Clinical relevance of St. John's wort drug interactions revisited.

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Author(s) Nicolussi, Simon; Drewe, Jürgen; Butterweck, Veronika; Meyer zu Schwabedissen, Henriette E
Author(s) at UniBasel Meyer zu Schwabedissen, Henriette ;
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The first clinically relevant reports of St. John's wort (SJW) interacting with drugs, altering their bioavailability and efficacy, were published around millennium: In 2000, a pharmacokinetic interaction between SJW and cyclosporine caused acute rejection in two heart transplant patients. Since then, subsequent research has shown that SJW altered the pharmacokinetics of several drugs such as digoxin, tacrolimus, indinavir, warfarin, alprazolam, simvastatin, or oral contraceptives. Research revealed that these interactions were caused by pregnane-X-receptor (PXR) activation. SJW is a potent activator of PXR and hence inducer of cytochrome P450 enzymes (most importantly CYP3A4) and P-glycoprotein. The degree of CYP3A4 induction correlates significantly with the hyperforin content in the product. Twenty years after the first occurrence of clinically relevant pharmacokinetic drug interactions with SJW, this review revisits the current knowledge on the mechanisms of action and on how pharmacokinetic drug interactions with SJW could be avoided.

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