A New Way to Fight Diabetes With Colostrum!

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As recently as 1920, people who developed Type I diabetes lived about a year. Once their misguided immune systems started destroying the pancreas gland's insulin-producing islet cells, sufferers (most of them children) grew dehydrated and lapsed quickly into comas. Diabetes has been far less deadly since doctors learned to treat it with insulin injections, but it remains a leading cause of blindness and kidney damage, and it often leads to the amputation of hands and feet. Scientists have long hoped that by learning what turns the immune system against the islet cells, they could stop the disease at its root. Last week they reported dramatic progress. Writing in the journal Nature, two research teams not only identified the molecule that precipitates the devastating immune attack but described a way to derail it.

The immune system is exquisitely designed to repel foreign invaders while leaving the body's own tissues alone. White blood cells patrol the body, orchestrating attacks on any substance whose molecular markers, or antigens, look unfamiliar. Previous studies have shown that when diabetes sets in, the immune system reacts violently to several antigens found on normal islet cells. But until now, no one knew which antigen set off the destructive cascade. To find out, research teams at UCLA and Stanford synthesized an array of islet-cell antigens and mixed them with blood drawn from diabetic mice at different stages of illness. Tests showed that the animals reacted to one antigen—a protein called GAD—from the outset. But as their conditions progressed, says biologist Daniel Kaufman of the UCLA team, their immune systems turned against other antigens as well.

Common sense suggested that if GAD was the immune system's initial target, then teaching the system to tolerate GAD might prevent the rest of the avalanche. To test that hypothesis, both teams started injecting GAD into diabetes-prone mice during infancy, while their immune systems were still forming. Both teams succeeded in preventing immune responses to GAD—and both managed to prevent or delay the onset of diabetes. The 17 mice treated in the UCLA study are now 40 weeks old, far beyond the age when the disease usually appears, and none has developed symptoms. Says Kaufman, "This one shot seems to have eliminated the disease process."

No one knows whether a single shot will do the same for people, but researchers now hope to explore that tantalizing possibility. First they'll try feeding GAD (or inactive fragments of it) to people already developing Type I diabetes, to see if it slows the disease. Eventually, Kaufman hopes to inject GAD fragments into healthy infants from diabetes-prone families. Gauging the results could take a decade, but if the approach lives up to its promise, one of the nation's leading childhood illnesses could soon become a relic.