tailed, sign test). The mean change of amplitude from the mean of the three baseline values was -2.40 ± 1.40 μv after 12 hours and -3.86 ± 2.50 μv after 36 hours of abstinence. These were highly significant decreases (respectively, P was .0039 and .0054, one-tailed, Wilcoxon matched-pairs signed-ranks test). The prompt return to the baseline level immediately after one cigarette or one pipeful was similarly significant (P was .0054, one-tailed, Wilcoxon test). Examined separately, the mean amplitude envelopes of the AVEP for all four intensities of the flash also showed significant drop from the mean baseline at 12 and 36 hours of abstinence, and increase after smoking (P was .0038 to .0329, one-tailed, Wilcoxon test).

Peak events (events) of the AVEP were identified according to an algorithm (*7*). Briefly, events VI and III are first identified as the most positive and negative peaks within the latency ranges of 125 to 250 msec and 65 to 105 msec, respectively. Then event IV is the most positive peak, after III and before VI, of 75 to 140 msec latency and event V is the most negative peak, after IV and before VI, of 90 to 165 msec latency. The effects on the mean amplitude of the IV-V complex in the eight subjects for whom both events could be so identified are shown in Fig. 1. The drop in the IV-V amplitude response at 36 hours was inversely proportional to flash intensity and was significant only for the dimmest and next to dimmest flashes (respectively, P was .0087 and .0344, one-tailed, Wilcoxon test). After smoking, the increases at these two lowest intensities approached the 5 percent level of significance (respectively, P was .1038 and .0643, one-tailed, Wilcoxon test); the small decreases for the two brighter flashes after smoking were insignificant ($P > .3372$, one-tailed, Wilcoxon test). The slope of the linear regression line of IV-V amplitude with log intensity is another measure which reflects the above relationships. The slope showed a mean decrease or "reduction effect" (*11*) after smoking (-1.89 ± 1.31); decrease occurred in every case, a significant finding ($P = .004$, one-tailed, sign test). The possibility of attributing these slope changes to systematic reduction effects of retesting alone is minimal in view of previous findings that showed some significant "augmentation effects" (*11*), but no reduction effects under short-term retesting conditions (*12*). The increase of slope after 36 hours of abstinence ($+ 0.93 \pm 1.76$) was not significant (P was .1038, one-tailed, Wilcoxon test).

The AVEP curves in Fig. 2 are illustrative. Both subjects show decrease of amplitude envelope and waveform changes at both intensities during withdrawal and prompt return to baseline amplitude and waveform after smoking. Changes in relative size of the IV-V complex are seen between the two intensities shown in Fig. 2. These reflect the increase of the four-intensity, amplitude-intensity, linear regression slope during withdrawal as well as decrease after smoking. They are marked in subject 2. Subject 1, incidentally, also showed increase of the P-300 wave (*13*) during abstinence, but this component showed no systematic changes for the subject group.

There were no significant changes of latencies for peak events IV and V, the 12 scales of the MACL, the 7 subjective behavior scales, or the KFA. That the behavior and mood rating scales and the KFA failed to detect differences after smoking could possibly be attributed to a wearing off of the effects by the time these tests were administered (after the 8.8-minute AVEP test). However, this appears unlikely, considering that these tests also failed to detect differences between the abstinent and baseline conditions. Therefore, the data seem to suggest that the AVEP is a more sensitive measure of the effects of tobacco on the brain than these other instruments.

The difference between the AVEP in tobacco-deprived and satiated conditions obtained by means of two different indices suggest that this substance alters the manner in which the brains of the smokers process sensory stimuli. The changes in the amplitude envelope are convergent with EEG and subjective data indicating a general alerting effect. In addition, the data regarding the IV-V complex suggest a differential effect that favors responsiveness to weak over strong stimuli. The latter is of special interest in relation to the theory of a perception-personality dimension of "augmentation-reduction" developed by Petrie (*14*), which has been supported by studies of the AVEP (*8, 9, 11*). In the context of that theory these data might suggest that smoking selectively enhances perception of weak stimuli among smokers. Further studies of the AVEP might help delineate a specific perception-related psychobiological factor that makes tobacco especially attractive and addicting to persons who become smokers.

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Irradiated Food

A petition has been filed with the German authorities to request permission to irradiate fresh ocean fish destined for human consumption. The purpose of irradiation is to prolong the storage life of iced fish and the petition foresees irradiation on board ship with a dose of 100 krad. There is ample experimental evidence that this dose, applied soon after catch, will

double the useful storage life of fish without adverse effect on organoleptic acceptability.

There is an immediate need for such a process in Germany because of the long distances that our fishing fleet must travel in order to reach the good fishing areas situated in the northwest Atlantic Ocean. The additional storage life obtained through irradiation will be of advantage both to the fishing industry and to the consumer. The industry will benefit from the possibility to extend the duration of fishing voyages by several days thus improving utilization of the ships; and the consumer will benefit through improved hygienic quality of the fish when it is sold at the retail outlet.

Although spores of *Clostridium botulinum* could survive the process, the hazard from the presence of this organism would not be increased because (i) fish caught on the high seas rarely harbor *C. botulinum*, (ii) normal spoilage flora, which compete with the growth of *C. botulinum*, are not completely eliminated with the low dose (100 krad) that is proposed, and (iii) the fish will be stored in melting ice (at a temperature that is too low to permit toxin production).

There is ample evidence that nutritional quality (vitamins, essential amino acids) is not significantly affected at the radiation dose level proposed.

The remaining question to be answered before authorization of irradiated fish for general human consumption can be granted concerns the toxicological safety of irradiated fish. Several long-term feeding studies in animals have already been completed. In the United States, cod and tuna irradiated at 2.8 and 5.6 Mrad have been fed to mice, rats, and dogs; clams irradiated at 0.4 and 0.8 Mrad have been tested in mice, rats, and chickens. In the United Kingdom, cod irradiated at 0.6 Mrad has been tested in mice and rats. Haddock, irradiated at 0.2 Mrad, has been fed to mice in Canada. In the Federal Republic of Germany, marinated herring irradiated at 0.16 and 0.48 Mrad has been tested on rats. None of the criteria on which toxicological evaluation is usually based, such as growth, tumor frequency, fertility, hematology, and so on, were affected as a result of incorporation of irradiated fish into the diet.

Recommendations of a Food and Agriculture Organization of the United Nations, International Atomic Energy Agency, World Health Organization (FAO/IAEA/WHO) Expert Committee are that irradiated foods should be permitted only if they have been tested in at least two species in life-span studies, and that data regarding chronic toxicity must be obtained in a nonrodent species; the radiation dose applied to the food should be as nearly as possible the same as that likely to be used for treatment of that food in commercial practice (*1*). Judged by these criteria, cod, irradiated at 5.6 Mrad, could be permitted, whereas cod, irradiated at only 0.1 Mrad, could not be permitted since no feeding studies have been undertaken with cod treated at this low dose. Presumably, the argument against permitting fish irradiated at the lower dose rests on the assumption that injurious substances might be present in fish irradiated at 0.1 Mrad which are not present after irradiation with 2.8 or 5.6 Mrad. Indeed, this reasoning has been put forward in the report of an FAO/IAEA/WHO Expert Committee which states: "There is evidence to suggest that the concentration of radiolytic products may increase with increasing dose until a certain level is attained and that the concentration may then remain at this plateau or even decrease as the radiation dose is increased" (*2*).

There is considerable evidence that increasing radiation doses result in increased amounts of radiolysis products. A linear increase in products with increasing dose was reported for H_2S in irradiated beef and pork (*3*), for malondialdehyde in irradiated starch (*4*), and for hydrocarbons in irradiated beef (*5*). Malondialdehyde concentration increased not quite linearly with radiation dose in milk powder and five other foodstuffs (*6*). An increase in values approximating a plateau level above 3 Mrad was observed when lipid peroxide values were determined in irradiated beef and pork (*7*) and when deoxy sugars were estimated in irradiated starch (*8*). Extensive gas chromatographic studies on total volatiles of irradiated fish revealed "that the concentration and number of compounds detected increased with radiation dose. . . . All of the compounds identified in the low-dose irradiated samples (0.1 to 0.6 Mrad) were also identified in the megarad-treated samples" (*9*).

There does not appear to be a great deal of evidence that radiolysis products in foods may decrease as the radiation dose is increased. Gas chromatographic analysis of water-methanol extracts of irradiated cod and haddock showed that the size of the peaks increased with increased radiation dose in the range 0 to 2.8 Mrad, but unidentified peaks after 5.8 Mrad were smaller than corresponding peaks after 2.8 Mrad [figure 4 of (*10*)]. This may be an indication that some compounds resulting from lower-dose irradiation are partly fragmented into smaller molecules by radiation doses above about 3 Mrad; but other explanations are also conceivable. I have found no reports that in the range below 3 Mrad a given substance may be present in any food in a higher concentration than after irradiation of the same food with a higher dose.

On the basis of results from a great deal of research on the chemical changes induced by ionizing radiation in various foods, I find it difficult to accept that the criteria laid down in previous years with respect to the radiation dose used to treat the food used in animal feeding studies are entirely valid. In the hope of receiving advice on this point, I would like to ask readers of *Science* to send me any information which indicates that the results of feeding studies with diets irradiated at higher dose should not be extrapolated to foods irradiated at a lower dose.

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