

REPLY

GBG, Hormones, Genes, and Anomalous Dominance: A Reply to Commentaries

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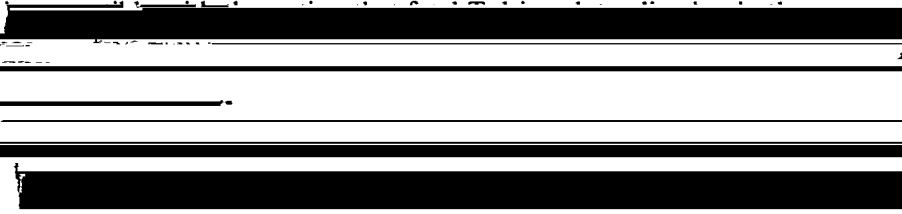
AND

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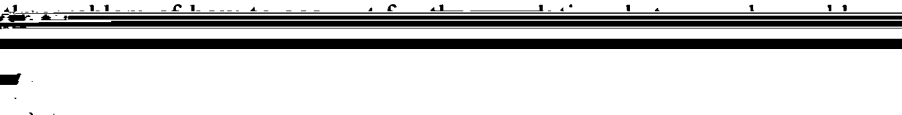
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The present issue includes three further commentaries on our assessment of the Geschwind-Behan-Galaburda (GBG) model of cerebral lateralization (Bryden, McManus, & Bulman-Fleming, 1994). Like the commentaries accompanying our original article, these papers sometimes

one. If one examines Fig. 1 of the target article (Bryden, McManus, & Bulman-Fleming, 1994), where we diagram the GBG model as we understand it, fetal testosterone (T) is an unobserved intervening variable; the observations made by GBG and those we have examined in our assessment of the model are correlations between the numerous variables along the right-hand side of the figure. Logically, it is possible that it is some factor ("X") other than T that produces all of these effects. We agree with Berenbaum and Denburg that studies such as that of Grimshaw (1993; see also Grimshaw, Bryden, & Finegan, 1995) lead to findings



GBG suggest, but this only serves to replace T with X and does not solve



association between RD and ANA even though this is not due to genetic association at the *individual level*. The conclusion must be that some aspect of the common family environment is responsible for the association. One possibility is that, as Geschwind suggested, the fetus may have an anomalous endocrine environment provided by the mother. If that environment were under maternal genetic control, and if it resulted in RD and ANA in the offspring, then there would be an association be-

co-segregation, since the characteristics are being transmitted maternally rather than through the individual's own genotype. We emphasize that such a hypothesis is pure speculation but it does encourage us to think

of only .31 for the relation between handedness and language lateralization, hardly strong support for the position that handedness is a better measure of brain lateralization than dichotic listening.

Ultimately, we feel that Van Strien misses the point. While it would be nice to carry out good MRI studies, the ultimate point is one of linking brain anatomy and physiology to *behavior*, and one therefore needs good behavioral measures. Certainly none of us would wish to deny that there is a neural substrate for functional asymmetries, as Van Strien suggests, but we remain to be convinced that the GBG model tells us anything about that substrate. It may be that the new imaging studies will provide