that beds younger than Lower Miocene were laid down along the north and south coasts. McGuinness (*6*) mentions a possible NNW–SSE axis of depression along the extreme western edge of Puerto Rico, and perhaps this sagging preserved later Miocene deposits along the west coast, whereas the higher arching to the east resulted in the erosion of contemporaneous deposits. Therefore it is conceivable that deposition took place along the north, south, and west coasts of the island. No known Tertiary rocks occur along the east coast, but Tertiary is presumed to underlie the shallow Vieques Sound, and Lower Miocene strata appear at the surface in southern and eastern Vieques Island, which, according to Meyerhoff (*5*), show a gradational fauna—i.e., gradational between the north and south coast faunas of Puerto Rico. The eastward tilting of both the St. John and the Caguana peneplains has carried the Tertiary down to lower levels in eastern Puerto Rico and in the islands farther east.

In Jamaica the White Limestone of Upper Eocene–Middle Miocene age forms a framework around the Cretaceous rocks of the Blue Mountains, extending as high as some 4000′, with Blue Mountain Peak rising to 7300′. It is the opinion of Trechmann (*9*) that the White Limestone once formed a covering over the Cretaceous of the highest areas, although today the north and south coast outcrops of this limestone are 24 miles apart. He believes that in post-Middle Miocene time, the Blue Mountains were raised at least 3000′. In Puerto Rico, remnants of the older, or St. John, peneplain occur at an elevation of 3000′ in the Adjuntas–Lares region and descend to 1500′ in eastern Puerto Rico and to 1000′ in the western part of the island. This peneplain was elevated in earth movements associated with world-wide diastrophism toward the end of the Miocene. It is not unreasonable, therefore, to postulate that the Tertiary formations of the north, south, and west coasts of Puerto Rico, which seem comparable to the Upper White Limestone–Bowden Marl of Jamaica in regard to faunal content and age, also covered the island of Puerto Rico.

Both these explanations are based on the supposition that Tertiary *in situ* occurs at elevations as high as 2160′. The possibility of Tertiary *in situ* beneath the surface in the Río Guaba region cannot be excluded, even although none has been found, and we are dealing here with boulders, not outcrops. As remarked previously, the subangular boulders appear to belong to the upper part of the San Sebastián formation, and there are indications in the vicinity that the Cretaceous boulders, cobbles, and pebbles may have formed the basal material of the same formation. The presence of these "exotic" upper boulders now lying in a milieu of basal material can be accounted for by gravitational movement down the steep Cretaceous surfaces, in the manner of scree formation.

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Quantitative Studies on Proteolipide as Incitant of Disseminated Encephalomyelitis in Mice

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The identification of the agent present in brain tissue that produces experimentally disseminated (allergic or isoallergic) encephalomyelitis that bears certain resemblance to the human demyelinating encephalitides has been advanced recently by the finding that proteolipide (*1*), a new type of lipoprotein, can reproduce the affection in mice (*2*).

In view of the fact that there has been no uniformity of opinion hitherto on the chemical nature of the incitant of the experimental disease (*3*), it was thought desirable to report additional evidence for consideration of brain proteolipide as the etiological factor of the experimental encephalomyelitis in mice. The evidence is based on a quantitative study of its encephalitogenic potency and derives, first, from the correlation of its concentration with the degree of reaction in mice and, second, from the inability of brain tissue of newborn mice to produce encephalomyelitis, for their tissues do not contain proteolipide (*4*). With respect to the first, the data collected concern (*a*) the number of reactors after injection and (*b*) degree of the reaction; (*c*) the number of days from the first injection to onset of signs; (*d*) the number of injections needed to induce a reaction and (*e*) the minimal time required to bring about a maximum reaction, presented in Table 1 as number of days times number of injections. In Table 1 will be found a summary of one example of a repeated experiment.

The preparation of mouse brain proteolipide followed the method of Folch and Lees (*1, 2*), and the proteolipide was obtained from 150 fresh brains (which weighed 60 g) of 2–4 months old W-Swiss mice. An additional test was made with proteolipide

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TABLE 1

THE QUANTITATIVE STUDY OF THE ENCEPHALITOGENIC ACTIVITY OF PROTEOLIPIDE AND NEWBORN MOUSE BRAIN

Material	No. with signs			No. symptom-free but with CNS lesions	Reactors		B/A	Cumulative time and no. injections to onset			C×D/A
	A	B						C		D	
	No. of mice	Non-paralytic	Paralytic		Total no.	Percentage		No. mice	Days	No. injections	
Proteolipide from 150 brains*	20	0	20	0	20	100	1	6 8 9 10 11 13 20	6 9 14 15 16 17 20	1 2 2 3 3 3 3	3.0
Proteolipide from 25 brains*	21	6†	11	4	21	100	½	2 4 6 11 12 14 16 17	6 9 14 17 18 22 27 28	1 2 2 3 3 4 4 5	6.7
Whole, normal adult mouse brain (control)	20	13†	5	2	20	100	¼	4 5 6 7 9 11 12 14 18	5 8 16 19 23 26 28 33 37	1 2 3 3 4 4 5 5 6	11.1
Whole, normal newborn mouse brain	20	0	0	0	0	0	—	0	60	6	—

* These separate residues, after proteolipide extraction, of the 150 and of the 25 brains were inactive in 35 mice. The RD_{50}, dry weight, for whole brain = 0.40 mg/ml ($=10^{-2.75}$); for proteolipide, 0.013 mg/ml ($10^{-2.92}$).

† These showed neurological signs, such as paresis, tremors, weakness, excitability (*5*) but no extensive paralysis, as in Column B.

diluted 1:6, to equalize the weight of tissue used as control. The latter comprised 25 whole brains, or 10 g of tissue, secured from normal adult mice; 65 whole brains of 4-day old Swiss mice, yielding 10 g of tissue, were also included (Table 1). Each of these materials was mixed with the Freund-type adjuvant and homogenized in a Waring blendor for 2 min, as already described (*2, 3*). The methods, the use of H-line W-Swiss mice (*6*) as animals of choice for inoculation, and other details of procedure have been stated before (*3, 5*).

The tabulated results indicate clearly that proteolipide deriving from mouse brain induced encephalomyelitis in mice to a greater degree than did whole adult mouse brain. There is a correlation between the concentration of proteolipide and the degree of reaction noted in inoculated animals. Finally, newborn mouse brain from which no proteolipide is obtainable, treated in the same manner as adult brain, failed to bring about the disorder. It has already been shown that all the encephalitogenic power of mouse brain resides in the proteolipide fraction (*2*). The present results would therefore appear to confirm this finding.

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Fluorescence in Ultraviolet Light in the Study of Boron Deficiency in Celery

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A better understanding of boron deficiency in celery, *Apium graveolens* L. var. *dulce* Pers., has been attained by observing symptoms of brown checking and