CYP2C9 Substrates and Inhibitors

How drugs affect and are effected by this important enzyme

NEZU
Biotech GmbH

Dear Client,

first and foremost, thanks for downloading this free resource. All graphics in this report are predictions made by our AI systems. They show the profile of this ADMET endpoint for thousands of marketed drugs. We divided it by indication, and as you can see, it changes a lot from one category to another. Therefore, try to find the closest indication to the compounds you are currently developing.

CYP2C9 substrate and **CYP2C9 inhibitor** endpoints answer two different questions. The substrate endpoint asks whether cytochrome P450 2C9 metabolizes a compound. The inhibitor endpoint asks whether the compound reduces CYP2C9 activity toward other substrates. A compound can be a substrate, an inhibitor, both, or neither.

Substrate status is commonly tested using human liver microsomes, hepatocytes, or recombinant CYP2C9. Researchers measure compound depletion or CYP2C9-dependent metabolite formation by liquid chromatography–mass spectrometry. Results may include intrinsic clearance in microlitres per minute per milligram of microsomal protein.

Inhibition is tested by incubating several concentrations of the compound with CYP2C9 and a marker substrate. Diclofenac 4′-hydroxylation and S-warfarin 7-hydroxylation are established marker reactions. Reduced metabolite formation indicates inhibition.

CYP2C9 is expressed mainly in the liver. It oxidizes many structurally diverse drugs before elimination or further metabolism. Strong CYP2C9 substrate dependence makes a compound’s exposure sensitive to enzyme inhibition, induction, and genetic variation. This dependence can alter clearance and metabolite formation. It does not establish whether the metabolites are active or toxic.

A high substrate prediction means CYP2C9 metabolism is more likely. A high inhibitor prediction means CYP2C9 inhibition is more likely. Inhibition can increase exposure to co-administered CYP2C9 substrates, but clinical relevance depends on unbound inhibitor concentration and dosing.

Both endpoints must be interpreted with metabolic stability, metabolite identification, plasma protein binding, and exposure estimates. CYP induction, CYP2C9 genotype, and inhibition of other enzymes also require separate assessment.

We generated these insights using advanced AI systems designed to analyze complex biotech data and highlight potential trends. While we strive for accuracy, AI models

can occasionally make mistakes or miss context, so please treat these predictions as informative guidelines rather than absolute facts.

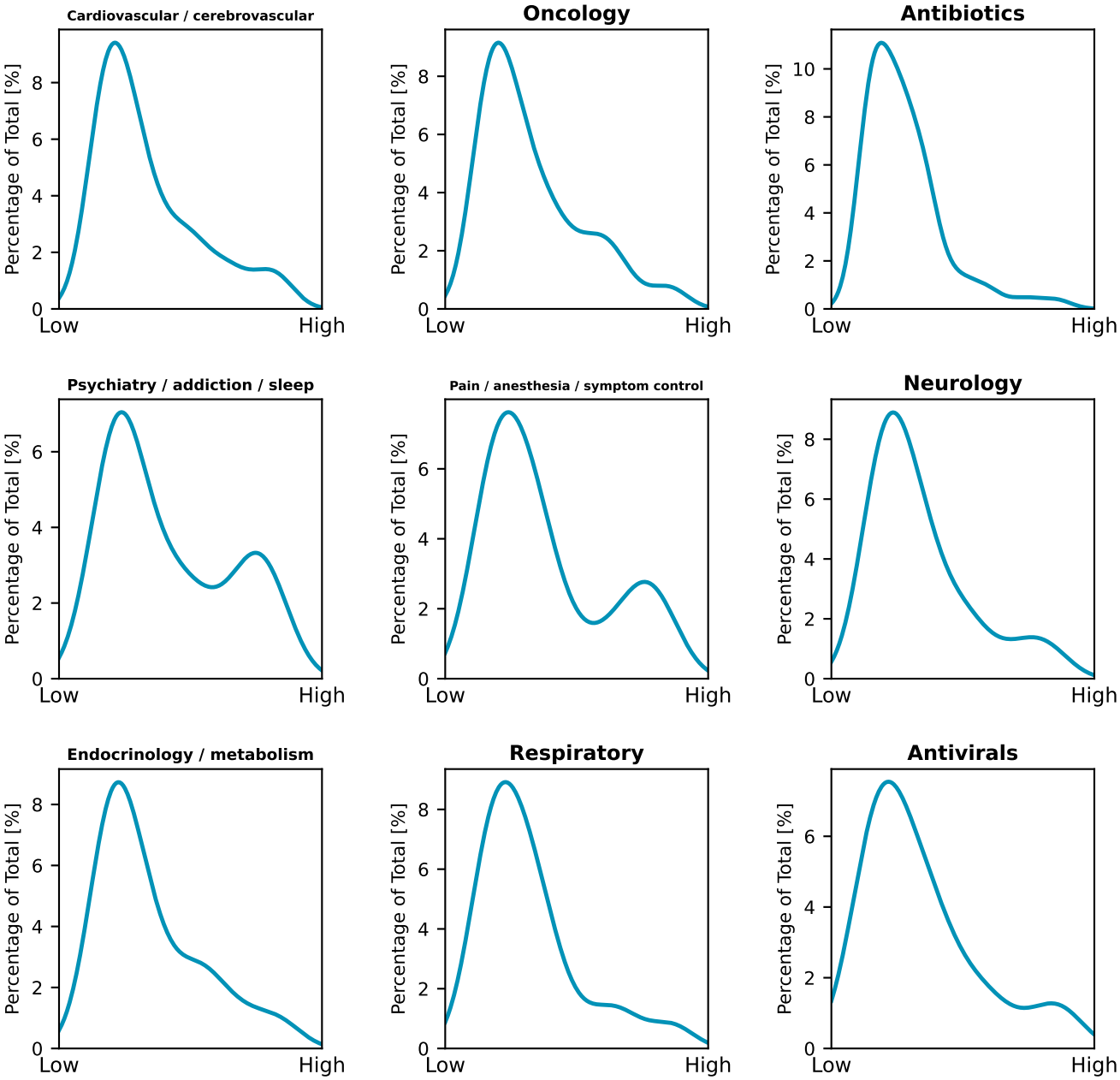
This report is provided for general informational and educational purposes only, not for medical diagnosis, treatment decisions, or financial advice. Because individual circumstances and data can vary, we cannot be held responsible for any decisions made or losses incurred based on these predictions.

Now that we got this out of the way, we genuinely hope these results are useful for your research.

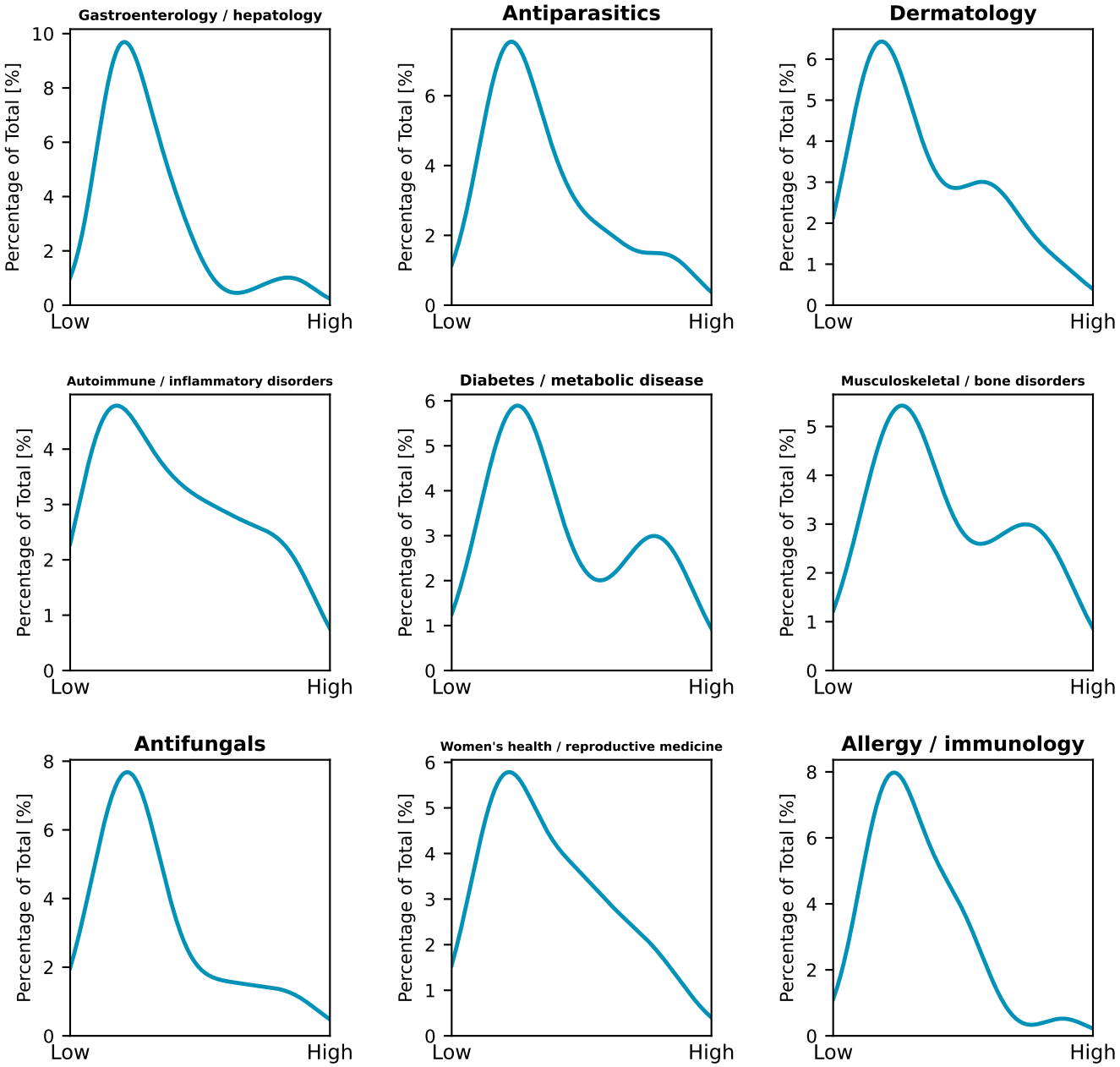
Sincerely,

Your friends at Nezu Biotech GmbH.

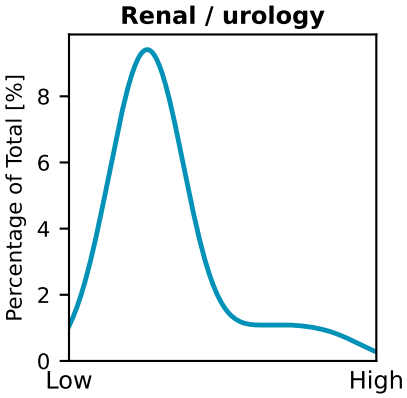
CYP2C9 Substrate

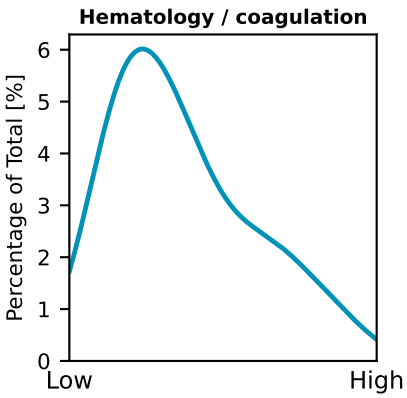


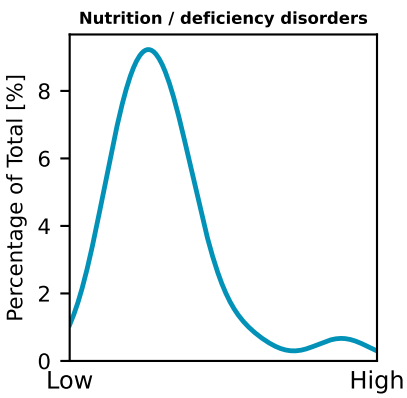
CYP2C9 Substrate

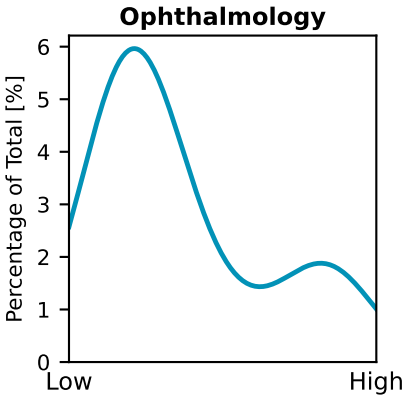


CYP2C9 Substrate



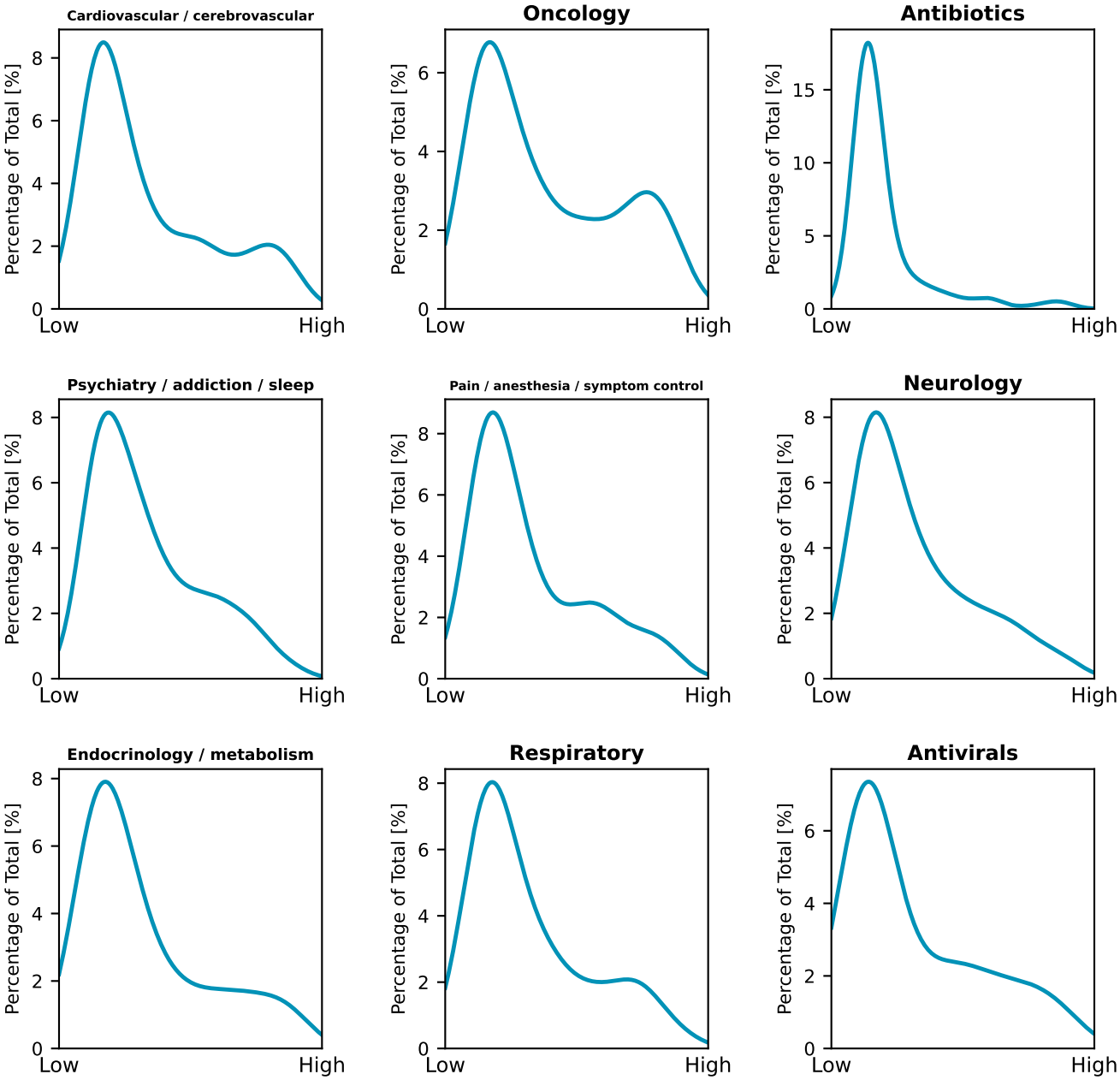




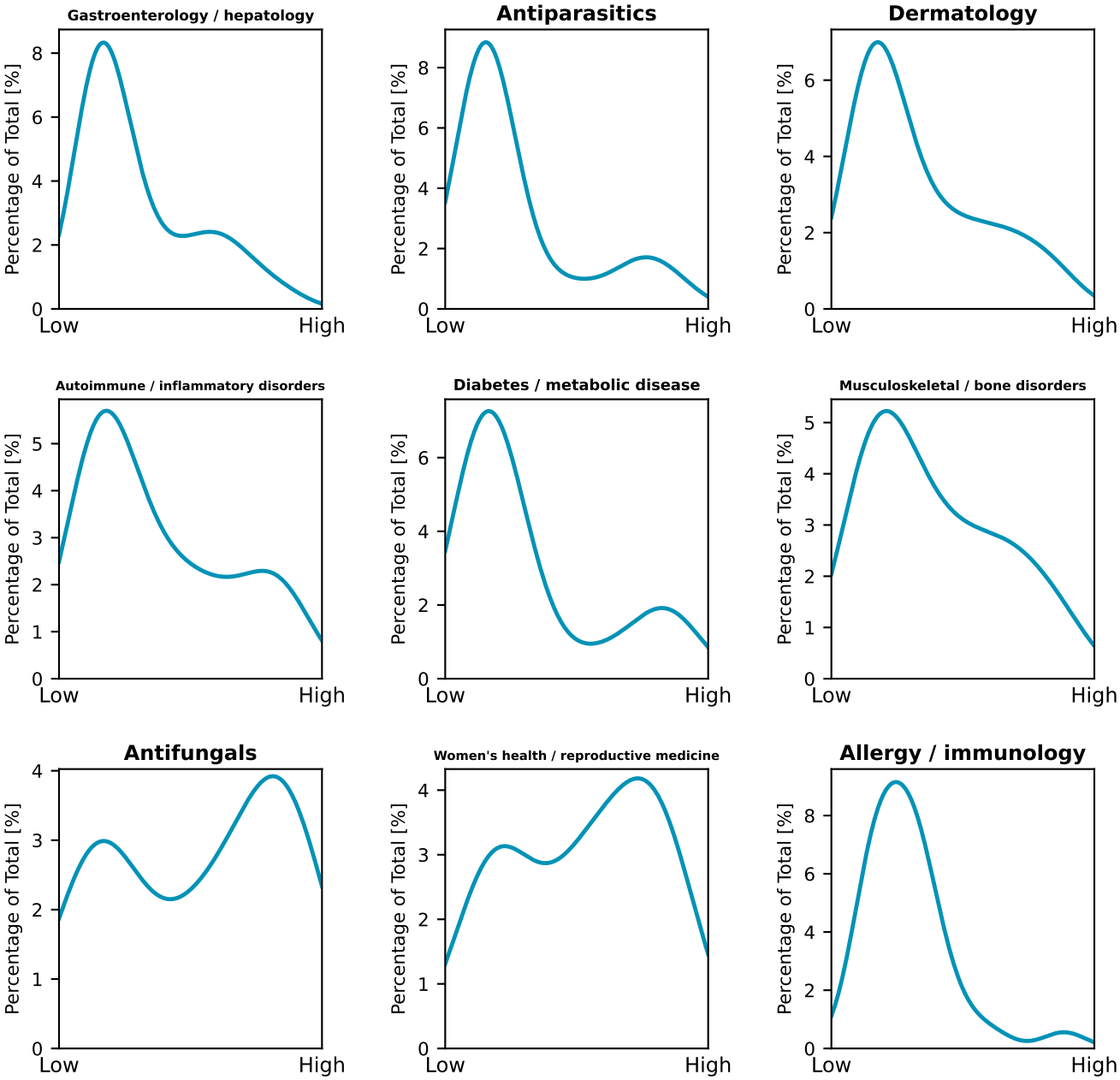


Derived from thousands of marketed drugs

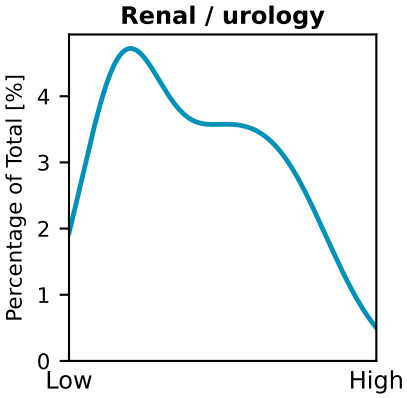
CYP2C9 Inhibitor A

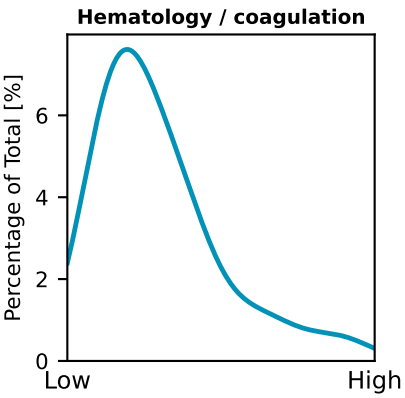


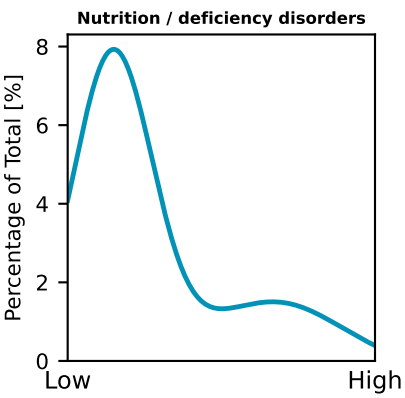
CYP2C9 Inhibitor A

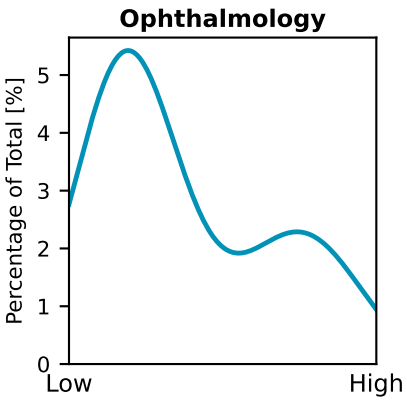


CYP2C9 Inhibitor A









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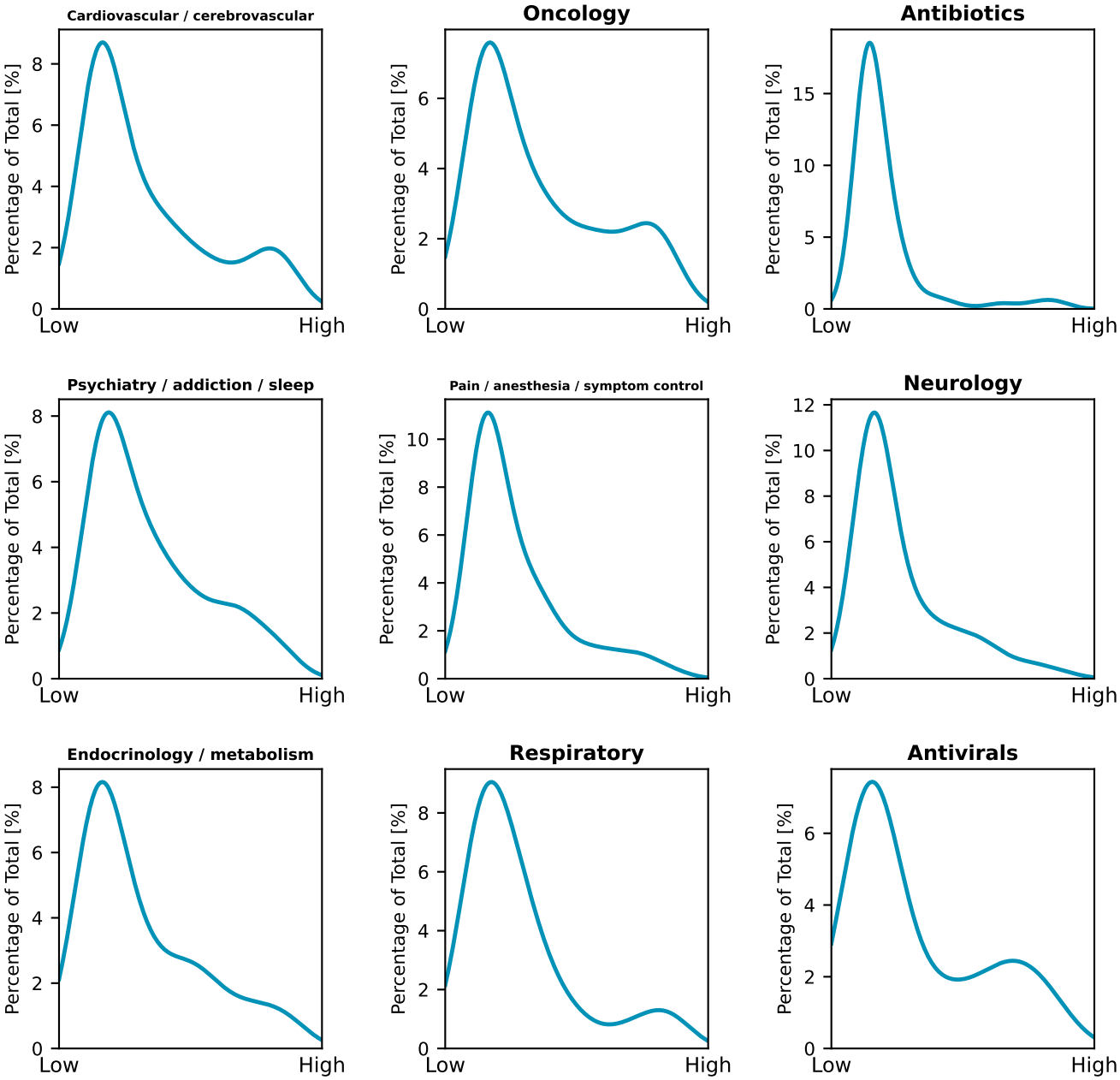
CYP2C9 Inhibitor B



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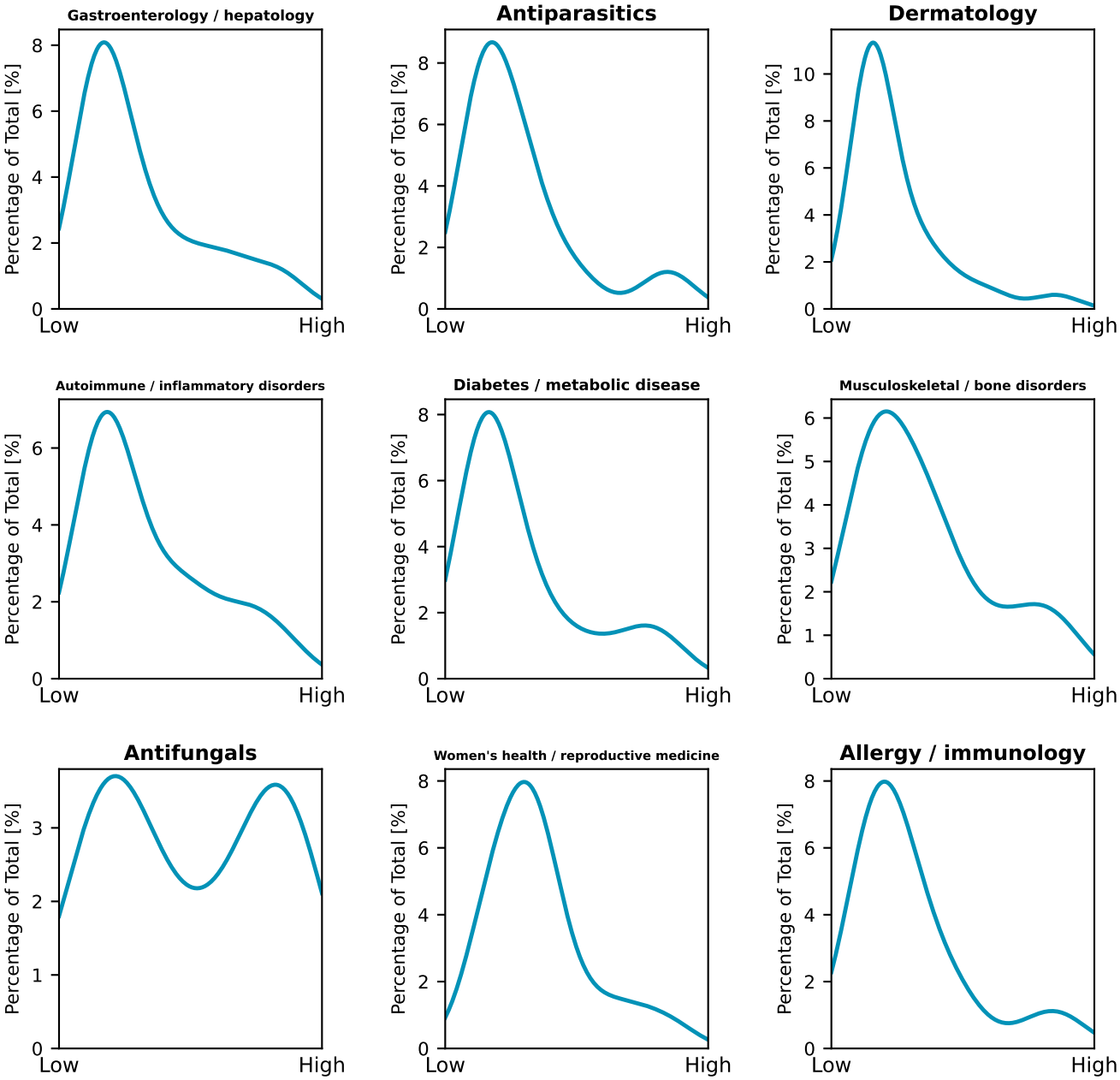
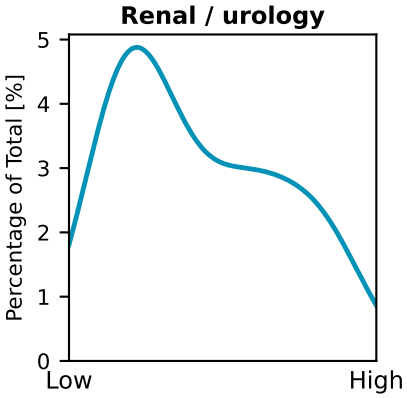
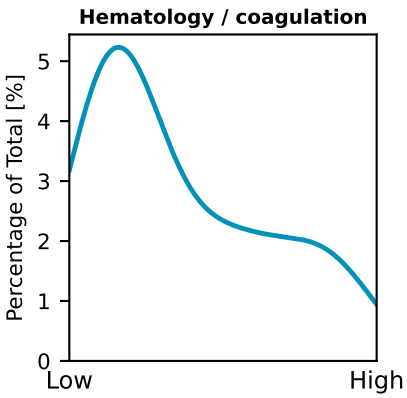
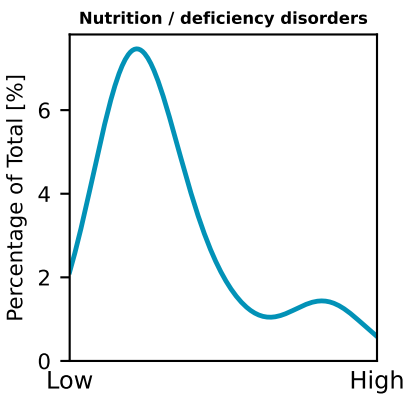
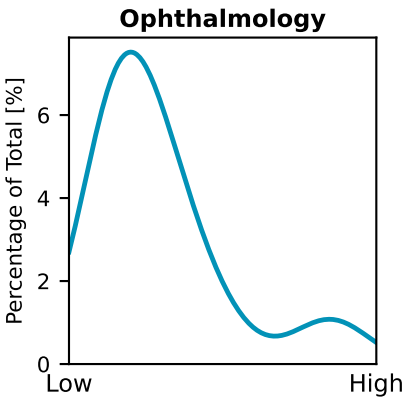


CYP2C9 Inhibitor B









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