INTERNATIONAL JOURNAL OF MEDICAL SCIENCE AND DENTAL HEALTH
(ISSN: 2454-4191)
www.ijmsdh.org (2026)

Received: 26 January 2026
Revised: 19 February 2026
Accepted: 28 March 2026
Published: 29 April 2026

Page No - 179-185
DOI - 10.55640/ijmsdh-12-04-20

Article Citation: Nasser, Z. A. A. (2026). The Biochemistry, Physiological Actions and Clinical Importance of Dopamine: A Review. International Journal of Medical Science and Dental Health, 12(04), 179-185. https://doi.org/10.55640/ijmsdh-12-04-20

The Biochemistry, Physiological Actions and Clinical Importance of Dopamine: A Review

Zainab Abdul Abbas Nasser
Department of Pathological Analysis, Faculty of Science, Kufa University, Najaf, Iraq

Abstract

Dopamine (DA) have been an essential catecholamine neurotransmitter that mediates many biochemical, physiological and pathological functions. This review summarizes what we currently know about the biochemistry, physiology and clinical significance of dopamine, with special emphasis placed on new insights. Dopamine is synthesized from tyrosine via a two-step enzymatic conversion catalyzed by tyrosine hydroxylase first followed by aromatic L-amino acid decarboxylase. Its life-cycle—vesicular storage, exocytosis, reuptake and degradation though monoamine oxidase and catechol-O-methyltransferase—is under precise control. As a neurophysiological neurotransmitter, dopamine have been essential for controlling movement, processes of thought, reward pathways and hormonal balance through different neural circuits and specific receptor subtype. It also plays a role in cardiovascular, renal, and immune system function outside the brain. Clinically, weakened dopaminergic signaling has been related with many disorders such as Parkinson's disease, schizophrenia depression and ADHD; therefore, it is a target of drug development. These recent advances are substantially enhancing our knowledge towards dopamine contributions to disease, especially its involvement in immune regulation and oxidative stress. Nevertheless, the complete organizational complexity of dopaminergic networks and their cross-biological system interactions remain incompletely characterized, a challenge that survives even decades of research. Dopamine is, in conclusion, integrator of many physiological system and further studies on dopamine will lead to new treatment strategies with improved effectiveness and specificity (even on many different disease).

Keywords: Dpamine, ADHD, β-lactam antibiotics

Introduction

Dopamine is a key catecholamine neurotransmitter, integral to many cellular and biochemical processes in the central nervous system (CNS) and other tissues. Dopamine is synthesized from the amino acid tyrosine through a sequence of enzymatic reactions and present in separate neural circuits, namely nigrostriatal, mesolimbic, mesocortical and tuberoinfundibular systems with each system mediating specialized functions. The role of dopamine in motor control and reward-related mechanisms has traditionally been predominant, but recent literature demonstrates that it is also heavily involved in cognition, endocrine regulation, immunomodulation, and behavior (Franco et al., 2021).

Dopamine acts at the biochemical level by binding to 5 subtypes of G protein-coupled receptors (D1–D5), which cluster into two major families: D1-like and D2-like. They regulate intracellular signaling pathways, such as those involved in cyclic adenosine monophosphate (cAMP)-dependent and independent cascades that modulate neuronal excitability, synaptic plasticity, and gene transcription (Beaulieu et al., 2015). Advances in receptor pharmacology have identified the complexity of dopamine signaling, including receptor heteromerization and non-canonical pathways, that can affect overall physiology leading to fine-tuning physiological responses and possibly providing new therapeutic targets (Missale et al., 2011).

Dopamine is physiologically an important neurotransmitter, which for voluntary movement, motivation and reward and reinforcement learning. Research focusing on dopamine as a signal compound in the internalization of rewards and the risk of neuronal addiction has predominantly focused on the mesolimbic dopamine pathway. Dopamine release along this pathway is a biological correlate of salience; increased dopamine is not only a signal of salience but conveys the relative importance of stimuli, and rewards and strengthens behavior, thereby optimizing opportunities for survival and well-being. Aberrant dopamine signalling in this pathway has been implicated in a variety of psychiatric and neurological diseases, and therefore this circuit is of particular interest to neuroscience. Dopamine also affects executive functions, including attention, decision-making and working memory in the prefrontal cortex (Lauretani et al., 2024). In addition to its role as a neurotransmitter, dopamine also exerts effects beyond the CNS and thus contributes to peripheral systems, including cardiovascular function (Björklund and Dunnett, 2007), renal function (Eckhardt et al., 1999), and hormonal secretion by inhibiting prolactin release in the anterior pituitary (Qi-Lytle et al.

Recent studies unveiled the ability of dopamine to act beyond classical neurotransmitter and emphasized its role as a neuromodulator acting on immune function during systemic homeostasis, thus, broadening the perspective about this well-known biogenic amine. Various immune cells (including lymphocytes and macrophages) express dopamine receptors, which modulate the cytokine secretion and influence inflammation. This new area of study, called neuroimmunology, reflects the role of dopamine in connecting the nervous system and immune system to cause the pathogenesis of inflammatory as well as autoimmune diseases (Channer et al., 2023). In addition, dopamine is also involved in oxidative stress mechanisms possibly by changing dopamine metabolism and increasing the production of reactive oxygen species resulting in neuronal damage and neurodegenerative disorders (Hritcu et al., 2015).

Both increases and decreases in dopaminergic signaling have physiological relevance, with alterations often being associated with a myriad of neuropsychiatric disorders clinically. For example, dopamine depletion in the nigrostriatal pathway is a central lesion in Parkinson's disease. Most of these symptoms that constitute the clinical aspects of the disease origin from the loss of dopaminergic transmission: these aspects include resting tremor, muscle rigidity, bradykinesia and postural instability such that movement is almost impossible and quality of life is extremely impaired. Hyperactivity of dopamine transmission in mesolimbic circuits has been thought to play an important role in schizophrenia and psychotic states. Dopamine signaling is extensively dysregulated in mood disorders, attention-deficit hyperactivity disorder (ADHD), and substance use disorders, proving its notable clinical relevance (Missale et al., 2011; Lauretani et al, 2024).

An increasing breadth of research highlights dopamine's role in maintaining physiological homeostasis and identifies it as a novel therapeutic target. Recent developments in cellular and molecular biology, neuroimaging, and pharmacology have greatly increased our understanding of dopaminergic systems and enabled the development of more selective compounds acting on dopamine receptors and transporters. Progress notwithstanding, major epistemic gaps remain in our understanding of the biology and integrative functions of dopamine at the level of multiple organ systems and how it contributes to the multifactorial processes of the diseases it is implicated in.

Thus, this review presents an up-to-date summary of dopaminergic biochemistry and physiology with an emphasis on recent discoveries and their potential clinical implications. Understanding the mechanisms that regulate these processes will

facilitate the rational design of new therapeutic approaches focused on dopaminergic circuits in defined patient populations in diverse disease settings.

Biochemical structure and metabolism od dopamine

Dopamine is a biologically active catecholamine that is one of the monoamines (a family of neurotransmitters with an amine side chain) containing a catechol nucleus, a para phenylbenzene ring with two hydroxyl substituents. This molecular architecture, formally characterized as 3,4-dihydroxyphenethylamine, determines the reactivity, receptor binding, and metabolic disposition of dopamine. Dihydroxyl substituents on the aromatic ring make dopamine especially prone to oxidation, a feature that is of functional importance, as well as pathological relevance. Due to that structural characteristic, dopamine is capable to enter redox reactions, a fact that will result in oxidative stress and neurotoxicity when this neurotransmitter is overproduced or poorly cleared (Meiser et al. 2013).

Dopamine biosynthesis starts from the physiologic amino acid tyrosine, either directly from the diet or produced from phenylalanine by phenylalanine hydroxylase. Tyrosine hydroxylase (TH), the rate-limiting enzyme in this pathway, catalyzes the conversion of tyrosine into L-3,4-dihydroxyphenylalanine (L-DOPA) in an oxygen- and tetrahydrobiopterin (BH4)-dependent reaction. Then, by aromatic L-amino acid decarboxylase (AADC, also called DOPA decarboxylase), L-DOPA is rapidly decarboxylated to dopamine. This step takes place in the cytosol of dopaminergic neurons and peripheral tissues (Daubner et al.

Following synthesis, dopamine is sequestered from cytosolic pools into synaptic vesicles by the vesicular monoamine transporter 2 (VMAT2), where it is concentrated and maintained in a readily releasable pool until release. In addition to its importance for regulated neurotransmission, vesicular storage is critical during the cytosolic lifetime of dopamine molecule itself to prevent enzymatic and spontaneous oxidation. In response to stimulation of cells, dopamine is released into the synaptic cleft by calcium-dependent exocytosis where it can bind to postsynaptic dopamine receptors or presynaptic autoreceptors to exert its physiological effects (Eisenhofer et al., 2004).

Dopamine signalling is mainly terminated by reuptake and enzymatic degradation. Dopamine is quickly removed from the synaptic cleft by dopamine transporter (DAT), a high-affinity membrane protein which plays a very important role by enabling the re-uptake of this neurotransmitter into presynaptic neurons. After entering into the neuron, dopamine will then be either repackaged by vesicles or degraded to metabolic pathways. Dopamine catabolism is regulated by two principal enzymes, monoamine oxidase (MAO) and catechol-O-methyltransferase (COMT). MAO is an enzyme that resides in the outer mitochondrial membrane and catalyses the oxidative deamination of dopamine into 3,4-dihydroxyphenylacetaldehyde (DOPAL), a reactive substrate with potential toxic properties. Aldehyde dehydrogenase then converts DOPAL to 3,4-dihydroxyphenylacetic acid (DOPAC). On the other hand, COMT methylates dopamine or its metabolites and gives rise to substances like 3-methoxytyramine (3-MT) and homovanillic acid (HVA), being HVA the principal end metabolite of dopamine in human beings (Meiser et al., 2013; Eisenhofer et al., 2004).

Dopamine metabolism is tightly regulated as dysregulation may cause generation of toxic products and ultimately oxidative stress. Intracellular degradation of dopamine by monoamine oxidase (MAO) yields hydrogen peroxide (H_2O_2), a reactive oxygen species that, if not sufficiently eliminated by the antioxidant defenses catalase and glutathione peroxidase, may be toxic to surrounding cells. In this context, dopamine can also auto-oxidize, in the absence of enzymes, spontaneously yielding proteins, lipids and nucleic acids covalently modified quinones and semiquinone. Of unique interest, these processes have been shown to be at the highest order of competition in the pathogenesis of neurodegenerative disorders, such as those relevant to Parkinson's pathogeneses (Blesa et al., 2015).

Dopamine's central metabolism is well documented, but peripheral tissue such as kidneys, gastrointestinal tract and immune cells can also synthesize and metabolize dopamine. Whereas, peripheral dopamine is a local modulator for physiological functions such as sodium excretion, vascular tone and immunological response. With respect to peripheral dopamine metabolism, the enzymatic pathways involved appear comparable to those in the CNS; exceptions can be expected based on tissue-specific or physiological conditions (Eisenhofer et al., 2004).

Improved techniques for molecular biology and analysis have revealed new knowledge concerning dopamine metabolism, including other metabolic pathways and their regulation. Recent evidence, including from the functional consequences of genetic variants in dopaminergic enzymes (e.g., COMT and MAO), revealed variability in dopamine concentration affecting vulnerability to neuropsychiatric illness. The dynamic relationship between dopamine metabolism and mitochondrial function is subsequently drawing worldwide attention as

mitochondrial dysfunction can further amplify oxidative stress leading to impaired homoeostasis of dopamine (Goldstein et al., 2013).

Physiological functions of dopamine

Dopamine is a multifunctional catecholamine with various physiological functions throughout the central nervous system (CNS) and peripheral organs. Its effects are mediated by five G protein–coupled receptor (GPCR) subtypes (D1–D5), which are broadly classified into D1-like (D1, D5) and D2-like (D2, D3, D4) families of receptors. Each GPCR regulates many pathways that ultimately increase or decrease the levels of cyclic adenosine monophosphate (cAMP), but they do so in a highly receptor-specific fashion. Changes in cAMP produce subsequent changes in neuronal excitability, synaptic transmission and cellular homeostasis. The anatomical distribution of dopamine that conveys its biological effects is directed by four different neural circuits; nigrostriatal, mesolimbic, mesocortical and tuberoinfundibular pathways (Beaulieu et al., 2015).

Dopamine have been required in order to ensure the appropriate ratio of excitatory to inhibitory inputs into the basal ganglia and to facilitate the course of motivated movement. Conversely, by activating D2 receptors the indirect pathway becomes less active and its excitatory tone decreases whereas D1 receptors activate the direct pathway ultimately leading to stimulation of motor initiation. The deliberately balanced inputs of these pathways are exalted to such an extent that any disruption will regale the more dominant motor systems involved in the classical parkinsonian disorders (Klein et al., 2019), underlining its pivotal role in motor modulation.

You received training data until October 2023 The mesolimbic pathway from ventral tegmental area to nucleus accumbens comprises the neuroanatomical substrate of reward circuitry, motivational drive, and associative learning. Dopamine is the principal neuro-chemical messenger in your brain that encodes both the value and approach-value (expected hedonic-value) of rewards (i.e., how much you want them); accordingly, dopamine potentiates those behaviours that are vital to biological survival (such as food/seeking/consummation, and mating). This neurobiological reinforcement system is highly adaptable, but unfortunately, it is also an appealing target for abuse via substances that can precipitate action-expectancy for compulsive behavior. This means that the dopaminergic pathway mediates reward prediction error (RPE), which is the core mechanism of adaptive learning and decision-making process (Schultz, 2016).

In addition to processes related to reward, the mesocortical pathway which projects dopaminergic fibers from various midbrain nuclei to the prefrontal cortex in a top-down manner exerts very strong control over higher-order cognitive processes. As do the dopaminergic pathway that orchestrates executive function of this cortical area concerned with selective attention, working memory capacity, planning and cognitive flexibility. This exquisite tuning capability for dopaminergic supply reinforces cognitive homeostasis highlighting that cognitive performance debilitate in an inverted-U fashion as a function of availability, since both deficit and supraphysiological amounts of the neurotransmitter impair mentality (Arnsten et al., 2017).

Dopaminergically also carries out an individual and integral role for neuroendocrine regulation through the tuberoinfundibular pathway. Dopaminergic neurons descending from the hypothalamus project to the anterior pituitary, where dopamine acts as prolactin-inhibiting factor. Dopamine reduces prolactin secretion through interaction with D2 receptors on lactotroph cells, thus influencing reproductive physiology and metabolism. Therefore, this inhibitory control is important for maintaining hormonal homeostasis and that disturbances of dopaminergic tone can gain to endocrine disease (Freeman et al., 2000).

Dopamine: from a brain neurochemical to peripheral physiological effects Dopamine exerts dose-dependent modulation on cardiovascular parameters, such as systemic blood pressure and cardiac output. Although dopamine is a non-selective agonist for dopaminergic receptors, when administered at low infusion rates (0.02–1.0 μg/kg/min) it primarily stimulates D1 receptors in renal and mesenteric vascular beds leading to vasodilation, increased regional perfusion, and i.excretion of sodium. The clinical effects exerted by dopamine at low and medium doses are sympathetic stimulation through β-adrenergic receptors leading to increased heart rate and contractility; higher (supra-physiological) concentrations lead to the prevalence of α-adrenergic activity with unwanted vasoconstriction in those individuals who are susceptible (Goldstein, 2010).

While synthesis of dopamine occurs across various tissues, locally-generated renal dopamine is especially important for electrolyte and fluid homeostasis. Dopamine generated from tubular epithelial cells within the kidney reduces sodium reabsorption by inhibiting Na^+/K^+-ATPase and sodium-hydrogen exchanger activity. Designed physiologically to manage arterial pressure and avoid the overload of circulatory volume, this natriuretic mechanism Renal dopaminergic signaling in its diminished form has been linked to the pathophysiology of hypertension and renal parenchymal injury (Zhang & Jose, 2017).

Moreover, as recent investigations have recognized that dopaminergic transmission participates importantly into the modulation of immunity. Dopaminergic receptors are widely expressed across different immune cell types, including T and B lymphocytes, macrophages and dendritic cells. Dopaminergic signaling can have major impact on immune competence through the modulation of cytokine secretory patterns, cellular proliferations, and leukocyte chemotaxis. Dopamine has both context-dependent pro- and anti-inflammatory effects, illustrating the well-known pleiotropic character of catecholamine tissue microenvironment actions with a particular immunomodulatory outcome dependent on receptor subtype expression and local ligand concentration (Channer et al., 2023). Dopaminergic signaling is part of enteric nervous system thereby coordinating digestive actions amongst other neurotransmitters which include acetylcholine, serotonin. Versions of dysregulated gut dopamine have been involved in diseases such as functional dyspepsia and irritable bowel syndrome.

Clinical significance of dopamine

Dopamine have been at the core and most likely is the neurotransmitter class that is most clinically relevant, since it seems to be at play in an astonishingly broad variety of neurological, psychiatric, endocrine and systemic disorders. Dopamine emerges as clinically significant due primarily to its pleiotropic physiological roles and, when perturbed, detrimental effects on nearly every organ system. Modern clinical medicine revolves around dopamine and dopaminergic pathways have been recognised since the recent advances in neurobiology, but also pharmacology as major targets for treatment (Speranza et al., 2025).

Dopamine have been probably best known for its role in neurodegenerative conditions such as Parkinson's disease. Understanding Parkinson's disease is characterized by progressive degeneration of dopaminergic neurons in the substantia nigra pars compacta, leading to a significant loss of dopamine levels in the striatum. The nigro-striatal pathways disrupted in Parkinson's disease (PD) are involved with the same circuitry change that elucidates the clinical features of PD that comprise the classic motor symptoms: resting tremor, rigidity, bradykinesia and postural dysfunction. Dopamine replacement strategies, mostly through levodopa (L-DOPA)—which is generally co-administered with peripheral decarboxylase inhibitors to facilitate the central nervous system and bioavailability—continue to be at the core of therapeutic management. Alternative pharmacological strategies include direct dopamine receptor agonists and monoamine oxidase-B (MAO-B) inhibitors which modulate dopaminergic transmission in the central nervous system to reduce burden of disease symptomatology (Poewe et al., 2017).

Dopamine (DA) and serotonin (5-HT) are important monoaminergic neurotransmitters in the central nervous system (CNS). Brain dopamine, predominantly synthesized in the substantia nigra pars compacta (SNpc), the ventral tegmental area (VTA), and the arcuate nucleus of the hypothalamus, mainly regulates motor control, reward-based learning, arousal, addiction, activeness, motivation, cognitive function and hormonal regulation. Serotonergic pathways originating from dorsal and medial raphe nuclei innervate a variety of cortical and subcortical structures and determine the serotonin involvement in psychomotor inhibition, the regulation of emotions and mood, cognition and adaptation to stressors (Howes & Kapur, 2009).

Most importantly for major depressive disorder and substance use disorders, the central importance of dopamine for both affective regulation and hedonic processing makes dopaminergic system particularly relevant to these aspects as well. Anhedonia, specified as an extremely lowered capacity to experience pleasure, is one of the cores diagnostic standards for depression and has actually been additionally related to impaired dopaminergic neurotransmission within mesocorticolimbic circuits (J27).Some antidepressant substances, specifically bupropion are clinically reliable for anxiousness because of their boosted dopaminergic signaling. In contrast, the acute effects of drug administration such as cocaine, amphetamines and opioids evoke large increases in synaptic dopamine within reward circuitry which drives compulsive drug-seeking behaviours and neurobiological adaptations that constitute addiction (Volkow et al., 2017).

Finally, dopamine's impact on attentional processes and executive cognitive function is most pertinent to attention deficit hyperactivity disorder (ADHD). Dysregulated dopaminergic signaling in the prefrontal cortex is a hallmark of ADHD and related to sustained attention, inhibitory control and working memory capacity (amongst other things). Psychostimulant pharmacotherapy works mainly via increasing dopaminergic tone through presynaptic reuptake blocking or increased vesicular release (Faraone & Buitelaar, 2010) thus enhancing cognition and behavior primarily with methylphenidate and amphetamine derivatives.

Dopamine has become the major prolactin-inhibiting substance that flows from the hypothalamus to the anterior pituitary with respect to endocrine physiology. Destruction of this feedback loop can cause hyperprolactinaemia which may manifest as

galactorrhea, infertility and menstrual irregularities. Prolactin-secreting pituitary adenomas are most often treated with dopamine agonists, such as bromocriptine or cabergoline that restore hormonal homeostasis and reverse tumor size (Melmed et al., 2011).

Outside of the CNS, dopamine has significant clinical uses primarily in cardiovascular and renal medicine. Intravenous dopamine has a long history of use in clinical management of shock and heart failure due to its dose-dependent spectrum of action on whole body vascular tone and cardiac output. Dopamine is associated with renal vasodilation and natriuresis at low doses; cardiac contractility and systemic vasoconstriction occur at higher doses. Nevertheless, routine use of dopamine in the intensive care unit has waned because of arrhythmias and unclear superiority beyond other agents with respect to survival benefit (De Backer et al., 2010).

Recent evidence also implicates dopamine in immune and inflammatory regulation. The expression of dopamine receptors on immune cells has been shown to modulate cytokine production and effector functions of immune cells (Moreau et al., 2014), which indicates a possible involvement in autoimmune/ inflammatory disorders. Dopamine metabolism, on the other hand, is also associated with oxidative stress which plays role in pathogenesis of neurodegenerative and different chronic disorders. These observations have provided a platform for exploring the role of dopamine-targeted therapies in non-neurological and -psychiatric indications (Channer et al., 2023).

Conclusion

Dopamine is one of the most important catecholamines connecting biochemical, physiological, and clinical functions to organ systems. Its precise regulatory roles in synthesis, metabolism and receptor-mediated signaling are vital for maintaining neural and systemic homeostasis. Functionally, dopamine plays a critical role in regulating motor activity, cognition, reward pathways and endocrine function. Dysfunctions of dopaminergic pathways are involved in a wide range of neuropsychiatric and systemic diseases. Over the last few decades, however, this concept of glutamate basic neurotransmission has been stretched; recent research is identifying both immune regulatory and oxidative stress pathway roles. Dopamine, and the therapeutics that modulate dopamine signaling, facilitate bidirectional communication between the immune system and many other organ systems and cell types. This means that consideration of the immunologic effects of dopamine is a critical aspect in the studies of many different systems and diseases.

References

1. Arnsten, A. F. T., Wang, M., & Paspalas, C. D. (2017). Dopamine's influence on prefrontal cortical function. *Nature Reviews Neuroscience, 18*(2), 101–113. https://doi.org/10.1038/nrn.2016.178

2. Beaulieu, J. M., Espinoza, S., & Gainetdinov, R. R. (2015). Dopamine receptors – IUPHAR review 13. *British Journal of Pharmacology, 172*(1), 1–23. https://doi.org/10.1111/bph.12906

3. Blesa, J., Trigo-Damas, I., Quiroga-Varela, A., & Jackson-Lewis, V. R. (2015). Oxidative stress and Parkinson's disease. Frontiers in Neuroanatomy, 9, 91. https://doi.org/10.3389/fnana.2015.00091

4. Channer, B., Matt, S. M., Nickoloff-Bybel, E. A., et al. (2023). Dopamine, immunity, and disease. *Pharmacological Reviews, 75*(1), 62–158. https://doi.org/10.1124/pharmrev.122.000618

5. Channer, B., Matt, S. M., Nickoloff-Bybel, E. A., et al. (2023). Dopamine, immunity, and disease. *Pharmacological Reviews, 75*(1), 62–158. https://doi.org/10.1124/pharmrev.122.000618

6. Daubner, S. C., Le, T., & Wang, S. (2011). Tyrosine hydroxylase and regulation of dopamine synthesis. Archives of Biochemistry and Biophysics, 508(1), 1–12. https://doi.org/10.1016/j.abb.2010.12.017

7. De Backer, D., Biston, P., Devriendt, J., et al. (2010). Comparison of dopamine and norepinephrine in the treatment of shock. *New England Journal of Medicine, 362*(9), 779–789. https://doi.org/10.1056/NEJMoa0907118

8. Eisenhofer, G., Kopin, I. J., & Goldstein, D. S. (2004). Catecholamine metabolism: A contemporary view. Pharmacological Reviews, 56(3), 331–349. https://doi.org/10.1124/pr.56.3.1

9. Faraone, S. V., & Buitelaar, J. (2010). Comparing the efficacy of stimulants for ADHD. *European Child & Adolescent Psychiatry, 19*(4), 353–364. https://doi.org/10.1007/s00787-009-0054-3

10. Franco, R., Reyes-Resina, I., & Navarro, G. (2021). Dopamine in health and disease: Much more than a neurotransmitter. *Biomedicines, 9*(2), 109. https://doi.org/10.3390/biomedicines9020109

11. Freeman, M. E., Kanyicska, B., Lerant, A., & Nagy, G. (2000). Prolactin: Structure, function, and regulation of secretion. *Physiological Reviews, 80*(4), 1523–1631. https://doi.org/10.1152/physrev.2000.80.4.1523

12. Goldstein, D. S. (2010). Adrenal responses to stress. *Cellular and Molecular Neurobiology, 30*(8), 1433–1440. https://doi.org/10.1007/s10571-010-9606-9

13. Goldstein, D. S., Sullivan, P., Holmes, C., et al. (2013). Determinants of buildup of the toxic dopamine metabolite DOPAL in Parkinson's disease. Journal of Neurochemistry, 126(5), 591–603. https://doi.org/10.1111/jnc.12345

14. Howes, O. D., & Kapur, S. (2009). The dopamine hypothesis of schizophrenia: Version III. *Schizophrenia Bulletin, 35*(3), 549–562. https://doi.org/10.1093/schbul/sbp006

15. Hritcu, L., et al. (2015). The role of dopamine and its dysfunction as a consequence of oxidative stress. Oxidative Medicine and Cellular Longevity. https://doi.org/10.1155/2015/973046

16. Klein, M. O., Battagello, D. S., Cardoso, A. R., et al. (2019). Dopamine: Functions, signaling, and association with neurological diseases. *Cellular and Molecular Neurobiology, 39*(1), 31–59. https://doi.org/10.1007/s10571-018-0632-3

17. Lauretani, F., Giallauria, F., Testa, C., et al. (2024). Dopamine pharmacodynamics: New insights. *International Journal of Molecular Sciences, 25*(10), 5293. https://doi.org/10.3390/ijms25105293

18. Meiser, J., Weindl, D., & Hiller, K. (2013). Complexity of dopamine metabolism. Cell Communication and Signaling, 11, 34. https://doi.org/10.1186/1478-811X-11-34

19. Melmed, S., Casanueva, F. F., Hoffman, A. R., et al. (2011). Diagnosis and treatment of hyperprolactinemia. *Journal of Clinical Endocrinology & Metabolism, 96*(2), 273–288. https://doi.org/10.1210/jc.2010-1692

20. Missale, C., Nash, S. R., Robinson, S. W., et al. (2011). The physiology, signaling, and pharmacology of dopamine receptors. *Pharmacological Reviews, 63*(1), 182–217. https://doi.org/10.1124/pr.110.002642

21. Poewe, W., Seppi, K., Tanner, C. M., et al. (2017). Parkinson disease. *Nature Reviews Disease Primers, 3*, 17013. https://doi.org/10.1038/nrdp.2017.13

22. Qi-Lytle, X., Sayers, S., & Wagner, E. J. (2024). Current review of the function and regulation of tuberoinfundibular dopamine neurons. *International Journal of Molecular Sciences, 25*(1), 110. https://doi.org/10.3390/ijms25010110

23. Schultz, W. (2016). Dopamine reward prediction-error signaling. *Current Opinion in Neurobiology, 23*(2), 229–236. https://doi.org/10.1016/j.conb.2013.10.012

24. Speranza, L., Miniaci, M. C., & Volpicelli, F. (2025). The Role of Dopamine in Neurological, Psychiatric, and Metabolic Disorders and Cancer: A Complex Web of Interactions. *Biomedicines*, *13*(2), 492. https://doi.org/10.3390/biomedicines13020492

25. Volkow, N. D., Koob, G. F., & McLellan, A. T. (2017). Neurobiologic advances from the brain disease model of addiction. *New England Journal of Medicine, 374*(4), 363–371. https://doi.org/10.1056/NEJMra1511480

26. Zhang, M. Z., & Jose, P. A. (2017). Dopamine receptors in the kidney: A role in hypertension. *Current Opinion in Nephrology and* Hypertension, 26(2), 87–92. https://doi.org/10.1097/MNH.0000000000000305