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RESPONSE

Response☆

We thank Dr. Watson and Professor Young for their comments on our study, which demonstrated a blunted cortisol response to apomorphine in patients with schizophrenia and which led to the conclusion that this constituted additional evidence for dopaminergic dysfunction in schizophrenia (Meltzer et al. 2001). However, Watson and Young err in their suggestion that our results are more related to elevated cortisol secretion than to dopaminergic dysfunction. First, basal cortisol levels were covaried in our analysis, so the differences that emerged with regard to cortisol secretion, our primary measure, between controls and patients with schizophrenia after apomorphine administration, were independent of any effects of this measure of hypothalamic-pituitary-adrenal (HPA) axis activity. Second, they base their conclusion on evidence that we believe has little or no relevance to our study. Namely, Watson et al. (2000) found that acute and chronic treatment with hydrocortison, 100 mg p.o., in normal volunteers blunted the growth hormone (GH) response to GH releasing hormone (GHRH). They concluded that cortisol acts at the pituitary level to suppress GH release. They noted no effect of the hydrocortisone on prolactin response to GHRH and confined the relevance of their findings to the effect of corticosteroids on *GH release* from the pituitary. The rationale for their current suggestion that HPA axis abnormalties per se could explain the *cortisol* response to *apomorphine* and, indeed, *all* measures of neurotransmitter-evoked pituitary hormone responses, is not apparent to us. In particular, effects of massive increases in cortisol levels to suppress GH release do not necessarily apply to the complex cascade of events that follow the administration of a dopamine agonist and that lead ultimately to increased CRF, ACTH, and cortisol secretion.

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