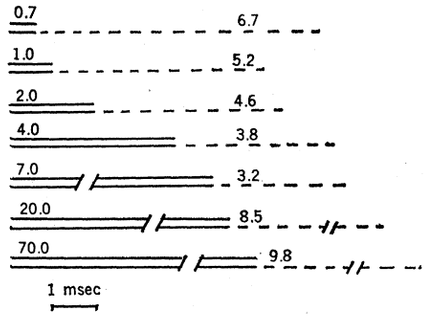

Fig. 2. Duration of the masking aftereffect (------) exerted by the binaural stimulus (═══) on its unilaterally prolonged part. Mean values.

similar to the monaural threshold shifts recorded in the same experiments as a function of stimulus duration and represented by the dotted line in Fig. 1 (mean values). On this curve the threshold for a sound of 1.0-msec duration is taken as a base line to allow a comparison with the lateralization curve for the same initial duration. Both curves coincide almost completely. If the threshold for a sound of some other duration were taken as a base line, then the dotted line would run close to the corresponding lateralization curve. Some discrepancies of the two parameters which arose in several individual experiments were probably due to a redistribution of monaural thresholds for the right and left ears caused by binaural interaction (*2*).

Changes of the thresholds dependent on sound duration are much more drastic for shorter signals than for longer ones. It has been shown by others (*1*), as well as in this study, that a shortening of auditory stimuli from 100 to 1.0 msec is accompanied by a rise of the auditory threshold by 24 to 26 db. A similar rise is produced by a very slight reduction of stimulus duration from 1.0 to 0.5 msec only (Fig. 1).

As stated above, a unilateral prolongation of the signal beyond the limit of about 1.5 msec had only a slight effect on the position of the sound image. But it brought to light a masking action of the binaural effect—the subject perceived a single fused sound and did not hear separately the subsequent monaurally prolonged part of the stimulus. The extent of this inhibitory aftereffect was similar to that found for the interaural successive masking (*3*); it amounted to about 4 to 10 msec (Fig. 2).

When one of the two stimuli was made still longer, its protracted part became free from the masking influence and was heard separately in addition to the binaural signal. But, despite the prevalence of its duration over that of the contralateral signal, it was only necessary to augment the intensity of the latter by 2 to 4 db to restore the masking of the longer stimulus. In other words, contrary to the relations recorded at the first stage of the experiment, it was not the relative duration of the stimulus, but its relative intensity which became specifically effective.

Thus, during the unilateral prolongation of the noise burst, three successive effects were recorded. At first the position of the sound image was greatly affected. In this phase the relative duration of the stimulus exerted a much stronger influence on the binaural interaction than its relative intensity. At a later stage the position of the sound image did not change anymore, but the binaural signal had a masking aftereffect on the monaurally prolonged signal. Finally, the latter was heard separately from the preceding binaural stimulus. In this phase it was the relative intensity of the sound that mainly influenced the binaural interaction.

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Unilateral Inhibition of Sound-Induced Convulsions in Mice

Abstract. *Loud sound repeatedly presented to one ear after an initial bilateral exposure produces a lateralized inhibition of convulsibility in SJL/J mice. Inhibition of the right side does not affect the convulsible state of the left side. Processes leading to inhibition and convulsibility may develop independently within the same animal.*

Mice of many strains convulse to loud sounds. Some seize during the first exposure to noise; others convulse only to subsequent stimulation (*1, 2*). An assignable genetic defect underlies initial seizure susceptibility in one strain (*3*). Normally resistant mice may become convulsible within 30 to 36 hours after as little as a 5-second exposure to intense sound (*2*). The onset of convulsibility may be delayed if mice are stimulated repeatedly at 6- or 12-hour intervals after first exposure to sound (priming). This inhibition is absent or reduced when intervals are longer than 18 hours (*2*). The induced convulsible state can be lateralized and may be limited to one half of the brain. Mice primed with one ear open later convulse only when stimulated through the ear open at priming (*4*). In this study I examined repeated unilateral stimulation following bilateral priming. The results show that inhibition to audiogenic seizures can be localized to the side of acoustic input, that the processes leading to convulsibility and inhibition may develop independently and coincidentally within the same animal, and that unilateral inhibition confers limited protection against convulsing in mice tested bilaterally.

Table 1. Proportions of audiogenic seizures in R and L mice, primed at 0 hr, according to the side of input receiving auditory stimulation. R, right ear open; L, left ear open; 0, both ears flooded with glycerin.

Postpriming stimulation 12, 24, and 36 hr	Test 48 hr	Proportion of clonic convulsions
	Experiment 1	
R	R	2 of 11
R	L	12 of 15
L	R	10 of 12
L	L	0 of 12
	Experiment 2	
0	R and L	26 of 27
R	R and L	13 of 16
L	R and L	10 of 16

Male mice of the highly inbred SJL/J strain aged 21 ± 3 days were obtained from the production branch of the Jackson Laboratory. Each mouse was primed by placing it in a test chamber and exposing it to 30 seconds of bell ringing at a sound level of 95 db above 0.0002 dyne/cm². In the first experiment 50 mice were bilaterally primed, and at 12-hour intervals thereafter each was reexposed to bell ringing for 30 seconds. In half the mice (group R-), the right ear was open and the left was flooded with glycerin at each exposure. In group L-, the left ear only was open. Immediately after each exposure the ear previously open was

filled with glycerin. At 48 hours postpriming, each group was subdivided and tested for convulsibility with either the right ear open (-R) or the left (-L). The results are presented in Table 1. The proportions of clonic convulsions observed in unilateral inhibition groups, R-R and L-L, and in control groups, R-L and L-R, were 2 of 23 and 22 of 27, respectively ($\chi^2 = 23.5$, $P<0.0001$). This indicates that inhibition can be localized to the side of acoustic input, and that within the same animal the processes leading to inhibition and induced convulsibility can develop independently and coincidentally. Mice of the unilateral inhibition groups which did not convulse at 48 hours were retained and tested with either the right or the left ear open at 120 hours postpriming. In R-R-R and L-L-L groups 10 of 10 mice convulsed, and in R-R-L and L-L-R groups 10 of 11 mice convulsed. Thus, the protective effect of unilateral inhibition was impermanent.

To determine whether inhibition at one site conferred immunity to convulsibility during bilateral testing, the following experiment was performed. SJL/J mice were bilaterally primed at 21 days and randomly allocated to R or L inhibition groups, or to a control group whose members had both ears flooded with glycerin. After priming, subjects were reexposed to sound stimulation at 12, 24, and 36 hours. At 48 hours postpriming, all mice were tested with both ears open. As is seen in Table 1, 96 percent of the mice in the control group convulsed whereas 72 percent of those in the inhibition groups convulsed ($\chi^2 = 4.59$, $P < 0.05$). In this within-subject test of competition, the side of input associated with inhibition afforded a slight reduction in the risk of seizure to bilateral stimulation. Although unilateral inhibition did not confer dramatic immunity to bilateral convulsibility, it did significantly lengthen the latencies to clonic convulsions. Whereas 10 of 26 mice of the control group had fast convulsions, only 1 of 23 mice of the inhibition groups convulsed with latencies shorter than 18 seconds ($\chi^2 = 6.32$, $P < 0.025$).

Although the nature of this inhibition is unsettled, certain of its features are known. The phenomenon is a poststimulation refractory state having a relatively long time constant and is not an interference or retrograde process as was previously suggested (*2*). The inhibition is not simply a temporary deafening due to acoustic trauma. Within 2 minutes after a 1-minute exposure to bell ringing, as well as prior to later exposure, mice reliably exhibit pinna reflexes and startle responses to the presentation of soft clicks. In addition, unconditioned galvanic skin responses to sine wave stimuli are detectable in mice tested 15 minutes after cessation of bell ringing.

The locus of the inhibition phenomenon, like that of the sensitization process, is lateral and possibly peripheral. The selective inhibition of convulsibility in one ear does not spread to the contralateral side, confers limited protection to bilaterally induced convulsions, and dissipates within 72 hours after cessation of stimulation.

Dextral-convulsive or sinistral-convulsive mice may be prepared acoustically either by restricting the priming stimulus to one ear or by unilateral reexposure to sound after bilateral priming. These mice, having convulsive "split personalities," provide useful new tools for studies of the physiological effects of intense sound.

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Earthquakes and Nuclear Detonations

The report by Emiliani *et al.* (*1*) asserts some statistical results which would be important if well substantiated. To the undersigned, their evidence appears inadequate. Since it is likely that the conclusions, if unchallenged, will be accepted as authoritative, and misapplied by readers not well versed in the subject, critical remarks are offered.

We do not question triggering of minor seismic events by Nevada test shots, at distances up to about 20 km. We do question the alleged correlation out to 860 km. This radius extends completely over the active areas of California, Nevada, and Utah; it includes two highly seismic zones, one off the northwest coast of California, the other extending from the Imperial Valley into the Gulf of California.

In the interval studied, 15 September 1961 to 29 September 1966, bulletins of the Pasadena laboratory report approximately 1330 earthquakes in southern California and in adjacent Mexico down to 32°N. Many smaller events, especially in Mexico, were registered but not reported. Bulletins from Berkeley (University of California) list comparable numbers of earthquakes for central and northern California. To the total should be added events in Nevada, Utah, Colorado, and so on. The list of Emiliani *et al.* included only 1109 events that probably represent no more than 30 percent of the information available in print, and a much smaller percentage of the earthquakes known to have occurred in the area.

The process by which the partial list was selected should have no relation to hours of occurrence; but the results would meet with more confidence if more data had been used. It is common experience in seismology that deviations from expected means, which look significant when small numbers of events are studied, decrease or disappear when more data are included.

Although much stress is laid on the correlation out to 860 km, it is not documented. Totals are shown only for the entire area. Totals are stated to have been counted for successive annuli; these should be reported, or at least totals should be given separately for the larger radii, say from 400 to 860 km.

Two simple significance tests have been neglected: (i) incidence of earthquakes in 8-hour intervals following the nuclear tests should be compared with incidence in corresponding intervals preceding them (*2*), and (ii) the whole counting process should be repeated after dates and hours when no shots were fired, selected systematically (say by adding 3 months to the day and hour of each actual firing time used).

The procedure lumps earthquakes of all sizes together; necessarily the great majority are small, so that any definite results refer to these. However, if any large regional earthquakes chance to fall in the selected time intervals, their small aftershocks will add to the count. We presume that collapse events have