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Science, Spontaneously

By Laura Pratt
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It was one of those "If life gives you lemons" stories.

Thirty-two years ago, a commercial laboratory in Ottawa that was selling laboratory animals to research labs observed that several of the rats in its colony were losing weight and dying. And then the phone rang. "We never ordered diabetic rats from you," said the scientist—a client—on the other end. Horrified, the commercial lab tried to destroy the animals and save its professional livelihood. But when word of all this reached a group of Toronto scientists working in Type 1 diabetes research, they raced to the fore and yelled, "Stop!" The world, they breathlessly explained, had been waiting for easy-to-manipulate small animals that would spontaneously develop a disease similar to its human counterpart. With this explosive mistake, the biobreeding (BB) rat was discovered. And diabetes research took a vaulting leap forward.


Dr. Philippe Poussier

Dr. Philippe Poussier, a senior scientist in molecular and cellular biology at Sunnybrook Research Institute who is among those in mid flight, has put these animals to excellent use, of late. It is courtesy of a BB rat and—a similarly spontaneous-disease-developing small animal—a NOD (nonobese-diabetic) mouse, that this researcher and his collaborators have been able to increase by a sizeable increment the state of Type 1 diabetes research. They have done so by identifying two susceptibility genes in animals that are associated with the human disease. Until recently, just a handful of genes had been identified as being predispositional for Type 1 diabetes. This news from Poussier and his collaborators bumps up the numbers significantly.

Insulin-dependent diabetes is a destruction of a person's insulin-producing cells which, as a consequence, renders her unable to control the entry of blood sugar into the cells, where sugar is necessary to provide energy. Sugar accumulates in the blood of a person with diabetes, and energy storage rapidly diminishes. In the absence of these cells' natural ability to produce this hormone, someone with Type 1 diabetes is forced to rely on insulin injections for the rest of his life. The weight of this burden—and its attendant psychological, economic and medical encumbrances—informs the imperative to pursue research in this area vigorously.

Employing an animal that spontaneously develops a disease very similar to its human foil, Poussier focuses his research on attempts to distinguish genetic variants in diabetes-stricken individuals. It is a formidable task. Consider that, in cystic fibrosis, there is one mutation that develops the disease; diabetes is thought to result by as many as 25 times that, and develops not only from genetic prompting, but environmental factors, as well. "We are trying," says Poussier, "to understand how the environment influences the frequency with which people who are genetically predisposed to the disease will develop it—or won't."

Poussier, then—working in conjunction with scientists at McMaster University in Hamilton and SickKids in Toronto, among other institutions—identifies and marks genes as being "disease-susceptible." That the molecular and cellular biologist has done so irrefutably in two cases is, he demurs, very satisfying.

After all, of the presumed 20 to 25 genes that are controlling the development of Type 1 diabetes in the rat, mouse and human, precious few have been locked in on. What's more, the few genes that have been identified appear to be involved in animal species and in humans—a significant correlation.

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