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The duration and intensity of pharmacological action of most lipophilic drugs are determined by the rate they are metabolized to inactive products. The [Cytochrome P450 monooxygenase system](https://en.wikipedia.org/wiki/Cytochrome_P450_monooxygenase_system) is the most important pathway in this regard. In general, anything that *increases* the rate of metabolism (*e.g.*, [enzyme induction](https://en.wikipedia.org/wiki/Enzyme_induction_and_inhibition)) of a pharmacologically active metabolite will *decrease* the duration and intensity of the drug action. The opposite is also true (*e.g.*, [enzyme inhibition](https://en.wikipedia.org/wiki/Enzyme_induction_and_inhibition)). However, in cases where an enzyme is responsible for metabolizing a pro-drug into a drug, enzyme induction can speed up this conversion and increase drug levels, potentially causing toxicity.

Various *physiological* and *pathological* factors can also affect drug metabolism. Physiological factors that can influence drug metabolism include age, individual variation (*e.g.*, [pharmacogenetics](https://en.wikipedia.org/wiki/Pharmacogenetics)), [enterohepatic circulation](https://en.wikipedia.org/wiki/Enterohepatic_circulation), [nutrition](https://en.wikipedia.org/wiki/Nutrition), [intestinal flora](https://en.wikipedia.org/wiki/Intestinal_flora), or [sex differences](https://en.wikipedia.org/wiki/Sex_difference).

In general, drugs are metabolized more slowly in [fetal](https://en.wikipedia.org/wiki/Fetal), [neonatal](https://en.wikipedia.org/wiki/Neonatal) and [elderly](https://en.wikipedia.org/wiki/Elderly) [humans](https://en.wikipedia.org/wiki/Human) and [animals](https://en.wikipedia.org/wiki/Animal) than in [adults](https://en.wikipedia.org/wiki/Adult).

Genetic variation ([polymorphism](https://en.wikipedia.org/wiki/Polymorphism_%28biology%29)) accounts for some of the variability in the effect of drugs. With N-acetyltransferases (involved in *Phase II* reactions), individual variation creates a group of people who acetylate slowly (*slow acetylators*) and those who acetylate quickly, split roughly 50:50 in the population of [Canada](https://en.wikipedia.org/wiki/Canada). This variation may have dramatic consequences, as the [slow acetylators](https://en.wikipedia.org/w/index.php?title=Slow_acetylators&action=edit&redlink=1) are more prone to dose-dependent toxicity.

[Cytochrome P450 monooxygenase system](https://en.wikipedia.org/wiki/Cytochrome_P450_monooxygenase_system) enzymes can also vary across individuals, with deficiencies occurring in 1 – 30% of people, depending on their ethnic background.

Dose, frequency, route of administration, tissue distribution and protein binding of the drug affect its metabolism.

*Pathological factors* can also influence drug metabolism, including [liver](https://en.wikipedia.org/wiki/Liver), [kidney](https://en.wikipedia.org/wiki/Kidney), or [heart](https://en.wikipedia.org/wiki/Heart) diseases.

*In silico* modelling and simulation methods allow drug metabolism to be predicted in virtual patient populations prior to performing clinical studies in human subjects.[[15]](https://en.wikipedia.org/wiki/Drug_metabolism#cite_note-pmid17268485-15) This can be used to identify individuals most at risk from adverse reaction.