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6. Arousal from hibernation typically requires approximately 2 hours. During this time, internal temperature increases from hibernating levels (near ambient temperature, 5°C) to 36° to 38°C, and behavioral activity ensues.
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8. Euthermic animals were anesthetized (Nembutal, 65 mg/kg, intraperitoneally), and pellets were implanted stereotaxically under aseptic conditions with a unilateral cannula guide (22-gauge, stainless steel tube). The tip of the guide was positioned 1 mm above the right lateral ventricle at coordinates AP, 7.1; L, 1.8; H, 9.5 [as given in S. A. Joseph, K. A. Knigge, L. M. Kalejs, R. A. Hoffman, P. Reid, *Edgewood Arsenal Special Publication 100-12* (1966)]. After at least 1 week of recovery from surgery, morphine sulfate (55 μg/μl in sterile 0.9 percent NaCl) was infused into the lateral ventricle with a miniature osmotic pump (Alza model 2001) connected to an injection cannula (26-gauge stainless steel tube) by a short length of PE-20 polyethylene tubing. The tip of the injection cannula projected 1 mm beyond the tip of the guide tube into the ventricular space. Morphine was delivered at a rate of 1 μl/hour for a period of up to 72 hours. For experiments on euthermic animals, the pump was implanted subcutaneously in the interscapular region, with the animals under light ether anesthesia; in hibernating animals, it was not implanted in the animal but rather was immersed continuously in a container of 0.9 percent NaCl maintained at 37°C to provide sustained pumping at a rate equal to that in euthermic animals. After the conclusion of each experiment, the infusion site was verified by injecting India ink through the injection cannula, killing the animal with a Nembutal overdose, and examining thionine-stained, serial coronal sections cut at 20 μm.
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Induced Hearing Deficit Generates Experimental Paranoia

Abstract. *The development of paranoid reactions was investigated in normal people experiencing a temporary loss of hearing. In a social setting, subjects made partially deaf by hypnotic suggestion, but kept unaware of the source of their deafness, became more paranoid as indicated on a variety of assessment measures. The results support a hypothesized cognitive-social mechanism for the clinically observed relationship between paranoia and deafness in the elderly.*

Clinical observation has uncovered a relationship between deafness and psychopathology (*1–3*). In particular, when deafness occurs later in life and the hearing loss is relatively gradual, paranoid reactions are often observed (*4–14*). Delusions of persecution and other paranoid symptoms, first noted by Kraepelin (*6*) in 1915, seem especially prevalent among the hard-of-hearing elderly (*7–9*). Audiometric assessment of hospitalized, elderly patients (with age and other selection factors controlled statistically) has revealed a significantly greater degree of deafness among those diagnosed as paranoid than among those with affective disorders (*10–12*).

Maher (*15*) suggested that one process by which deafness may lead to paranoid reactions involves an initial lack of awareness of the hearing defect by the person, as well as by interacting others. Paranoid thinking then emerges as a cognitive attempt to explain the perceptual anomaly (*16*) of not being able to hear what people in one's presence are apparently saying. Judging them to be whispering, one may ask, "about what?" or "why me?" Denial by others that they are whispering may be interpreted by the hard-of-hearing person as a lie since it is so clearly discrepant with observed evidence. Frustration and anger over such injustices may gradually result in a more profound expression of hostility.

Observers, without access to the perceptual data base of the person experiencing the hearing disorder, judge these responses to be bizarre instances of thought pathology. As a consequence, others may exclude the hard-of-hearing person, whose suspiciousness and delusions about their alleged plots become upsetting (*17*). Over time, social relationships deteriorate, and the individual experiences both isolation and loss of the corrective social feedback essential for modifying false beliefs (*18, 19*). Within a self-validating, autistic system, delusions of persecution go unchecked (*20*). As such, they eventually become resistant to contrary information from any external source (*21*). In this analysis, paranoia is sometimes an end product of an initially rational search to explain a perceptual discontinuity, in this case, being deaf without knowing it.

We now report an experimental investigation of the development of paranoid reactions in normal subjects with a temporary, functional loss of hearing. Across a variety of assessment measures, including standard personality tests, self-reports, and judgments of their behavior by others in the situation, these subjects became significantly more paranoid than did subjects in two control conditions. The effect was transient and limited to the test environment [by the specificity of the instructions, by extensive postexperimental interviews (debriefing procedures), and by the healthy "premorbid" status of each participant]. Nevertheless, qualitative observations and objective data offer support for the role of deafness-without-awareness as a causal factor in triggering paranoid reactions. Although the subjects were young and had normal hearing, these results have obvious bearing on a possible cognitive-social mechanism by which deafness may eventuate in paranoia among the middle-aged and elderly.

Participants were 18 college males selected from large introductory classes. In the selection process, each student (i) demonstrated that he was highly hypnotizable according to the Harvard Group Scale of Hypnotic Susceptibility (*22*) and the Stanford Scale of Hypnotic Susceptibility, form C (*23*); (ii) evidenced posthypnotic amnesia; (iii) passed a test of hypnotically induced partial deafness; (iv) scored within the normal range on measures of psychopathology; and (v) attended at least one of two hypnosis training sessions before the experiment.

Six participants were randomly assigned to the experimental treatment in which partial deafness, without awareness of its source, was hypnotically induced. The remaining participants were randomly assigned to one of two control groups. In one of these groups, partial deafness with awareness of its source was induced to demonstrate the importance of the knowledge that one's difficulty in understanding others is caused by deafness. In the other control group, a posthypnotic suggestion unrelated to deafness was experienced (a compulsion to scratch an itchy ear) along with amnesia for it, to establish whether merely carrying out a posthypnotic suggestion with amnesia might be sufficient to yield the predicted results. Taken together, these two groups provide controls for experimental demand characteristics,

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subject selection traits (hypnotic susceptibility), and the rational basis for the experienced sensory anomaly (*24*).

During group training sessions, each subject was instructed in self-hypnosis and completed consent and medical history forms, a number of Minnesota Multiphasic Personality Inventory (MMPI) scales (*25*), and our clinically derived paranoia scale (*26*). In the experimental session, subjects were hypnotized, after which they listened through earphones to deep relaxation music and then heard taped instructions for one of the three treatments. The use of coded tapes randomly selected in advance by one of the researchers (L.K.) made it possible for the hypnotist (P.Z.), experimenter, (S.A.), observers, and confederates to be ignorant of the treatment assignment of the subjects. All subjects were given the suggestion to begin experiencing the changed state when they saw the posthypnotic cue ("FOCUS") projected on a viewing screen in the laboratory. In order to make the task socially realistic and to conceal the purpose of the experiment, each subject was led to believe he was participating, along with two others (who were confederates), in a study of the effects of hypnotic training procedures on creative problem solving. Because of the hearing defect that subjects were to experience, all instructions and tasks were projected automatically by timed slides, the first of which was the posthypnotic cue. While working on a preliminary anagram task, the two confederates engaged in a well-rehearsed, standard conversation designed to establish their commonality, to offer test probes for the subject's deafness, and to provide verbal content that might be misperceived as antagonistic. They recalled a party they had both attended, laughed at an incident mentioned, made a funny face, and eventually decided to work together, finally asking the subject if he also wanted to work with them.

The instructions had previously suggested that group effort on such tasks is usually superior to solitary responding. The subject's behavior was videotaped, observed directly by two judges from behind a one-way mirror, and scored independently by the confederates immediately after the session. After this conversation, the three participants were asked to develop stories about pairs of people in ambiguous relationships [Thematic Apperception Test (TAT)]. On the first task, they had the option of working together or of working alone. Thus, an interdependence among confederates and the subject was created [important in the natural etiology of paranoia (*17, 19, 21*)], which centered around developing a common creative solution. On the second TAT task, participants had to work alone.

After these tasks were completed, each confederate was instructed by the slides to go to a different laboratory room while the subject stayed in the room to complete evaluation forms, including the MMPI and others. Extensive debriefing followed (*27*), and to remove any tension or confusion, each subject was rehypnotized by the experimenter and told to recall all the events experienced during the session. Subjects were reevaluated in a 1-month follow-up.

Major results are summarized in Table 1, which presents group means and one-tailed *t*-test values derived from a single a priori planned comparison that contrasted the experimental group with the two control groups taken together (*28*). This analysis followed standard analysis of variance tests. As predicted, the experience of being partially deaf, without being aware of its source, created significant changes in cognitive, emotional, and behavioral functioning. Compared with the control groups, subjects in the deafness-without-awareness treatment became more paranoid, as shown on an MMPI paranoia scale of Horn (*25*, p. 283) and on our clinically derived paranoia scale (*26*). Experimental subjects also had significantly elevated scores on the MMPI grandiosity scale of Watson and Klett (*25*, p. 287)—one aspect of paranoid thinking. Experimental subjects perceived themselves as more irritated, agitated, hostile, and unfriendly than control subjects did and were perceived as such by confederates ignorant of the treatment. When invited to work with confederates on the TAT task, only one of six experimental subjects elected to do so; in contrast, 9 of 12 control subjects preferred to affiliate ($z = 4.32$, $P < .001$).

The TAT stories generated by the subjects were assessed in two ways. Subjects' own ratings of the creativity of their stories indicated that experimental subjects judged their stories to be significantly less creative than did subjects in either of the control groups. Second, the stories were scored (reliably by two judges) for the extent to which subjects evaluated TAT characters. An evaluative-judgmental outlook toward other people is a hallmark of paranoia. The experimental subjects used significantly

Table 1. Mean scores on dependent measures distinguishing experimental from control subjects.

Dependent measures	Treatment			t (15)	P
	Deafness without awareness ($N = 6$)	Control			
		Deafness with awareness ($N = 6$)	Post-hypnotic suggestion ($N = 6$)		
Paranoia measures*					
MMPI–Paranoia	1.50	.33	− .17	1.838	< .05
MMPI–Grandiosity	1.33	− .83	− 1.00	1.922	< .05
Paranoia clinical interview form	.30	− .09	− .28	3.667	< .005
TAT					
Affective evaluation	83.35	16.65	33.50	2.858	< .01
Self-assessed creativity	42.83	68.33	73.33	3.436	< .005
Self-rated feelings					
Creative	34.17	55.83	65.83	2.493	< .05
Confused	73.33	39.17	35.00	2.521	< .05
Relaxed	43.33	81.67	78.33	2.855	< .01
Agitated	73.33	14.17	15.33	6.586	< .001
Irritated	70.00	25.00	7.00	6.000	< .001
Friendly	26.67	53.33	56.67	2.195	< .05
Hostile	38.33	13.33	13.33	2.047	< .05
Judges' ratings					
Confused	40.83	27.08	17.67	1.470	< .10
Relaxed	34.17	54.59	65.42	2.839	< .01
Agitated	51.25	24.59	13.75	3.107	< .005
Irritated	45.84	18.92	11.25	3.299	< .005
Friendly	23.34	48.34	65.00	3.385	< .005
Hostile	18.75	5.00	1.67	2.220	< .05

*These measures were taken before and after the experimental session; reported means represent difference scores (after minus before).

more evaluative language, both positive and negative (for example, right-wrong, good-bad) ($t = 2.86$, $P < .01$) than controls did. In addition, they differed significantly ($z = 5.00$, $P < .001$) from the controls in their greater use of positive evaluative language. Experimental subjects reported feeling no more suspicious than did control subjects. These last two findings weaken the possible criticism that the results were based simply on anger induced by the experimental manipulation.

Both groups experiencing a hearing deficit reported, as expected, that their hearing was not keen, but reported no other sensory difficulties. Those who were partially deaf without being aware of the source of the deafness did experience greater confusion, which is likely to have motivated an active search for an appropriate explanation. Over time, however, if their delusional systems were allowed to become more coherent and systematized, the paranoid reaction would be less likely to involve confusion. Ultimately, there is so much confidence in the proposed paranoid explanatory system that alternative scenarios are rejected.

Despite the artificiality of our laboratory procedure, functionally analogous predicaments occur in everyday life. People's hearing does deteriorate without their realizing it. Indeed, the onset of deafness among the elderly is sometimes actively denied because recognizing a hearing deficit may be tantamount to acknowledging a greater defect—old age. Perhaps self-deception about one's hearing deficit may even be sufficient, in some circumstances, to yield a similar response, namely, a search for a more personally acceptable alternative that finds fault in others rather than in oneself. When there is no social or cultural support for the chosen explanation and the actor is relatively powerless, others may judge him or her to be irrational and suffering from a mental disorder. Although our subjects were young and had normal hearing, these findings have obvious bearing on a possible cognitive-social mechanism by which deafness may lead to paranoia among the middle-aged and elderly.

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Exposure of Rats to Alcohol in utero Alters Drug Sensitivity in Adulthood

Abstract. *Pregnant rats were intubated with alcohol (ethanol, 3 grams per kilogram) twice daily throughout gestation. Control animals received solutions of isocaloric sucrose. At birth, offspring were placed with untreated surrogate dams. Beginning at 6 months of age, the offspring were tested for their thermogenic responsiveness to various drugs and to cold. Prenatal exposure to alcohol resulted in tolerance to alcohol and cross-tolerance to pentobarbital and diazepam but did not result in cross-tolerance to chlorpromazine, morphine, and* d*-amphetamine and did not affect responsiveness to cold. This pattern of effects suggests that prenatal exposure to alcohol produces specific long-term effects on the neural mechanisms underlying drug tolerance.*

Fetal alcohol syndrome refers to a pattern of anomalies—growth retardation, morphological abnormalities, and behavioral disorders—in the offspring of female chronic alcoholics (*1*). Nearly all of these anomalies have also been produced in animals prenatally exposed to alcohol (*2*), supporting the validity and utility of animal models of this syndrome.

We report that, in rats, prenatal exposure to alcohol modifies adult responsiveness to alcohol and to drugs for which alcohol causes cross-tolerance but does not alter responsiveness to drugs for which alcohol does not induce cross-tolerance. This pattern of effects suggests that alcohol exposure in utero alters specific cellular mechanisms underlying tolerance in the brain. Because of its sensitivity as an indicator of drug action, temperature regulation was used to assess tolerance (*3*). Considerable information is available concerning the neuroanatomical and neurochemical bases of thermoregulation (*4*), which could provide foci for further studies.

Twenty-five Long-Evans rats (Blue Spruce Farms), pregnant and about 100 days old, were divided into two groups. Group A rats ($N = 15$) were intubated with 3 g of ethanol (8 percent by volume) per kilogram twice daily throughout pregnancy, beginning on day 1 of gestation. This dose is behaviorally teratogenic in rats (*5*). Group C rats ($N = 10$) were intubated with a solution of isocaloric sucrose. The group C animals were given the same amount of food (Teklad 10 percent) and water as received by the group A animals. All animals were individually housed in Plexiglas cages in a

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