

## Insulin, Glucagon and Feed Intake Regulation: Ruminant and Rat Models

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### Abstract

Hormones belong to metabolic factors that control and regulate feed intake in ruminants and rats. Estrogen depresses feed intake. Insulin is an important hormone possessing both long-term and short-term effects on ruminant nutrient partitioning and feed intake. Insulin is associated with overconsumption in rats. The role of glucagon in feed intake regulation in ruminants has not been thoroughly described. Some evidence exists that exogenous glucagon reduces feed intake in sheep. Intravenous glucagon induced satiety in humans. Intraperitoneal glucagon has not affected feed intake in rats. Research is needed to elucidate how to manipulate animal and human endocrinology to optimize feeding strategies and systems in food-producing animals and to improve human health.

**Keywords:** Feed Intake; Regulation; Hormone; Ruminant; Rat

## Introduction

The objective of this mini review article was to illuminate hormones-mediated feed intake regulation in ruminant and rat models. A special emphasis was made on estrogen, insulin and glucagon. This was to help formulate nutritional strategies to optimize metabolism and performance in food-producing animals and to improve human physiology and health. Nutrient partitioning is mediated by a variety of hormones [1]. Hormones are involved in both short-term and long-term regulation of feed intake [2,3]. The greatest fluctuations in body metabolism, nutrient partitioning, and feed intake usually occur around calving in large ruminants when levels of metabolic and reproductive hormones are highly variable in periparturient cows [3,4].

## Discussion

Estrogen depresses feed intake by acting primarily on the paraventricular nucleus of the hypothalamus [5,6]. Insulin is another important hormone possessing both long-term and short-term effects on ruminant nutrient partitioning and feed intake [7,8]. The long-term roles of insulin in feed intake control relate to pregnancy and lactation [1,9]. This occurs mainly during mid- and late-lactation when the cow tends to gain weight. Insulin is involved in both up- and down-regulation of feed intake in mammals. When compared to prepartum levels, insulin secretion drops substantively shortly after parturition [3]. Without the postpartum drop in insulin secretion rate, the cow would be unable to use body reserves and deal with insufficient dry matter intake (DMI). The low postpartum insulin will additionally enable the cow to gradually increase DMI.

The short-term insulin effects on nutrient metabolism and partitioning, which are more relevant to the findings of dairy cattle studies, initiate upon or even shortly before feeding or nutrient ingestion [10]. Seeing the fresh feed can cause a surge in insulin secretion in sheep and cattle [10-12]. The higher postprandial insulin surge leads to greater glucose uptake by peripheral tissues. By increasing the peripheral glucose uptake, the postprandial insulin secretion may contribute to satiety [13]. A higher postprandial insulin secretion and thus the increased peripheral glucose use may induce satiety signals. In contrast, insulin can stimulate feed intake in response to insufficient nutrient supply [14]. Insulin has also been reported to be associated with overconsumption in the rat [15]. It might be possible that the postprandial rise in insulin secretion in high-producing cows demanding much energy and nitrogen to sustain milk yield, may not necessarily depress DMI. Instead, due to high nutrient demand by the mammary gland, the postprandial insulin secretion might facilitate nutrient uptake by increasing feed intake.

Although studied in humans in as early as 1955, the role of glucagon in feed intake regulation in ruminants has not been delineated [3,16,17]. Intravenous glucagon induced satiety in humans. In contrast, intraperitoneal glucagon stimulated glucogenolysis but did

not affect feed intake in rats [18]. Glucagon stimulates hepatic glucose production via both glycogenolysis and gluconeogenesis [7]. The hepatic glucose release does not appear to be the exclusive pathway whereby glucagon may affect satiety [19]. For example, Geary and Smith [20] showed that increased blood glucose did not end the meal. The peritoneal use of rabbit glucagon antibodies in rats to reduce gluconeogenesis and blood glucose increased feed intake [20,21]. These data show that reduced blood glucose can induce hunger, but increased blood glucose may not induce satiety. Also, some evidence exists that exogenous glucagon reduces feed intake in sheep [22]. Thus, the effect of glucagon on feed intake control seems to be mediated by other agents than only glucose. Future research is needed to elucidate the impact of glucagon on feed intake in dairy cows.

## Conclusion

Hormones contributions to feed intake in ruminants and rats were reviewed. Research is needed to clarify how to manipulate animal and human endocrinology to optimize nutritional strategies and systems in food-producing animals and to improve human health.

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