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RESPONSE II TO GASSER'S LETTER

To the Editor: Until radioassay and monoclonal antibodies bring about a simplification of H-Y serology (as they have with H-2 serology), the available H-Y serology techniques, used in the right way, serve perfectly well. Small differences are not alarming or equivocal when based on repeated "blind" tests, with alternative assays of the same materials and with the same reagents. No critical interpretations have been made outside this context.

There is no "range" of H-Y in normal females, excepting obligate heterozygotes as in the polled goat, and no "significant" H-Y in "normal" females has ever been registered. Normal males are strongly H-Y positive, but it would be astonishing if there were not some variation of expression, as there is even for H-2 (and *Sxr* mice are outbred and so are liable to genotypic variation). What probably is crucial is the amount of H-Y needed to induce a testis; below that threshold, the likelihood of aberrant development is presumably maximal.

The warning against superfluous male antigens also must be viewed with caution. Conservatism of H-Y was the prime basis for proposing its function in sex determination. Given the span of time since isolation of the mammalian Y chromosome, any female antimale response should focus on the most salient conserved molecular feature. A family of functionally related molecules sharing "group-specific" H-Y could account for such conditions as incomplete testes in patients with diminished expression of H-Y. The families of immunoglobulin and of glycoprotein-70, which also exhibit sessile residence in the plasma membrane, combined with the propensity for secretion, particularly connote such a scheme.

STEPHEN S. WACHTEL[1] AND RON G. ROSENFELD[2]

[1] Sloan-Kettering Institute, New York, NY 10021.
[2] Department of Pediatrics, Stanford University Medical Center, Stanford, CA 94305.