

Dopamine

Mechanism of action

Dopamine's [mechanism of action](#) depends on the dosage given, and it can cause [vasodilation](#), increased urinary flow, increased cardiac output, or increased [blood pressure](#), depending on the dose.

Dose

Rx Start with 2–5 mcg/kg/min and titrate.

In a dose (mcg/kg/min) 0.5–2.0 (sometimes up to 5) dopaminergic renal, mesenteric, coronary, & cerebral vasodilatation, (+) inotrope

In a dose (mcg/kg/min) 2–10 β_1 positive inotrope

In a dose (mcg/kg/min) > 10 α , β & dopaminergic releases nor-epi (vasoconstrictor)

Indications

Specifically indicated for the correction of hemodynamic imbalances present in the [shock](#) syndrome due to [myocardial infarction](#), [trauma](#), endotoxic [septicemia](#).

Dopamine is primarily a [vasoconstrictor](#) (β_1 effects usually overridden by α -activity). 25% of dopamine given is rapidly converted to [norepinephrine](#) (NE). At doses > 10 mcg/kg/min one is essentially giving NE.

Used to treat low blood pressure, low heart rate, and cardiac arrest, especially in acute neonatal cases. It is also indicated for congestive heart failure, trauma, renal failure, and shock. Reactions to intravascular [contrast medium](#) if [shock](#) develops: add dopamine, start at 5 mcg/kg/min

[Hypotension](#) with [bradycardia](#) (vasovagal reaction).

Dopamine is ineffective for treating central neurological deficits such as Parkinson's disease, but its amino acid precursor, L-DOPA, can be administered systemically

see [Dopamine overdose hypothesis](#).

Dopamine (contracted from 3,4-dihydroxyphenethylamine) is a [hormone](#) and [neurotransmitter](#) of the catecholamine and phenethylamine families that plays a number of important roles in the human brain and body. Its name derives from its chemical structure: it is an amine that is formed by removing a carboxyl group from a molecule of L-DOPA.

[Dopaminergic neurons](#) of the [midbrain](#) are the main source of [dopamine](#) (DA) in the mammalian

central nervous system.

Degeneration primarily of pigmented ([neuromelanin](#)-laden) dopaminergic neurons of the pars compacta of the substantia nigra, resulting in reduced levels of dopamine in the [neostriatum](#) (caudate nucleus, putamen, globus pallidus).

Side Effects

May cause significant [hyperglycemia](#) at high doses.

Function

[Prolactin](#) is the only [pituitary hormone](#) predominantly under inhibitory control from the [hypothalamus](#) by prolactin releasing inhibitory factors (PIFs), with dopamine being the primary PIF.

[Parkinsonism](#) may be primary or secondary to other conditions. All result from a relative loss of [dopamine](#) mediated inhibition of the effects of [acetylcholine](#) in the [basal ganglia](#).

In the brain, dopamine functions as a neurotransmitter—a chemical released by nerve cells to send signals to other nerve cells. The brain includes several distinct dopamine systems, one of which plays a major role in reward-motivated behavior. Most types of reward increase the level of dopamine in the brain, and a variety of addictive drugs increase dopamine neuronal activity. Other brain dopamine systems are involved in motor control and in controlling the release of several other important hormones.

The [frontal lobe](#) contains most of the [dopamine](#)-sensitive neurons in the cerebral cortex. The dopamine system is associated with reward, attention, short-term memory tasks, planning, and motivation. Dopamine tends to limit and select sensory information arriving from the [thalamus](#) to the fore-brain. A report from the National Institute of Mental Health says a gene variant that reduces dopamine activity in the prefrontal cortex is related to poorer performance and inefficient functioning of that brain region during working memory tasks, and to slightly increased risk for schizophrenia.

Recent animal research indicates that dopamine and serotonin, neuromodulators traditionally linked to appetitive and aversive processes, are also involved in sensory inference and decisions based on such inference. Bang et al. tested this hypothesis in humans by monitoring sub-second striatal dopamine and serotonin signaling during a visual motion discrimination task that separates sensory uncertainty from decision difficulty in a factorial design. [Caudate nucleus](#) recordings (n = 4) revealed

multi-scale encoding: in three participants, [serotonin](#) tracked sensory uncertainty, and, in one participant, both dopamine and serotonin tracked deviations from expected trial transitions within our factorial design. Putamen recordings ($n = 1$) supported a cognition-action separation between the caudate nucleus and putamen-a striatal sub-division unique to primates-with both dopamine and serotonin tracking decision times. These first-of-their-kind observations in the human brain reveal a role for sub-second dopamine and [serotonin](#) signaling in non-reward-based aspects of cognition and action ¹⁾.

Dopamine agonist

[Dopamine agonist](#).

Dopamine receptor

see [Dopamine receptor](#).

Dopamine transporter

see [Dopamine transporter](#)

¹⁾

Bang D, Kishida KT, Lohrenz T, White JP, Laxton AW, Tatter SB, Fleming SM, Montague PR. Sub-second Dopamine and Serotonin Signaling in Human Striatum during Perceptual Decision-Making. *Neuron*. 2020 Oct 5:S0896-6273(20)30715-7. doi: 10.1016/j.neuron.2020.09.015. Epub ahead of print. PMID: 33049201.

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