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Influence of Time-restricted Meal Intake on Blood Pressure Variables

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Abstract

Time-restricted eating (TRE) has garnered attention for its potential metabolic benefits by aligning meal timing with circadian rhythms and facilitating extended fasting periods. This narrative review explores the mechanistic underpinnings of blood pressure regulation within the context of diurnal rhythms and the potential implications of TRE on blood pressure outcomes. While research on TRE's direct impact on blood pressure remains limited, existing studies suggest associations between narrower eating windows and reductions in blood pressure levels, independent of weight loss. Mechanistically, TRE may influence blood pressure through various pathways, including alterations in sympathetic activity, hormonal regulation, and sodium retention. Animal studies and preliminary human trials support the hypothesis that aligning meal timing with circadian rhythms may promote cardiovascular health by modulating blood pressure rhythms. However, conflicting findings and methodological variations across studies underscore the need for further research, particularly larger clinical trials with diverse populations and comprehensive methodologies such as ambulatory blood pressure monitoring. Integrating TRE into broader lifestyle interventions may offer a holistic approach to optimize cardiovascular health through circadian alignment and metabolic regulation.

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INTRODUCTION

From a mechanistic perspective, Time-Restricted Eating (TRE) is believed to offer metabolic advantages by aligning the timing of meal intake with circadian rhythm and potentially boosting catabolism through an extended fasting period. Notably, commencing and concluding TRE protocols at earlier hours appear to result in more substantial reductions in blood pressure. Furthermore, narrower eating windows are linked to lower blood pressure levels. It is important to highlight that while subsequent weight loss does not continuously correlate with decrease in blood pressure, decreased insulin levels may play a crucial role in lowering blood pressure.[1]

TRE entails partaking food exclusively within a daily time-restricted window of eating period without intentionally restricting calories. It is crucial to differentiate TRE from intermittent fasting (IF), in which the total or partial caloric restriction occurs on specific days of the week and the fasting duration or timing of food intake is not consistently maintained everyday. TRE is considered to be a subtype of intermittent fasting, emphasizing that it is the time of eating, rather than the content of caloric intake or quantity, that is the primary determinant. Notably, the absence of intentional caloric restriction in TRE may enhance adherence.[2,3]

However, the investigation into the impact of TRE on blood pressure variables remains incomplete, as no studies specifically addressing blood pressure as a primary outcome have been conducted. The well-established diurnal rhythm of blood pressure is widely recognized, and disruptions in this daily variability are closely related to effects on cardiovascular morbidity and mortality rates.[4-6]

Mechanism of Diurnal Rhythm of Blood Pressure

Blood pressure shows a diurnal rhythm, with relatively higher levels during the day as opposed to the night. Blood pressure has a proclivity to decrease during sleep by 10–20%, experiencing a distinct morning surge following the time of waking up.[7,8] It's essential to note that the diurnal rhythm of blood pressure observed does not necessarily mimic the autogenous circadian rhythm which is essentially an internally regulated, roughly 24-hour cycle persisting without external cues. Behavioural and physiological factors play a more significant role in shaping the diurnal blood pressure rhythm rather than this internally regulated circadian rhythmicity.

The autogenous circadian rhythm of blood pressure showcases peaks in systolic (SBP) as well as diastolic (DBP) BP at around 21:00.

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Peak-to-trough amplitude of approximately 3–6 mmHg is observed for SBP and 2–3 mmHg for DBP. This pattern has also been observed in studies involving healthy, normotensive adults subjected to inpatient protocols. These protocols, such as the "constant routine protocol" involving complete and consistent wakefulness with constant posture and isocaloric snacks which are given at an interval of 2 hours in dim light, or the "forced desynchrony protocol" which entails[9,10] recurring sleep/wake cycles equally distributed along a non-24-hour cycle in dim light, effectively delineate the autogenous circadian rhythm from environmental cues, e.g., exposure to light and behavioural cues, e.g., physical activity, sleep, food intake zeitgebers.

The observed diurnal rhythm is primarily influenced by behavioural and physiological factors. Physiological regulators of blood pressure, include sympathetic activity, levels of cortisol hormone, cardiac vagal tone, vascular tone, and renal sodium retention, each factor exhibiting its own unique autogenous circadian effect. Sympathetic activity, which is measured by the amount of circulating adrenaline and noradrenaline levels, demonstrates a circadian trough at night and peaks in daytime, with maximum levels in the afternoon. Plasma cortisol peaks in morning and reaches a trough at night. Cardiac vagal tone assessed through heart rate variability, peaks in the morning and dips at midday. Urinary sodium excretion follows a circadian pattern, increases during the day, and is relatively decreased at night, influenced by both circadian and sleep effects.[11-13]

Additionally, in murine models, clock proteins such as Per1, Per2, Clock, and Cry1/2 are shown to play a crucial role in controlling sodium retained by the kidneys, subsequently affecting blood pressure rhythm.[14-18] Studies done on animal models are indicative of the fact that genes are responsible for maintaining the functionality of vascular smooth muscle cells and also show circadian oscillations. Deleting Bmal1 in murine vascular smooth muscle cells has an impact on the amplitude and timing of blood pressure oscillations, exterminating the variable component of circadian rhythmicity of pulse pressure. [20]

Behavioural factors, including sleep/wake cycles and types of fasting/feeding, have pivotal roles in the regulation of blood pressure rhythms. Night-time sleep is related to decreased blood pressure while waking up from sleep is linked to increased blood pressure. The beginning of sleep involves a decrease in sympathetic drive, a recumbent body position, and a decrease of core body temperature, regulated by the circadian rhythm, these three factors result in progressive vasodilation and culminate in a lowering of arterial blood pressure.[21,22] Notably, a study measuring rate of blood flow to the subcutaneous adipose tissue of the lower leg in normotensive lean individuals observed heightened peripheral blood flow and dampened peripheral vascular resistance in the course of night time sleep. The blood pressure shows variation all along the night in accordance with the different stages of sleep, with the least value of blood pressure observed during deep sleep and the maximum value seen during lighter stages of sleep.[24] The morning surge in blood pressure upon waking is probably attributed to the presence of lighter sleep stages, the redistribution of blood volume from the skin to central arterial circulation which occurs because of a circadian-mediated increase in core body temperature, and heightened sympathetic activity.[22]

Another significant behavioural factor influencing BP rhythms is the fasting/feeding pattern. Both types of fasting whether short-term (48 hours) or long-term (4–41 days) done under medical supervision have been shown to reduce both systolic and diastolic blood pressure. Fasting is associated with natriuresis, a phenomenon that can be offset simply by administration of glucose whether in patients with normal blood pressure or in hypertensive individuals.[27,28] The exact mechanisms behind this occurrence of sodium depletion remain unknown but are speculated to involve the effect of insulin on renal sodium retention and decrease of insulin hormone level which occurs during fasting. Insulin has been theorized to have a direct role in increasing blood pressure through various methods, which include stimulating sodium reabsorption in the kidney, trophic effects on migration and proliferation of vascular smooth muscle cells, upregulation of angiotensin system, and regulation of the sympathetic nervous system in a manner synchronous with tissue specificity, remains a subject of controversy regarding its causal relationship with hypertension.[29]

The clinical importance of the diurnal blood pressure rhythm lies in its alteration in individuals with cardiometabolic diseases, potentially serving as an indicator or mediator of heightened mortality and morbidity. On a more specific note, deviations such as the absence of a nocturnal blood pressure decline ("non-dipping BP") and increased night time blood pressure as opposed to daytime blood pressure ("reverse dipping") are linked to an elevated risk of cardiovascular disease and mortality. In hypertensive patients, those individuals with metabolic syndrome exhibit an increased occurrence of non-dipping nocturnal BP, indicating potential erroneous regulation of renal water and excretion of sodium. A reverse dipping of the blood pressure rhythm is associated with an increased incidence of cardiovascular events and mortality in patients of type 2 diabetes.

One proposed mechanism involves aligning the timing of meal intake along with autogenous circadian rhythm to benefit and realign overall health, often termed the "circadian alignment" hypothesis. Diverse studies performed in animals illustrate the vital consequences of this all-pervading rhythm in regulating metabolism. Studies show that mice with either whole-body or even tissue-specific deletion or dysfunction of alleles of various circadian rhythm-regulating genes exhibit malfunction in various physiological processes of glucose homeostasis, metabolism of lipids, and weight regulation.[30]

Observations in human populations further support this hypothesis, with a higher incidence of obesity and metabolic syndrome noted among night shift workers.[31] These findings point to the fact that dysfunction or dysregulation of circadian rhythm, characterized by a delinking or desynchrony of our autogenous inherent circadian rhythm and the timing of various behaviours like time of eating/food intake, taking rest or sleeping, and time of sport or physical activity, might contribute to the increased occurrence of obesity, metabolic dysregulation and resultant metabolic disorders, and even, cardiovascular disease.

Adjusting the time of the eating window to an earlier time of the day aligns with human metabolism, which is naturally more active during that period. Hypothetically, such a shift may have potential benefits for blood pressure, as an earlier intake of salt aligns with the diurnal upregulation of urinary sodium excretion by kidneys. Conversely, the metabolic benefits of TRE may be attributed to increasing the routine fasting period, thereby increasing catabolism. According to

the "fasting" hypothesis, narrowing the eating window according to an individual's baseline, irrespective of when it occurs either early in the day or late, may be associated with any notable effects of TRE on BP, potentially involving fasting-induced excretion of sodium in urine and a decrease in the levels of insulin.

Murine subjects, commonly utilized in studies involving Time-Restricted Feeding (TRF), exhibit nocturnal behavior, with their metabolic peak coinciding with the dark (night-time) period. According to a study, the temporal distribution of dietary intake can impact blood pressure (BP) rhythms. In murine models subjected to unrestricted 24-hour feeding, wherein heightened consumption of food and water endogenously takes place during the active/dark phase, acme of MAP is observed in the active/dark phase, while the nadir of MAP is evident in the inactive/light phase. Significantly, capacity timing of food to synchronize rhythms of blood pressure continues even in conditions devoid of the light/dark cycle (constant darkness) and in Bmal1 knockout mice, characterized by the absence of a key transcription factor in regulation of the circadian rhythm in cells.[32]

A study was done in which feeding was regulated and the rhythms of blood pressure in diabetic db/db mice were studied. The investigation revealed that db/db mice display diminished rhythms in mean arterial pressure (MAP), primarily attributed to elevated MAP levels in the inactive/light phase (non-dipping), simultaneously occurring alongwith heightened intake of food in this period. It demonstrated that implementing an 8-hour or 10-hour Time-Restricted Feeding (TRF) regimen during the active/dark phase prevented the progression of non-dipping BP in mice with diabetes and reinstated dipping of blood pressure in those with pre-existing non-dipping patterns. Mechanistically, TRF selectively inhibited sympathetic activity during the inactive/fasting phase in mice with diabetes, as proved by a decrease in heart rate, an increase in heart rate variability heightened spontaneous baroreflex sensitivity, and a reduction in urinary noradrenaline and normetanephrine levels. Favourable results of time-restricted feeding on blood pressure in diabetic patients could have been brought about via a reduction in sympathetic vascular tone during the inactive/light phase induced by fasting. Findings from time-based intake of meals in murine models point towards the fact that the time of meal intake could independently influence blood pressure rhythm, and curtailing feeding to the active/dark phase leads to lower blood pressure during the inactive/light phase, in accordance to principles of "fasting" hypothesis.

Review of Previous Studies: Current Evidence

Studies to date have indicated a reduction in blood pressure (BP) in specific instances of TRE. For example, a randomized crossover trial involving men with prediabetes demonstrated significant improvement in blood pressure and insulin sensitivity with 6-hour early time-restricted eating, even without weight loss.[33] Additionally, a non-randomized study focusing on patients of metabolic syndrome revealed enhanced cardiometabolic profiles, including lower blood pressure, in individuals adopting a 10-hour time-restricted eating pattern for 12 weeks.[34] A couple of other studies, which were not randomized, included obese/overweight adults[35,36] reported significant reductions in blood pressure.[37-45]

Conversely, various studies in overweight/obese adults did not show substantial changes in blood pressure. Drawing a coherent conclusion from these findings proves challenging because of enough variations in samples taken, duration and timing of eating window, and the timing of outcome measurement. Moreover, nearly all TRE studies reported concurrent weight loss, complicating the determination of whether blood pressure reduction is solely attributed to TRE. Furthermore, a lacuna is concomitant decrease in intake of food while practising any time of time dependent feeding pattern, introducing the possibility of observed decrease in blood pressure might be primarily associated with caloric restriction.

Compelling proof[33] supporting the decrease of blood pressure via time-based food intake is derived from a study conducted by Sutton *et al*. This randomized, cross-over, eucaloric, and isocaloric controlled feeding study demonstrated that TRE effectively lowered morning blood pressure without weight loss. It implemented a 5-week TRE protocol alongwith a 6-hour eating window, finishing dinner not beyond 15:00 (3:00 pm), and compared it to a control condition of 12-hours. The participants, consisting of 8 men with prediabetes, exhibited normal morning systolic and diastolic blood pressures at baseline and not on any anti-hypertensive treatment. TRE resulted in a noteworthy reduction in systolic and diastolic blood pressure by 11 ± 4 mmHg and 10 ± 4 mmHg (mean ± SD), respectively, measured in the morning. This significant blood pressure decrease was unexpected, particularly considering the minimal weight loss observed (approximately 1 kg for both conditions). One potential way to explain this could be that an extended fasting period may be associated with TRE (18-hour overnight fast) might have contributed to the decreased blood pressure recorded the subsequent morning compared to the 12-hour overnight fast in the group without intervention. In summary, clinical studies on TRE present varied results regarding blood pressure outcomes and noteworthy differences in various study designs and varied TRE protocols could be the reason for this difference.

Future Perspectives

To deepen our comprehension of TRE's potential impact on blood pressure (BP) outcomes, additional methodology-based research in larger sample sizes and diverse population are required. Utilizing 24-hour ambulatory BP monitoring could prove instrumental in exploring the varied effects of TRE during diverse times of the day. Gaining insights into the chronobiology of BP rhythm holds the potential to unveil innovative approaches for BP control and the mitigation of cardiometabolic morbidity and mortality risk, especially in individuals with metabolic syndrome.[1] Lastly, integrating TRE into broader lifestyle modifications, such as adjusting the sleep timing and/or time of physical activity, present a practical strategy to synergistically influence BP rhythms. This holistic perspective has the potential to enhance the overall efficacy of TRE in promoting cardiovascular health.

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