Health Service of Ecuador has described (*9*) the isolation of Venezuelan equine encephalomyelitis from *Culicoides*. The outbreak in Ecuador involved both men and horses.

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Effect of Gravity on Flowering of Soybeans

The antagonistic effects of synthetic auxin on the flowering of short-day plants have been demonstrated by numerous workers. High levels of natural auxin within plants have also been shown to delay flowering. Fisher and Loomis (*1*) and Fisher (*2*) suggested that high concentrations of naturally produced auxin at the lower nodes of soybean are antagonistic to floral induction. They found that, with plants on long photoperiods, flowering could be induced earlier by removing young, auxin-producing leaves after 3 to 5 trifoliate leaves had fully expanded. The complete loss of auxin-producing tissue through detopping, however, allowed active vegetative growth of the axillary buds. Such growth gave rise to high auxin levels at the lower nodes, thereby markedly delaying flowering.

Van Overbeek and Cruzada (*3*) showed that pineapple plants that were tipped on their sides flowered earlier than those that were grown upright. Pineapples do not behave like most short-day plants, since in them auxin has been shown to induce, rather than to inhibit, flowering (*4*). Apparently, then, the high auxin content in the apex of the horizontally grown plants induced earlier flowering, the auxin being concentrated in the apical regions by gravitational force. It was therefore thought that in soybeans, plants that begin to flower at the lower nodes, a similar method of growth might also cause an accumulation of auxin at the tip and subsequent lower auxin levels at the basal nodes, which would allow earlier floral induction.

Flambeau soybeans, on 18-hour photoperiods, were treated as follows: (i) in a control series, plants were allowed to grow normally; (ii) to make the plants grow downward, lead weights were placed around the stems near the tips of plants that had two mature trifoliate leaves; and (iii) lead weights were placed around the stems, near the tips, as in treatment ii, but vegetative suckers were removed as soon as they were 1 cm long. As the tips grew, the lead weights used in treatments ii and iii were moved toward the apex on the inverted stems. By the time seven or eight trifoliate leaves had fully expanded, 25 g of lead was required to keep the tips of the plants from turning upward. As the plants became older, the tips of the stems showed symptoms typical of the injury induced by an excess of externally applied auxin. Cellular enlargement and proliferation in the cortex were marked. The leaves continued to position themselves normally, resulting in a twisting of the petiole close to the stem. Enlargement of the petiole was pronounced in leaves that appeared after the sixth leaf was mature. Suckers usually grew from nodes 2 and 3.

Inverted plants flowered earlier and at lower nodes (Table 1) than the controls. Removing the suckers from the inverted plants stimulated the earliest flowering.

Table 1. Flowering of Flambeau soybeans on 18-hour photoperiods.

Treatment	Percentage flowering (days)					Lowest flowering node
	45	50	55	60	65	
Control	0	0	0	58	100	6.9
Inverted	0	44	55	78	100	4.7
Inverted, suckers removed	22	67	78	100	100	4.0

These data show that flowering in soybeans can be geotropically influenced, and they provide further support for a theory of auxin control in flowering. Apparently the accumulation of auxin in the tip region, through gravitational force, caused a reduced level of auxin at the basal nodes, and thereby induced earlier flowering (*5*).

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Triplet States of Biologically Active Molecules

In a recent communication concerning the probable importance of excited-state mechanisms in biological systems, and in particular the role of triplet-state energy transfer in oriented or partly oriented aqueous media, A. Szent-Györgyi (*1*) suggests that the mode of action of many drugs may involve interference with energy-transfer processes. As evidence for this possibility, Szent-Györgyi cites the fact that 2-4-dinitrophenol is physiologically active at similar concentrations to those at which it will quench the phosphorescence of riboflavin; he also cites other very suggestive but not conclusive *in vitro* experiments on the fluorescence of aqueous dyestuffs.

In this connection, I wish to report some observations made in 1953 but not yet published, on phosphorescence from narcotized tissue. We had been investigating (*2*) sensitive methods of detecting carcinogens by low-temperature fluorescence spectroscopy and the *in vivo* conditions of formation of carcinogen-protein complexes. We decided to look briefly at the low-temperature emission spectra obtainable from spontaneous tumor tissue. Aqueous tissue homogenates and ether extracts crystallized in an excess of naphthalene (which provided ordered host material) were prepared, and the emission spectra were observed at 90°K under irradiation from a mercury arc. The samples used were human tumor tissue from 15 to 20 patients. All

showed strong, long-lived ($> 10^{-4}$ sec) emission spectra, usually in the green-yellow region of the spectrum, but this phosphorescence was tracked down in every case to the anesthetics that had been used before operation. Despite the subsequent use of samples from patients who had been subjected to a restricted anesthetic program, the anesthetic emission was always so strong that the search for any fluorescence characteristic of tumor tissue was abandoned.

The emission was observed with substances as widely different as barbiturates, morphine, hyoscine, sodium pentothal, syncurine, and *d*-tubocurarine chloride, and it was stronger than that which could be obtained from the drugs alone in concentrations as high as the maximum possibly present within the tissue.

This observation lends strong support to the view that the molecules of anesthetic act as traps for energy absorbed elsewhere in the tissue. I have already put forward the idea, in connection with a discussion of certain aspects of radiation damage (*3*), that most of the biologically important prosthetic groups act as such traps. The molecules of an anesthetic may thus provide traps competitive with those normally operative, and this leads to the prediction that radiation damage may be less severe on irradiation of anesthetized tissue than it is on unanesthetized tissue.

It is noteworthy that, while the triplet-level explanation is probably not directly applicable to small molecules with anesthetic properties (cyclopropane, ether), these molecules, with no exceptions so far as I am aware, are in turn oxidized to products that show phosphorescence with an exceptionally high quantum yield. We are now embarking on a more quantitative study of these phenomena.

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Blocking Effect of Brain Extract on Lysergic Acid Diethylamide Reaction in Siamese Fighting Fish

The method used to study the blocking effect of beef brain extract is based on the criteria described by Abramson and Evans (*1*) for the reaction of the Siamese fighting fish to lysergic acid diethylamide (LSD-25). However, these criteria were modified for the purposes of this investigation (*2*) and are listed in Table 1. The main criterion utilized was the nose up-tail down position of the fish, at an angle of approximately 45 deg or more. Beef brain extract was prepared in a fashion similar to that described by Florey and McLennan (*3*). In general, the experiments reported here were all run similarly.

Table 1. Inhibition of LSD-25 effect by brain extract. The readings recorded in rows 1a, 2a, and 3a were made 1 hour after brain extract was added to the container, but before LSD-25 was added. The readings in rows 1b, 2b, 3b, and 5b were made 1 hour after LSD-25 was added.

Treatment	Response (No. of fish)			
	Nose up, tail down	Kink in tail	Top of water, tail up	Bottom of container
	Experimental containers			
1a Brain extract (2 mg/ml)	0	0	4	0
1b LSD-25 (2 μg/ml)	4	4	1	2
2a Brain extract (0.2 mg/ml)	0	0	9	0
2b LSD-25 (2 μg/ml)	8*	9	0	2
3a Brain extract (0.02 mg/ml)	0	0	2	4
3b LSD-25 (2 μg/ml)	9	10	0	1
	Water control			
4a No brain extract	0	0	3	2
4b No LSD-25	0	0	2	4
	LSD-25 control			
5a No brain extract	0	0	3	3
5b LSD-25 (2 μg/ml)	10	10	0	0

* One fish dead.

Five bottles containing 200 ml of distilled water each were used to observe 50 fish simultaneously, with ten fish in each bottle. Readings were made with two different methods every 15 minutes for a period of 4 hours or more. The effects of brain extract (2 mg/ml, 0.2 mg/ml, and 0.02 mg/ml) on the reaction of the fish to 2 μg of LSD-25 per milliliter of solution were compared with a water control (no brain extract) and an LSD-25 control (no brain extract). Table 1 presents, in general, the method of bioassay currently employed.

Until LSD-25 was added, none of the fish showed the typical nose up-tail down position except as a normal movement. Fifteen minutes after the LSD-25 was added to the five vessels, all the fish in the LSD-25 control showed the nose up-tail down position and continued to do so throughout the experiment. Essentially similar results were obtained in the bottle containing 0.02 mg of crude brain extract per milliliter. In the bottle containing 2 mg of brain extract per milliliter, by contrast, not only was the initial excitatory phase absent, but there was also a lag, with one fish showing the nose up-tail down behavior at 45 minutes and only four fish showing this behavior at 1 hour. Table 1 gives illustrative data obtained at the end of the first hour.

Using unpublished reaction-time curves, it can be readily shown that the fish in the bottle containing 2 mg of brain extract per milliliter acted as if approximately 0.2 μg of LSD-25 per milliliter were present. In other words, the brain extract blocked the appearance of the LSD-25 effect, and when this effect finally did make its appearance it resembled that of a much weaker solution of LSD-25 than was actually added.

The question naturally arose: Is the serotonin present in brain extract responsible for the action of the brain extract? Two hours after the fish were exposed to 2 mg of serotonin per milliliter, 2 μg of LSD-25 was added. Serotonin does not block the LSD-25 reaction.

Negative results were also found with histamine and γ-aminobutyric acid, as well as with the following amino acids: *l*-hydroxyproline; *l*-serine; 3,5-diiodo-*l*-tyrosine; *dl*-α-aminobutyric acid; *dl*-cysteine hydrochloride; *l*-lysine hydrochloride; *dl*-methionine; *l*-tyrosine; *dl*-valine; *dl*-tryptophan; *l*-leucine; *dl*-phenylalanine; acetyl glycine; *l*-arginine hydrochloride; *dl*-threonine; *l*-histidine hydrochloride; *l*-glutamic acid; *l*-valine; acetyl-*dl*-phenylaline; glycine; and *l*-proline. There was possibly slight blocking in *l*-histidine hydrochloride, for all the fish did not react to LSD-25 immediately.

Lack of material (the result of the difficulty of preparing the crude brain extract in quantity) has prevented our determining where the blocking substance acts or what it is. Conceivably, it could act in several places: (i) outside the fish, forming a loose compound with LSD-25; (ii) at the gill membrane; or (iii) inside the fish itself, as a true pharmacologic inhibitor. The data indicate that some type of equilibrium is set up in which the inhibitory action is dependent on the concentration of the LSD-25 blocking substance present in the liquid.