor-infiltrating leukocytes to a certain extent, and also increase the survival time of tumor-bearing mice by nearly three times [6]. The single-cell sequencing data of human melanoma biopsy samples also supports this mechanism. The depletion characteristics of RAMP1-expressing $CD8^+$T cells in the tumor are usually more significant, and the high expression of RAMP1 may be correlated with the poor prognosis of patients [6]. In addition to this, the study by Vats et al. also observed that sensory nerve fibers exert an inhibitory effect on the formation of tertiary lymphoid structures within melanoma. After removal of local sensory innervation, high endothelial venues in tumor tissue can gradually mature, effectively promoting the recruitment of immune cells and the orderly construction of tertiary lymphatic structures [7].

A similar neuroprotumorigenic effect was also observed in gastric cancer models. Zhi et al., through multiple sets of gastric cancer mouse models, found that in vivo nociceptive CGRP-positive peptideergic neurons can expand in number depending on nerve growth factor. Such neurons can directly form structural connections with gastric cancer spheres, and the CGRP released by them can bind to RAMP1 receptor on the surface of tumor cells, thereby promoting tumor proliferation and distant metastasis [8]. Drug removal of sensory neurons or intervention with CGRP inhibitor remmagepam can effectively curb tumor growth and prolong the survival time of model animals [8].

Recent studies in lung adenocarcinoma have further refined this regulatory network. A related study in 2026 pointed out that tumor progression can locally amplify the degree of invasion of nociceptive sensory nerves and continuously stimulate the release of CGRP. These neuropeptides can act on macrophage subsets in the tumor microenvironment, hinder the recruitment process of CXCL13 positive fibroblasts, and eventually interfere with the assembly and formation of tertiary lymphoid structures [11]. After local removal of sensory innervation, the formation ability of tertiary lymphoid structures in tumors can be restored, which can effectively activate the specific immune response mediated by B cells and T cells, and then inhibit tumor proliferation. For animal models exposed to cigarette smoke, drug blockade of CGRP signal can also improve the sensitivity of tumors to immunotherapy [11]. Based on the results of multiple studies on melanoma, gastric cancer and lung adenocarcinoma, the CGRP-RAMP1 pathway can be regarded as a conservative core axis of tumor immune escape mediated by nociceptors-tumor. This pathway may rely on a variety of cellular mediators such as $CD8^+$T cells, natural killer cells, and tumor-associated macrophages, and play a differential tumor-promoting role in different tumors [6-8, 11].

3. Anti-tumor neuro-immune mechanisms

3.1. Sympathetic nervous system: context-dependent local restriction of pro-tumor myeloid cells

In addition to the well-known immunosuppressive effects of the sympathetic nervous system, it has also been observed to produce anti-tumor activity in some studies. The realization of this kind of effect is related to the receptor subtypes and the anatomical distribution pattern of the nerves involved. An antitumor phenotype is more likely to be observed when sympathetic fibers function locally in the tumor lesion, and systemic effects mediated by circulating catecholamines are less likely to confer the same effect.

Some experimental evidence shows that sympathetic axons can regulate the homeostasis of myeloid cells in the tumor microenvironment and limit the accumulation of pro-tumor myeloid cells

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under certain conditions. Liu et al. carried out axonal innervation optical reconstruction experiments based on the cutaneous melanoma model, and found that sympathetic axons in the local tumor could regulate the number and spatial distribution pattern of tumor-promoting myeloid cells in the tumor microenvironment to a certain extent [3]. Studies have shown that tumor growth tends to be accelerated after surgical resection or drug depletion of sympathetic axons. Optogenetic stimulation of local sympathetic nerve fibers can effectively slow down the process of tumor proliferation. rescue experiments further confirmed that α2-adrenergic receptor agonists could reverse sympathetic exhaustion induced tumor growth acceleration. The functional activity of specific receptor subtypes, rather than the overall adrenergic tone, most likely dominates this biological effect [3]. This neuroprotective mechanism also showed obvious tumor volume dependence. With the increase of tumor volume, the density of sympathetic nerve fibers in the lesion will gradually decrease. The systemic adrenalin-mediated β-adrenergic pathway can play a role in promoting tumors at this stage, and different stages of tumor progression may be dominated by two sets of completely different neuroregulatory circuits [3].

Studies on pancreatic ductal adenocarcinoma show similar local neural regulation. Guillot et al. demonstrated with the help of 3D imaging that sympathetic axis bursting was able to reduce the infiltration of CD163-positive tumor-promoting macrophages within the tumor. Surgical sympathectomy will increase the population proportion of such macrophages and promote tumor proliferation and distant metastasis [12]. Mechanistically, sympathetic nerve fibers may inhibit the abnormal accumulation of M2-polarized myeloid cells in the tumor microenvironment. This protective regulation phenomenon is not shown in breast cancer and prostate cancer, and may even be masked by other pathway effects. The differences in the expression of cell receptor subtypes and the basic immune status of the tumor microenvironment may be the core reasons for the functional heterogeneity between tumors.

Sympathetic innervation may also exert additional antitumor effects by regulating the formation of tertiary lymphatic structures. The study by Riffard et al. found that loss of sympathetic innervation hinders the generation of tertiary lymphoid structures during acute pulmonary inflammatory responses. Sympathetic nerve signals may be able to build an effective tissue structure scaffold and provide basic conditions for the body to produce anti-tumor lymphatic immune responses [13].

3.2. Parasympathetic nervous system: vagal anti-tumor activity in pancreatic cancer

Parasympathetic vagal signaling has shown pro-tumor activity in prostate cancer and gastric cancer, and it has also been suggested that this signaling pathway can inhibit the progression of at least one type of solid tumors. The differentiation of functional phenotypes is largely determined by the expression profile of receptor isoforms in different organs, and the neurotransmitters themselves do not change at a fundamental level.

The anti-tumor effect of parasympathetic signaling has been most fully verified in pancreatic ductal adenocarcinoma, and this regulatory mode is significantly different from the pro-tumor phenotype in prostate cancer and gastric cancer. Renz et al. used gene-edited mice as a study model and found that the subphrenic vagotomy procedure accelerated the lesion progression of pancreatic ductal adenocarcinoma. Systemic administration of carbamylmethacholine, a muscarinic agonist, can reshape the normal tumor growth rhythm and effectively alleviate the malignant progression of tumors caused by vagus nerve loss [5]. For the established pancreatic ductal adenocarcinoma lesions, carbamylmethacholine intervention can also prolong the survival time of model mice to a certain extent [5]. These studies suggest that vagal innervation of pancreatic tissue transmits a

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sustained tumor-suppressive signal through muscarinic receptors. Most likely, this pathway relies on TNF-α in the tumor microenvironment to complete immune regulation. Drugs mimic the physiological activity of vagus nerve and can reproduce this kind of nerve-mediated tumor protective effect [5].

4. Cross-cancer summary: neural effect direction by nerve subtype and tumor type

The studies reviewed above reveal a consistent pattern: no single nerve subtype has a fixed pro- or anti-tumor designation across cancer types. Table 1 summarizes the documented directional effects:

Table 1. Context-dependent neuro-immune regulation: pro-tumor and anti-tumor effects by nerve subtype and cancer type

Cancer Type	Sympathetic	Parasympathetic	Sensory (CGRP/SP)
Breast cancer	Pro-tumor (β-AR → M2-TAM, metastasis) [2]	Anti-tumor (Nat Neurosci 2019)	Context-dependent
Prostate cancer	Pro-tumor (early; β2/β3-AR → tumor initiation) [4]	Pro-tumor (late; M1R → invasion/metastasis) [4]	Limited data
Melanoma	Anti-tumor early → Pro-tumor late (α2/β-AR switch) [3]	Limited data	Pro-tumor (CGRP→CD8 exhaustion, ↓TLS) [6, 7]
Pancreatic cancer	Anti-tumor (α-AR → ↓M2-TAM) [12]	Anti-tumor (muscarinic→tumor suppression) [5]	Pro-tumor (inflammation → early progression)
Gastric cancer	Limited data	Pro-tumor (M3R→Wnt signaling) [10]	Pro-tumor (CGRP–RAMP1→tumor growth) [8]
Lung adenocarcinoma	Promotes TLS (some conditions) [13]	Limited data	Pro-tumor (CGRP→↓TLS→immune evasion) [11]

The receptor subtype engaged, rather than the nerve subtype per se, is a consistent determinant of directional outcome. β-adrenergic receptors are predominantly linked to pro-tumor outcomes, while α2-adrenergic receptors show anti-tumor activity in melanoma and pancreatic cancer [3, 12]. Muscarinic M3 receptors drive gastric tumorigenesis while vagal M-receptor signaling broadly suppresses pancreatic cancer [5, 10].

5. Conclusion

Various research evidences in this paper show that peripheral nerves can actively participate in the regulation of anti-tumor immunity. The classification of neural subtypes alone cannot determine their overall effect on tumor progression. Sympathetic nerve, parasympathetic nerve and sensory nociceptor fibers can mediate bidirectional immune regulatory pathways, which can not only help tumor immunosuppression, but also enhance the body's anti-tumor immune response. The combination of various neurotransmitters, receptor subtypes, target immune cell types, primary tumor organs and disease progression stages may jointly determine the final direction of neuromodulation in tumors.

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A number of common regulatory laws can integrate the conclusions of this review to establish a logical and coherent neuroimmune regulation framework. CGRP shows stable immunosuppressive properties in a variety of tumor systems, and is the most consistent tumor-promoting neuropeptide. This molecule can be regulated by a variety of cellular mediators such as $CD8^+$T cells, macrophages and $CXCL13^+$ fibroblasts, and the specific form of action may change with the difference of tumor microenvironment [6-8, 11]. The sympathetic nervous system shows significant temporal plasticity, and its functional output will dynamically switch with tumor progression. In the same tumor, as the tumor burden continues to increase, the host-dominated adrenergic signaling pathways will gradually change. In the early stage, the α2-adrenergic receptor-mediated protective anti-tumor signaling is the main signal, and in the later stage, the β-adrenergic receptor-dominated tumor-promoting pathway will be the main signal. The tumor regulatory function of acetylcholine also has obvious organ heterogeneity. This neurotransmitter can promote the invasion of prostate cancer by activating the M1 receptor, participate in driving the occurrence and development of gastric cancer by means of the M3 receptor, and inhibit the malignant progression of pancreatic ductal adenocarcinoma through a unique muscarinic signaling pathway [4, 5, 10]. Taken together, it can be found that the final direction of the neuroimmune response depends more on the combination of receptor subtypes activated by the lesion than on the original classification of nerve fibers. This mechanistic feature may explain the existing clinical phenomenon. Non-selective neuromodulation interventions, such as broad-spectrum β-receptor blocking drugs, show different efficacy in clinical trials of different cancer types, and this heterogeneous outcome may be due to the non-specific interference of drugs on the dual neural circuits of tumor promotion and inhibition in vivo [14, 15].

There are still some research limitations in this review. At present, most of the evidence for the mechanism of neuroimmune regulation comes from animal mouse models, and the evidence for direct causal research in human tumor tissues is still relatively scarce. This study only covered some common cancer types, but not all cancer types. At the same time, there is still a lack of systematic and complete explanation of the synergistic or antagonistic interaction between different neural subtypes in the tumor microenvironment. Future studies can further analyze the spatial distribution relationship between nerve fiber subtypes and immune cell populations within a single tumor, and clarify the dominant receptor subtype combination pattern in different disease stages. Relevant research results may promote the transformation of neuroregulatory mechanisms and provide more scientific and reasonable targeted intervention strategies for the field of immuno-oncology [16].

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