during purification (*10*). Indeed, the low RNA/DNA ratio (1:3) reported by Smith and Stoker (*11*) for *Coxiella burnetii* may reflect such losses and suggests the physicochemical lability of rickettsial RNA.

The differences between the results obtained at 36°C and at 4°C suggest the participation of enzymatic processes in the loss of nucleic acids from the rickettsiae. Since the omission of glutamic acid did not influence the experimental results, "energy metabolism" does not seem to be of importance. The present findings are perhaps analogous to the degradation of RNA in resting cultures of *Escherichia coli* H, which has been reported by Stephenson and Moyle (*12*).

The loss of nucleic acids from *R. mooseri* upon incubation at 36°C may be one of the reasons for the concomitant inactivation of the biological properties of this organism.

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4 October 1957

Barium-140 Radioactivity in Foods

We wish to report the presence of the fission products barium-140 and lanthanum-140 in certain foodstuffs in the United States during periods of nuclear weapons tests. The presence of these nuclides in the amounts observed in no way constitutes a hazard; it carries none of the potential implications associated, for example, with strontium-90. It is of practical concern primarily to those engaged in measurements of radioactivity near the natural levels, and when an accurate summation of total radiation is desired. As in the case of cesium-137 (*1*), measurements of barium-140/lanthanum-140 may also be of value in the study of the fallout process.

Barium-140 has a half-life of 12.8 days, and its daughter lanthanum-140 has a half-life of 40.2 hours. Because of their somewhat similar biochemistry and the short half-life of lanthanum, secular equilibrium is likely to be maintained in biological systems, and both nuclides will follow the chemistry of barium. The biochemical behavior of barium-140 is similar to that of calcium and of strontium-90, but the short half-life of barium-140 and the larger biological discrimination factors against it render it potentially much less dangerous than strontium-90.

The presence of barium-140 was first noted in some deer in New Mexico during the summer of 1956—presumably the result of the United States nuclear test Operation Redwing. It was detected and identified (by its half-life) by means of the Los Alamos human counter (*2*), a large 4π liquid scintillation counter designed especially for the measurement of radioactivity at natural levels in people and foodstuffs. A threefold increase was noted in the gamma activity in the spectral region from 1 to 2 Mev (normally potassium-40 only). This corresponds roughly to a barium-140 activity of 0.03 μc per 70 kg, which is 6 percent of the maximum permissible amount for man, on the basis of the "large population" value (*3*). A cow taken directly from the New Mexico range showed a similar amount of barium-140, but commercial beef did not, perhaps because of different feeding habits or because of the time lag between slaughtering and the appearance of the meat on the retail market. While the apparent potassium-40 activity of milk samples showed a few instances of slight increases during 1956, it was not possible to identify the excess activity.

Barium-140 appeared in several United States milk samples during the months of June, July, and August, 1957, presumably as a result of distant fallout from the test operation in Nevada, Operation Plumb-bob, and perhaps from test operations of the U.S.S.R. Identification of the excess activity in the potassium region of the spectrum was again possible on the basis of the measured half-life. Confirmation of the assignment of the activity to barium-140 was obtained by spectral analysis, for which an 8- by 4-inch sodium iodide crystal in a steel room similar to the installation developed by Marinelli and his coworkers at the Argonne National Laboratory (*4*) was used. All five of the prominent barium-140/lanthanum-140 gamma peaks were identified.

Table 1 summarizes dates and concentrations of barium-140 in powdered milk for those locations at which the barium-140 gamma activity exceeded that of natural potassium-40. While detectable increases in activity in the upper energy

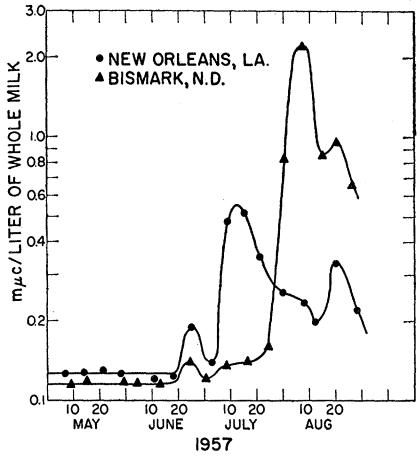

Fig. 1. Typical curves of upper channel activity versus date, for Bismarck, N.D., and New Orleans, La.

channel occurred at other sampling points during the summer months, at none of them did the increase reach this level. Thirty-seven points are routinely sampled.

The dates reported in Table 1 are those on which the samples (50 to 100 pounds of nonfat-dry milk solids) were received at Los Alamos. In general, the delay between production and arrival is about 1 week, and for accurate correlation with meteorological data, the actual production date must be ascertained.

Table 1. Barium-140 peak levels in milk.

Date received (1957)	Estimated barium-140 content (mμc/lit of whole milk)
Bismarck, N.D.	
7 Aug.	2.2
20 Aug.	0.85
Idaho Falls, Idaho	
10 June	0.40
5 Aug.	0.32
26 Aug.	0.54
Payette, Idaho	
19 June	0.12
9 Aug.	0.46
Louisville, Ky.	
21 July	0.27
9 Aug.	0.46
New Orleans, La.	
15 July	0.36
20 Aug.	0.19
Willows, Calif.	
10 June	0.31
Ladysmith, Wis.	
16 Aug.	0.23
Des Moines, Iowa	
9 Aug.	0.18
Ogden, Utah	
19 June	0.06
14 Aug.	0.15
Monroe, Utah	
11 June	0.09
25 July	0.12

Typical curves of upper channel activity versus date are shown in Fig. 1 for Bismarck, N.D., and for New Orleans, La. The constancy of the potassium-40 assay is indicated by the reproducibility of the results for the first 6 weeks.

The peak concentrations given in Table 1 can be compared with the International Commission on Radiological Protection's maximum permissible concentration for barium-140/lanthanum-140 in drinking water of 300 mμc/lit (*3*). The latter value is for continuous exposure for an indefinite period of time, while the exposure resulting from weapons testing is of short duration. Unlike strontium-90, barium-140 cannot present a cumulative hazard because of its very short half-life. Barium-140 has not been observed in any human subjects, although a search has been made for it.

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28 October 1957

Action of Blood-Borne Gamma-Aminobutyric Acid on Central Synapses

When substances are identified in the brain, it is natural at the same time to inquire into their function. Thus they become candidates for various roles, including that of potential neurohumoral transmitters. Such, indeed, has been the case with serotonin (*1*), and such is now the case with gamma-aminobutyric acid (GABA). Bazemore, Elliott, and Florey (*2*) have identified the latter as an active principle of factor I, which Florey and McLennan (*3*) had extracted from mammalian brain and had shown to have inhibitory actions.

One of the readiest methods of acquiring preliminary information of this sort is to paint a solution of the material upon the exposed cerebral cortex. The high doses thus applied and the unusually high concentration gradients that result serve to uncover any possible actions. Effects achieved in this highly abnormal way are undeniable but difficult to interpret in terms of physiological function, even when specificity can be asssured. Although the usefulness of this method, as in the topical application of strychnine (*4*) to fire brain areas, in order to map them, has gained it considerable respectability, this should not be extended to other uses. Thus, Kato (*5*), in studying conduction in nerve, found it convenient to make use of mechanical stimulation by a miniature mallet, but there was no suggestion that this was a normal way to activate or that this mechanical stimulus played a part in propagation of the nerve impulse. Nevertheless, the actions of GABA have been studied almost exclusively by topical application.

We have, therefore, wished to study the effects of blood-borne GABA and have resorted to the method we have previously used to help establish the roles of acetylcholine, adrenaline and nor-adrenaline (*6*), and serotonin (*1*) as neurohumoral transmitters in mammalian brain. This has been the relatively close arterial injection in the common carotid artery serving effectively to bring across the blood-brain barrier relatively small doses which, therefore, on dilution in the systemic blood stream become subthreshold for peripheral actions and consequently exhibit the cerebral actions in isolation or in relatively pure form—that is, not complicated by the peripheral actions and the resulting barrage of afferent impulses which bombard the brain. In this manner, by activating cortical synapses through the transcallosal pathway and recording the response as evoked cortical potentials in the lightly anesthetized cat, we have demonstrated that GABA, when delivered through the natural route (that is, when it is blood-borne), can, like adrenaline, nor-adrenaline, and serotonin, inhibit synaptic transmission. It does this in doses of 50 to 500 μg/kg (Fig. 1); thus it has a potency of about 1/50 that of serotonin, intermediate between that of nor-adrenaline and adrenaline, the series being nor-adrenaline, 1; GABA, 7×; adrenaline, 15×; serotonin, 300×. Unlike the effects of topical application reported by Purpura and Grundfest (*7*) the surface negative evoked response is usually reduced without affecting the positive wave or inverting the negative wave into a positive one.

Comparison with serotonin brings out further significant differences. The time course of the GABA action is faster in all respects. As the continuous plot of the surface negative evoked responses shows, the time of onset and the duration of action are remarkably short. The latter suggests an enzymatic destruction of GABA as was supposed by Florey and McLennan (*3*) or a binding into an inactive state by adsorption as believed by Elliott (*8*). Successful interference with this enzymatic or this binding process would result in abnormal accumulation of GABA, which would be evi-

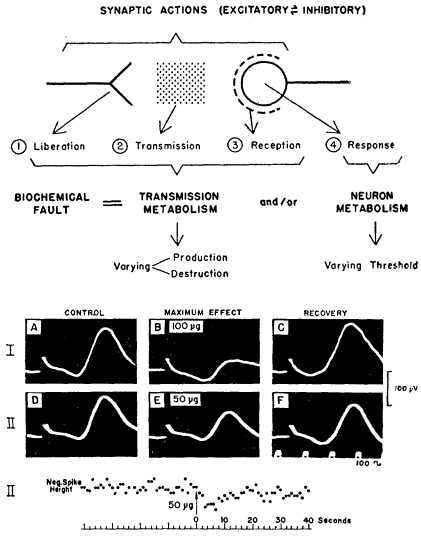

Fig. 1. Cerebral synaptic action of gamma-aminobutyric acid in a two-neurone intercortical (transcallosal) system. (Top) Potential factors in disturbed synaptic equilibrium. (Bottom) Potentials evoked in the cerebral cortex of the cat by electrical stimulation of the contralateral cortex every second. Gamma-aminobutyric acid was injected into the ipsilateral common carotid artery.

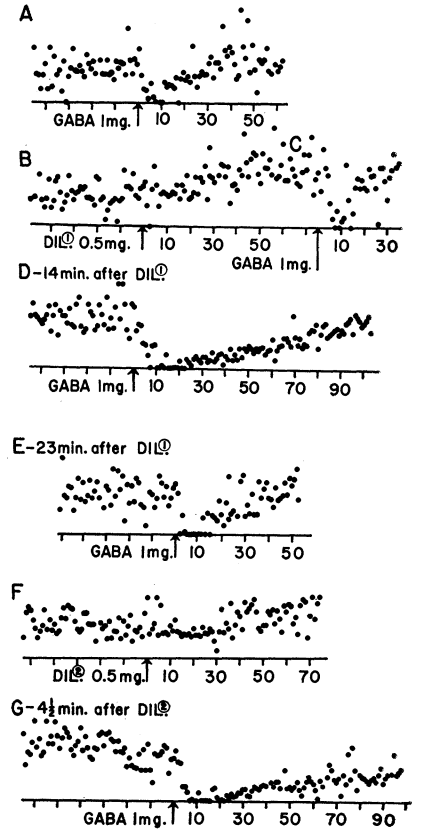

Fig. 2. Augmentation of GABA cerebral synaptic inhibition by dilantin. Negative cortical spike heights from transcallosal system potentials evoked by contralateral cortical stimulation (one per second). Injections were made into the ipsilateral common carotid artery.