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Effect of Excessive Insulin on the Pancreatic Islets of Young Rats.*

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Hypertrophy of the pancreatic islets in diabetes mellitus is usually explained as a compensation resulting from hypoinsulinism. In the thyroid gland experimental evidence has been obtained by Loeb[1] and his associates that excess hormone is inhibitory. Such observations lend strength to the idea that cell regeneration is a response to functional need.

By the method previously used by one of us[2] the mitotic activity of the islet cells was determined in serial sections of the chrome-formol fixed tissue. The results in detail are shown in the table.

TABLE I.

Age at Injection Days	Age when Killed Days	Injections in Units	Mitoses per 100 Islets	Mitoses per 100 Islets in Control
4	7	1(3x)	4.8	32.0
4	7	1(2x), 2(1x)	10.0	
5	7	1(1x), 2(1x)	2.5	28.9
4	9	1(3x), 2(2x)	3.6	20.8
3	9	1(3x), 2(2x)	10.3	
4	9	1(4x), 2(1x)	9.7	
2	9	1(2x), 2(4x)	6.9	
4	10	1(2x), 2(3x)	7.6	
5	10	1(1x), 2(3x)	3.5	8.2
3	11	1(2x), 2(2x) 3(1x), 5(1x)	1.8	5.7
4	11	1(4x), 2(1x) 3(1x)	3.6	13.8
4	20	1(4x), 2(2x) 3(7x), 7(2x)	0.7	3.7

The injections were made at 1 or 2 day intervals with the last and largest dose about 18 hours before autopsy. Usually the pancreases from the 2 or 3 animals were mounted together and from 100 to 800 islets were enumerated. The controls were rats of the same age and they often were litter mates. The insulin treatment inhibited the proliferative activity of the islet cells although the growth of the animals continued normal and their weights were sometimes greater than those of the controls.

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[1] Loeb, L., *J. Med. Res.*, 1920, **41**, 481.

[2] McJunkin, F. A., and Breuhaus, H. C., *Arch. Path.*, 1931, **12**, 900.