CYP1A2 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.

CYP1A2 substrate status asks whether cytochrome P450 1A2 recognizes and metabolizes a compound. A substrate uses CYP1A2 as a metabolic pathway. This affects the compound itself, including its clearance and metabolite formation.

Substrate status is assessed using human liver microsomes, human hepatocytes, or recombinant CYP1A2. Researchers incubate the compound with the biological system and the required enzyme cofactor. They measure parent-compound disappearance or metabolite formation by liquid chromatography–mass spectrometry. Selective chemical inhibition or recombinant-enzyme comparisons show how much metabolism depends on CYP1A2.

CYP1A2 substrate metabolism occurs mainly in the liver. A high value indicates a greater probability that the compound is a CYP1A2 substrate. It does not indicate rapid clearance. Substrate status is not inherently unfavorable. Concern only increases when CYP1A2 accounts for a large fraction of total clearance.

CYP1A2 inhibition asks whether a compound reduces CYP1A2 activity. An inhibitor affects the metabolism of other CYP1A2 substrates. It does not need to be metabolized by CYP1A2 to inhibit the enzyme.

Inhibition is commonly tested using human liver microsomes or recombinant CYP1A2. Researchers incubate a CYP1A2 probe substrate, often phenacetin, with increasing concentrations of the test compound. They measure the reduction in probe-metabolite formation.

A strong inhibitor reduces CYP1A2-mediated clearance of susceptible co-administered drugs. This increases their exposure when CYP1A2 is an important elimination pathway. The clinical effect depends on inhibitor concentration and unbound exposure.

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.

You have the vision. We can help make it reality.

Click here to book a demo. Tell us about your molecules.

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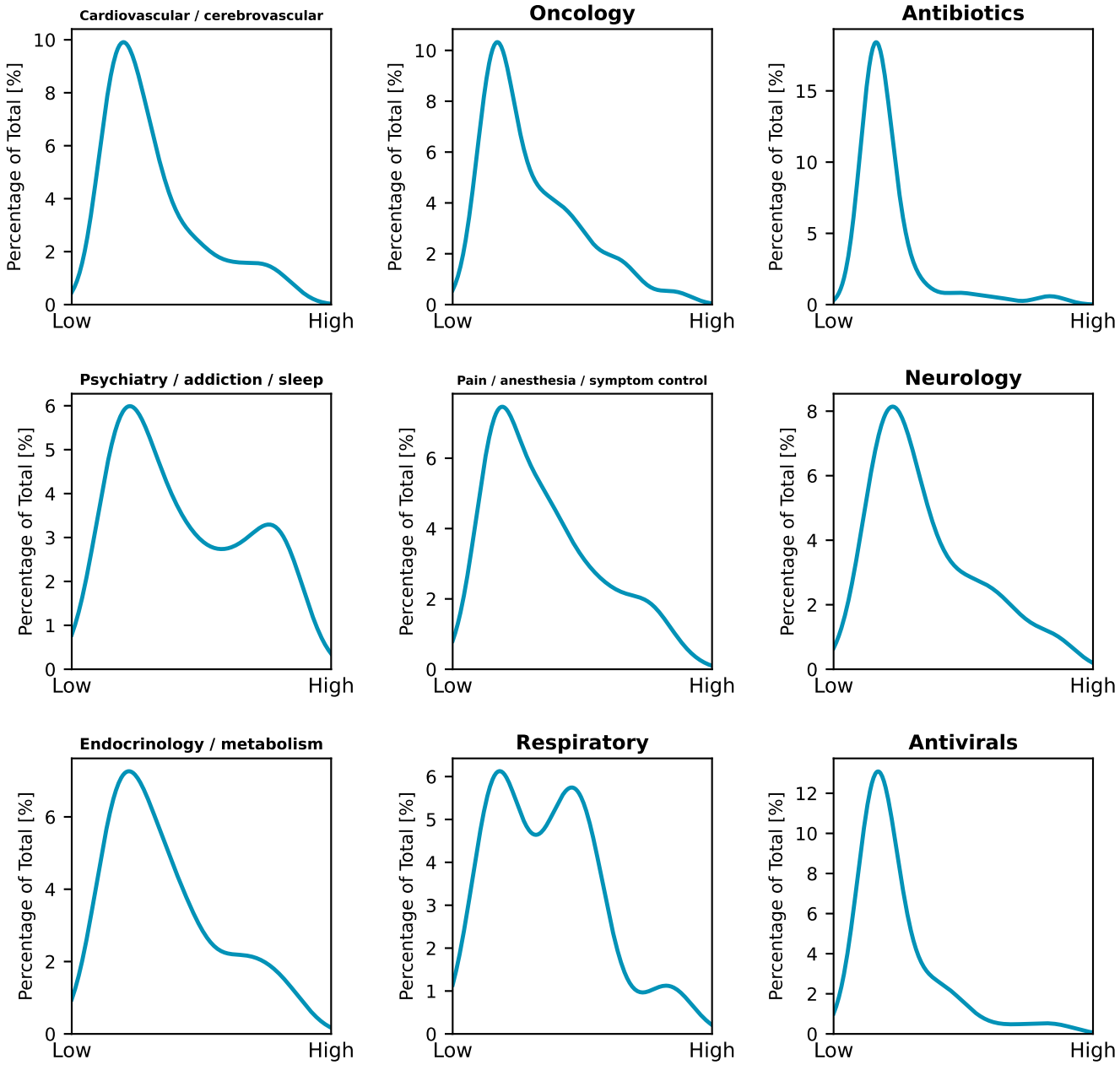
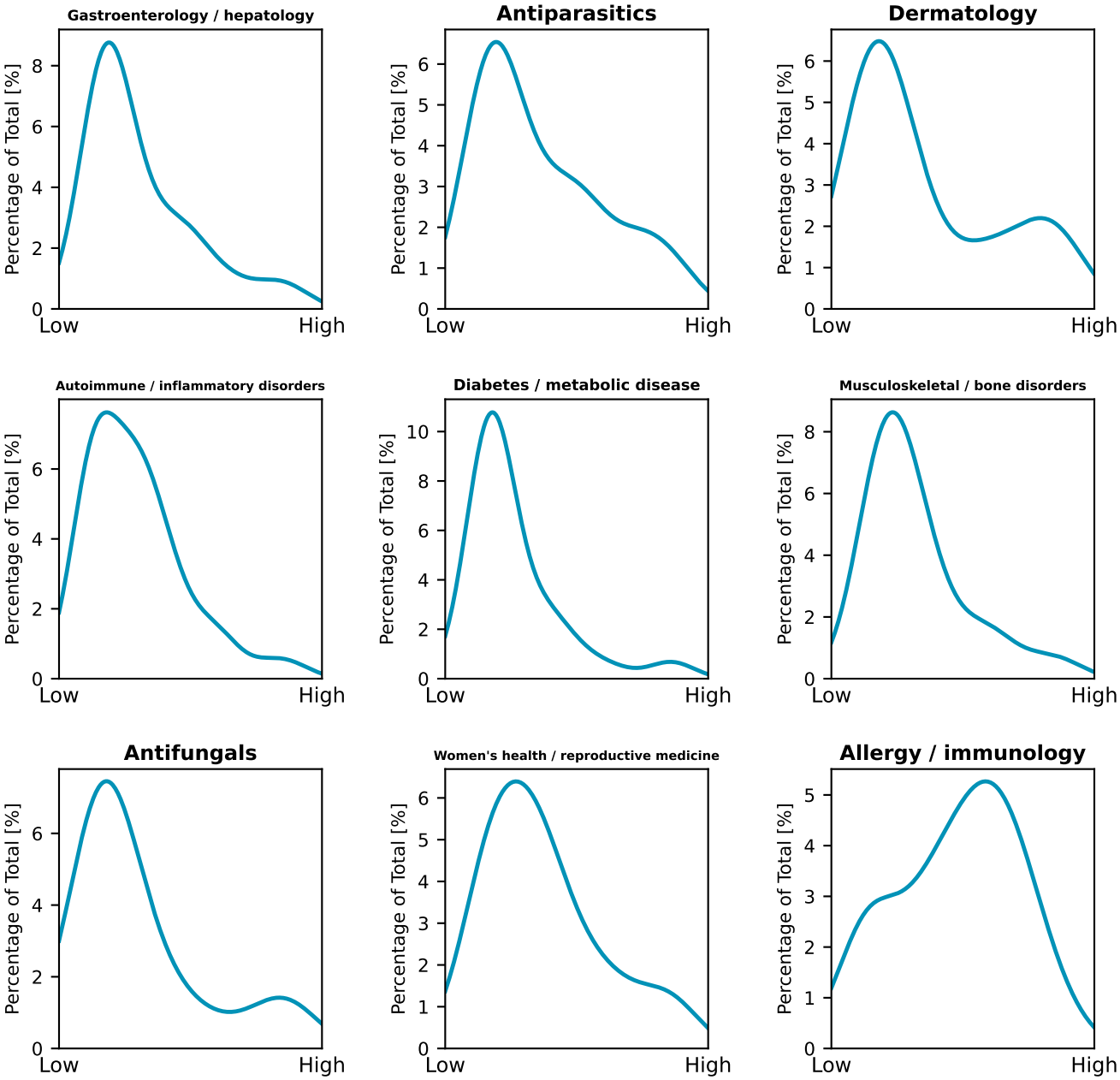
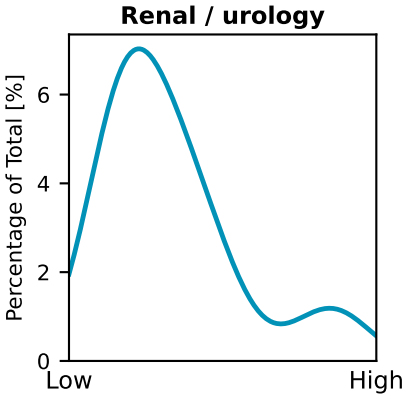
CYP1A2 Substrate

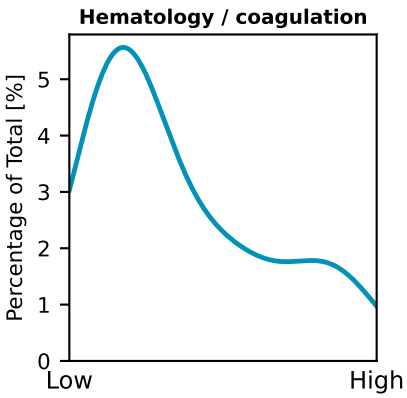


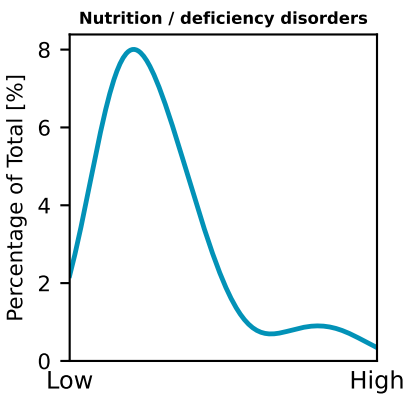
CYP1A2 Substrate

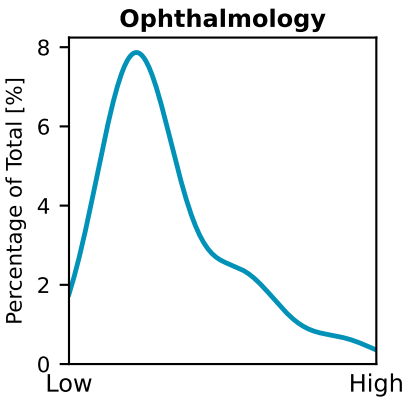


CYP1A2 Substrate



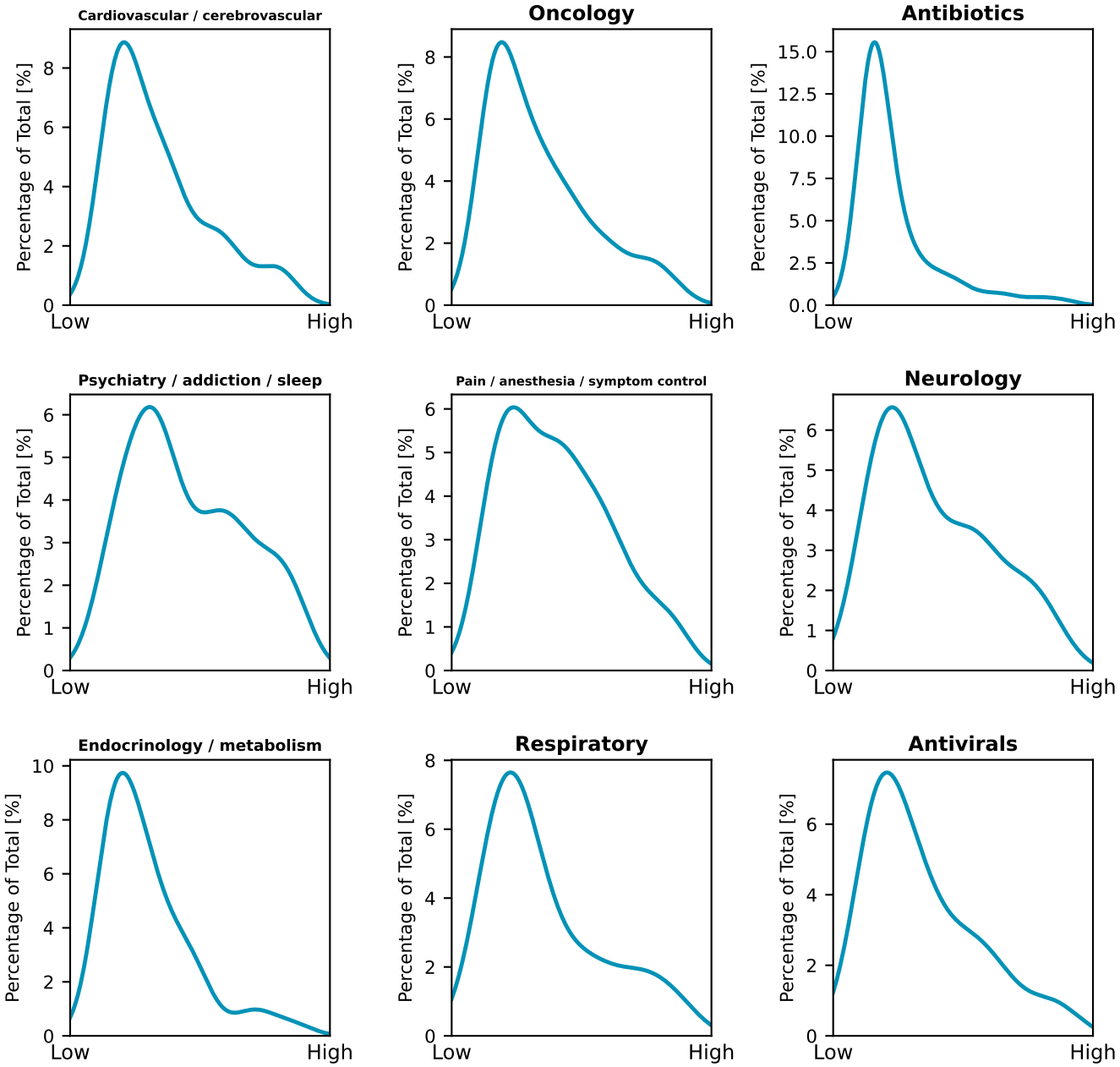




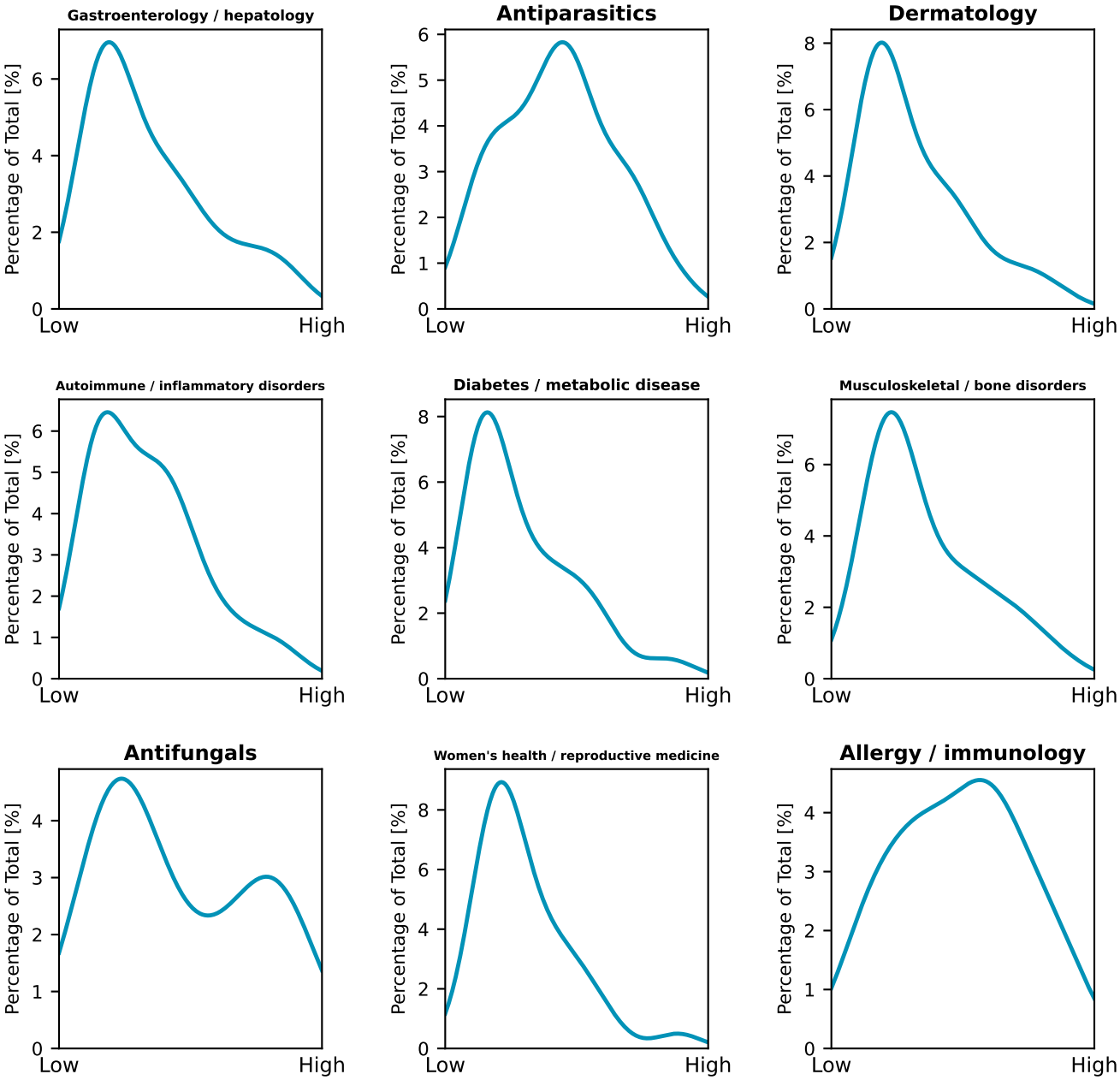


Derived from thousands of marketed drugs

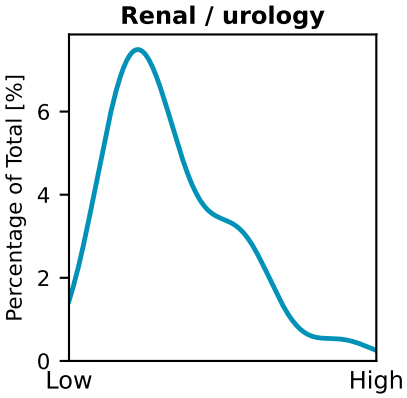
CYP1A2 Inhibitor A

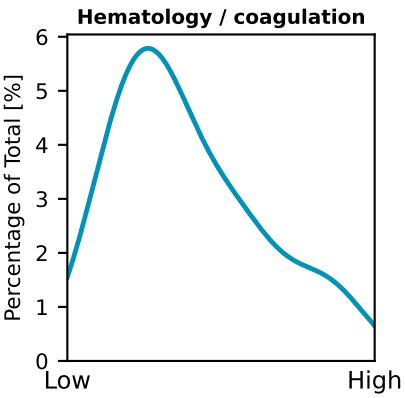


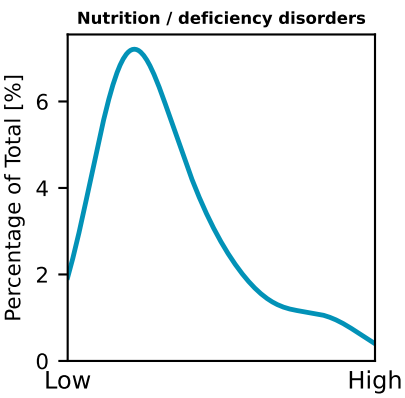
CYP1A2 Inhibitor A

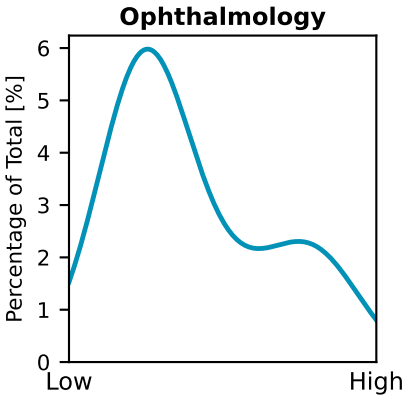


CYP1A2 Inhibitor A









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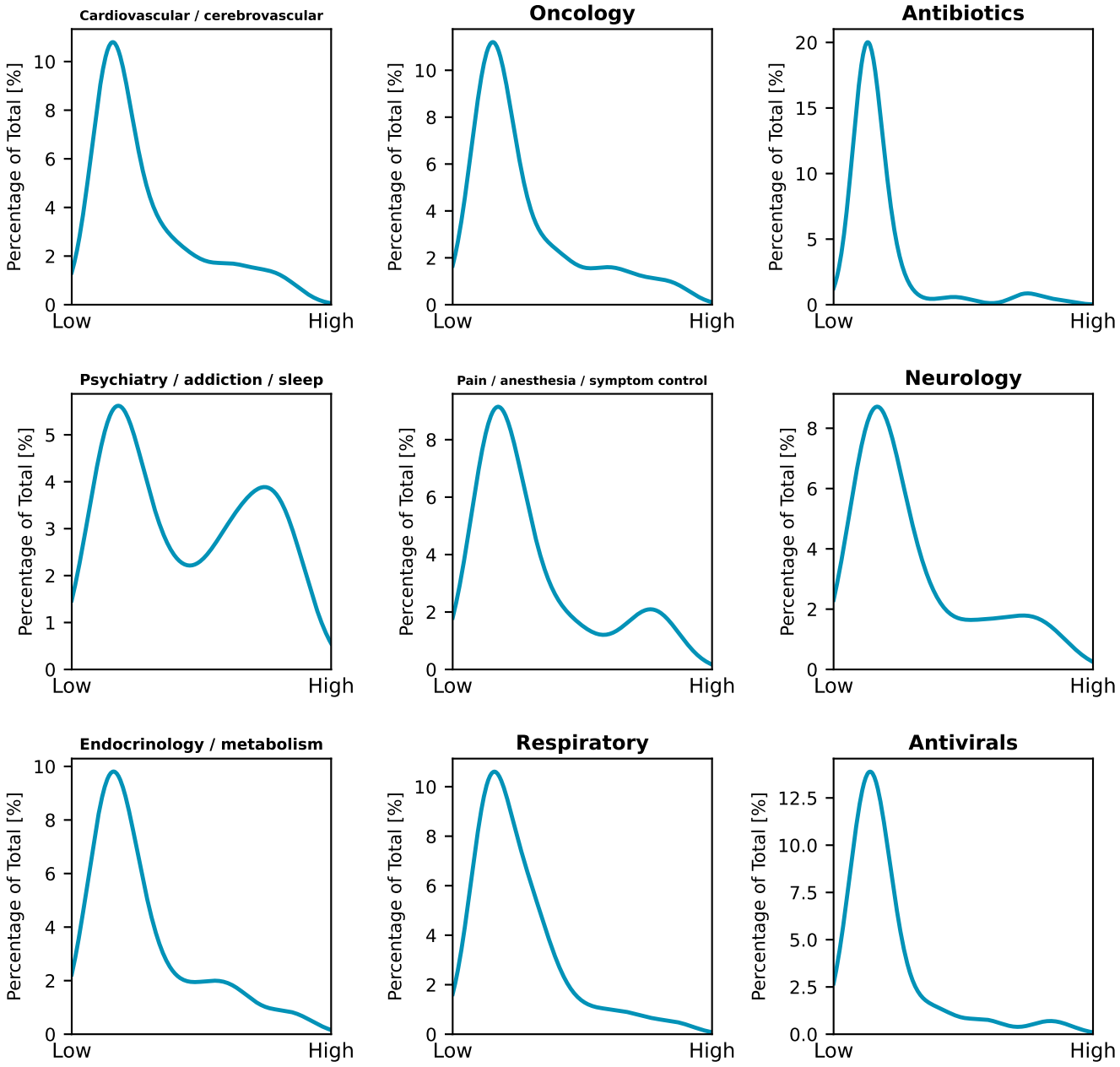
CYP1A2 Inhibitor B



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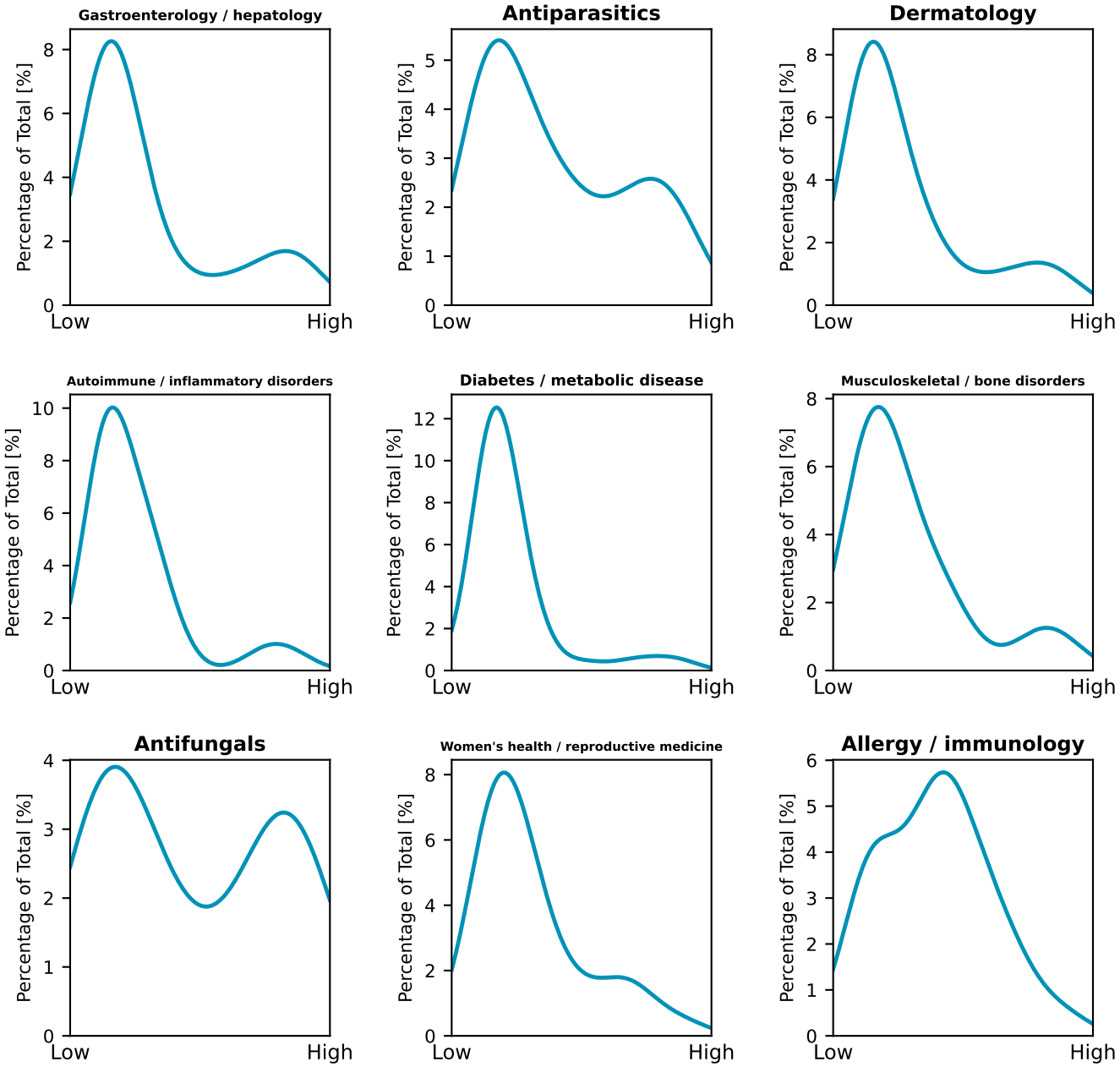
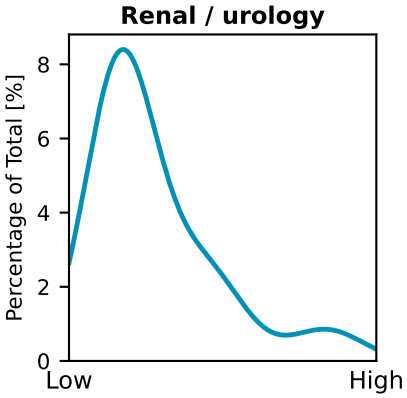
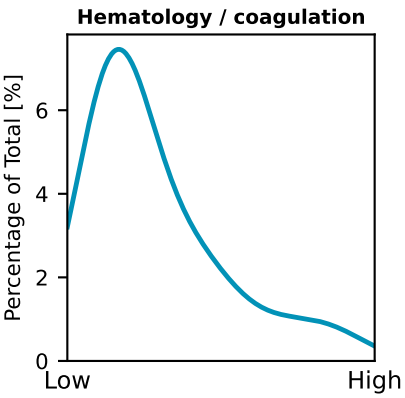
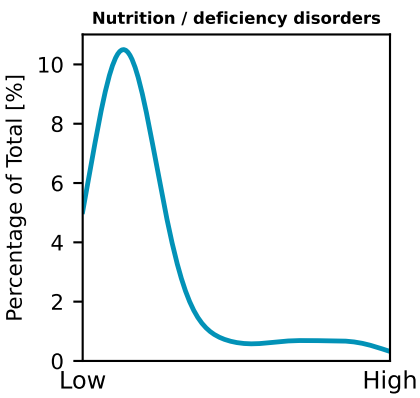
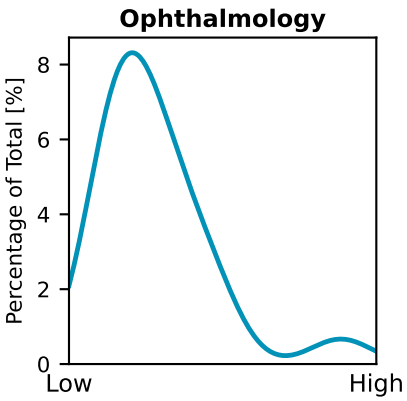


CYP1A2 Inhibitor B









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