

Common Pathway Hypothesis

The common pathway hypothesis is a conceptual model in [neurosurgery](#) and [psychiatric_neuromodulation](#) proposing that different anatomical targets for [deep_brain_stimulation](#) (DBS) in neuropsychiatric disorders may ultimately converge on a shared functional circuit responsible for symptom improvement.

Origin and Application

Initially formulated to explain why distinct DBS targets—such as the [subthalamic_nucleus](#), [internal_capsule](#), or [superolateral_medial_forebrain_bundle](#)—can lead to similar therapeutic outcomes in conditions like [treatment-resistant_obsessive-compulsive_disorder](#) (TR-OCD) and [treatment-resistant_depression](#).

The hypothesis suggests that different anatomical entry points modulate a core subcortical-cortical network, yielding therapeutic effects through a final common pathway.

Role in DBS for OCD

In the context of OCD:

The hypothesis has been used to justify multiple DBS targets including the [anteromedial_subthalamic_nucleus](#) and the [superolateral medial forebrain bundle](#).

The [ocd_response_tract](#) (ORT) has been proposed as the anatomical correlate of this common pathway.

Criticism and Reappraisal

Recent studies, such as Coenen et al. (Mol Psychiatry, 2025), challenge the oversimplification of the common pathway hypothesis:

The sIMFB appears to encompass a wider range of OCD sub-networks, suggesting a more distributed and nuanced connectomic model.

The overlap between ORT and specific targets like the amSTN may not fully account for clinical efficacy.

The idea of a singular common tract may mask the functional diversity and anatomical specificity of DBS effects in OCD.

Alternative Views

[network_neuroscience](#) and individualized [connectomic_analysis](#) are emerging as more precise tools to understand DBS mechanisms.

These approaches emphasize targeting personalized dysfunctional circuits rather than relying on a single tract or common endpoint.

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