really something other than hormones. Gastrointestinal peptides would be a more appropriate term."

Regardless of their nomenclature, gut hormones are more than a scientific curiosity. As Stephen R. Bloom of the Royal Postgraduate Medical School in London points out, the gut hormone motilin is involved in indigestion. While motilin's normal role is to empty the stomach of food and to keep the stomach free from bacterial growth, it sometimes forces stomach acid up into the esophagus where it creates indigestion. In fact, symposium speakers concurred that as gut hormone research progresses, more and more gut hormones will probably be implicated in various digestive diseases and perhaps offer clues as to how to treat those diseases more effectively than they are being treated today.

Meanwhile, an analog of one gut hormone looks promising as an anti-obesity drug, reports Gerard P. Smith of New York Hospital-Cornell Medical Center in White Plains, N.Y. In 1937, a gut hormone called interogastrone was found to inhibit food intake in animals. The results were confirmed 30 years later with more pure extracts. The pancreatic hormone glucagon was also found to exert such effects. Why do these hormones cut appetite? Because food releases them from the digestive track into the circulation, they may be satiety signals. In any event, they signaled

Smith and his colleague James Gibbs that they might, if injected as pharmaceuticals, turn out to be effective anti-obesity drugs.

So Smith and Gibbs started injecting various gut hormones into experimental animals, and as Smith reported at the Miles symposium, the one that works best, in either its natural or synthetic analog



Dockray: Similarities to classic hormones.

state, is cholecystokinin (CCK). CCK is normally made in the small intestine and acts on the gall bladder and pancreas. But higher concentrations of CCK are found in the brain than in the intestine, so CCK may have a central nervous system role in controlling behavior such as food intake. In fact, Rosalyn Yalow of the Bronx, N.Y., Veterans Administration Hospital has found that CCK is much less prevalent in the brains of a strain of genetically obese mice than in the brains of normal mice (SN: 1/27/79, p. 57). When Smith and Gibbs injected a CCK analog into hungry dogs, the animals quickly lost their appetites and stopped eating. When the researchers injected the analog into hungry monkeys, they, too, rapidly lost their appetites. And there were no toxic side effects. This past year, the investigators also found that the analog could cut appetite in hungry volunteers. Currently they are attempting to see whether the analog can also reduce appetite in moderately obese men and women.

If the CCK analog once again produces the desired effects, it may eventually receive the U.S. Food and Drug Administration's approval as an effective and safe anti-obesity drug. And if so, it would be the first one to become commercially available, since current anti-obesity treatments, such as liquid protein diets, amphetamines and intestinal bypass surgery, tend to produce more undesirable side effects than they do weight loss.

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SCIENCE NEWS, VOL. 117