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Pharmacogenomics of methadone maintenance treatment.

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Abstract

Methadone is the major opioid substitution therapy for opioid dependence. Dosage is highly variable and is often controlled by the patient and prescriber according to local and national policy and guidelines. Nevertheless many genetic factors have been investigated including those affecting its metabolism (CYP2B6-consistent results), efflux transport (P-gp-inconsistent results), target μ-opioid receptor (μ-opioid receptor-inconsistent results) and a host of other receptors (DRD2) and signaling elements (GIRK2 and ARRB2; not replicated). None by themselves have been able to substantially explain dosage variation (the major but not sole end point). When multiple genes have been combined such as ABCB1, CYP2B6, OPRM1 and DRD2 a greater contribution to dosage variation was found but not as yet replicated. As stabilization of dosage needs to be made rapidly, it is imperative that larger internationally based studies be instigated so that genetic contribution to dosage can be properly assessed, which may or may not tailor to different ethnic groups and each country's policy towards an outcome that benefits all.

KEYWORDS: dependence; drug abuse treatment; enantiomers; metabolism; methadone; opiate; pharmacogenomics; receptors; response variability; transport

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