electrical pulses to tissue slices perfused with Krebs–Ringer medium as described by Srivasan, Neal and Mitchell. Slices (120 mg) prepared from the cortex of young adult rats (about 150–200 g body weight) were pre-incubated with 1.6 × 10⁻⁵ M ³⁵S-taurine for 15 min in 20 ml of oxygenated Krebs–Ringer medium together with amino-oxo-acetic acid (10⁻³ M). (The latter compound inhibits the metabolism of GABA and was included to keep conditions identical to those of the GABA experiments, although it has no known effect on taurine metabolism.) After 30 min the slices were collected by rapid filtration and transferred to a perfusion cell through which taurine-free oxygenated Krebs–Ringer medium was pumped. Samples were collected for radioactivity determination before and after stimulation by rectangular pulses of 60 Hz (duration 2 ms; current 1 mA; voltage 40–80 V). Fig. 1 shows that there is a rapid efflux of ³⁵S from the tissue comparable with that found in similar conditions for GABA. To show the release of ³⁵S to be taurine, brain slices and medium, after and before stimulation, were extracted with 10% (v/v) trichloroacetic acid and washed four times with ether. Extracts were separated on ascending paper chromatography in butanol: acetic acid : water (12 : 3 : 5 by volume) and by high voltage electrophoresis in formic-acetic acid buffer, pH 1.86, at 7 kV. In all cases a single peak of radioactivity coincided with that of a standard sample of taurine.

Fig. 2. Uptake of ³⁵S-taurine into rat cortical slices. Brain slices (10 mg wet weight of cortex) were incubated in 10 ml of oxygenated Krebs–phosphate medium at 37°C. After 15 min ³⁵S-taurine was added to the medium to a final concentration of 5 × 10⁻⁷ M. Slices were collected by filtration and rinsed before determining radioactivity at various times of incubation with taurine. Estimations (C) are recorded together with s.e.m. for at least four determinations at each point.

If taurine is released on stimulation, as our experiments suggest, an efficient process for the termination of its action should be available. Since the enzymatic conversion of taurine to isethionic acid in rat brain is too slow to fulfill this role, we have looked for a transport system that could efficiently remove taurine from the extracellular fluid of the brain. In preliminary experiments uptake of very low concentrations of ³⁵S-taurine into slices of rat brain cortex has been found with no detectable metabolism of taurine. Fig. 2 shows the uptake of taurine at 5 × 10⁻⁷ M into brain slices; the curve has been fitted to the mean result at each concentration. Further work will be necessary to establish the detailed kinetics of the process and the exact form of the uptake curve. These experiments indicate that uptake is a possible mechanism for the inactivation of taurine and the provision of calculated Kₘ supports the view. For this uptake process was estimated by measuring the uptake of taurine concentrations of 2.5 × 10⁻⁷ M and 10⁻³ M in the medium into brain slices. The amount of radioactivity in the tissue after 10 min of incubation was used to calculate the rate of uptake and a value of Kₘ = 5 × 10⁻⁵ M was obtained (compared with GABA where Kₘ = 2.2 × 10⁻⁵ M). Such a process has also been considered likely to apply at synapses where GABA is thought to be released.

It would be premature to conclude that taurine is a neurotransmitter or modulator in the nervous system, for much work on specific antagonists and its localization within identifiable neurons is still essential. Our work does indicate an interesting similarity in release and uptake mechanisms between GABA and taurine, and there are sufficient resemblances in localization and biosynthesis of the two amino-acids to warrant further study.

We thank Dr J. F. Mitchell and Mr C. Rayner for their help in the release experiments. L. K. K. holds an MRC studentship.

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DDT in California Sea Lions

We wish to report extraordinary concentrations of DDT residues* in California sea lions, Zalophus californianus, which inhabit year round the coastal waters of California and Baja California, Mexico. These waters receive agricultural runoff from California valleys where DDT has been used extensively1–4, and where residues have been increasing in the primary stages of some coastal pelagic food chains5.

Tissue samples of blubber, and in some cases, brain liver and muscle, were collected from animals of both sexes and various ages between September 19 and December 10, 1970. These were four apparently healthy adults (one male and three females) shot at San Miguel Island, 56 km from Santa Barbara, and one adult male killed near Año Nuevo Island, 31 km north of Santa Cruz, California, six dying males stranded on beaches between Santa Cruz and San Francisco which were killed in the laboratory, and fourteen fresh male carcasses found on or near Año Nuevo Island. Tissue samples weighed approximately 50 g, were frozen immediately after collection, and stored at −68°C until chromatography was performed according to the methods of Stanley and LeFavour6 and Kadoum7.

DDT residues, while present in all samples, predominated in blubber (Table 1). The breakdown of total DDT residues

* DDT residues refer to all the constituents of commercial DDT and its metabolites: p, p'-DDT (1, 1, 1-trichloro-2, 2-bis(p-chlorophenyl) ethane), p, p'-DDD (1, 1-dichloro-2, 2-bis(p-chlorophenyl) ethane), and p, p'-DDE (1, 1-dichloro-2, 2-bis(p-chlorophenyl) ethylene).
was similar for all animals in both blubber and brain (Table 2). An analysis of DDT residues according to sex and age revealed no significant differences.

### Table 1 DDT Residues in Healthy, Sick, and Dead California Sea Lions: Means and Standard Deviations. Number of Specimens in Each Group (Parentheses) and Ranges

<table>
<thead>
<tr>
<th>Group</th>
<th>Total DDT residues</th>
<th>Blubber</th>
<th>Brain</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>p.p.m. wet weight</td>
<td>p.p.m. lipid weight</td>
<td></td>
</tr>
<tr>
<td>Healthy—killed</td>
<td>906±640</td>
<td>8.6±5.8</td>
<td>1,220±860</td>
</tr>
<tr>
<td></td>
<td>(5)</td>
<td>(4)</td>
<td>(5)</td>
</tr>
<tr>
<td></td>
<td>41 to 1,929</td>
<td>0.2 to 16</td>
<td>47 to 2,436</td>
</tr>
<tr>
<td>Sick—killed</td>
<td>689±164</td>
<td>13±2</td>
<td>1,022±336</td>
</tr>
<tr>
<td></td>
<td>(6)</td>
<td>(5)</td>
<td>(6)</td>
</tr>
<tr>
<td></td>
<td>400 to 903</td>
<td>8.9 to 16</td>
<td>552 to 1,387</td>
</tr>
<tr>
<td>Fresh carcasses</td>
<td>1,006±645</td>
<td>14±11</td>
<td>1,687±1,280</td>
</tr>
<tr>
<td></td>
<td>(14)</td>
<td>(6)</td>
<td>(14)</td>
</tr>
<tr>
<td></td>
<td>258 to 2,678</td>
<td>2.6 to 34</td>
<td>333 to 5,077</td>
</tr>
<tr>
<td>Totals: all specimens</td>
<td>911±582</td>
<td>12±8</td>
<td>1,452±1,104</td>
</tr>
<tr>
<td></td>
<td>(25)</td>
<td>(15)</td>
<td>(25)</td>
</tr>
<tr>
<td></td>
<td>41 to 2,678</td>
<td>0.2 to 34</td>
<td>47 to 5,077</td>
</tr>
</tbody>
</table>

To investigate whether some of the high concentrations in carcasses were due to tissue decomposition after death, blubber and brain samples were collected from a sick animal immediately after death, 2 days later, and once again 5 days after death. During these 5 days, the carcass was left outdoors and covered with burlap sacks. Total DDT lipid values in serial order were 1,267, 1,167 and 1,268 p.p.m. for blubber and 146, 147 and 147 p.p.m. for brain. Evidently, concentrations in these tissues did not fluctuate significantly during this time period.

### Table 2 Percentage of DDT and its Metabolites in Blubber and Brain: Means and Standard Deviations

<table>
<thead>
<tr>
<th>p.p.m. lipid weight</th>
<th>Blubber (N=26)</th>
<th>Brain (N=15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>DDE</td>
<td>93.9±1.9</td>
<td>94.1±3.4</td>
</tr>
<tr>
<td>DDD</td>
<td>3.9±1.3</td>
<td>3.3±1.1</td>
</tr>
<tr>
<td>DDT</td>
<td>2.2±1.1</td>
<td>2.6±1.2</td>
</tr>
</tbody>
</table>

DDT residues in the muscle of four healthy adults averaged 1.2±0.8 p.p.m. wet weight and 418±246 p.p.m. lipid weight. Mean concentrations in the liver of four healthy adults and three subsadulbs which had been killed in the laboratory, combined, were 17±10 p.p.m. wet weight and 677±229 p.p.m. lipid weight.

The DDT residues in the blubber and brains of these sea lions are higher than those reported in other marine mammals.8–12 Maximal values equal those in brown pelicans, Pelecanus occidentalis.14–18 We do not know the effects of high DDT concentrations on California sea lions, but studies on other animals suggest several possibilities—reproductive failure19–21, decreased resistance to stressful infectious agents,22,23, and an acute toxic effect on the central nervous system.24

Abortions in California sea lions have increased since 1968 on several rookeries off the coast of California and Baja California, Mexico.21 A systematic census on San Nicolas Island yielded a count of 135 aborted foetuses in 1969 and 442 in 1970, more than a three-fold increase from one year to the next.22 We counted 283 one-week-old pups at Isla San Benito and Isla San Martin, Mexico, between May 25 and May 28, 1971—133, or 47% of them were dead.

Between August 25 and November 30, 1970, Mrs J. Schonewald of the California Academy of Sciences received 420 reports of sick or dead sea lions observed between Monterey and Fort Bragg, California. An epidemic was publicized nationally (for example, San Francisco Chronicle, October 14, 1970, and New York Times, October 15, 1970) with death being attributed to the bacterium, Leptospira, which was isolated in some of the animals.

It is doubtful that the increased mortality resulted from an acute toxic effect of DDT since concentrations in carcasses were not significantly higher than in the living animals. Second, concentrations of DDD and DDT, combined, in the brains of carcasses are far below the lower limit of 30 p.p.m. suggested as the direct cause of death in birds.24

The potential danger of organochlorine pesticides to pinnipeds warrants our concern, for it involves not only these marine mammals but man, whose diet often includes fish and cephalopods.

We thank J. Carr, R. Gantt and R. C. Hubbard for collecting samples; J. Phillips and D. Murphy for chromatography; D. M. Robinson for transportation, and R. Hingardner for helpful suggestions. This study was supported in part by a grant from the National Science Foundation.

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Light Flashes produced in the Human Eye by Extremely Relativistic Muons

It is generally agreed that the light flashes observed by astronauts on Apollo missions 11–14 were produced by the passage of heavy cosmic ray nuclei through the eye. The exact mechanism has been a subject of some controversy, however, most workers leaning towards either Čerenkov radiation or direct ionization and excitation of molecules at the retina. Light flashes similar to the Apollo observations have been reported to result from irradiations of the eyes by X-rays,7 neutrons,5,6,9,10 and slow alpha particles, all of which are more likely to involve ionization and excitation than Čerenkov radiation. Early indications that the human eye might be sensitive to individual relativistically charged particles4 have not been borne out by calculations3 or by recent observations5.

This report describes a test of the ability of the human eye to detect Čerenkov radiation when it is generated with sufficient intensity by the passage of a burst of relativistic charged particles through the vitreous humour and retina of the eye. The small values of LET and total dose involved in producing the bright extended flashes described below seem to rule out any significant contribution from direct ionization and excitation. All observations were made during two short periods of exposure to a beam of 6 ± 2 GeV muons at the Brookhaven AGS. Red X-ray dark adapting goggles were worn for at least 0.5 h before exposure and observations were made in a completely darkened state under improvised cloth and leather hoods with the muons entering the eye through the side of the head. The muons arrived spread over pulses of 440 ms duration with a time interval of 2 s between pulses. The flux density of muons in each pulse was approximately $3 \times 10^5$ muons cm$^{-2}$.

The phenomena appeared as bright diffused flashes which appeared and disappeared with the beam pulses and seemed to fill the periphery of the field of view but never the entire field of view. When I turned away from the beam so that it entered from the rear of the head, the flashes were no longer clearly distinguishable. The coincidences between announced flashes and the beam pulses were checked as well as my ability to tell when my eyes were centred in the active beam.

Localized areas of the retina function as units at threshold levels of signal. The areas of these summation units range from $10^{-6}$ cm$^2$ at the fovea to about $9 \times 10^{-4}$ cm$^2$ on the periphery with an average of roughly $8 \times 10^{-5}$ cm$^2$. Signals generated in a unit within an interval of 0.1 s are effectively in coincidence. The number of such coincidences required to induce a visual sensation is somewhere between two and twenty, ten being typical of most measurements.

There is some possibility that the flashes observed in this experiment are similar in origin to the haze reported in X-ray,7 and neutron5 irradiations of the eye, phenomena that would certainly not involve Čerenkov radiation. But the flux rate that resulted in bright extended flashes corresponds to the passage of three muons per pulse through a $9 \times 10^{-4}$ cm$^2$ summation unit on the periphery or less than 1 in each 0.1 s interval of beam. This rate of dose, 0.026 Mr in 0.1 s, is a factor of ten smaller than the X-ray doses over the same or shorter intervals that were required for even a slight haze5 despite the similarly low LET associated with both radiations. Moreover, the 10 s persistence reported for the neutron produced haze5 was not observed in this experiment.

On the other hand, the number of "visible" Čerenkov photons in each 0.1 s of pulse that can be expected to accompany the muons through the retinal summation units has been calculated12 as 40 on the periphery, 0.1 at the fovea and about 4 for a unit of average area. If we assume the probability of absorbing each photon incident on the periphery to be 20%, the value measured at $10^{-20}$ eccentricity14,15, then a threshold value of ten absorbed photons should be exceeded in the muon experiment for 26% of the peripheral units in each 0.1 s interval of the 0.44 s pulse and essentially nowhere else on the retina. Although calculations of this type are necessarily crude as a result of our incomplete knowledge of the peripheral region of the retina, they do indicate Čerenkov radiation as the most plausible mechanism for producing the flashes described above.

I thank members of the Columbia, Harvard, National Accelerator Laboratory, and Rochester University collaboration who designed and operated the muon facility at the Brookhaven AGS for help, Professor L. Lederman for helpful suggestions and Dr Paul Bottino for help with the observations.

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