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Acute Metabolic and Neurocognitive Adaptations to Short-Term Fasting

Daniel Bricker*, Julian Alberto, Zlatan Pecar, Dario Pecar, Syed Asad

Universal Neurological Care, Jacksonville, Florida, USA
Email: *daniel.bricker@universalneurocare.com

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Abstract

This study examined the metabolic, cognitive, and electrophysiological adaptations to a 48-hour water-only fast in a cohort of adults (n = 10). Blood glucose, β-hydroxybutyrate (BHB), Trail Making Test A (TMTA) and B (TMTB) performance, and auditory P300 event-related potentials were measured at baseline, 24 hours, and 48 hours. Glucose decreased progressively from 104.5 ± 10.2 mg/dL at baseline to 69.2 ± 7.9 mg/dL at 48 h (−34%), while BHB rose from 0.27 ± 0.15 mmol/L to 2.73 ± 0.81 mmol/L, indicating robust nutritional ketosis. TMTB completion time improved by 22% over the same period (95.4 ± 12.7 s to 73.2 ± 10.8 s), suggesting enhanced executive function during early ketosis. P300 latency and amplitude remained stable across all time points, indicating preserved cortical processing speed and attentional resource allocation. Exploratory sex-stratified analysis revealed greater ketone elevation in males at 48 h (3.64 ± 0.54 mmol/L) versus females (1.99 ± 0.62 mmol/L), without corresponding differences in cognitive or electrophysiological measures. These findings demonstrate that prolonged fasting elicits a predictable metabolic shift toward ketosis without impairing, and potentially improving executive performance, underscoring the need for further research into the cognitive effects of acute nutritional ketosis.

Keywords

Short-Term Fasting, Nutritional Ketosis, Metabolic Adaptation, Executive Cognitive Function, Cognitive Flexibility, Electroencephalography, Neurocognitive Resilience

1. Introduction

Fasting has emerged as a widely investigated nutritional intervention with broad implications for human metabolic health, neurocognitive performance, and dis-

ease resilience [1]. When caloric intake is withheld, the body undergoes a metabolic shift from primary glucose oxidation to the utilization of fatty acid-derived ketone bodies, most prominently β-hydroxybutyrate (BHB) [2]. Although BHB is technically one of several ketone bodies, it is the most stable and physiologically abundant during fasting and is therefore the principal biomarker for assessing the degree of nutritional ketosis in both research and clinical settings [3]. This metabolic transition, often termed metabolic switching, typically begins within the first 12 - 24 hours of fasting and deepens over time, promoting mitochondrial efficiency, reducing oxidative stress, and activating autophagic pathways [4]. These cellular-level adaptations have been associated with neuroprotective effects and improved bioenergetic stability in the central nervous system [5].

While prolonged fasting and intermittent fasting have been extensively studied for their effects on metabolic markers, weight regulation, and systemic inflammation [6], less is known about the short-term neurocognitive and electrophysiological consequences of fasting during the initial 48-hour window—the period in which the body transitions fully into a ketone-dominant energy state. Theoretically, the early depletion of hepatic glycogen and reduction in plasma glucose could impair cognitive performance, particularly in tasks requiring rapid information processing [7]. However, emerging evidence suggests that BHB may act as an efficient alternative cerebral fuel, sustaining or even enhancing neural network activity in domains such as executive function, cognitive flexibility, and sustained attention [8].

To investigate these effects in a controlled, time-sensitive manner, we conducted a 48-hour water-only fasting protocol in a healthy adult cohort. For the purposes of methodological clarity, participants were instructed to cease caloric intake one hour prior to baseline testing. Although non-caloric fluids (e.g., water, black coffee, unsweetened tea) were permitted, all caloric beverages and food sources were strictly prohibited until the completion of the 48-hour period. Each participant underwent identical time-of-day testing at baseline, 24 hours, and 48 hours to minimize circadian influence on metabolic, cognitive, and electrophysiological outcomes [9].

This study is unique in its integration of metabolic, cognitive, and cortical electrophysiological measures. Capillary glucose and BHB levels were collected to confirm metabolic switching [10]. The Trail Making Test A (TMTA) and Trail Making Test B (TMTB) were used to assess visual scanning speed, psychomotor processing, executive function, and set-shifting ability [11]. Neural processing speed and cortical activation were measured via WAVi EEG-derived P300 latency and voltage during an auditory oddball paradigm, widely regarded as a robust, noninvasive index of cortical efficiency [12].

Our primary objective was to determine whether the metabolic changes associated with short-term fasting would impair, preserve, or enhance cognitive performance and cortical processing efficiency. We hypothesized that despite significant reductions in glucose and elevations in BHB, both cognitive and electrophysio-

logical measures would remain stable, and that certain executive functions might show improvement during nutritional ketosis. Additionally, exploratory analyses examined whether males and females differed in their magnitude of ketone production, given prior evidence of sex-specific variation in substrate utilization during fasting [13].

2. Method

2.1. Participants

Ten healthy adults (five male, five female; mean age 33 ± 11 years, range 20 - 53 years) were enrolled from a wellness clinic population. Participation was voluntary, with individuals expressing interest in understanding the physiological effects of fasting. The study was conducted as an exploratory pilot investigation rather than a formal clinical trial. The inclusion criteria required absence of metabolic, neurological, or cardiovascular disease and no use of medications or supplements known to influence glucose regulation, ketogenesis, or cognitive function. All participants provided written informed consent.

All participants were free from metabolic disorders, neurological disease, cardiovascular illness, and any condition known to interfere with glucose regulation, ketone production, or cognitive performance. None were taking medications or supplements that could alter the metabolic or neurocognitive variables under study. Recruitment was achieved through local outreach and word-of-mouth referrals. Prior to enrollment, all participants provided informed consent in accordance with the ethical principles set forth in the Declaration of Helsinki.

2.2. Study Design and Fasting Protocol

The study employed a repeated-measures design, with each participant serving as their own control across three time points: baseline, 24 h, and 48 h of continuous fasting [14]. Baseline testing occurred following an overnight fast, with participants instructed to cease caloric intake one hour before arrival to ensure a minimally post-absorptive state. From baseline until the 48 h endpoint, participants abstained from all calories but were permitted water, black coffee, or unsweetened tea; artificial sweeteners and additives were prohibited due to potential effects on insulin and ketone production [15]. Follow-up sessions were scheduled at the same time of day as baseline to minimize circadian variability, and all assessments were conducted in a controlled environment. Fasting compliance was verified via time-stamped glucose and β-hydroxybutyrate (BHB) readings at each visit, with expected biomarker trajectories (falling glucose, rising BHB) confirming adherence.

2.3. Metabolic Measures

Capillary blood glucose and β-hydroxybutyrate (BHB) concentrations were obtained at each time point using a handheld meter [16]. BHB, the primary circulating ketone body, is widely recognized as the most reliable peripheral biomarker of

nutritional ketosis [17]. While the term ketones is commonly used in public discourse, physiologically this refers collectively to BHB, acetoacetate, and acetone. For the purposes of this study, the BHB measurement served as the definitive index of ketone availability to the brain. Blood samples were collected via single-use lancets, with values recorded to the nearest 0.1 mmol/L for BHB and 1 mg/dL for glucose [18]. The same testing device and operator were used for all measurements to maintain methodological consistency.

2.4. Caffeine Intake

Participants were permitted to maintain habitual caffeine intake (black coffee or unsweetened tea). Daily caffeine use was recorded but not standardized across participants. This factor is acknowledged as a limitation of the study design.

2.5. Cognitive Performance Measures

Cognitive performance was evaluated using the Trail Making Test (TMT), a validated neuropsychological instrument sensitive to subtle cognitive changes in healthy individuals [19]. Part A (TMTA) required participants to connect a series of numbers in ascending order as quickly as possible, providing a measure of visual scanning ability, psychomotor processing speed, and sequencing efficiency [20]. Part B (TMTB) incorporated an additional executive demand by requiring alternation between numbers and letters in ascending sequence (e.g., 1-A-2-B), thereby taxing cognitive flexibility, divided attention, and set-shifting capacity [21]. In both tasks, performance was quantified as the time in seconds required to complete the sequence, with shorter times representing superior performance.

2.6. Electroencephalographic Measures

Neural processing speed and cortical activation were assessed via event-related potentials (ERPs) recorded during an auditory oddball paradigm using the WAVi Research Brain Measurement System [22]. This protocol reliably elicits the P300 component, a positive deflection in the ERP waveform occurring approximately 300 milliseconds after the presentation of a target stimulus [23]. The P300 latency reflects the speed of cognitive evaluation, while the amplitude, measured in microvolts, is interpreted as an index of cortical activation and attentional resource allocation [24].

Recordings were obtained with participants seated comfortably, eyes open, and fixated on a visual point to minimize ocular artifacts [25] [26]. Data were band-pass filtered between 0.1 and 30 Hz, and epochs containing movement or electromyographic noise were excluded prior to waveform averaging. Latency values were measured in milliseconds from the onset of the auditory stimulus to the peak of the P300 waveform, and amplitude values were measured from baseline to the peak of the positive deflection.

An auditory oddball paradigm was used to elicit the P300 component. Data were band-pass filtered (0.1 - 30 Hz) and baseline corrected (−200 to 800 ms rel-

ative to stimulus onset). Epochs containing blinks, eye movements, or muscular artifacts were excluded, and at least 30 artifact-free trials were averaged for each participant.

2.7. Statistical Analysis

Statistical analyses were performed using GraphPad Prism (version 10). Because this was an exploratory pilot study with a small sample, analyses focused on within-subject changes across baseline, 24 h, and 48 h. Repeated-measures ANOVA was used for glucose, β-hydroxybutyrate (BHB), Trail Making Test (TMTA and TMTB), and P300 parameters. All values are reported as mean ± SD, with significance set at $p < 0.05$ but interpreted cautiously given the pilot nature of the study.

3. Results

3.1. Metabolic Adaptations

The metabolic data demonstrated a clear and progressive shift from glucose-dominant metabolism toward ketone utilization over the course of the 48-hour fasting period. Mean fasting glucose at baseline was 104.5 ± 10.2 mg/dL, consistent with euglycemia following an overnight fast. By 24 hours, glucose had declined to 87.7 ± 8.6 mg/dL, representing an approximate 16% reduction, and by 48 hours, levels reached 69.2 ± 7.9 mg/dL, marking a total decrease of nearly 34% from baseline. This inverse relationship between glucose and ketones is clearly depicted in Figure 1, illustrating the coordinated metabolic transition toward nutritional ketosis.

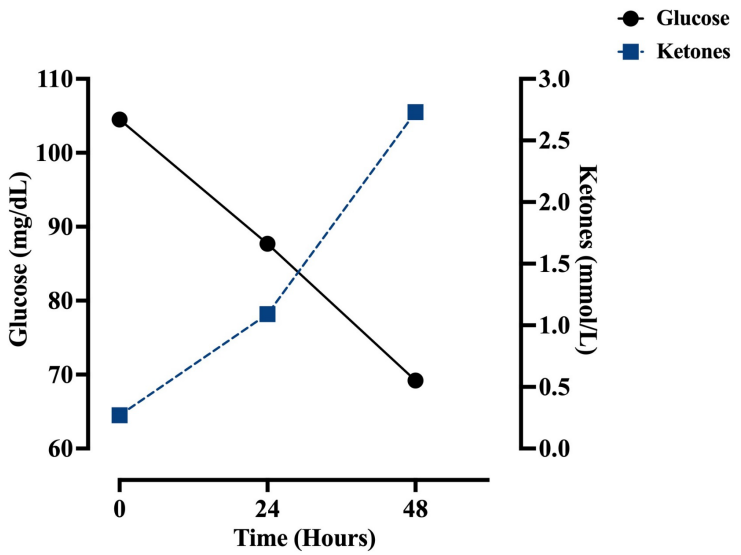

Figure 1. Blood glucose and β-hydroxybutyrate levels at baseline, 24 h, and 48 h.

Concomitantly, β-hydroxybutyrate (BHB) levels rose sharply, reflecting the anticipated metabolic switch to fatty acid oxidation and ketone production [27]. The figure displays baseline BHB values averaged 0.27 ± 0.15 mmol/L, indicative of

minimal ketosis. At 24 hours, concentrations increased to 1.09 ± 0.38 mmol/L, and by 48 hours, levels reached 2.73 ± 0.81 mmol/L, signifying entry into a robust state of nutritional ketosis [28]. This observation aligns with prior literature on sex-based differences in lipid mobilization and ketone production during prolonged fasting, though the present sample size precludes definitive conclusions [29].

3.2. Cognitive Performance: Processing Speed and Executive Flexibility

The Trail Making Test performance revealed a differential pattern across the two tasks, suggesting selective effects of fasting on cognitive domains. TMTA completion time, which primarily reflects processing speed and visual scanning, increased modestly from a baseline mean of 42.1 ± 6.8 seconds to 48.3 ± 7.2 seconds at 24 hours, and further to 50.2 ± 7.5 seconds at 48 hours. As shown in the left panel of Figure 2, this mild slowing, while not reaching statistical significance in this small cohort, may reflect transient adaptation to reduced glucose availability in tasks heavily reliant on psychomotor speed.

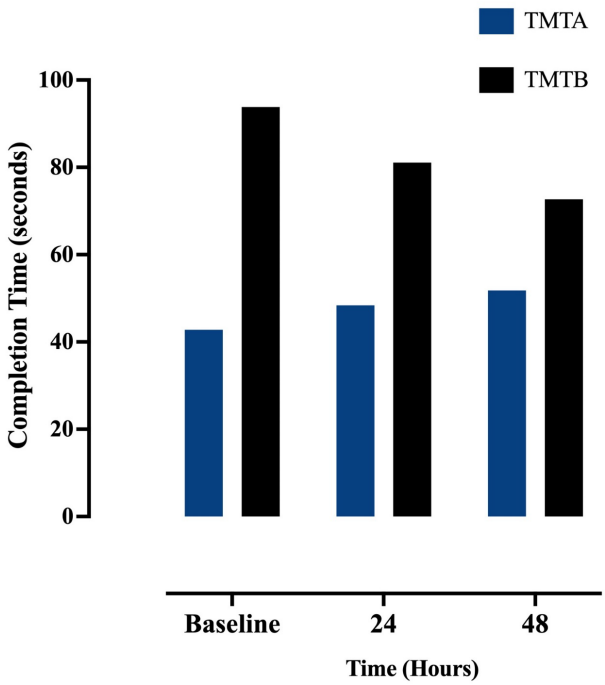

Figure 2. Divergent effects of fasting on psychomotor speed and executive flexibility.

In contrast, TMTB performance improved markedly over the same period. Baseline completion time averaged 95.4 ± 12.7 seconds. As the right panel of the figure shows, at 24 hours, participants completed the task in 82.5 ± 11.9 seconds, and by 48 hours, mean completion time had dropped to 73.2 ± 10.8 seconds, representing a 22% improvement relative to baseline [30]. Given that TMTB perfor-

mance is strongly dependent on cognitive flexibility, working memory, and set-shifting capacity, this enhancement suggests that early nutritional ketosis may selectively support executive function [31].

3.3. Cortical Processing Efficiency Indexed by P300

Electrophysiological markers remained stable across the fasting interval [32]. As shown in the left panel of **Figure 3**, the P300 latency measured 272 ms at baseline, 266 ms at twenty-four hours, and 267 ms at forty-eight hours. Voltage similarly exhibited negligible net change (14.5 μV at baseline, 13.9 μV at twenty-four hours, 14.5 μV at forty-eight hours), which is detailed in the right panel of **Figure 3**. The absence of a systematic latency prolongation argues against any fasting-related slowing of cortical stimulus evaluation, and the preserved voltage suggests stable attentional resource allocation and network engagement [33]. Together with improved TMTB performance, these findings indicate that neural efficiency was maintained, if not functionally optimized for executive control, during early ketosis.

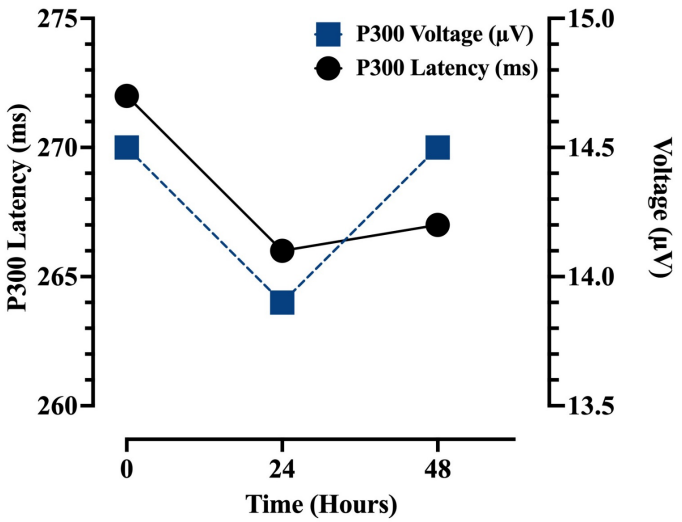

Figure 3. Cortical processing efficiency indexed by P300 during 48 h of fasting.

3.4. Ketone Response by Sex

Given the marked rise in ketones and the suggestion of sex differences at forty-eight hours, BHB trajectories rose separately for males and females [34]. As **Figure 4** notes, males displayed a steeper slope from twenty-four to forty-eight hours, culminating in higher mean ketones at the study endpoint. Despite this metabolic divergence, behavioral and electrophysiological measures did not differ qualitatively by sex in this sample. This pattern implies that a wider physiological range of ketone exposure, at least within the bounds observed here does not necessarily translate to measurable differences in executive performance or P300 indices over forty-eight hours in healthy adults.

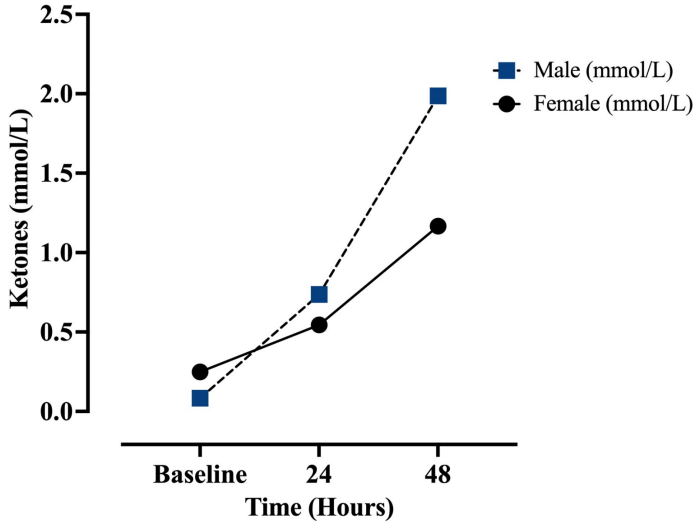

Figure 4. Sex-specific β-hydroxybutyrate (BHB) response across 48 h of fasting.

4. Discussion

The present pilot study examined the interplay between systemic metabolic adaptation, executive function, and cortical processing efficiency during a 48-hour water-only fast in healthy adults. Despite its modest scale, the findings contribute several novel insights into the acute neurocognitive consequences of early nutritional ketosis.

As anticipated, fasting induced a marked metabolic shift characterized by a ~34% reduction in blood glucose and a tenfold rise in β-hydroxybutyrate (BHB). This transition is consistent with the classic progression from glycogen depletion to hepatic ketogenesis [35] [36]. Importantly, our findings support emerging evidence that BHB functions not only as an efficient cerebral substrate but also as a signaling molecule that promotes mitochondrial efficiency, reduces oxidative stress, and enhances neurotrophic pathways [37].

Cognitively, a dissociation emerged between processing speed and executive flexibility. Psychomotor speed (TMTA) slowed modestly, likely reflecting reduced glucose availability, whereas executive set-shifting (TMTB) improved by 22% over 48 hours. This selective enhancement suggests that prefrontal networks supporting higher-order cognition remain robust, and may even be facilitated under conditions of early ketosis. These results extend prior work demonstrating that ketone availability can sustain or enhance higher-order cognition even when glucose is reduced [38]-[40].

Electrophysiological outcomes further underscore this resilience. Both P300 latency and amplitude remained stable, indicating preserved cortical stimulus evaluation speed and attentional resource allocation. Given the sensitivity of P300 latency to neural slowing in pathological states, its stability here is a notable finding. To our knowledge, few studies have directly examined event-related potentials during short-term fasting, and our results provide early evidence that cortical ef-

ficiency is maintained under acute caloric deprivation [41]-[43].

Exploratory sex-stratified analysis revealed higher ketone levels in males relative to females at 48 hours, consistent with prior reports of sex-specific metabolic responses [44]. However, these differences did not correspond to variation in cognitive or electrophysiological measures, suggesting that cortical function is resilient across a physiological range of ketone exposure during short-term fasting.

From a translational standpoint, these data challenge the longstanding assumption that glucose restriction necessarily impairs brain function [45]. Instead, our findings indicate that acute fasting preserves cortical efficiency and may selectively enhance executive flexibility. This has relevance for diverse real-world contexts, including intermittent fasting regimens, endurance sports, and occupational settings where short-term caloric deprivation occurs. By integrating metabolic, cognitive, and electrophysiological markers, this study adds preliminary evidence that early ketosis is a cognitively sustainable state in healthy adults.

5. Conclusions

In summary, this pilot investigation demonstrates that a 48-hour water-only fast induces rapid and pronounced metabolic switching from glucose to ketone utilization without detrimental effects on executive cognition or cortical processing efficiency. In fact, executive flexibility, as indexed by TMTB performance was enhanced, suggesting that ketone availability may selectively support higher-order cognitive processes during acute caloric deprivation [46].

These findings contribute to a growing body of evidence challenging the view that the human brain is strictly dependent on continuous glucose supply for optimal performance. Instead, the data support the concept of metabolic flexibility, wherein the brain readily adapts to alternative substrates without functional compromise [47].

While the small sample size and absence of a control group limit generalizability, the robustness of the metabolic changes and the preservation of neurocognitive function provide a compelling rationale for larger, controlled studies. Future research should explore mechanistic underpinnings of ketone-facilitated executive function, extend fasting durations beyond 48 hours, and assess the role of individual metabolic phenotypes, including sex differences in modulating the neurocognitive response to fasting [32].

In conclusion, early-stage nutritional ketosis appears to be a cognitively sustainable state in healthy adults, with potential applications in both performance optimization and therapeutic contexts. The metabolic resilience and neurocognitive stability demonstrated here highlight fasting as a physiological state worthy of further clinical and translational exploration [48].

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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