Eating Timing and Diabetes

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Received: January 29, 2014
Published: February 17, 2014

Citation: Nikkhah A (2014) Eating Timing and Diabetes. Int J Diabetol Vasc Dis Res. 2(1e), 1. doi: http://dx.doi.org/10.19070/2328-353X-140006e

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Eating timing is a feasible strategy that has received seriously inadequate considerations in public health and education [1]. Human body tolerates less glucose as evening begins, mainly because glucose is demanded most during more active times or day-time [2,3]. This editorial develops and strengthens a recommendation to avoid large evening and night food meals rich in sugars, starches, and fats to help reduce risks of visceral adiposity, diabetes mellitus, hypertension, and cardiovascular abnormalities.

Optimal understanding of human physiology requires appreciation of comparative interspecies physiology. Optimal animal physiology is understood with premium perception of ruminant physiology with vastly exclusive complex systems biology [4]. Ruminants as irreplaceable human food producers are metabolically and economically suitable models to study cell, organ and whole body physiology [5,6]. Evening vs. morning feeding in lactating cows has increased eating rate and postprandial rumen and peripheral metabolism [7,8]. Evening eating, in addition, increased milk production in dairy cows [9] and improved feed efficiency in beef cattle [6]. These recent discoveries emphasize the highly significant nature of feeding and eating timing in regulating nutrient partitioning and metabolism [10]. However, ruminants are different from human in sphacnic and peripheral metabolism. As such, different effects of eating timing on cell nutrrophiogenicomics could be expected in human vs. ruminants.

Glucose concentrations rise at the end of ‘resting period’, which is ‘dark period’ in human. The glucose rise just before the onset of the activity period is known as ‘dawn-phenomenon’ [11]. The blood glucose peak coincides with circadian rises in corticosterone levels. The glucocorticoid peak contributes to the elevated glucose output and insulin requirement [12]. Growth hormone related increases in hepatic glucose production are a main cause of the dawn glucose rise. Moreover, nocturnal melatonin secretion induced by darkness increases postprandial insulin requirements [13]. Reduced nocturnal glucose tolerance may partly be mediated by increased melatonin secretion. Reduced glucose tolerance reflects reductions in glucose demands because glucose is demanded the least during inactive times or night-time. As such, reduced glucose tolerance could be an evolutionary preparation for the resting body to cope with the darkness. Avoiding large night meals would allow melatonin, insulin and other intermediates to more efficiently optimize nocturnal metabolism. Shift-workers with perturbed suprachiasmatic nuclei and eating time driven rhythms of nutrients and hormones metabolism require special diurnal and nocturnal nutritional regimes [14].

Integrating animal models discoveries lead to an implication to synchronize external cues with internal human physiology to maximize nutrient efficiency and optimize health. Persistent education and insight dissemination will help to practically incorporate eating timing as a feasible lifetime strategy into successful local and global nutritional and public health policies and programs.

References