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Chronological Changes During 10-Day Intermittent Fasting with Low Energy Intake on High Intensity Aerobic Performance and Lipid Constituents

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***Abstract*—Athletes generally undertaken intermittent fasting via reducing their daily caloric intake for certain period of time. However, such practice may impair high intensity exercise performance mainly due to disturbances in fuel availability. Meanwhile, if such practice was continuous, body will make changes to meet the exercise demand. Hence, this study aimed to examine changes in lipid constituents throughout 10 days (D) of intermittent fasting (IFA) and normal diet (CON) while performing a series of intense exercise. Twenty male collegiate athletes (aged 20.3 ± 2.7 years; 2.8 ± 0.2 L.min-1 VO2peak) were randomly allocated to either IFA group which consumed 1590 ± 79 kcal.day-1 (40% total caloric omission), or CON group with 2672 ± 139 kcal.day-1 (normal intake). Participants performed high-intensity exercise at 90% VO2peak cycling to exhaustion, 2 h after breakfast in the morning on D0, D2, D4, D6, D8, and D10 before blood samples were collected. The high intensity exercise time-to-exhaustion performance declined over the 10 days, however, showed a trend of recovery at the end of the experimental period. Compared to D0, plasma triglycerides decreased on D6-10 (P<0.01) whilst total cholesterol (TC) increased in IFA on D2-10 (P=0.01). LDL increased in IFA on D2-10 (P=0.01) in contrast to reductions in CON on D8-10 (P=0.01), whilst HDL increased in IFA on D6-10 (P=0.01). Loss in body weight was consistent with the changes in lipid constituents. In conclusion, the practice of intermittent fasting must exceed 10 days to ensure that high-intensity performance does not deteriorate although changes in HDL is evidence in the later phase of the intervention period.**

Keywords—ramadan fasting, caloric restriction, aerobic performance, lipids

I. INTRODUCTION

Fasting is a common practice where omission of caloric and fluid ingestion for a specified period in a day which may last for several hours since the first meal in view of achieving health and exercise performance benefits. It is well known fact that acute practice of intermittent fasting impairs exercise performance while chronic practice doesn't seem to affect performance, however, it is less known on the changes that occur during the interphase period. Studies have shown that lipid constituents, can be regulated by modification of macronutrient's compositions of either eucaloric (i.e. high or low carbohydrate to fat ratio) or hypocaloric conditions [1,2]. Apart from diet, prolonged endurance exercise at low to moderate intensity, either acute or chronic, positively improves lipid constituents and its ratios by elevating HDL and these modulations are believed due to the high amount of energy expenditure from the activity performed [3,4]. On the contrary, high intensity exercise which typically brief and exerts small energy expenditure, is shown feeble to any lipid constituent modification [3,5,6]. Therefore, it seems that energy expenditure has more influence on lipid mobilisation compared to exercise intensity. However, question whether series of high intensity exercise during intermittent fasting, which cumulatively exert larger amount of energy expenditure promote similar effects like prolonged exercise, remained unanswered.

Earlier studies suggest that high-intensity exercise is associated with a rapid breakdown of muscle glycogen, which implies that this type of exercise is dependent on carbohydrate availability [7,8]. It is therefore plausible that reducing the caloric intake reduces carbohydrate availability and is thus a limiting factor in sustaining a high-power output. However, in a later study, it is shown that the metabolic rate is not substantially affected by intermittent fasting and caloric deprivation, due to continued oxidative metabolic mechanisms that meet energy need [9]. The study supports the finding of Cahill et al. (1966) which suggests that the rate of carbohydrate utilisation during resting decreases under the fasted state while the energy demand is met by an increased rate of fat oxidation [10]. Acute fasting (1–4 days) is shown to be detrimental to high-intensity exercises [11,12]. On the other hand, prolonged intermittent fasting (10–56 days) reported no change in similar exercise performance [13,14]. However, contrasting findings exist in studies involving 7 days of fasting, where performance is either attenuated [15] or unchanged [16]. It can be deduced that recovery in high-intensity exercise performance involving fasting of more than 7 days could be due to adaptations in fuel utilisation.

Many studies have shown that practice of depriving daily caloric consumption for 3-7 days, modifies plasma lipid constituents via lipolysis where increases in total cholesterol and LDL while HDL and triglyceride were noted unchanged [1,17]. Although it is consistent to say that depriving daily caloric consumption leads to lipid mobilisation in the circulation as early as inception, little is known about how exercise could better metabolise this readily lipids thus increase HDL formation during caloric deprivation. Low to moderate intensity exercise (50-70% of VO_2max) which exerts lipid oxidation, is actually a key factor to promote changes in lipid constituents (i.e. lowering triglycerides, total

cholesterol and LDL whilst increasing HDL) [4,18]. Although it has less well documented, the effect of short term high intensity exercise is feeble on lipid constituents. Works from Lira and colleagues reported that, there were no changes in lipid constituents after an acute supramaximal exercise at approximately 115% VO_2max [5]. However, taking into context of whether the mobilisation of lipid constituent from intermittent fasting practice helps in the formation of HDL under this mode or exercise, is less documented.

The main purpose of this study was to observe the chronological changes throughout 10 days of intermittent fasting to shed some light on the relationship between intermittent fasting, high-intensity exercise performance and fuel adaptation. It was hypothesised that there would be a reduction in high-intensity cycling time-to-exhaustion at the beginning stage of intermittent fasting and that this impairment would become negligible by Day-10 due to adaptation in fuel utilisation. In addition, higher total cholesterol and lipoproteins would be observed at the inception of intermittent fasting, however, with 10 days of frequent high intensity exercise, although at low energy expenditure, plasma HDL would increase higher with intermittent fasting diet compared to normal diet.

II. METHODS

A. Participants

Twenty male (n = 20) participants with BMI ranging between 24 ± 2 $kg.m^{-2}$, aged 20.3 ± 2.7 years, and VO_2peak 2.8 ± 0.2 $L.min^{-1}$ were randomly allocated into the study treatments of either intermittent fasting (IFA) or control (CON). During the period of the study, participants were obliged to stay at a residential college to allow the researcher to closely supervise and control their nutritional intake, physical activity and sleep time. Ethics approval was obtained from the Institutional Ethics Committee and all participants signed the informed consent forms and health screen questionnaires on their first visit.

B. Preliminary measures

The maximal oxygen consumption (VO_2peak) was measured using an incremental exercise test employing the Astrand graded exercise testing cycle ergometer protocol (Monark 928E Pro VO_2 Astrand Testing Bike, Sweden) on their first visit. Briefly, the test began with a workload of 100 W, which increased by 50 W every 2 min until volitional exhaustion. Expired air was assessed using metabolic chart (COSMED, Quark CPET, Italy) and VO_2peak was defined as the highest oxygen uptake obtained during the last 30 sec interval during the test. A week later, on visit 2, all participants were familiarised with the exercise protocol testing.

C. Dietary intervention

On visit 3, which was 2 days after familiarisation, all participants started their baseline experimental session (Day-0). Participants performed the prescribed exercise protocol with blood samples were collected immediately after the exercise. During this visit, participants were provided with normal standardised daily dietary intake of 53% carbohydrate, 19% protein and 28% fat, based from their estimated caloric requirements using Harris-Benedict equation (IFA = 2600 ± 120 $kcal.day^{-1}$; CON = 2670 ± 139 $kcal.day^{-1}$). For the next 10 days following Day-0, participants were exposed to their allocated group treatments of either IFA or CON. For IFA, energy intake was only 60% of participant's estimated caloric requirements (1590 ± 79 $kcal.day^{-1}$), with 40% energy restriction achieved via omission of lunch (12:00), while for CON, the diet was continuation from Day-0. Caloric intake and water (2.30 ± 0.23 $L.day^{-1}$ at 35 $mL.kg^{-1}$ body mass) was provided in four (IFA) or five (CON) meals over the course of the day depending on the experimental condition: (breakfast: 06:00, lunch: 12:00, snack 1: 17:00, dinner: 18.00 and supper: 21:30) as summarised in Table 1.

TABLE I. DAILY MACRONUTRIENT AND ENERGY INTAKE DURING THE EXPERIMENTAL SESSION.

	CHO (g)	**FAT (g)**	**PRO (g)**	**Water (ml)**	**Total energy (kcal)**
Breakfast					
CON	66.1 ± 4.8	10.6 ± 1.1	34.9 ± 2.2	440 ± 28	498 ± 24
IFA	60.8 ± 3.2	9.7 ± 0.6	32.1 ± 1.4	498 ± 24	459 ± 19
Lunch					
CON	99.1 ± 13.8	15.8 ± 1.7	52.4 ± 3.1	660 ± 42	748 ± 36
IFA	0	0	0	0	0
Day-2, -4, -6, -8 and -10					
Snack					
CON	33.2 ± 2.2	5.3 ± 0.2	17.4 ± 1.8	220 ± 14	249 ± 12
IFA	30.5 ± 1.9	4.9 ± 0.2	16.1 ± 1.0	449 ± 28	230 ± 9
Dinner					
CON	99.1 ± 13.8	15.8 ± 1.7	52.4 ± 3.1	660 ± 42	748 ± 36
IFA	81.1 ± 10.0	12.9 ± 1.6	42.8 ± 2.9	897 ± 56	612 ± 19
Supper					
CON	33.2 ± 2.2	5.3 ± 0.2	17.4 ± 1.8	220 ± 14	249 ± 12
IFA	30.5 ± 1.9	4.9 ± 0.2	16.1 ± 1.1	449 ± 28	230 ± 9

The values are shown as mean ± SD, CON, control group (n = 10); IFA, intermittent fasting group (n = 10); CHO, carbohydrate; PRO, protein.

D. High Intensity Exercise Programme (time-to-exhaustion test)

Two hours (2 h) after having a standardised breakfast at around 7.00 am, participant's body weight (InBody370 Body Composition Analyser, Korea) and urine specific gravity (USG) (Atago refractometer PAL 10-S, Japan) were obtained prior to the commencement of the exercise test at 9.00 am. Participants then performed a 2 min warm-up at 30% of $\dot{V}O_2$peak followed by 1 min at 60% of $\dot{V}O_2$peak with a cadence of 70 ± 5 r.min^{-1} on a cycle ergometer (Monark 928E, Sweden) under standard environmental conditions of 24 ± 1°C and 85 ± 1% relative humidity. Immediately following this set of warm-up, the high intensity cycling exercise was executed where the load was increased to a constant workload based on participants 90% of VO_2peak with a cadence of 60 ± 5 r.min^{-1} until exhaustion (when cadenced dropped below 55 r.min^{-1}). Time of the exercise was recorded. Three min (3 min) following completion of exercise, 3 ml of blood sample were withdrawn from participant's median cubital vein. The blood was then transferred into an EDTA vacutainer (BD Vacutainer SST- II Advance) and spun at 3500 rpm and 4°C for 15 min. Plasma glucose (pre and post-test), TG, TC, HDL and LDL were then measured using Colorimetric/Fluorometric Assay Kits (Biovision Inc, USA) whilst ratios of TC/HDL and LDL/HDL were computed based on the lipid constituents data.

E. Statistical Analyses

Data were analysed using SPSS software (version 20; IBM Corp., Armonk, NY, USA). However, first, the Shapiro–Wilk test was carried out to determine the normality of the sample, the result of which showed that all the data were normally distributed. A *t*-test was used to compare each group's body mass and USG at baseline (Day-0). For the analysis of the interaction for each group's body mass, USG, and HIT performances as well as lipid constituents in each experimental session, a mixed factor repeated measures a 2 × 6 (group × time) ANOVA was used to identify the significant changes of each day (Day 2, 4, 6, 8 and -10) compared to the baseline (Day-0). The Bonferroni post-hoc test was used to identify the pairwise difference. All statistical significances were set at $P < 0.05$.

III. Results

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A. Baseline Measurement

The baseline (Day-0) measurement of body mass for IFA: 67.51 ± 3.47 kg; CON: 66.64 ± 6.43 kg; $P = 0.792$] showed no difference between group. There was no difference between groups in the USG as well ($P = 0.253$), which indicates that participants were in a well-hydrated state during the sessions. High intensity exercise performance between group ($P = 0.251$) also showed no significant difference.

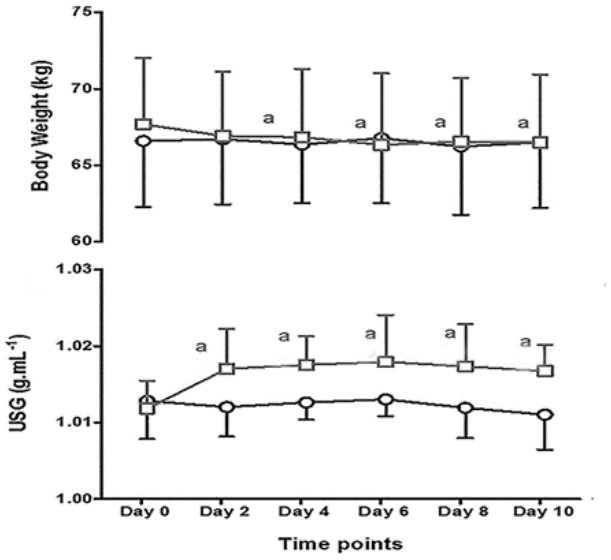

Fig. 1. The top graph indicates body weight changes and the bottom graph shows urine specific gravity changes throughout 10 days of intermittent fasting. The values shown are M ± SD. [a] denotes significant different from Day-0 ($p < 0.05$). The circle (○) represents CON and the square (□) represents IFA.

For body mass over the 10 days, interaction and time effects ($P < 0.001$) were observed. Generally, body mass reduced in the IFA group on Day-4, -6, -8 and -10, compared to the baseline ($P < 0.001$) with an overall percentage reduction of -1.55% (Day-0: 67.51 ± 3.47 kg; Day-10: 66.46 ± 4.31 kg). Body mass in the CON group was unchanged throughout the session ($P > 0.05$). There were time ($P < 0.0001$), group ($P = 0.004$) and main interaction ($P < 0.0001$) effects for time to exhaustion, as shown in Table 2. Cycling time in the IFA group was noted to reduce on Day-2 and throughout the experimental session compared to the baseline. However, there was a trend of recovery during the later phase of this session. During post exercise, the glucose concentration showed a time effect ($P < 0.001$) with no group ($P = 0.651$) or interaction effects ($P = 0.080$). The post-high intensity exercise glucose concentration was higher in the IFA group on Day 4-8 compared to the Day-0 ($P < 0.05$) as shown in Figure 2.

TABLE II. CHANGES IN EXERCISE PERFORMANCES, AFTER EXERCISE.

	Day-0	Day-2	Day-4	Day-6	Day-8	Day-10
High intensity exercise (min)						
CON	10.53 ± 2.23	10.34 ± 2.29	10.35 ± 1.33	11.36 ± 1.43	11.35 ± 1.29	10.38 ± 1.38
IFA	11.14 ± 1.20	7.55 ± 1.20[a]	7.56 ± 2.26[a]	8.27 ± 2.23[a]	9.28 ± 2.30[a]	9.29 ± 2.40[a]

The values shown are mean ± SD, CON; control group, IFA; intermittent fasting group. [a] denotes significant difference from Day-0 value ($p < 0.05$).

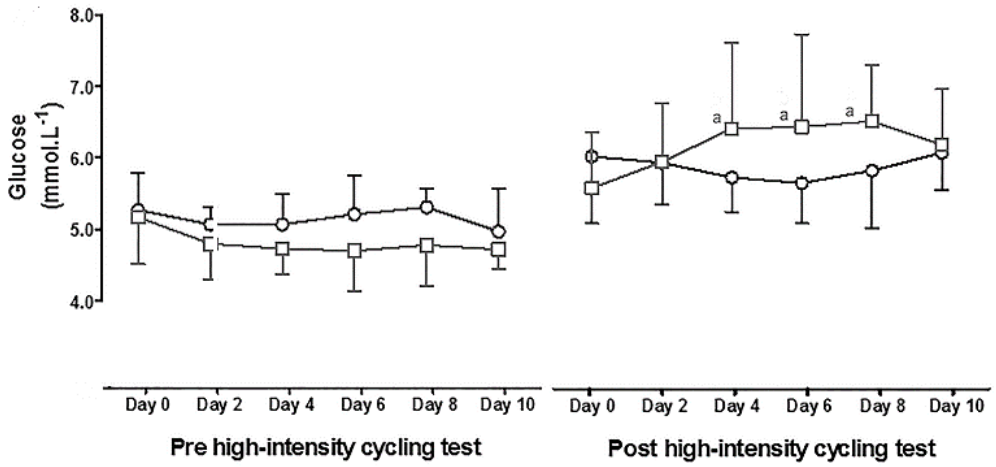

Fig. 2. Glucose levels at pre and post high-intensity exercise. The values shown are M ± SD. [a] denotes significant different from Day-0 ($p < 0.05$). The circle (○) represents CON and the square (□) represents IFA.

As presented in Figure 3, there were time ($P < 0.001$) and treatment ($P = 0.004$) effect in plasma TG, where there were considerable reductions in IFA on Day-6-10 compared to Day-0 ($P < 0.01$). While for plasma LDL, there were time ($P = 0.003$) and treatment ($P = 0.03$) effect where increases in IFA were noted on Day 2-10 compared to Day-0 ($P = 0.01$). In contrast, LDL concentrations in CON were noted reduced on Day 8-10 ($P = 0.01$). Meanwhile for HDL, there was time effect in IFA ($P = 0.002$) where HDL concentrations were elevated on Day 6-10 compared to Day-0 ($P = 0.01$). Although there were no general interaction and group effect in TC, elevations were noted in IFA throughout the experimental period (Day 2-10) compared to Day-0 ($P = 0.01$). For ratios of lipid constituents, within treatment analysis of TC/HDL showed significant increases in IFA on Day 2-10 compared to Day-0 ($P = 0.01$). For LDL/HDL ratio, within treatment analysis showed increments in IFA on Day 2-8 ($P = 0.01$) whilst in CON on the contrary, showed reductions on Day 8-10 ($P = 0.01$).

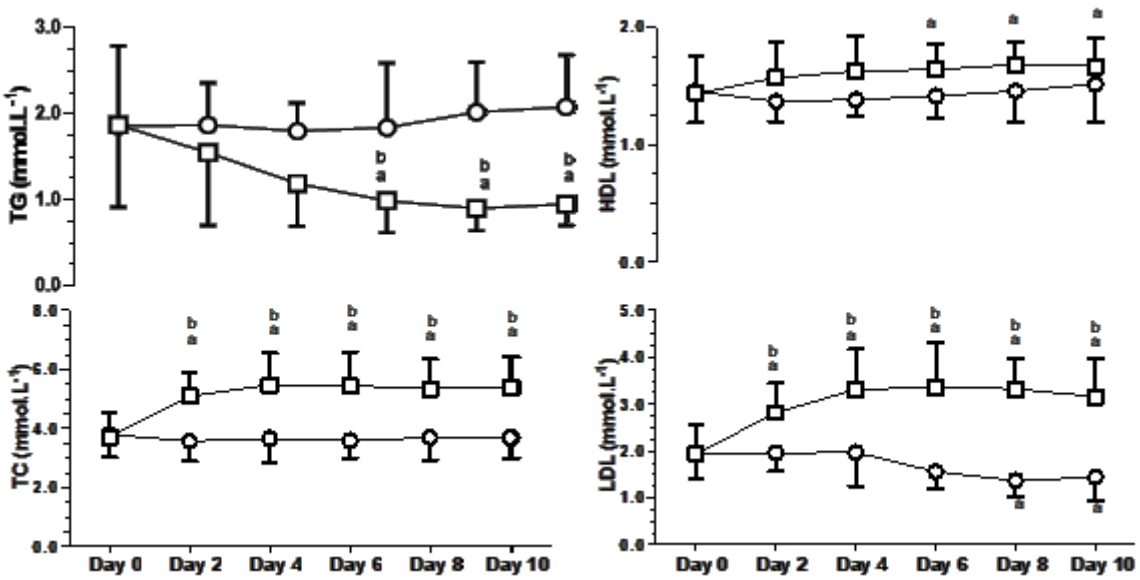

Fig. 3. Values are and presented as means ± SD. Blood samples for triglycerides (TG), total cholesterol (TC), high density lipid (HDL) and low density lipid (LDL) were collected 3 minutes following exercise testing. D0 is the baseline, while D2, D4, D6, D8 and $D10_0$ are the experimental. Intermittent fasting (IFA) represent as □ (*square*), while normal diet (CON) represent as ○ (*circle*). [a] denotes significant difference within group compared to D0 ($P < 0.05$), and [b s]ignificant difference between group compared to CON ($P < 0.05$).

IV. DISCUSSION

In this study there are two major findings: (i) in high-intensity cycling exercise, time-to-exhaustion is reduced throughout the 10-day period, but a trend of recovery is observed towards the end of the experiment, (ii) most lipid constituents showed changes over time from the dietary modifications (i.e 40% caloric cut) and short term high intensity exercise preceded and these changes are distinctive of the IFA and CON treatments. Post-exercise TC and LDL increased during the early phase of the experiment in the IFA treatment, while TG progressively reduced, HDL increased significantly at later phase. Interestingly, LDL/HDL in IFA treatment reduced indicating desirable lipoprotein ratios in both treatments at later phases of the experiment, reflecting the beneficial effect of exercise training on lipid constituents.

In the high-intensity exercise, participants' cycling time-to-exhaustion dropped throughout the 10 days of intermittent fasting. This suggests that depriving energy intake by 40% during intermittent fasting influences exercise performance. Since this mode of exercise is mainly dependent on anaerobic glycolysis and glycogenolysis, the reduction in performance in the IFA group is likely due to a drop in glycogen availability. This will slow down the muscle's energy filling rate during highly glycogen-demanding exercises causing a failure in maintaining the cycle cadence. Under similar caloric conditions, performance has also been reported to be negatively affected by prolonged high-intensity exercise in both acute (1 to 3 days) [19] and longer (7 days) periods of caloric deprivation [15]. However, a progressive trend of improvement in performance throughout the experiment was noted, although it remained lower than at baseline. This, again, could be due to fuel adaptation under intermittent fasting where gluconeogenesis prevents a drop in muscle and hepatic glycogen. Therefore, if a longer period of intermittent fasting were to be carried out, it is likely that the performance would recover. This notion is in line with the study by Ferguson et al. (2009), which showed that, after three weeks of fasting, aerobic performance was not affected due to an improvement in the power to weight ratio at 90–100% of VO_2max [20]. Meanwhile, post-exercise plasma glucose was elevated in both groups to replenish the amount utilised during the high intensity exercise which relies mainly on endogenous carbohydrate stores as a fuel source can produce an increase in plasma glucose within a few minutes. It has also been suggested that during high-intensity exercise, hormones such as catecholamine noticeably increase, causing the production of glucose to rise seven to eightfold while its utilisation is only increased three to fourfold [21].

The progressive reduction in body mass observed in IFA was as predicted and in line with published data [22]. Negative energy balance and exercise demand are the main reasons implicated in the reduction in body mass. As the energy intake is reduced, the utilisation of stored energy in the form of body fats becomes fundamental in sustaining basal metabolic rate. This is seen through the daily energy expenditure and meeting of the energy requirement for the exercise test [23].

In this study, TC showed increment in the IFA treatment throughout the experiment period. As body mass reduction was also observed in IFA, these increases are believed to be associated with fat breakdown and cholesterol mobilization due to the deprived caloric intake treatment given [17]. Fasting and caloric deprivation is commonly accompanied by substantial lipolysis [24], which explains the raised plasma lipids and cholesterol levels released from peripheral cells [18]. Correspondingly, earlier studies also reported an increase in TC as early as 3 and 7 days following subtotal starvation *per se*, among healthy non-obese human participants [1,17]. Likewise, LDL which is a major carrier of cholesterol increased in the IFA treatment. This could be due to the reduction of LDL uptake by the liver which occurred almost instantaneously at the onset of depriving caloric intake [8]. Supported by few earlier studies, deprived caloric consumption is associated with decreased insulin levels which upsurges the LDL receptor-binding [25]. This will then, trigger the LDL receptor gene expression in the liver and lead to LDL in the blood to be observably uplifted. Meanwhile, plasma TG showed changes over time in the IFA treatment, where progressive reductions were observed throughout the experiment period. Although exercise might play an important role in reducing plasma TG [26], in this study, the gradual reduction observed is believed predominantly due to the negative energy balance. As such, negative energy balance has been shown to trigger the release of glucagon and cortisol hormones which promote lipolysis which in turn progressively breaks plasma TG [27].

In present study, raised TC and LDL due to intermittent fasting during the early phase progressively turns preferable at the later phase, where LDL was eventually reduced in each treatment while HDL raised. In line with this finding, a study where participants performed high intensity training at 85% maximum heart rate, and at near anaerobic threshold for 9 to 12 weeks showed increased HDL and HDL2, respectively [27]. On the same note, 32-min high intensity interval running at 90 and 60% VO_2max for 8 weeks showed reductions in TG and VLDL [28]. In contrast, a study by Lira et al. (2010) demonstrated that a single supramaximal exercise (4-min run at 115% of VO_2max) or a 10-min resistant exercise (110% of 1-RM) did not show any change in lipid constituents [5]. These lack in changes could be due to high intensity exercise which usually exert lower energy expenditure therefore lessen the chance for lipid and its constituents to modify [5,29]. As for present study, no profound changes on lipid constituents were observed throughout the early phase. This would probably due to low energy expenditure from the activity where participants only performed short duration high intensity exercise which makes slimmer chance for any lipid constituents to trigger changes. However, lipid constituents' changes observed at the later phase seems to be modulated by series of testing sessions. Therefore, positive changes in TC, LDL and HDL noted at the later phase are believed resulted from cumulative energy expenditure contributed by each of the session performed. In other words, lipid constituent modifications might not solely be dependent on a single exposure to the high intensity exercise, but rather associated

with the amount of energy expenditure or the exercise performed regularly [30].

The finding of this study implies that 10 days of caloric intake deprivation of around 40% during intermittent fasting has detrimental effects on high-intensity time-to-exhaustion, however, a trend of recovery was noted towards the final day. It is speculated that a longer period (in excess of 10 days) might be required to eliminate the effect of fasting. The recuperation in performance, is suggestive of fuel adaptation to meet the exercise demand when participants are under sub-caloric conditions. Metabolically, the observation in performance is explained by the reduction in TG and increases in TC and LDL. In addition, cumulative training effect of high-intensity cycling in this fasted state also shows a favourable response in HDL towards the end of the 10-day period. However, there are few limitations worth consideration as the participant's physical activity level is categorised as active, hence the findings may not be applicable to general male population, furthermore, to athletes who often practice caloric restriction.

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