

Original Studies**Seasonal Variation in Glucocorticoid Activity in Healthy Men¹****Brian R. Walker², Ruth Best, Joseph P. Noon, Graham C. M. Watt and David J. Webb***University of Edinburgh, Department of Medicine, Western General Hospital (B.R.W., R.B., J.P.N., D.J.W.), Edinburgh EH4 2XU; and University of Glasgow, Department of General Practice, Woodside Health Centre (G.C.M.W.), Glasgow G20 7LR, Scotland, United Kingdom*Address all correspondence and requests for reprints to: Brian R. Walker, British Heart Foundation Senior Research Fellow, University of Edinburgh, Department of Medicine, Western General Hospital, Edinburgh EH4 2XU, Scotland, United Kingdom. E-mail: B.Walker@ed.ac.uk.

Many endocrine systems are subject to seasonal variation. However, studies of the hypothalamic-pituitary-adrenal axis in man have been limited to patients with psychiatric illness with conflicting results. We studied 105 healthy men, age 24–33 yr, during a 15-month period that included two winters. We measured cortisol and its metabolites by gas chromatography/mass spectrometry in plasma and urine and the intensity of dermal blanching after overnight topical application of beclomethasone dipropionate.

There were no differences between subjects studied during the two winter periods, but marked differences between subjects studied in winter and summer. In winter, 0900-h plasma cortisol concentrations were higher (73 ± 10 ng/mL, $n = 41$ vs. 35 ± 4 , $n = 25$ in summer; $P < 0.01$), total cortisol metabolite excretion was lower (678 ± 67 μ g/mmol creatinine vs. 900 ± 98 ; $P < 0.05$), the ratio of metabolites of cortisol to those of cortisone was higher (3.0 ± 0.2 vs. 2.1 ± 0.1 ; $P < 0.01$), and dermal glucocorticoid sensitivity was higher (7.2 ± 0.4 arbitrary units vs. 5.6 ± 0.5 ; $P < 0.02$). Although blood pressure and fasting insulin/glucose relationships were not measurably different between seasons, these correlated with dermal vasoconstriction and cortisol metabolite excretion rate.

We conclude that plasma cortisol and tissue sensitivity to glucocorticoids are higher in winter, but cortisol production rate is reduced. This could be explained by a reduction in cortisol clearance rate: urinary free cortisol/cortisone ratios were not different but A-ring-reduced metabolites of cortisol were higher in winter, suggesting that conversion of cortisone to cortisol by hepatic 11 β -hydroxysteroid dehydrogenase 1 is enhanced. It is an intriguing possibility that increased glucocorticoid activity contributes to the increased prevalence of disease during the winter.

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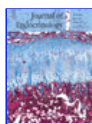
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