Journal of Addictive Behaviours and Therapy

Open access Perspective

Future of Pharmacogenetics as an Opioid Agonist Therapy

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INTRODUCTION

Opioid agonists are a heterogeneous group of compounds with varying effects, the most important medically being the relief of acute pain. Opioid agonists activate mu-opioid receptors and, to a lesser extent, delta-opioid receptors, resulting in analgesic effects. Opioid agonists inhibit incoming pain signals in the pain-sensitive thalamo-spinal and reticular tracts by activating mu-opioid receptors at the spinal cord (minor component) and supraspinatus levels. Most analgesic effects are thought to occur in these pathways in the mu-opioid receptors rich thalamus, somatosensory cortex, and association cortex. Receptors in the periaqueductal grey matter and greater raphe nucleus activate descending pain control pathways to potentiate analgesia.

DESCRIPTION

The same mu-opioid receptors are found in the ventral tegmental area. VTA neurons release dopamine in nerve endings in the ventral striatum and medial prefrontal cortex. Activation of this circuit is a common component of reward or euphoric experiences, and this circuit is activated by drugs of abuse such as opioid agonists, ethanol, benzodiazepines, cocaine, nicotine, and cannabis. Opioid agonists enhance dopaminergic transmission in the ventral striatum and medial prefrontal cortex to produce a reward. This enhanced dopaminergic transmission in the ventral striatum and medial prefrontal cortex is due to activation of the mu-opioid receptors, located in γ-aminobutyric acid (GABA) interneurons that inhibit the VTA. Mu-opioid receptors activation results in the inhibition of GABAergic inhibitory interneurons, allowing VTA dopaminergic neurons to release mu-opioid receptors e dopamine in the ventral striatum and medial prefrontal cortex. Activation of this dopaminergic pathway is key to the rewarding properties of opioid agonists. Mu-opioid receptors increase the activation of dopaminergic VTA neurons by attenuating tonic inhibition of GABAergic interneurons. Mu-opioid receptor is also found in the ventral striatum and medial prefrontal cortex and is thought to enhance opioid euphoria.

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CONCLUSION

Genetic variability may be a factor in the complex phenotype of therapeutic response to pharmacotherapy in opioid dependence. It should be remembered that drug therapy for opioid dependence is best provided in conjunction with regular (weekly) counselling, treatment of comorbidities, and social support. Appropriate management of opioid-dependent patients should include effective treatments for comorbidities (nicotine and alcohol dependence, anxiety, mood disorders).

ACKNOWLEDGEMENT

None

CONFLICT OF INTEREST

The author's declared that they have no conflict of interest.

Received:	03-October-2022	**Manuscript No:**	IPJABT-22-15002
Editor assigned:	05-October-2022	**PreQC No:**	IPJABT-22-15002 (PQ)
Reviewed:	19-October-2022	**QC No:**	IPJABT-22-15002
Revised:	24-October-2022	**Manuscript No:**	IPJABT-22-15002 (R)
Published:	31-October-2022	**DOI:**	10.35841/ipjabt-6.5.32

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Citation Rose E (2022) Future of Pharmacogenetics as an Opioid Agonist Therapy. J Addict Behav Ther. 6:32.