vasodilate the tail. Stability of the internal body temperature is thereby assured within the limits possible through changes in vasomotor state (uppermost tracing in Fig. 1A).

Thresholds for initiation of other thermoregulatory effector processes, such as shivering and panting, have been demonstrated to vary with both the ambient (skin) temperature and the local temperature either of the preoptic hypothalamus (*16*) or of other thermosensitive sites such as the spinal cord (*17*) as controlled by implanted thermodes. The form of such functions often resembles the relation presented in Fig. 2A. Recent research in our laboratory has determined how tail and foot vasodilation can be triggered by heating thermodes implanted in the hypothalamus of squirrel monkeys restrained in cool environments (*12*). Some of these results appear in Fig. 2B in a form that facilitates direct comparison with the adjacent microwave data. The striking resemblance lends credence to the hypothesis that low-intensity microwaves, absorbed in the vicinity of thermosensitive neural tissue in the hypothalamus and elsewhere (for example, posterior hypothalamus, midbrain, spinal cord, or deep viscera), can provoke immediate and dramatic changes in thermoregulatory effector response systems. Theoretical analyses (*18*) suggest that internal hot spots could occur under our experimental conditions (10 mW/cm^2, 2450-MHz microwaves) that would locally elevate temperature as much as 0.5°C. A possible neural mechanism would integrate many small afferent signals from diverse structures throughout the body into a strong effector command. The thermoregulatory neural substrate exhibits the diversity and integrative function appropriate to such a mechanism (*19*). Further, researches into the consequences of multiple thermal inputs confirm that the magnitude of the thermoregulatory effector response can be directly related to the number and sign of localized temperature changes occurring at discrete thermosensitive sites within the body (*20*).

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8. The Lucite restraining chair was mounted 1.85 m from the front edge of a 15-dB standard-gain horn antenna inside a lighted chamber 1.83 by 1.83 by 2.45 m. The interior chamber walls were covered with 20-cm pyramidal microwave absorber (Advanced Absorber Products type AAP-8) to minimize reflections. The long axis of the monkey's body was aligned with the electric vector of the incident plane wave (*E* polarization). Air (± 0.5°C) circulated at 1.1 m/sec through the anechoic space. The animal was under constant surveillance by television camera during the 4- to 5-hour test sessions; sessions were conducted in the presence of a continuous 73-dB (sound pressure level) masking noise to prevent auditory cues to the presence or absence of microwaves.
9. Thermocouples with 0°C reference junctions were constructed in special configurations from 36-gauge copper-constantan wire. Leads were shielded and held out of alignment with the *E* vector. Any thermocouple electromotive force showing abrupt changes greater than 4-μV coincident with microwave onset or termination was discarded as inadmissible datum. Field measurements (*10*) revealed no perturbations of the microwave field by the fine wires at the monkey's location.
10. Microwaves generated by a Cober (model S2.5W) source were fed to the antenna through standard waveguide components. Calibrations to determine far-field uniformity were made with a broadband isotropic radiation detector (Narda model 8306 B). Field intensity was mapped at 12-cm intervals across a 1 by 1.5 m plane passing through the center of the restraining chair location orthogonal to the incident microwave. The maximum nonuniformity of the central 50 by 50 cm of this plane, 8 percent with the chair absent, increased an additional 5 percent with the chair present. Power densities specified in this report were measured with the Narda probe positioned with chair present, at the location of the monkey's head.
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21. Supported by grant 77-3420 from the Air Force Office of Scientific Research. We thank H. Graichen and S. J. Allen for valuable assistance and J. D. Hardy for his continuing encouragement.

15 October 1979; revised 10 December 1979

Elevated Blood Acetaldehyde Levels in Alcoholics and Their Relatives: A Reevaluation

The first evidence of elevated blood acetaldehyde concentrations in alcoholics (1.4 to 2.4 μM) compared with controls (1.1 to 1.9 μM) after ethanol intake was reported by Truitt (*1*). Concentrations of 11 to 45 μM in alcoholics and 4 to 30 μM in controls were reported by Korsten *et al.* (*2*). Schuckit and Rayses (*3*) reported elevated blood acetaldehyde concentrations in healthy young subjects with alcoholic relatives (65 to 78 μM) compared to control subjects (41 to 48 μM). This study indicated that the previously observed blood acetaldehyde dif-

0036-8075/80/0321-1383$00.50/0

ferences were not necessarily the result of alcoholism, and they suggested the possibility that the elevated acetaldehyde concentrations were involved in the etiology of alcoholism.

The data and the interpretations reported in the studies described above, however, must be reevaluated because of recent information regarding the determination of acetaldehyde in human blood (*4*, *5*). These studies indicate that human acetaldehyde levels can be accurately measured only if the blood is immediately deproteinized (to avoid rapid disappearance of acetaldehyde initiated by the blood sampling) and after correction for artifactual acetaldehyde formation (during treatment of the blood).

The rapid disappearance artifact may explain the low acetaldehyde values reported by Truitt (*1*), who was aware of the need for the correction for artifactual formation of acetaldehyde (*6*). However, the data of Korsten *et al.* (*2*) almost certainly reflect the artifactual formation reaction. These workers believed that they would avoid this problem by using thiourea (*7*). However, it was later demonstrated that thiourea does not inhibit acetaldehyde formation in blood (*8*). Thus their acetaldehyde levels most likely reflect a combination of the artifactual reactions and what was left of the acetaldehyde formed in vivo. In the case of the report by Schuckit and Rayses (*3*), the reliability of the acetaldehyde values is open to question because of a number of factors. Unfortunately, their analytical procedure (*9*), including freezing and thawing whole blood (which results in hemolysis) followed by heating in a head-space vessel, does not take into account either disappearance or formation reactions. Thus it seems likely that most in vivo acetaldehyde had disappeared and what was left was mainly artifactually formed acetaldehyde. Hemolysis greatly elevates acetaldehyde formation in human blood (*6*, *8*), which probably explains the high overall blood acetaldehyde concentrations found by these investigators.

The fact that the mechanism for the reported blood acetaldehyde differences might well be found in the analytical procedures used does not necessarily negate the importance of these results. Even with inadequate methods, significant differences were found.

So, what do these acetaldehyde differences reflect? There are three major possibilities in explaining the differences. The first is that they could reflect real differences in in vivo acetaldehyde concentration. A second possibility is that the acetaldehyde formation reaction (or reactions) is more prominent in the blood of alcoholics and their relatives. The third is that the amount of acetaldehyde disappearing from human blood during preparation for analysis differs in alcoholics and their relatives as compared to controls. Because of the lack of further data it is too early to speculate which, if any, of these alternatives provides an explanation for the acetaldehyde differences. In further studies, formation and disappearance of acetaldehyde during the analytical procedure should be controlled.

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19 January 1979; revised 28 June 1979

When compared to controls, alcoholics challenged with alcohol show an increase in acetaldehyde in the blood. The same finding has been noted in nonalcoholic young men with alcoholic close relatives (when compared to controls). As indicated by Eriksson (*1*), the importance of these findings should not be overlooked since, despite methodological difficulties (which we alluded to), subject and control bloods were handled by identical procedures (*2*). Imperfections in methods would tend to add to the variance in each group and thus, if anything, obscure rather than magnify the difference between groups.

The assay of acetaldehyde from blood has always been difficult. In February 1979 we began to process bloods with immediate proteinization, adding thiourea, and analyzing fresh samples while correcting for artifactual acetaldehyde formation as recommended by Eriksson (*1*). Results to date on 15 pairs of nonalcoholic young men with alcoholic family histories compared to controls indicates a replication of the increased acetaldehyde in the group with positive family histories. As would be predicted from Eriksson's comments, the absolute levels of acetaldehyde, however, were less than half of those reported (*2*).

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