

Editorial





# Insulin chronophysiology: a nutritional wisdom

## Philosophy and discussion

Insulin is called the storage hormone because it stimulates glucose entry into the peripheral fat and muscle cells. As substantiated globally, much less glucose crosses into the portal vein in ruminants when compared to non-ruminants. In consequence, insulin would not have as significant impacts on hepatic glucose metabolism in ruminants as it would in non-ruminants. <sup>1-3</sup> Nervous system, gut peptides, other pancreatic secretions, and nutrient absorption are the main candidates in stimulating insulin release from the pancreas. The effects of the nervous system on insulin release regulation take place via sympathetic and parasympathetic neurons. The vision, odor, and flavor of the food can induce insulin secretion via activating the parasympathetic neurons in humans.

The previous substantiated science led to the proposal that neural impulses and gastrointestinal hormones are involved in the post-feeding insulin response to feed delivery in ruminants as well. Secretin and pancreozymin (cholecystokinin) stimulated insulin release in sheep, and blood insulin rose sooner than did blood glucose, suggesting that glucose was not a major cause of the initial rise in post-feeding insulin release. Nevertheless, the ultimate increase in blood glucose may contribute to maintaining the high post-feeding insulin concentration.<sup>1,4</sup> In goats fed ad libitum for a 3h period daily, a post-feeding rise in blood insulin occurs that probably is caused via VFA stimulation of the pancreatic β-cells. A post-meal rise in blood insulin was observed, however, no such a peak was noticed in blood VFA. Thus, these suggest that the nervous signals (rather than VFA) either directly or through the secretion of gut hormones may result in the post-meal insulin response. A similar post-feeding rise in blood insulin occurs in lambs fed in two equal morning and evening meals.<sup>1,2</sup>

When monitored every 15min for 48h in six lactating cows fed once daily at 0900 h, blood insulin exhibited distinct diurnal rhythms in all cows, peaking at 1745h and falling to a nadir overnight or during the dark phase i.e., 2300-0700h. 1.3 The 24h patterns in peripheral blood insulin are closely linked to the 24h patterns in feed intake. In cows fed forage and concentrate separately, blood insulin rises sharply upon concentrate delivery at 0600h and 1430h and declines shortly thereafter, remaining lower overnight. 1.2 Similarly, feed intake showed two major peaks (after both concentrate delivery), being considerably lower between 0200 to 0600h. In that trial, the forage was offered ad libitum at 0600h. The fact that blood insulin was higher in the afternoon but lower overnight would support the circadian patterns of peripheral insulin observed. Overall, the 24h patterns in peripheral blood insulin relate to the dietary content of readily fermentable carbohydrates.

Future research must be directed to uncover delicate details of specialized circadian rhythms of animal models' insulin metabolism and levels. In addition, interaction of such rhythms with lifestyle indices of mainly eating behaviour and exercise must be discovered.

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#### Akbar Nikkhah

Department of Animal Sciences, University of Zanjan, Iran

Correspondence: Akbar Nikkhah, Chief Highly Distinguished Professor, Department of Animal Sciences, Faculty of Agricultural Sciences, University of Zanjan, Foremost Principal Highly Distinguished Elite-Generating Scientist, National Elite Foundation, Iran, Email anikkha@yahoo.com

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Such models may be utilized to gain insight into human metabolism. These are key to profitable livestock production and improving human health in today's interrupted unnatural human life.<sup>5-8</sup> Insulin management improvement serves food-producing ruminants and humans as a postmodern wisdom.

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## **Conflict of interest**

Author declares that there is no conflict of interest.

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