

Pdf Xchange Editor Serial key code for this software can be found in this website [www.pdfexchange.com](http://www.pdfexchange.com)Effect of long-term cyclosporin A therapy on the eicosanoid metabolism and function in man. There are extensive data on the generation of prostanoids by blood monocytes in the course of a whole range of inflammatory disorders, and on the activation of such cells by lipopolysaccharides, and it is generally considered that the influence of cyclosporin A (CsA) on the immune system is mediated, in part, through its inhibitory action on monocyte/macrophage function. In order to study the effect of long-term CsA treatment on monocyte activation, we examined the profiles of prostanoid production by these cells in a group of 12 alloantibody positive renal allograft recipients treated with the drug (mean CsA trough levels: 86.2 +/- 8.5 ng/ml) for 22.6 +/- 7.7 months (range 14-38 months). These plasma concentrations of CsA were significantly lower than those found in patients undergoing short-term therapy or those in a group of normals (193.4 +/- 18.6 ng/ml), but higher than those found in patients receiving conventional, low-dose immunosuppression (33.8 +/- 16.8 ng/ml). We found that the production of six of the major prostanoids (thromboxane B2, 6-keto-prostaglandin F1 alpha, prostaglandin E2, 12-hydroxy-heptadecatrienoic acid, 13-hydroxy-octadecadienoic acid and 15-hydroxy-eicosatetraenoic acid) was elevated in the presence of exogenous arachidonate (10(-6)M) in normal subjects (140-150%). All six prostanoids were also significantly increased in six patients, with further significant elevations in one patient with renal rejection, two patients with active disease and three patients with stable renal function.(ABSTRACT TRUNCATED AT 250 WORDS)Rising c-Fos expression underlies the temporal regulation of rat alpha 2-adrenoceptors after in vivo stresses. We examined c-Fos expression during the development of long-term brain changes induced by in vivo stresses using a highly sensitive and specific monoclonal antibody. We





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