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The molecular aspects of oxidative & nitrosative stress and the tryptophan catabolites pathway (TRYCAT) as potential causes of depression

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Abstract

Depression is the most common mental disorder in the world. It is estimated that 350 million people suffer from depression worldwide. Depressive disorders will have become the second most frequent health problem globally by the year 2020, just behind ischemic heart disease. The causes of depressive disorders are not fully known. Previous studies showed that impaired tryptophan catabolites pathway, oxidative and nitrosative stress may play an important role in the pathogenesis of depression. Patients with depression have lower plasma levels of superoxide dismutase and glutathione peroxidise in comparison to controls. Moreover, depressed patients are characterized by decreased plasma levels of zinc, coenzyme Q10, albumin, uric acid, vitamin E and glutathione. Abnormal nitric oxidative production and nitric oxide synthase activity are also associated with depression. A dysfunction of the tryptophan catabolites pathway, indicated by increased levels of tryptophan 2,3-dioxygenase and indoleamine 2,3-dioxygenase, is also involved in the development of depression. Furthermore, increased levels of kynurenine and quinolinic acid might cause depression. Moreover, studies to date indicate that 8-oxyguanine, malondialdehyde, and 8-iso-prostaglandin F2α may serve as possible biomarkers. Additionally, regulation of defective mechanisms may provide a promising direction for the development of new and effective therapies.