Nutritional science of glucose and lactate

Abstract
This article describes nutritional and physiological dynamics of glucose and lactate in human and animal models. Considering the utmost significance of these two nutrients in energy and protein assimilation and metabolism, interdisciplinary concepts are developed to increase and update the current knowledge and insight on nutritional importance of glucose and lactate.

Keywords: nutrition, glucose, lactate, metabolism

Introduction
Blood glucose in human is known to vary diurnally in humans. The diurnal variation in blood glucose is attributed to diurnal patterns of feed intake, hepatic gluconeogenesis, glucose tolerance, and blood levels of hormones such as adrenalin, glucagon, and corticosterone. Altogether, the diurnal variations in blood glucose of humans and rats are orchestrated by the internal clock located in the suprachiasmatic nucleus of the hypothalamus. For instance, blood glucose rises just before the beginning of the activity period, which is the early morning in humans and evening in rats. The early morning rise in blood glucose is known as “down phenomenon” occurring in non-diabetics. Such an early morning rise in blood glucose may be an arrangement by the suprachiasmatic nucleus preparing the body for the forthcoming activity period.1,4

Glucose tolerance or insulin sensitivity decreases as day progresses, reaching a nadir around midnight. The diurnal regulation of glucose metabolism is meal-independent in humans and rats. When compared to humans, blood glucose is lower in ruminants partly because dietary carbohydrates undergo rumen fermentation before facing abomasal and intestinal assimilation. As a result, little intact or partially hydrolyzed soluble carbohydrates (5-30% of that consumed) enter the duodenum. Assuming an arrival of 1kg starch in the small intestine, less than 60% has the potential to be fully digested before entering the large intestine. In addition, the gut uses a considerable amount of glucose. Thus, the glucose absorbed via the small intestine may not contribute to more than 20% of net hepatic glucose output.1,2,5

Usually, about 20-35% of the total VFA produced in the rumen is propionate. Ruminants rely on gluconeogenesis mainly from propionate and to a variable extent from alanine and glutamine, lactate, and glycerol to meet their glucose demands. Gluconeogenesis occurs mostly in the liver and some in the kidney. In non-ruminants including human, feeding can cause a rapid surge in the intestinal glucose absorption, and therefore, an abrupt glucose appearance in the peripheral blood. Post-feeding blood glucose response is expected to be higher and earlier in non-ruminants than in ruminants. Apart from substrate availability, the post-feeding blood glucose response to feeding can cause a rapid surge in the intestinal glucose absorption, and therefore, an abrupt glucose appearance in the peripheral blood. Post-feeding blood glucose response is expected to be higher and earlier in non-ruminants than in ruminants. Apart from substrate availability, the post-feeding blood glucose response to food presentation and intake is regulated by the interactive effects of hormones such as insulin and glucagon.1,2,6

L-lactate is the predominant lactate isomer produced in the rumen. However, D-lactate is also produced when high-grain diets are fed. Hepatic L-lactate flow originates from either the gut or non-splanchnic muscle tissues. The gut L-lactate is produced mostly by the rumen microorganisms and transferred to the liver to be used as a gluconeogenic or energy-generating substance. In addition to the rumen, the non-splanchnic muscle tissues generate L-lactate as the end-product of anaerobic glucose oxidation. The resultant L-lactate travels to the liver to be converted to glucose which is then transferred back to the muscles to sustain normal muscle metabolism. It is notable that the hepatic use of L-lactate (for oxidation or gluconeogenesis) is twice that of D-lactate. In vitro studies have shown that L-lactate is used for gluconeogenesis with a lower rate which is not responsive to insulin, when compared to propionate. Thus, post-feeding insulin rise will probably not greatly reduce gluconeogenesis from lactate. L-lactate may be the second most significant contributor to gluconeogenesis in fed ruminants (13-17.5%). In fattened cows, however, L-lactate contributed as much as 74.4% to the hepatic glucose synthesis. These data indicate the significance of lactate in energy use by both mammary and non-mammary tissues.2,5-7

Acknowledgements
Thanks to the Ministry of Science Research and Technology, and National Elite Foundation for supporting the author’s global initiatives and programs of optimizing science edification in the third millennium.

Conflict of interest
Author declares that there is no conflict of interest.

References
