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The effect of complete fasting on urinary oxidative stress indicators in middle-aged individuals – preliminary studies

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Abstract:

Background and Study Aim: Fasting as a metabolic intervention is gaining popularity, yet the impact of multi-day food deprivation on redox homeostasis remains understudied. The aim of this case study was knowledge about impact of an 8-day fast on selected urinary markers of oxidative stress and renal function in trained, middle-aged individuals.

Material and Methods: Six healthy, regularly training individuals participated in the study: 4 men and 2 women aged 45–60. Participants underwent an 8-day, supervised fast. Morning urine and blood samples were collected before and immediately after the intervention, and body composition analysis was performed. Protein concentration, total oxidative status (TOS), malondialdehyde (MDA), total antioxidant capacity (TAC), and the activity of key antioxidant enzymes: superoxide dismutase (SOD) and its isoforms (CuZnSOD, MnSOD), catalase (CAT), glutathione peroxidase (GPx), and glutathione S-transferase (GST) were determined. Changes in body weight and composition, as well as serum cortisol (C) and beta-hydroxybutyrate (ß-HB) levels, were also analysed.

Results: Statistically significant increases in urinary protein concentration ($p<0.05$), TOS ($p<0.05$), MDA ($p<0.05$), and CuZnSOD activity ($p<0.05$) were observed. Cohen's d effect size analysis confirmed a large or very large magnitude of these changes (d: 1.54, 2.52, 0.90, and 2.42, respectively). No statistically significant changes were observed in CAT, SOD, MnSOD, GPx, GST activity, or TAC values. The intervention also resulted in significant increases in serum C ($p<0.01$) and ß-HB ($p<0.001$) concentrations.

Conclusions: 8-day fasting in trained middle-aged men and women induces a state of increased oxidative stress and proteinuria, indicating significant metabolic and renal burden. The antioxidant system's response is selective and noncompensatory, suggesting a potential pro-oxidant redox imbalance. These results, although based on a small sample, provide evidence of the complex metabolic effects of long-term fasting and emphasize the need for a cautious approach to its use.

Keywords: proteinuria, redox balance, urine markers,

Dictionary:

Fasting – *noun* the practice of going without food [37].

Oxidative stress – *noun* damage to cells caused by free radicals produced in aerobic metabolisation [37].

Homeostasis – *noun* the process by which the functions and chemistry of a cell or internal organ are kept stable, even when external conditions vary greatly [37].

Protein – *noun* a compound that is an essential part of living cells and is one of the elements in food that is necessary to keep the human body working properly [37].

1. Introduction

Interest in various forms of fasting, from intermittent to long-term periods of complete food deprivation, has grown significantly in recent years, both in the context of popular health trends and serious scientific research [1-3]. They are seen as potential tools in the prevention and treatment of lifestyle diseases, as well as in the pursuit of extending healthy life [4, 5]. However, while short-term fasting and caloric restriction have been the subject of numerous studies [6], extreme interventions, such as multi-day water-only fasting, still pose many unknowns [7]. One of the key areas that can be impacted by such intensive intervention is the body's oxidation-reduction balance [8]. During prolonged fasting, fundamental metabolic changes occur: the body switches to drawing energy from endogenous reserves, primarily adipose tissue, leading to increased production of ketone bodies [9, 10]. These profound adaptations, although evolutionarily justified, can generate increased amounts of reactive oxygen species (ROS) as byproducts of increased metabolic processes. Oxidative stress, resulting from ROS overproduction or weakened antioxidant systems, is a factor of proven importance in the pathogenesis of many diseases and in the aging process [11]. On the other hand, research indicates that oxidative stress also plays a key role in the context of sports performance and recovery [12], which is particularly important in the case of the studied group of people who train regularly. Previous research by our team has indicated that similar metabolic interventions can significantly modify the profile of proinflammatory cytokines, suggesting a broad, systemic impact of fasting on homeostasis [13]. It seems reasonable to hypothesize that an 8-day fast is such a profound metabolic intervention that it leads to redox imbalance and causes measurable changes in kidney function [14, 15].

The aim of this study was knowledge about impact of an 8-day fast on selected urinary markers of oxidative stress and renal function in trained middle-aged individuals.

2. Materials and Methods

Study participants and ethical considerations

4 healthy men and 2 women participated in the study (case study, n = 6). Inclusion criteria were: age 45–60 years, regular physical activity (combat sports, yoga), no chronic diseases, and no ongoing medication. The group was selected based on their high level of discipline and body awareness, which minimized the risk of noncompliance with the research protocol.

Prior to the study, medical screenings were conducted to ensure volunteers did not have chronic illnesses or dependencies on psychostimulant substances. All individuals received comprehensive information regarding the research methodology and potential risks. The study protocol received approval from the Research Ethics Committee.

Experimental protocol

The study was conducted in accordance with the guidelines of the Declaration of Helsinki. All participants were informed of the study objectives, procedure, and potential risks, as well as the right to withdraw from the study at any stage, and then provided informed, written consent.

The research protocol included:

'Before' measurement: the day before the fast, venous blood samples and first morning urine samples were collected in the morning after fasting. Body composition analysis was also performed,

Intervention: participants underwent an 8-day fast, during which they drank only spring water ad libitum. The intervention was conducted under medical supervision,

'After' measurement: on the 9th morning, immediately after the fast, all measurement procedures from step 1 were repeated.

Laboratory and measurement methods

Body composition analysis – Tanita TBF 300A (Amsterdam, the Netherlands): a bioelectrical impedance analyser (BIA) was used to assess body weight, body fat (FAT), lean body mass (FFM), and total body water (TBW).

Urine markers: measurements were performed using the following equipment: a Perkin Elmer LS45 spectrofluorimeter (for MDA), a Shimadzu UV-1700 spectrophotometer (for CAT), and a PerkinElmer VICTOR-X3 reader (for TAC, TOS, GST, GPx, SOD).

Total Antioxidant Capacity (TAC): determined using a method based on the decolourization of the oxidized ABTS radical by antioxidants present in the sample [16]. Absorbance readings were taken at a wavelength of 650 nm.

Total oxidation status (TOS): determined using a method based on the oxidation of iron(II) ions to iron(III) ions in an acidic environment, which forms a coloured complex with xylene orange [17]. Readings were taken at a wavelength of 560 nm.

Malondialdehyde (MDA): the concentration was determined based on its reaction with thiobarbituric acid [18]. Readings were performed using a spectrofluorometer at an excitation wavelength of 515 nm and an emission wavelength of 552 nm, ensuring high specificity.

Superoxide dismutase (SOD) activity: enzyme activity was determined using a method in which superoxide anion reacts with hydroxylamine to form a nitroso ion [19]. SOD activity inhibits this reaction. Readings were taken at a wavelength of 560 nm.

Catalase activity (CAT): determined by a kinetic method, monitoring the absorbance changes resulting from hydrogen peroxide decomposition at a wavelength of 240 nm [20].

Glutathione peroxidase activity (GPx): determined by a method based on the coupled regeneration reaction of oxidized glutathione in the presence of glutathione reductase and NADPH [21, 22]. The kinetics of absorbance changes were monitored at a wavelength of 355 nm.

Glutathione S-transferase activity (GST): determined by a kinetic method, monitoring the absorbance changes at a wavelength of 355 nm [23].

Serum markers: serum cortisol and ß-hydroxybutyrate levels were quantified using the Cortisol-CLIA assay (SNIBE Co., Ltd., Shenzhen, China) and the RANBUD ß-HB kit (Randox Laboratories Ltd., Crumlin, UK), respectively.

Hematocrit: determined using the microhematocrit method. Venous blood was immediately heparinized and filled into capillary hematocrit tubes. Samples were then centrifuged for 5 min at 10,000 × g in a microhematocrit centrifuge (e.g., Microfuge 20R, Beckman Coulter). The hematocrit result was calculated as the ratio of the height of the pelleted red blood cells to the total height of the blood column and expressed as a percentage.

Statistical analysis

Data are presented as arithmetic means and standard deviation (M; SD or ±). The Student's t-test for paired samples or its nonparametric equivalent (Wilcoxon signed-rank test) was used to compare the results before and after fasting, depending on the data distribution. Statistical significance was set at $p<0.05$. To assess the practical significance of the observed changes, Cohen's d effect size was calculated, interpreting the values as small, medium, or large.

3. Results

Urinary oxidative stress markers

Statistically significant, more than three-fold increase in urinary protein concentration was observed ($p<0.05$), representing a very large effect size ($d = 1.54$). A significant increase in the concentration of pro-oxidant markers TOS ($p<0.05$) and MDA ($p<0.05$) was also noted, with very large ($d = 2.52$) and large ($d = 0.90$) effect sizes, respectively. Total antioxidant capacity (TAC) did not change significantly, although analysis of the effect size indicates a moderate downward trend ($d = −0.47$). Analysis of antioxidant enzyme activity (Table 1) revealed a statistically significant, multiple increase in extracellular superoxide dismutase (CuZnSOD) activity ($p<0.05$), with a very large effect size ($d = 2.42$). The activity of the remaining enzymes tested – CAT, SOD, MnSOD, GPx, and GST – did not change statistically significantly. A large effect size was observed for CAT ($d = −0.80$) and a moderate effect size for GST ($d = −0.42$), suggesting that the lack of statistical significance may have resulted from low study power.

Table 1. Concentration of selected oxidative stress markers in urine (Mean and SD)

Indicator	Before fasting	After fasting	Cohen's d (effect sizes)
Protein (mg/L)	98,69 ±38,69	**309,82 ±190,50***	1.54
CAT (IU/L)	2,69 ±1,48	1,77 ±0,66	−0.80
TAC (mmol/L)	2,64 ±0,70	2,30 ±0,74	−0.47
TOS (µmol/L)	25,19 ±4,99	**36,49 ±3,93***	2.52
SOD (NU/mL)	17,00 ±13,57	17,55 ±3,84	0.06
MnSOD (NU/mL)	17,49 ±14,05	13,89 ±3,51	−0.35
CuZnSOD (NU/mL)	0,19 ±0,47	**3,65 ±1,97***	2.42
GPx (IU/L)	133,11 ±119,74	104,99 ±113,24	−0.24
GST (IU/L)	1,18 ±1,48	0,63 ±1,10	−0.42
MDA (µmol/L)	5,28 ±1,21	**6,30 ±1,05***	0.90

*Difference before vs. after fasting; *$p<0.05$

Somatic and biochemical indicators

Analysis of somatic indicators revealed a significant decrease in body mass after the end of the fasting intervention, a reduction in total body fat mass, and a decrease in BMI. Other indicators, including percentage of body fat, FFM, and TBW, did not show significant changes (Table 2). Small to moderate effect sizes were observed for body mass (d = −0.40), BMI (d = −0.46), body fat (d = −0.39), FFM (d = −0.29), and TBW (d = −0.30). Simultaneously, a statistically significant increase in cortisol concentration ($p<0.01$) and a very large, over 15-fold increase in serum ß-hydroxybutyrate concentration ($p<0.001$) were observed, with very large effect sizes (d = 1.41 and d = 15.20, respectively) (Table 3).

Table 2. Selected somatic, physiology and morphology indicators (mean and SD)

Indicator	Before fasting	After fasting	Cohen's d (effect sizes)
Body mass (kg)	77.3 ±16.26	**71.0 ±15.01****	−0.40
BMI (kg/m²)	25.32 ±4.38	**23.45 ±3.65****	−0.46
Fat (kg)	18.53 ±5.67	**16.43 ±5.17***	−0.39
FFM (kg)	58.77 ±15.89	54.39 ±13.38	−0.29
TBW (kg)	56.72 ±2.05	55.42 ±2.12	−0.30
Max HR (bpm)	168.83 ±7.52	167.67 ±9.18	−0.14
Hematocrit (%)	44.83 ±2.32	44.83 ±2.71	0.00

*Difference before vs. after fasting; *p<0.05, **p<0.01

Table 3. Serum cortisol and beta-hydroxybutyrate (ß-HB) concentrati**ons (Mean ±SD)**

Indicator	Before fasting	After fasting	Cohen's d	p-value
Cortisol (ng/ml)	302.937 ±100.655	426.234 ±71.851******	1.419	**0.005**
ß-HB (mmol/l)	0.322 ±0.147	4.881 ±0.402*******	15.205	**0.000**

Difference before vs. after fasting**; ****p<0.01, *******p<0.001

4. Discussion

From a somatic perspective, the intervention led to a statistically significant reduction in body mass, total fat mass (kg), and BMI. These changes are consistent with previous reports showing that prolonged fasting or intermittent energy restriction induces rapid weight loss, primarily through mobilization of adipose tissue stores [24, 25]. The lack of significant change in fat percentage, despite a reduction in absolute fat mass, may reflect parallel decreases in fat-free mass and total body water, observed here as non-significant trends. Similar patterns have been described in therapeutic fasting studies, where reductions in lean tissue and hydration status accompany fat loss [26]. Clinically, even modest BMI reductions can improve metabolic risk profiles, including lipid and glucose regulation [27].

A statistically significant increase in urinary protein concentration (proteinuria) is one of the most concerning findings. This phenomenon raises questions about renal function under conditions of profound metabolic stress. This may be a transient

response to renal stress, resulting, for example, from impaired tubular protein reabsorption or compromised glomerular filtration barrier integrity [28]. Regardless of the mechanism, this signals that prolonged fasting poses a significant challenge to renal function.

The simultaneous increase in markers of oxidative stress – TOS and MDA – supports the hypothesis of increased oxidative processes. Increased TOS indicates a general increase in the concentration of oxidizing substances, while elevated levels of MDA, a product of lipid peroxidation, indicate cell membrane damage. This is a logical consequence of the intense mobilization and oxidation of fatty acids, as confirmed by the very large increase in ß-HB, the main ketone body [29]. This is also confirmed by the significant increase in cortisol, a classic marker of the stress response [30, 31].

The antioxidant system's response to this pro-oxidant state appears to be highly selective and insufficient. Other fasting studies also indicate complex changes in antioxidant systems; for example, fasting during Ramadan can modulate SOD, GPx, and CAT activity [32], and a 5-day fast affects genes related to redox regulation [33]. The only enzyme whose activity significantly increased was CuZnSOD. Its increased presence in urine can be interpreted in two ways: as an attempt at an adaptive stress response, but also as an indicator of the release of this enzyme from cells damaged by the stressor [34]. The lack of significant changes in the activity of other key enzymes (CAT, GPx, GST, MnSOD) is intriguing. However, analysis of Cohen's d effect sizes revealed that medium to large effects were observed for CAT ($d = -0.80$), TAC ($d = -0.47$), and GST ($d = -0.42$), suggesting that the lack of statistical significance may be due to low study power (small sample size) rather than a lack of a true biological effect. This means that the stress-buffering capacity of these systems may actually have been impaired.

This picture – increased oxidative stress coupled with a reduced antioxidant response [35] – suggests that an 8-day fasting may tilt the redox balance toward a pro-oxidant response. Special caution should be exercised, especially in populations with existing metabolic disorders, where fasting may paradoxically exacerbate oxidative stress [36]. It is possible that the activation of defense mechanisms affects only specific pathways or cellular compartments not fully represented by the analysed urinary indicators.

Potential applications of the results

Despite these limitations, the results provide important insights. They indicate that long-term fasting is an intervention with a powerful, yet potentially risky, impact on metabolism and organ function, particularly the kidneys. These data could inform the design of future, larger clinical trials and the development of safety monitoring protocols for individuals considering similar practices.

5. Conclusions

Eight-day fasting in trained middle-aged men leads to a statistically significant increase in oxidative stress markers (TOS, MDA) and the appearance of proteinuria, which indicates significant metabolic and renal burden.

The response of the body's antioxidant system to this stress is selective, not systemic – it manifests itself mainly through an increase in CuZnSOD activity.

The lack of a compensatory increase in total antioxidant capacity (TAC) with a simultaneous increase in pro-oxidant markers suggests a shift in the redox balance towards the pro-oxidant state.

The results of this study, although limited in scope, provide evidence that long-term fasting is a powerful metabolic intervention that should be undertaken with caution and awareness of potential risks.

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Ethics Approval: Approval was granted by the Research Ethics Committee of JDU in Czestochowa, Poland (Reference No. KE-U/9/2024).

Informed Consent Statement: This study used international research ethics guidelines, including the Declaration of Helsinki. All participating have given their informed consent.

Conflicts of Interest: The authors declare no conflicts of interest.

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