terone and a 100-fold excess of estradiol or DES does not diminish the amount of testosterone (approximately 20 percent) converted to 5β-reduced metabolites. Lack of effect is not due to assay conditions: spiroxenone (20-spirox-4-ene-3-one), a specific inhibitor of ring A reduction, decreased formation of 5β-reduced metabolites by more than 50 percent.

The classical model of steroid hormone action on brain target cells requires that the hormone binds to cytoplasmic receptors and is translocated to the cell nucleus where initiated genomic effects are translated into neuronal changes (*20*). Whether events of this type mediate androgen action on behavioral mechanisms is still uncertain. In the dove, there appears to be no decrease in nuclear uptake of testosterone that could account for behavioral insensitivity to androgen (*5*). However, our evidence indicates a significant increase in 5β-reductase activity, and such activity is likely to compete both with the conversion of testosterone to active metabolites and with the binding of testosterone to receptors. We suggest, therefore, that inactivation by 5β-reduction influences behaviorally effective androgen concentrations within target cells of the POA. This mechanism could be important in determining brain sensitivity to circulating androgen under changing hormonal conditions. At present, we do not know how 5β-reductase is controlled, but the evidence points to a role for an aromatization product.

J. B. HUTCHISON
T. STEIMER
MRC Unit on the Development and Integration of Behaviour, University of Cambridge, Madingley, Cambridge CB3 8AA, England

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9. Intramuscular 5β-DHT induces behavior resembling sexual patterns in chicks given appropriate sensory stimulation (J. Balthazart, G. Malacarne, P. Deviche, *Horm. Behav.*, in press). But there is no evidence for direct action of 5β-DHT on brain mechanisms of behavior in any species. In the dove, 5β-reduced metabolites, which are major products of testosterone catabolism, do not occur in hypothalamic cell nuclear fractions after [^{3}H]testosterone is injected.
10. The stereotyped perch call is uttered repetitively by the male in visual isolation. This behavior is specifically androgen-dependent; estrogen is ineffective (J. B. Hutchison, T. Steimer, R. Duncan, *J. Endocrinol.*, in press). As in male courtship behavior, the effectiveness of intrahypothalamic androgen on perch calling declines with time after castration (J. B. Hutchison and L. Innes, in preparation).
11. Brain samples from individual males were removed by means of a topographical dissection procedure and assayed for total (5β-DHT, 3α- and 3β-, 17β-diols) 5β-reductase activity [T. Steimer and J. B. Hutchison, *Brain Res.* **209**, 189 (1981)]. The intra-assay coefficient of variation was 11.3 percent (18 samples in duplicate) and conversion rates were linear with respect to time and amount of brain tissue used. The large excess of substrate used in this procedure avoids interference with high-affinity receptors and the effect of endogenous substrate. Group assay data were compared statistically by analysis of variance with planned comparison between group means (two-tailed). Brain areas were compared with the paired *t*-test (two-tailed). Males were randomly distributed to groups for treatment with either saline or hormone. All males were killed for assay during the same period so that the length of the postcastration period could be strictly controlled. A member of each group was killed for brain assay on each day to avoid possible interassay variation biasing the results. In view of the number of males used, initiation of treatment with hormones was staggered to ensure that each male received the same period of treatment. The experiments were carried out blind by assigning a code number to each assay sample which allowed identification of individual males after assay. Assays, behavioral observations, and dissection of samples were carried out by separate researchers. Testosterone propionate (300 μg per day), estradiol benzoate (300 μg), and DES (300 μg per day) were injected intramuscularly as a microcrystal suspension in 0.85 percent sodium chloride solution. Maintenance of birds, hormone administration, behavioral selection criteria, and castration procedures were described previously (*6*).
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13. Males were observed for vocal behavior (perch calling) for 10 minutes, beginning at 0900 hours. Behavior was recorded continuously by means of the WRATS computer-compatible system. The longest of the daily durations of calling (peak durations) were calculated for each bird.
14. When [^{3}H]testosterone of high specific activity is injected intramuscularly, uptake of radioactivity in the POA is very low compared to the pituitary in the dove (T. Steimer and J. B. Hutchison, in preparation), suggesting that relatively few cells take up the hormone.
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Aseismic Uplift in California

We disagree with several of the arguments cited by Jackson *et al.* in support of their view that "the inference of widespread aseismic uplift in southern California is not justified" (*1*). Specifically, the striking correlation shown in figure 1 of Jackson *et al.* (*1*) is an artifact of the construction, the rod calibration data are atypical, the cited regression techniques are of doubtful value, and the geologically and geodetically determined uplift rates are inappropriately compared.

First, figure 1 of the report by Jackson *et al.* (*1*) offers the most visually impressive suppport for their conclusion that signal (tilt) and topography are correlated at both short and long wavelengths. However, this illustration provides no support for this conclusion. For example, we show by means of the same method used in generating figure 1 of Jackson *et al.* (*1*) that a similarly strong correlation is produced through the application of a uniform tilt (and hence devoid of short wavelength components) to an actual terrain profile characterized by unequal bench mark spacing (*2*).

Second, the rod calibration data presented by Jackson *et al.* (*1*) exaggerate the magnitude of the normal rod error and, by implication, misrepresent the validity of the correction procedures designed to accommodate these errors. Rod calibration data are represented in figure 3 of the Jackson (*1*) report as rod "strain," a procedure that suggests that the error is distributed as a step function and thus distorts the error in the region of the calibration points. Moreover, by representing an error of 0.02 mm in the 0.2-m footpiece (or ungraduated part) of the rod as "strain," Jackson *et al.* (*1*) imply that the error over a nominal length of 1 m would amount to 0.10 mm (1×10^{-4}). However, the most misleading distortion introduced into this particular argument is the characterization of the identified rod, 312-268, as "a typical rod used in the southern California study" (*1*). The validity of the rod excess, which is derived from the calibration data and permits the conversion of the field measurements into corrected observed elevation differences, depends on the distribution of the error over the length of the rod. The less linear this distribution, the less valid the correction. Linear regressions of cumulative rod errors on cumulative nominal lengths computed for the first 100 calibrations in the National Geodetic Survey rod and instrument file read to the nearest 0.01 mm showed that these errors are indeed generally linearly distributed (*2*). Ninety percent of the standard deviations about the regression lines were 0.02×10^{-3} m (2×10^{-5}) or less; the 1965 and 1966 calibrations for rod 268 yielded the larg-

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est standard deviations (0.07×10^{-3} m) encountered. Hence rod 268 is the least typical of this representative group. Thus the implication that arises from the characterization of this rod as typical—namely, that geodetically determined elevation differences are generally contaminated by errors as large as those that might be produced through the use of rod 268—is unjustified.

Third, Jackson *et al.* (*1*) contend that regressions through the short wavelength residuals obtained from a fitting of the data to low-order polynomials provide a legitimate vehicle for estimating height dependent error (and thus a basis for "correcting" the data). Our experience, on the other hand, indicates that the regression coefficients are especially sensitive or unstable to both the rejection criteria and the order of the polynomial (*2*). Moreover, and perhaps even more important, short wavelength correlations over certain lines tend to be dominated by those correlations that occur within fractional parts of the line—suggesting that real movements control the correlation. Indeed, there is no way in which subsidence of residual lows can be distinguished from uplift of residual highs; both produce correlations of the same sign and hence lead to ambiguous interpretations of the analytical results.

Finally, the manner in which Jackson *et al.* (*1*) choose to compare geologically determined uplift rates with those based on geodetic measurements is again misleading. Clearly, short-term episodic events cannot be resolved on the basis of geologic observations, whereas geodetically determined aseismic episodic uplift in southern California computed on an annual basis could easily range up to 0.5 m/year or more. Useful comparisons should be based on the cumulative uplift developed through the full cycle of geodetically defined uplift and partial collapse, which for the Palmdale area produces an average value of about 4 to 5 mm/year—and, hence, in good agreement with those rates based on geologic studies.

ROBERT O. CASTLE
MICHAEL R. ELLIOTT
THOMAS D. GILMORE
ROBERT K. MARK
EVELYN B. NEWMAN
JOHN C. TINSLEY III
U.S. Geological Survey,
Menlo Park, California 94025

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Castle *et al.* do not seriously challenge the evidence for systematic errors on the leveling data. They do not explicitly deny the correlation between apparent uplift and topography, which has now been observed on additional profiles in southern California (*1*) and by other investigators (*2*). They do not mention the reversal in the sign of this correlation where rods were interchanged in 1964, which we presented as evidence that rod calibration errors cause part of the correlation (*3*). They offer subsidence caused by fluid extraction as an alternative explanation for the observed correlation. We concur that subsidence may contribute to the observed correlation [and to the inferred uplift (*4*)]. It does not explain the reversal in correlation cited above, nor the strong correlation in a long section between Saugus and Lebec where there was no fluid extraction (*1, 2*).

As to their objection to figure 1 in (*3*), we estimated the correlation between uplift and topography using classical regression techniques. These techniques are described in our report (*3*) and do not depend in any way on figure 1. The figure does show the reversal in sign of the correlation in 1964, which is not an artifact of the plotting technique.

We did not imply that rod errors are distributed as step functions, nor did this assertion play any role in our argument. We argued that many rods suffer nonuniform errors, with accumulated errors as large as 100 ppm (0.1 mm) over a 1-m section. The largest relative error for rod 312-268 is 0.11 mm in the top 1 m of the rod, not in the footpiece. There were many rods used in the southern California study (*4*) for which calibration data (*5*) indicate accumulated rod errors of 0.1 mm or more over 1-m sections. Examples are rods 312-368 and 312-387, used in 1955; 312-301 and 312-322, used in 1961; 312-248 and 312-254, used in 1964; and 317-0163 and 317-0263, used in 1965. Castle *et al.* compare rod 312-268 against rods calibrated "to the nearest 0.01 mm." Such calibrations were first begun in 1965, whereas the major episode of inferred aseismic uplift took place before 1964. Very few of the relevant rods were ever calibrated to the nearest 0.01 mm.

Their objection to our regression analysis is unfounded. We used standard, well-tested techniques. Our results were stable with respect to moderate changes in the data rejection criterion and the order of polynomial used to distinguish long wavelength from short wavelength effects. Using some different assumptions (*1*), we have reanalyzed our data, and the results are in good agreement with our previous results (*3*). Our results (*1*) for the profile from Saugus to Lebec are in close agreement with the independent results of Stein (*2*). The "experience" to which Castle *et al.* refer, if correctly presented (*6*), is based on the use of smaller data sets and much higher order polynomials than we employed. One should expect instability under such conditions. We tested our results for statistical significance, but Castle *et al.* do not report any such test. The correlations between uplift and topography are not due to faults in our analytical techniques; they are real, and they reveal serious systematic errors in the leveling data.

We do not claim that long-term averages of tectonic uplift, including the effects of earthquakes, should necessarily yield the same rates as short-term averages of aseismic uplift. However, the problem of reconciling geologic and geodetic data is one of the exciting challenges of earth science. We agree that short-term episodic events may not be resolved with geologic observations. We are not persuaded that aseismic uplift of tectonic origin could "easily range up to 0.5 m/year." The episodic uplift proposed by Castle *et al.* (*4, 7*) is neither predicted nor explained by any of the well-known models of mountain building. For this reason its existence would have "surprising and important implications," as we stated (*3*).

The central issue is the reliability of the leveling data used to infer the uplift. We have shown that much of the data are subject to systematic errors that are of the right sign and magnitude to explain most of the inferred uplift. Castle *et al.* have not directly addressed the evidence for these systematic errors. We stand by our conclusion that "inference of widespread aseismic uplift in southern California is not justified."

DAVID D. JACKSON
WOOK B. LEE, CHI-CHING LIU
Department of Earth and Space Sciences, University of California, Los Angeles 90024

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