Falcaria vulgaris attenuates morphine toxicity in prefrontal cortex in rats

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Background
Morphine is a major risk factor in the development of functional disorder of several organs. *Falcaria vulgaris* is a vegetable that contains antioxidant ingredients.
Objective
This study was designed to evaluate the effects of *F. vulgaris* against morphine-induced damage to the prefrontal cortex of rats.
Materials and methods
In this study, 64 Wistar male rats were randomly assigned to eight groups: sham group, morphine group (20 mg/kg once daily in the first 5 days and double per day in the following 5 days; on the 11th to 20th day, 30 mg/kg, doubles each day), *F. vulgaris* groups (50, 100, and 150 mg/kg), and morphine+*F. vulgaris* groups (50, 100, and 150 mg/kg). Treatments were administered intraperitoneally daily for 20 days. Ferric reducing/antioxidant power method was applied to determine the total antioxidant capacity. The number of dendritic spines was investigated by Golgi staining. Cresyl violet staining method was used to determine the number of neurons in prefrontal region. Moreover, Griess technique was used to determine serum nitrite oxide level.
Results
Morphine administration increased significantly nitrite oxide level and total antioxidant capacity and decreased the number of neuronal dendritic spines and neurons compared with the sham group ($P<0.05$). In the *F. vulgaris* and morphine +*F. vulgaris* groups, in all dosages, the number of neurons and neuronal dendritic spines increased significantly whereas nitrite oxide level and total antioxidant capacity decreased compared with the morphine group ($P<0.05$).
Conclusion
It seems that *F. vulgaris* administration improves brain's prefrontal region injury in rats due to morphine.

Keywords:
Falcaria vulgaris, morphine, prefrontal cortex

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Introduction

Opioids produce free radicals and cause apoptosis in some cells. Morphine is an opioid analgesic drug, and the main psychoactive chemical in opium [1]. Morphine is addictive and causes physiological dependence [2]. Morphine spreads rapidly in brain tissue within 10–20 s and attaches to the nicotinic acetylcholine receptors (nAChRs) [3]. Morphine rapidly passes through the blood–brain barrier and stimulates the mesolimbic dopamine system. This substance can regulate brain neurotransmitters, including catecholamine and serotonin A [4]. Dopaminergic structure shows a vigorous role in controlling memory and mainly rewards behaviors [5]. Morphine acetylcholine receptors are found in pathways, for instance, accumbens nucleus and ventral tegmental. Stimulation of these receptors increases dopamine release in accumbens nucleus and prefrontal cortex and induces feeling of joyfulness in the user [6]. However, morphine can induce oxidative stress in some organs including the brain [7]. Pathologic changes associated with neuronal apoptosis have been reported owing to the use of morphine [8]. Moreover, morphine can induce increased oxidative stress and neuronal apoptosis, destroy DNA, and produce reactive oxygen species [9]. This compound seems to activate the areas of the brain that play an important role in drug addiction and learning process. Among the brain areas greatly affected by morphine are mesocorticolimbic and brain's prefrontal regions [10]. The prefrontal cortex of brain shows a key role in character and state of mind [11]. The purpose for studying the prefrontal region is for the reason that of the function this cortex has in regulatory performance,