

Ritonavir

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Ritonavir is a medication used in the treatment of HIV (human immunodeficiency virus) infection. It belongs to a class of drugs known as protease inhibitors. Protease inhibitors work by inhibiting the activity of the HIV protease enzyme, which is necessary for the virus to replicate and produce mature, infectious viral particles.

Here are some key points about ritonavir:

HIV Treatment: Ritonavir is primarily used as part of combination antiretroviral therapy (cART) for the treatment of HIV infection. It is not used as a standalone medication but is often combined with other antiretroviral drugs to enhance their effectiveness.

Boosting Agent: While ritonavir has antiretroviral activity on its own, it is often used in lower doses as a “booster” for other protease inhibitors. Ritonavir inhibits certain enzymes in the liver that metabolize protease inhibitors, leading to increased and more sustained levels of the co-administered protease inhibitor in the blood.

Combination Therapy: Ritonavir is commonly used in combination with other antiretroviral drugs, such as nucleoside reverse transcriptase inhibitors (NRTIs) and integrase inhibitors, to create a potent and comprehensive treatment regimen for HIV.

Side Effects: Like any medication, ritonavir can cause side effects. Common side effects may include nausea, vomiting, diarrhea, fatigue, and changes in lipid levels. It's important for individuals taking ritonavir to be monitored regularly by healthcare providers for both efficacy and potential side effects.

Drug Interactions: Ritonavir can interact with a variety of other medications due to its inhibition of certain liver enzymes. These interactions may affect the metabolism and efficacy of co-administered drugs. Therefore, healthcare providers must be aware of a patient's complete medication regimen to manage potential interactions.

Shen et al. identified that elevated [synapsin 2a](#) (Syn2a) in the [infralimbic cortex](#) (IL) to basolateral [amygdala](#) (BLA) circuit disrupted presynaptic orchestration, leading to an excitatory/inhibitory imbalance in the BLA region and causing [extinction resistance](#). [Overexpression](#) or silencing of Syn2a

levels in IL neurons replicated or alleviated behavioral, electrophysiological, and biochemical phenotypes in resistant mice. They further identified the proline-rich domain H in the C-terminal of Syn2a was indispensable for the interaction with synaptogyrin-3 (Syngr3) and demonstrated that disrupting this interaction restored extinction impairments. Molecular docking revealed [ritonavir](#), an FDA-approved HIV drug, could disrupt Syn2a-Syngr3 binding and rescue [fear extinction](#) behavior in Syn2a-elevated mice. In summary, aberrant Syn2a elevation and its interaction with Syngr3 at the presynaptic site were crucial in fear extinction resistance, suggesting a potential therapeutic avenue for related disorders ¹⁾.

¹⁾

Shen XY, Zhang J, Huang HZ, Li SD, Zhou L, Wu SP, Tang C, Huang X, Liu ZQ, Guo ZY, Li X, Man HY, Lu YM, Zhu LQ, Liu D. Synapsin 2a/Synaptogyrin-3 interaction regulates fear extinction in mice. *J Clin Invest*. 2024 Jan 4:e172802. doi: 10.1172/JCI172802. Epub ahead of print. PMID: 38175724.

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