for the ovariectomized rats and 82.5 for the ovariectomized-adrenalectomized ones. These scores were not significantly different.

Figure 1 shows the effects of KCl application; KCl applied to the neocortex facilitated lordosis behavior in the rats with intact adrenal glands, but not in the adrenalectomized rats. Analysis of variance revealed a significant effect of adrenalectomy ($F = 221.5$; d.f. = 1,88; and $P < .001$), a significant time of testing effect ($F = 34.2$; d.f. = 4,325; and $P < .001$), and a significant interaction ($F = 85.6$; d.f. = 4,352; and $P < .001$).

Lordosis behavior on the estrogen-progesterone test which followed the KCl test is also shown in Fig. 1. Both groups showed frequent lordosis responses and the two groups did not differ significantly in this regard.

The results of experiment 1 suggested that the KCl facilitation of lordosis is mediated by the adrenal gland, possibly by adrenal progesterone. This is consistent with the findings of Feder and Ruf and of Resko (*4*), who showed that ACTH can stimulate adrenal progesterone secretion and can induce receptive behavior in estrogen-primed rats. To obtain further confirmation of this possibility, experiment 2 was designed to examine KCl-induced lordosis behavior in rats in which ACTH secretion was blocked by the synthetic glucocorticoid dexamethasone. Female rats were ovariectomized, implanted with cannulas, and screened for their response to estrogen as in experiment 1. All received 2 μg of estradiol benzoate daily for 2 days. On day 3, 15 animals received a subcutaneous injection of 500 μg of dexamethasone (9α-fluro-11β,17α,21-trihydroxy-16α-methyl-1,4-pregnadiene-3,20-one) per kilogram of body weight dissolved in propylene glycol in a concentration of 1.25 mg/ml according to the procedures of Paris *et al.* (*5*); six control animals received an injection of propylene glycol. One hour later all animals were tested for lordosis behavior. Eight dexamethasone-treated and six vehicle-treated animals were then administered 15 percent KCl through the cannulas; seven dexamethasone-treated animals received saline through the cannulas at the same time. All animals were then retested for behavior 15, 30, 60, and 90 minutes later. Following the last test all animals were given a subcutaneous injection of 500 μg of progesterone and were tested again 3 hours later.

As is shown in Fig. 2, KCl facilitated lordosis in estrogen-primed rats, but not in those pretreated with dexamethasone. Dexamethasone by itself had little effect on lordosis behavior. Analyses of variance indicated that the groups differed significantly ($F = 29.6$; d.f. = 2,18; and $P < .001$), that the group treated with KCl plus dexamethasone did not differ from the dexamethasone group ($F < 1.0$, not significant), and that the KCl plus dexamethasone group was significantly lower than the group treated with KCl only ($F = 50.0$; d.f. = 1,18; and $P < .001$). On the final test, after progesterone treatment, all groups showed high levels of lordosis behavior, and the groups did not differ significantly from each other.

In contrast to previous findings (*2*), the KCl-treated animals in the present studies showed only very slight motor impairment. The results of these studies suggest that the KCl facilitation of lordosis behavior is mediated by adrenal gland secretions rather than by the induction of a suppression of some neocortical inhibitory system. The data thus require a reconsideration of the currently popular model which postulates that progesterone acts by releasing neocortical inhibition of lordosis behavior. Based on the present conclusion, the only compelling data for the possibility of a neocortical inhibitory system derive from Beach's observation (*6*) of exaggerated lordosis behavior in some female rats that had sustained neocortical lesions.

The most reasonable interpretation of the present findings would seem to be that topical application of KCl to the neocortex acts as a stressor causing the release of ACTH and of adrenal steroids. The most likely adrenal hormone to be active is progesterone, since neither corticosterone nor a wide variety of metabolites of progesterone are as effective as progesterone in inducing lordosis in estrogen-primed rats (*7*). Moreover, it is known that a variety of agents, such as reserpine and Metopirone, can induce lordosis in estrogen-primed rats and do so concomitant with a rise in plasma progesterone levels and that both the behavioral and secretory processes can be blocked by dexamethasone (*5*). Presumably, KCl treatment is acting in the same way.

The present findings do not negate the possibility that lordosis behavior is under neocortical inhibition. The data only cause one to raise questions about the interpretation of experiments regarded as providing support for that hypothesis.

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Electronic and Catalytic Properties of Tungsten Carbide

Levy and Boudart (*1*) reported that tungsten carbide is much like platinum in its catalytic activity, whereas pure tungsten is not, and suggested that formation of the compound made the electron distribution of tungsten platinum-like. Subsequently, x-ray photoemission (*2*) and soft x-ray appearance potential (*3*) experiments on tungsten, platinum, and tungsten carbide were independently reported. Apparently conflicting conclusions were drawn in the reports of these experiments. Our purpose in this comment is to note that the two sets of experimental data are not inconsistent with one another and that they are both consistent with the essential point of Levy and Boudart's suggestion as we read it.

The photoemission study probed the occupied conduction bands of the three materials below the Fermi level, E_F. Because of transition probability weights, the *d*-like component of these bands is most heavily weighted in the spectra (*4*). The spectrum of tungsten carbide in the vicinity of E_F indicated a high density of *d*-like electron states at E_F, and in this respect the compound

is like platinum but unlike tungsten, which has a low density of d-like states there.

The appearance potential experiment probed about the same depth into the sample, but it probed the width of the unoccupied d-band states above E_F, shedding little light on the density of states at E_F. Platinum, with its almost filled d bands, displays only a narrow unoccupied bandwidth above E_F. Tungsten carbide was shown to have a broad unoccupied d band (or more likely, hybrid tungsten $5d$, carbon $2p$ bands) above E_F—broader, in fact, than the unoccupied d band of tungsten. In this sense tungsten carbide is unlike platinum. But being unlike in having a different unoccupied bandwidth is not inconsistent with being alike in having a high density of d states at E_F. Differences between tungsten carbide and platinum in such factors as crystal structure and electrons per atom inevitably break down any rigid band relation between unoccupied bandwidth and density of d states at E_F. A bulk property that we believe is important for strong catalytic activity is a high density of d states at E_F and, in this essential factor, tungsten carbide does appear to be like platinum.

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Analysis of Human Chronic Pain

Timmermans and Sternbach (*1*) essay to investigate the interrelations of personality variables and clinical measures of pain. The multivariate technique which best illuminates this sort of hypothesis is canonical correlation analysis (*2*), which, beginning with two clusters of measures and their matrix of intercorrelations, extracts maximally correlated pairs of subscales linear in the clusters separately. The subscales may be interpreted as either general factors (summations of diverse indicators) or specific "types" (systematic contrasts among the measures of a cluster). In the present instance, the two clusters are the pain variables and the personality measures. The computed canonical pairing of personality and pain scales would provide optimal evidence (as far as linear correlation-based computation can be evidence) for the influence of one upon the other, which the authors seek to estimate.

Unfortunately, the authors chose instead to perform a factor analysis using all of their variables together. This technique cannot express the formal distinction, crucial to the investigation, between the measure clusters and, as a result, there are several flaws in the data analysis as published.

1) The finding on which the authors rest—"a significant proportion of the variance is contributed by variables comprising a factor of *interpersonal alienation and manipulativeness*"—is null in the context of their goals. Whether or not there is information about type or intensity of pain to be gleaned from all the other measures, factor analysis is not searching it out; what it finds has no direct relevance to the establishment of such a relation. In the present case, factor 1 does not load on any variables of the pain cluster, so its estimation is useless for the clinical treatment of pain; similarly, factor 2, the pain factor, does not load on any of the personality variables—those variables have already been forcibly assigned to factor 1—or, in other words, is not at all predictable from the personality cluster. This mutual irrelevance is a specific goal of the rotation routine the authors chose to use, which pursues simple structure at the expense of just those intercluster correlations we are looking for—those which a canonical analysis would specifically display. It might happen that the canonical analysis would, in fact, extract just these two factors as its first pair, but we have no way of knowing.

2) Factor 2, the pain factor, is really only one item with two contrasts arbitrarily tossed in. For, with any score with which ratio is highly positively correlated, such as the second factor score, pain estimate minus ratio will necessarily correlate negatively if only the estimate and ratio are not too strongly associated in the population. But ratio loads high on this factor mostly because clinical is high on this factor and ratio has clinical for numerator. Without such redundancy factor 2 would not have emerged at all: with its effective latent root cut by two-thirds, it would have appeared at the end of the analysis as the unique variance it really is. This is clearly a flaw in the factor analysis. A different choice of redundancies, involving the estimate more, would quite change the content of factor 2. In a canonical analysis, however, it is strength of association with personality measures—not intracluster redundancy—that provides the assortment of pain variables into patterns of loadings. The resulting scales are resistant to this sort of confounding.

3) When one uses a priori certain functional combinations of variables, the results of a factor analysis can be quite misleading. In particular, the correlation between alternate versions of a construct, and thus their assignment to one factor or another, is strongly dependent on mathematical details of form. For instance, ratio and difference represent essentially the same concept: ratio is the antilogarithm of the difference of the logarithms of the contrasting clinical and maximum pain indicators. Yet ratio loads almost wholly on factor 2, while difference loads mainly on factor 3. One contrast between the two types of pain tolerance seems to be part of the pain factor, while an intellectually identical alternate version of the contrast is not. A canonical analysis strategy helps us avoid this paradox. Both difference and ratio are particular special contrasts among the pain indicators. Analysis without any transforms would suggest, by inspection of secondary factors, a selection of linear contrasts which are optimally informative. To determine the influence of the various possible ratios, we would switch contexts and look for similar contrasts among the logarithms of the pain indicators. Ratio and difference are contrasts belonging in different analyses; a single factor analysis only confuses them.

There is a canonical analysis routine