the benches on both sides seems to be that they are blocks that have been uplifted 20 m relative to the valley floor. As one approaches the rift walls, the scarps become higher and the blocks become more tilted, as one would expect from our spreading, uplifting hypothesis.

So far we have discussed only the features of the central valley of Gorda Rise. The features found on the outer flanks of the crestal ranges are somewhat more complex. Blocklike topography also occurs here but it is partially obscured by sediments and disrupted by what appear to be volcanic structures. Wherever blocks can be identified, they are tilted, the steep scarps facing inward toward the valley, and the gentle backslopes facing outward. A tilted-block form was found in abyssal hills 90 km west of the central valley.

Gorda Rise has been established as part of the oceanic-rise system. Menard (*5*) and Wilson (*6*) both consider it to be a northward continuation of the East Pacific Rise, and McManus (*4*), summarizing much of the data available for this area, comes to a similar conclusion although he also provides an alternative. Shor *et al.* (*7*) show that in crustal structure Gorda Rise resembles other parts of the oceanic-rise system, and Vine (*2*) shows that the magnetic-anomaly pattern is symmetric across it and is similar to the pattern mapped in other parts of the system. Present activity of Gorda Rise is indicated by high heat flow (*8*) and by earthquake activity (*9*).

Menard (*10*) has noted that portions of the oceanic-rise system fall into two distinct groups, depending upon their spreading rates. The portions that spread rapidly, typified by the southern East Pacific Rise, are characterized by smooth, hilly topography and a thin second layer. Portions that spread more slowly have steep mountainous topography, with one or more central rift valleys and a thicker second layer. Gorda Rise shows all the characteristics of the slowly spreading parts of the oceanic-rise system (*4*, *7*).

If Gorda Rise is typical, the detailed block structure (Fig. 2) should be seen on the rift-valley walls of other slowly spreading portions of the system. However, the usual survey, using a wide-beam echo sounder aboard a surface ship, would show the larger blocks as rounded hills, and the smaller ones as faint side echoes in an almost smooth slope. In their report on the Mid-Atlantic Ridge near 23°N, Van Andel and Bowin (*11*) describe a number of elongated parallel ridges and steps in the valley and on the slopes and peaks; nearly all their profiles show several steps in the inner walls of the rift. All these features might be revealed as tilted-block structures by techniques providing higher resolution. Indeed, Van Andel and Bowin suggest on petrographic grounds that the gross structure of the ridge originates in the uplifting and tilting of blocks; their Fig. 7 shows the ridge top and a lower step interpreted as uplifted blocks.

The Mid-Atlantic Ridge was studied in detail near 45°N by Loncarevic, Mason, and Matthews (*12*); their bathymetric chart and their various profiles clearly show a rounded bench, often 5 km wide, about halfway up the rift walls. This may represent a large, uplifted, tilted block, although Loncarevic (personal communication) states that no specific evidence was found to indicate block faulting.

For the northern East Pacific Rise, Menard and Mammerickx (*13*) have recontoured the ship soundings of several detailed surveys; they believe that the origin of the topography on this part of the rise system can best be described as a combination of volcanism and block faulting.

In conclusion, the detailed form of the topography indicates that the walls of Gorda Rise central rift valley are composed of faulted blocks. We believe that the best explanation of the height and shape of these blocks, the character of their tops, and the form of their sediments is that they originated in the valley floor and have been uplifted and displaced from the valley center to form the walls. Furthermore, other reports of detailed studies show the possibility that this mechanism is general for formation of the topography of the central valleys of slowly spreading portions of the oceanic-rise system.

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Lysergic Acid Diethylamide (LSD): No Teratogenicity in Rats

Abstract. *Lysergic acid diethylamide (LSD) in doses of 1.5 to 300 micrograms was given to 55 pregnant rats during periods of organogenesis and on the 4th or 5th day of pregnancy to 34 rats. Examination of the resultant 887 young for congenital defects showed no greater frequency than in controls. These experiments failed to prove that LSD is teratogenic in rats.*

The report of Cohen *et al.* (*1*) of chromosomal damage in human leucocytes, induced by lysergic acid diethylamide (LSD), prompted us to test this compound for teratogenicity in rats. A pilot experiment was planned to ascertain possible effects of the drug administered to pregnant Wistar rats during periods of embryonic organogenesis. Delysid (Sandoz; batch 65002), containing LSD at 0.1 mg/ml, obtained from the National Institute of Mental Health, was administered intraperitoneally or orally in single doses on the 7th, 8th, or 9th day of gestation, or in multiple doses from the 7th to the 12th day. The total dosage to individual rats ranged from 1.5 to 300 μg.

Fifty-five pregnant rats were treated; four litters were completely resorbed; 47 rats were killed on the 21st day of pregnancy and their young were removed; four were allowed to deliver and raise their young. The mean litter size of the 21-day fetuses was 10.2 $\pm$ 1.8 (controls, 10.0 $\pm$ 2.8); their mean weight was 3.53 $\pm$ 0.44 g (controls,

4.14 ± 0.71 g). The differences between experimental and control animals are not significant.

Of the 508 21-day fetuses, 504 (409 dissected and 95 cleared for skeletal examination) were normal and only four were considered abnormal. One had hydrocephalus, but 12 of its littermates were normal; its mother had been injected once on the 7th day with 50 μg of LSD. Another fetus had short extremities and syndactylism; there were nine normal littermates; the mother had been injected daily from the 7th to the 11th day with 50 μg of LSD. Two fetuses were small, weighing 1.7 and 2.1 g; they were from a litter of six, the other four being normal; the mother had been injected on the 7th and 9th days with 50 μg of LSD. There was no common pattern in the abnormalities of the four young and there was no dose-dependence.

The four treated rats that delivered had 44 apparently normal young, some of which could be raised and bred. Since an incidence of about 1 percent of congenital malformations in the offspring of untreated Wistar rats is not unusual, our pilot experiments did not prove that LSD is teratogenic in rats. Even dosage as great as 300μg to rats did not harm their embryos. Such dosage is comparable to human dosage; on a weight basis it is more than 200-times larger.

Later we learned of the results of Alexander *et al.* (*2*) who gave single injections of LSD to rats early in pregnancy (4th day of gestation) and observed resorption of one litter and some stillborn, stunted young in others. On the 4th or 5th day of pregnancy we gave 34 rats doses ranging from 1 to 100 μg of LSD (Delysid), and obtained from 32 females a total of 335 young. Of these, 296 were removed on the 21st day by cesarean section; with the exception of one fetus that was small, all were normal on external inspection, dissection, or clearing. Thirty-nine young were delivered and raised; they remain alive and healthy. Two of the 34 rats had no litters. A resorption rate of 5.9 percent is not different from that for pregnant Wistar rats injected with saline solution.

Although most of the doses administered in these experiments were very large (the highest single doses administered by us were about 80-times larger than those given by Alexander *et al.*), we found no abnormalities other than reduction in size of one of the young. Alexander *et al.* (*2*) used a dry LSD dissolved in saline, while we administered Delysid. The stocks of rats used in their and our experiments also must differ since they mention that their control offspring weighed an average of 64 g at 10 days; rats in our laboratory weigh about 19 g at 10 days. Donaldson (*3*) cites weights of 15 g for 10-day-old rats. If the LSD-treated rats weighed 44 to 46 g at 10 days, as Alexander *et al.* (*2*) state, it would appear that the drug did not reduce the weight of these animals but more than doubled it.

Thus we did not find LSD teratogenic during the organogenetic period and found no abnormalities in the offspring of rats injected on the 4th or 5th day of pregnancy, although the doses administered to some of the pregnant rats were as high as those used by human beings.

We draw no conclusions from the negative results with rats, concerning teratogenicity of LSD in man, since it is known that a drug teratogenic in one species may not be so in another; this general rule applies to results with mice (*4*) and hamsters (*5*) also.

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Carbon-14 Milk Constituents from Cows Fed Carbamate Labeled with Carbon-14 on the Carbonyl

Abstract. *Oral administration to a dairy cow of Furadan insecticide (2,2-dimethyl-2,3-dihydro-7-benzofuranyl* N*-methylcarbamate) labeled with carbon-14 on the carbonyl produced in the milk certain radioactive materials which were not Furadan metabolites. The data suggest that these products were natural milk constituents containing only the carbon-14 atom from the Furadan molecule. Carbon-14–labeled carbon dioxide formed by the hydrolysis of the carbamate insecticide is the apparent precursor of these radiolabeled constituents of the milk.*

Carbamic acid esters act as parasympathomimetic agents as a result of their ability to inhibit acetylcholinesterase. In summarizing their many clinical uses, Casida (*1*) also pointed out that their pesticidal applications were equally varied.

Metabolism of carbamate insecticides in mammals, plants, and insects has been explored by radiotracer techniques. The use of carbamates labeled on the carbonyl carbon with carbon-14 has become a standard procedure in investigations of the general metabolic fate of these chemicals (*2*). We can easily estimate the degree of hydrolysis by collecting and quantitating the labeled carbon dioxide released from the treated organism. Radioactive residues remaining in the organism, or in its excretory products in the case of animals, are considered as carbamate metabolites.

The possibility that rapid hydrolysis of a carbonyl-C^{14} carbamate in an animal might lead to the incorporation of labeled carbon dioxide into naturally occurring products has been considered. An investigation (*3*) in which eight carbonyl-C^{14} carbamates were administered to rats revealed that 25 to 77 percent of the dose was expired as labeled carbon dioxide.

In order to support the assumption that the expired radioactivity originated from hydrolysis of the carbamate or its metabolites, Krishna and Casida administered sodium carbonate-C^{14} to the rats; they reported that the radioactivity was rapidly and almost quantitatively eliminated as C^{14}-carbon dioxide. Four hours after the animals were treated, 96 percent of the dose was expired as C^{14}-carbon dioxide, 2 percent was excreted in the urine, and 3 percent remained in the body.

The nature of the radioactive material in the urine and body was not determined. The findings suggested that the use of carbamates labeled with carbonyl-C^{14} would not complicate a study of metabolism by allowing in-