# UA-Led Study on Hyperglucagonemia, Type 2 Diabetes Picked Up by Journal of Endocrinology

The *Journal of Endocrinology* published a paper with the University of Arizona's **Jennifer Stern, PhD**, as lead author that summarizes her research which suggests obesity's metabolic pathophysiology may be driven by inappropriate fasting-induced regulation of glucagon—a hormone that is abnormally elevated in obesity and type 2 diabetes.

Dr. Stern's article, **"Obesity dysregulates fasting-induced changes in glucagon secretion,"** appears in the August 2019 issue (Volume 242), of the journal, a publication of the Society of Endocrinology.


Dr. Stern, who joined the UA faculty last fall, is an assistant professor of medicine (in the Division of Endocrinology) and physiology. She is one of four faculty members who will represent the UA Department of Medicine at the third annual **Innovations & Inventions** research showcase. That event is hosted Oct. 17, 4:30-7 p.m., by the UA College of Medicine – Tucson in the HSIB Forum of the Health Sciences Innovation Building.

## Glucoregulatory Disturbances

Obesity is the leading cause of type 2 diabetes, a disease characterized by high blood glucose, also called hyperglycemia. There are two key hormones that regulate blood glucose concentration. Insulin acts to decrease blood glucose, while glucagon increases blood glucose. In people with obesity and type 2 diabetes, both insulin and glucagon are elevated compared to lean individuals. "The glucagon:insulin ratio is a better determinant of metabolic regulation than glucagon or insulin alone," says Dr. Stern. "Normally, in healthy individuals, the glucagon:insulin ratio increases with fasting. In contrast, re-feeding normally causes a decrease in the glucagon:insulin ratio."


## Glucagon Signaling in Obesity

Dr. Stern and colleagues evaluated glucagon homeostasis in lean and obese mice and people. They found that, in lean mice, fasting for an extended period of time caused a rise in glucagon secretion and an increase in the glucagon:insulin ratio. "This is what we expect to see in lean healthy mice, but our observations in the obese mouse was a stark contrast," Dr. Stern said. Fasting decreased both glucagon and insulin in obese mice, while refeeding increased both glucagon and insulin. As a result, in obese mice, the glucagon:insulin ratio was unaffected by nutritional state.

"Consistent with our findings in the mouse, in obese humans the glucagon:insulin ratio remained relatively static in response to fasting and re-feeding. Therefore, the glucoregulatory disturbance in obesity may be driven by inappropriate regulation of glucagon by fasting and a static glucagon:insulin ratio," Dr. Stern said.


The *Journal of Endocrinology* study is a follow-up to work Dr. Stern did that was published in the July 2018 issue of the journal *Diabetes*, **"Fasting-induced changes in glucagon secretion are dysregulated in obesity."** That journal is a publication of the American Diabetes Association.

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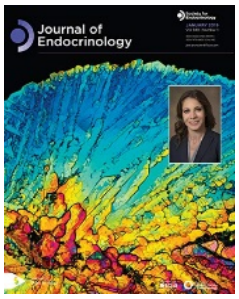


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September 13, 2019

The *Journal of Endocrinology* has published a paper with the UA's Dr. Jennifer Stern as lead author on research that suggests the metabolic glucoregulatory disturbance in obesity that can result in type 2 diabetes may be driven by inappropriate fasting-induced regulation of glucagon, resulting in a static glucagon:insulin ratio…

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