Detraining Increases Body Fat and Weight and Decreases $\dot{V}O_2$peak and Metabolic Rate

Michael J. Ormsbee[1] and Paul J. Arciero[2]

[1]*Human Performance Laboratory, Department of Nutrition, Food, and Exercise Sciences, The Florida State University, Tallahassee, Florida; and* [2]*Human Performance Laboratory, Health and Exercise Sciences Department, Skidmore College, Saratoga Springs, New York*

Abstract

Ormsbee, MJ and Arciero, PJ. Detraining increases body fat and weight and decreases $\dot{V}O_2$peak and metabolic rate. *J Strength Cond Res* 26(8): 2087–2095, 2012—Competitive collegiate swimmers commonly take a month off from swim training after their last major competition. This abrupt cessation of intense physical training has not been well studied and may lead to physiopsychological decline. The purpose of this investigation was to examine the effects of swim detraining (DT) on body composition, aerobic fitness, resting metabolism, mood state, and blood lipids in collegiate swimmers. Eight healthy endurance-trained swimmers ($\dot{V}O_2$peak, 46.7 ± 10.8 ml·kg^{-1}·min^{-1}) performed 2 identical test days, 1 in the trained (TR) state and 1 in the detrained (~5 weeks) state (DT). Body composition and circumferences, maximal oxygen consumption ($\dot{V}O_2$peak), resting metabolism (RMR), blood lipids, and mood state were measured. After DT, body weight (TR, 68.9 ± 9.7 vs. DT, 69.8 ± 9.8 kg; $p = 0.03$), fat mass (TR, 14.7 ± 7.6 vs. DT, 16.5 ± 7.4 kg; $p = 0.001$), and waist circumference (TR, 72.7± 3.1 vs. DT, 73.8 ± 3.6 cm; $p = 0.03$) increased, whereas $\dot{V}O_2$peak (TR, 46.7 ± 10.8 vs. DT, 43.1 ± 10.3 ml·kg^{-1}·min^{-1}; $p = 0.02$) and RMR (TR, 1.34 ± 0.2 vs. DT, 1.25 ± 0.17 kcal·min^{-1}; $p = 0.008$) decreased, and plasma triglycerides showed a trend to increase ($p = 0.065$). Our data suggest that DT after a competitive collegiate swim season adversely affects body composition, fitness, and metabolism. Athletes and coaches need to be aware of the negative consequences of detraining from swimming, and plan off-season training schedules accordingly to allow for adequate rest/recovery and prevent overuse injuries. It's equally important to mitigate the negative effects on body composition, aerobic fitness and metabolism so performance may continue to improve over the long term.

Key Words swimming, performance, blood lipids, resting metabolic rate, mood state

Address correspondence to Dr. Paul J. Arciero, parciero@skidmore.edu.

26(8)/2087–2095

Journal of Strength and Conditioning Research

Introduction

Competitive swimming is an intense and demanding sport that requires long hours of repetitive movement patterns, sometimes performed twice a day over the course of the season. At the high school and collegiate levels, swim coaches prepare their athletes to achieve peak swim fitness just before the end-of-season competitions to optimize performance. In turn, it is common for these athletes to intentionally take 4–6 weeks off from training after the last major competition in an attempt to promote optimal physiological and psychological recovery and prevent overuse injuries (34). This postseason recovery period is often referred to as the 'transition' phase of an athletes' overall training schedule. The balancing of intense training with strategically placed epochs of "recovery" is known as periodization of sports performance training, and the transition phase is instrumental in promoting continued gains in performance of athletes (18). Indeed, given the intense and repetitive nature of competitive swimming, a month off "out-of-the-water" after the season has ended is often recommended by coaches and trainers. Although this practice of a break from training in swimmers is essential for injury prevention, muscle recovery, and even psychological rejuvenation, it may have deleterious effects on markers of overall health such as body composition, cardiovascular fitness, and metabolism.

It is well documented that exercise training promotes positive adaptations to body composition (3,4), cardiovascular (3,35), and metabolic systems (3,4,9,16,27). Many of these beneficial effects occur after an acute bout of exercise or after a very short-term training period. For example, we have previously shown that body weight and fat mass (FM) decrease and insulin action increases significantly with as little as 10 days of exercise training in obese men and women (6), whereas a single acute bout of endurance exercise significantly increases insulin sensitivity in healthy young men and women (7). Recent evidence supports a maintenance of 'muscle memory' in previously trained muscle fibers that have undergone a period of inactivity with associated disuse atrophy (8). Although this 'muscle memory' allows a relatively rapid regain of strength, once training has resumed (31), the abrupt cessation of physical training

Table 1. Participant characteristics.*†

	Total (n = 8)
Age (y)	19.5 ± 1.0
Weight (kg)	68.8 ± 9.7
Height (cm)	170. ± 5.5
Lean mass (kg)	50.5 ± 11.1
Fat mass (kg)	14.7 ± 7.6
Body fat (%)	22.3 ± 11.5
$\dot{V}O_2$peak ($L \cdot min^{-1}$)	3.24 ± 0.9
$\dot{V}O_2$peak ($ml \cdot kg^{-1} \cdot min^{-1}$)	46.7 ± 10.3

*$\dot{V}O_2$peak = peak oxygen consumption.
†Values are mean ± *SD*.

abolishes any previous muscle strength gains in both older (11,16,17) and younger (16,19) individuals. In addition, we have previously shown that 6–10 days of inactivity are associated with reduced glucose tolerance, insulin action, and GLUT-4 transporter levels (5,36). Others have reported reductions in total aerobic capacity (10,14), deltoid muscle respiratory capacity, and muscle glycogen content compared with levels during peak season training (9). Still others have reported significant increases in body weight (4.8 kg) and body fat (BF; 4.3 kg) after 2 months of detraining (DT) in collegiate female swimmers ($\dot{V}O_2$max, 54.9 ± 5.8 $ml \cdot kg^{-1} \cdot min^{-1}$) (1). However, the full spectrum of potential physical and psychological changes that may occur from relatively brief periods of DT are not well understood in collegiate level swimmers, particularly with regard to body composition, aerobic fitness, metabolism, blood lipids, and mood state. Thus, the purpose of this investigation was to examine the effects of 35–42 days (~5 weeks) of swim DT on body composition, $\dot{V}O_2$peak, resting metabolic rate (RMR), mood state, and fasting lipid levels in collegiate level swimmers. We hypothesized that this period of swim DT, although necessary to help the body recover from the competitive swim season and prevent overuse injury, will result in increased body weight and BF and decreased $\dot{V}O_2$peak and resting metabolism in these collegiate athletes. These deleterious changes may make it harder for these athletes to regain their fitness and negatively impact their long-term health. As such, it may be more advantageous to devise transition phase schedules that not only allow for sufficient rest, recovery, and overuse injury prevention, but this also maintains cardiovascular and metabolic health in these athletes.

Table 2. Body weight and composition in the TR state and after approximately 5 weeks of swim DT.*†

	TR	DT
Weight (kg)	68.9 ± 9.7	69.8 ± 9.8
Exact p value	0.034	
Lean mass (kg)	50.5 ± 11.1	50.5 ± 10.8
Exact p value	0.99	
Body fat (kg)	14.7 ± 7.6	16.5 ± 7.4
Exact p value	<0.001	
Body fat (%)	22.3 ± 11.5	24.3 ± 11.0
Exact p value	<0.001	
Waist circumference (cm)	72.7 ± 3.1	73.8 ± 3.6
Exact p value	0.029	
Waist-hip ratio	0.76 ± 0.04	0.77 ± 0.03
Exact p value	0.10	

*TR = trained; DT = detraining.
†Values are mean ± *SD*.

Methods

Experimental Approach to the Problem

This study was designed to test the hypothesis that approximately 5 weeks (35–42 days) of swim DT (Independent variable) would result in significant decrements to body composition, aerobic fitness, RMR, and fasting blood lipids (dependent variables) in collegiate swimmers. Measurements during this study were conducted during 2 distinct phases: (a) during a habitually trained (TR) state approximately 48 hours after a typical exercise bout (during the week before end-of-season championships, mid-February) and (b) after 35–42 days of DT (just over a month after the season ended, mid-March). The participants were instructed to engage in no-swim training but instead engage in other forms of physical exercise such as light-moderate physical activity (<6.0 METS) and normal activities of daily living for the entirety of the DT period. Some of the activities that the participants engaged in during the DT period included running, cycling, and weight training. In addition, all the participants were instructed to maintain a food and beverage intake pattern similar to that of their TR state to avoid any confounding influence of nutritional intake during the DT period. Compliance was verified via weekly phone, email, and personal contact over the 35- to 42-day DT phase by research personnel. All the measurements were made over 2 consecutive days (visit 1 and visit 2) during both the TR and the DT phases of the study. The subjects were instructed to fast for 12 hours before each test day but stay very well hydrated before arriving at the laboratory. During visit 1 of both the TR and the DT phases, total body mass (TBM), RMR, and body composition were measured between 0600 and 0800 hours. Fasting blood samples were also collected for analysis of glucose and lipid concentrations immediately after the RMR measurement. The participants were instructed to abstain from caffeine, alcohol, and exercise for 24 hours before TR and DT visit 1. The participants were then instructed to arrive back to the laboratory that same day for visit 2 of each phase, at which time $\dot{V}O_2$peak was measured between 1500 and 1700 hours. All

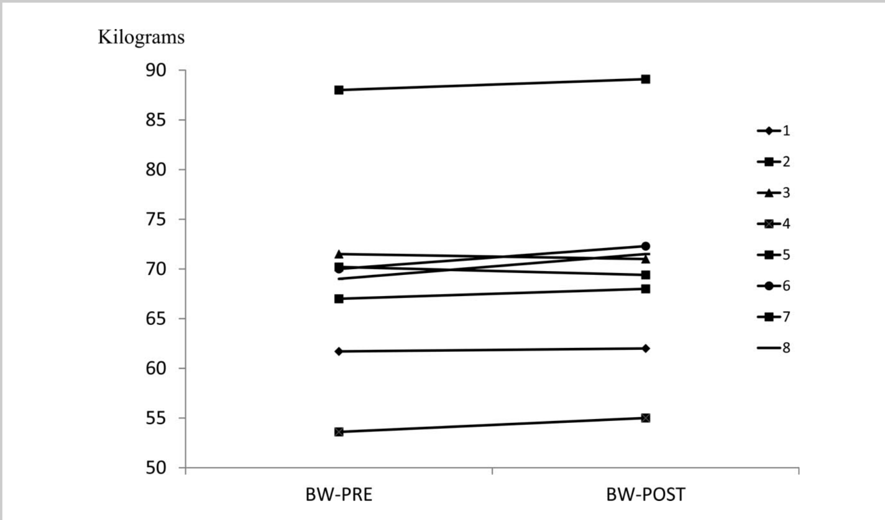

Figure 1. Individual changes in body weight (BW) in the trained (TR) state and after approximately 5 weeks of swim detraining (DT).

the participants were familiarized with all the testing procedures before their first TR visit 1. All the participants were asked to record all food and beverages for 2 days before TR visit 1 testing and then asked to consume the same food and beverages for 2 days before the DT visit 1 to remove the effects of varying nutrient and fluid intake on outcome variables.

Subjects

Eight healthy, Caucasian, Division III collegiate swimmers (4 female: age 19.5 ± 1.2 years, height 165 ± 1.6 cm, mass 64.2 ± 8.3 kg; 4 male: age 19.5 ± 1.0 years, height 174 ± 4.2, mass 73.5 ± 9.7 kg) participated in this study. All the participants were in excellent overall health; had no history of cardiovascular, metabolic, or hormonal disorders; and used no medications other than birth control, antidepressants, or occasional over-the-counter aspirin or ibuprofen. All the participants were members of the Skidmore College varsity swim team and had been training (>10 $h \cdot wk^{-1}$) and competing for 5 months before study participation. All the participants had been involved in competitive swimming for a minimum of 5 years before this study and routinely engaged in high-intensity exercise including swimming and resistance exercise training for the past 7 months. Participant characteristics are given in Table 1. Because of the similar response between male and female participants, the genders were averaged together ($n = 8$) for all the results reported in this study. This study was approved by Skidmore Colleges' human participants Institutional Review Board. All the participants were informed as to the experimental procedures and signed informed consent statements and medical history forms in adherence with the human subjects' guidelines of Skidmore College and with the current national and international laws and regulations governing the use of human subjects before any data collection.

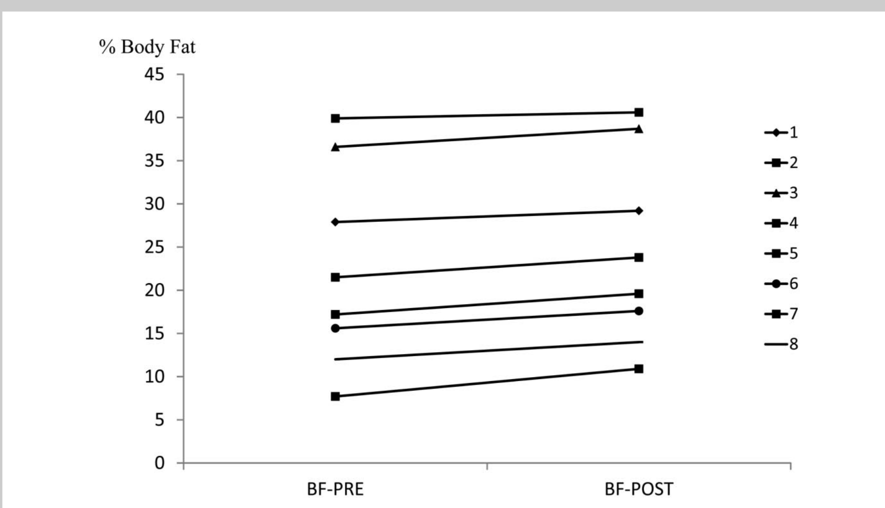

Figure 2. Individual changes in percent body fat (BF) in the trained (TR) state and after approximately 5 weeks of swim detraining (DT).

Procedures

Body Mass and Height. The TBM was measured after an overnight fast and urine void on the morning of visit 1 testing in both the TR and DT states. The participants were clothed in shorts and tee shirt and weighed to the nearest ±0.1 kg on a calibrated balance beam scale. Height was assessed to the nearest 0.10 cm using a sliding vertical scale stadiometer. Waist, hip, thigh, and arm circumferences were also measured using a flexible measuring tape using standardized protocols (12).

Table 3. Peak exercise testing in the TR state and after approximately 5 weeks of swim DT.*†

	TR	DT
$\dot{V}O_2$peak (ml·kg^{-1}·min^{-1})	46.7 ± 10.8	43.1 ± 10.3
Exact *p* value	0.02	
Maximum HR (b·min^{-1})	188 ± 7.3	189 ± 8.5
Exact *p* value	0.27	
Respiratory quotient	1.16 ± 0.1	1.15 ± 0.1
Exact *p* value	0.80	
Perceived exertion	18.6 ± 0.7	18.7 ± 0.9
Exact *p* value	0.79	
Time to exhaustion (min)	12.2 ± 2.7	11.1 ± 2.3
Exact *p* value	0.04	

*TR = trained; DT = detraining; HR = heart rate.
†Values are mean ± *SD*.

Resting Metabolic Rate. The participants were transported to the Human Performance Laboratory immediately upon waking in the morning by a research technician. Upon arrival, height and weight were recorded, and the participants were asked to lie down quietly in a supine position for 15 minutes before the testing began. After the rest period, RMR was measured between 0600 and 0730 AM for 30–45 minutes with a computerized open-circuit indirect calorimeter (VacuMed, Ventura, CA, USA). Briefly, the participants were positioned supine and fitted with a facemask (model 7900; Hans-Rudolph, Kansas City, MO, USA) connected to corrugated tubing that was in turn attached to the metabolic cart. A constant fraction of expired air was withdrawn, dried, and delivered to a zirconium-cell O_2 analyzer (Ametek, Pittsburgh, PA, USA) and an infrared CO_2 analyzer (Ametek). Energy expenditure (kilocalories per minute) was calculated from the equation of Weir (37). The test-retest intraclass correlation (r) for measurement of RMR in our laboratory is 0.90.

Body Composition. Total body composition was determined by dual energy x-ray absorptiometry (DXA; software version 4.1, model DPX; Lunar, Madison, WI, USA) with subjects in the supine position as previously described (3). Total body adiposity is expressed as %BF. The DXA test-retest intraclass correlation (r) and coefficient of variation (CV) for whole-body composition analysis in our laboratory with $n = 12$ is $r = 0.99$, CV = 0.64%, and $r = 0.98$, CV = 2.2%, for fat-free mass (FFM) and FM, respectively.

Blood Lipids. Twelve-hour fasted blood samples were obtained via a finger stick and subsequently analyzed for total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), triglycerides (TRGs), and glucose (GLU) concentrations (milligrams per deciliter) using the Cholestech LDX blood analysis system (Hayward, CA, USA). Measured total blood cholesterol, HDL-C, and TRG values were used to calculate low-density lipoprotein cholesterol (LDL-C) (17). Test-retest intraclass correlation (r) and CV in our laboratory with $n = 15$ is $r = 0.95$, CV = 3.2%, $r = 0.94$, CV = 2.5%, and $r = 0.97$, CV = 5.3% for TC, GLU, and HDL-C (milligrams per deciliter), respectively.

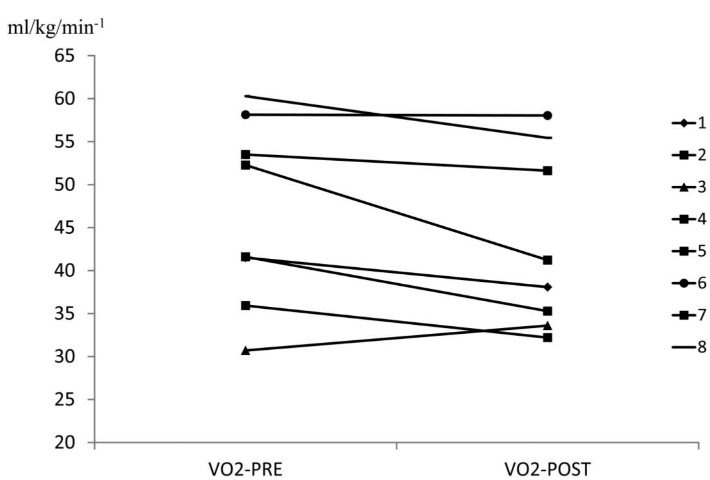

Figure 3. Individual changes in peak oxygen consumption ($\dot{V}O_2$peak) in the trained (TR) state and after approximately 5 weeks of swim detraining (DT).

Peak Oxygen Consumption and Time to Exhaustion. Peak oxygen consumption was determined by a progressive and continuous test to exhaustion on a cycle ergometer (Monark ergometer model 864) in a well-ventilated facility at neutral room temperature (18–20° C). Seat height was adjusted to an optimal height (~15° bend in the knee with leg in the maximal downstroke of the pedal). The same seat height was used for testing $\dot{V}O_2$peak in the both the TR and DT states. The initial work

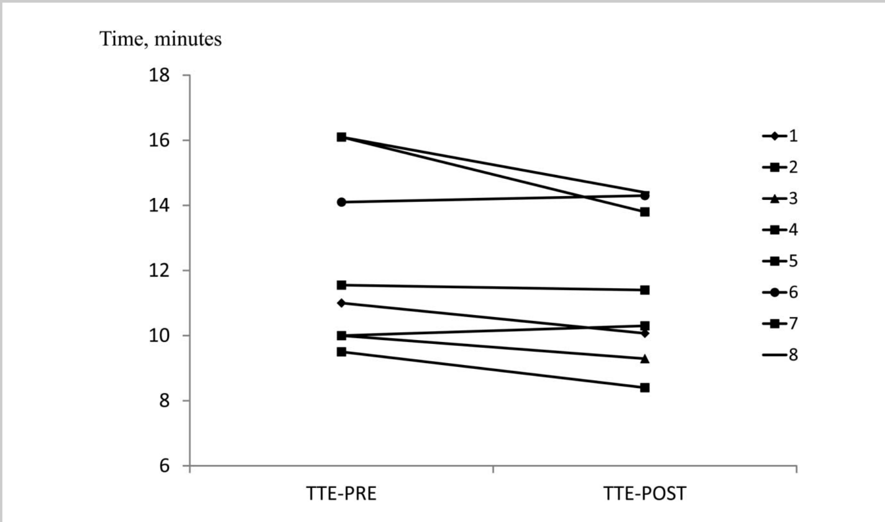

Figure 4. Individual changes in time to exhaustion (TTE) in the trained (TR) state and after approximately 5 weeks of swim detraining (DT).

rate for each subject was set to 50 W for the first 3 minutes and was increased 25 W every 2 minutes until volitional exhaustion or until the participants were unable to maintain 60 rpm. Expired gases were collected throughout the test, and O_2 and CO_2 levels were determined on electrochemical O_2 (Ametek) and infrared CO_2 analyzers (Ametek), respectively. The $\dot{V}O_2$peak was defined as the highest value obtained (60-second average). Heart rate was recorded during testing using a Polar heart rate chest strap and watch (Polar, Port Washington, NY, USA). Perceived exertion was measured using the Borg perceived exertion scale. The total time to exhaustion (TTE) during the $\dot{V}O_2$peak test was also recorded in both the TR and DT states.

Mood State. The profile of mood states (POMS) questionnaire (23), a standardized test that is sensitive to environmental stimuli (2), was administered to each subject in the TR and DT states. The POMS test consists of 65 adjectives that describe a person's mood state, based on a Likert scale ranging from 0 (not at all) to 4 (extremely). When these 65 adjectives are analyzed, 6 factors are derived: tension-anxiety, depression-dejection, anger-hostility, vigor-activity, fatigue-inertia, and confusion-bewilderment.

Statistical Analyses

Data were analyzed using IBM SPSS version 19.0 software (Chicago, IL, USA). All outcome variables were analyzed using a paired t-test to compare TR and DT values. All data are expressed as mean ± *SD*, unless noted otherwise. Statistical significance was determined at $p \leq 0.05$.

Results

Body Weight and Composition

Body weight increased significantly after 35–42 days of swim DT (Table 2; Figure 1). This was entirely because of a 12% increase in body FM and percent ($p < 0.001$), as lean mass was unchanged ($p = 0.99$). In fact, in all 8 subjects, there was an increase in BF (Figure 2). Waist circumference was significantly increased ($p = 0.03$; Table 2) though there were no changes in hip, thigh, or arm circumferences (data not shown).

Peak Oxygen Consumption

There was a significant 7.7% decrease in $\dot{V}O_2$peak during the 35- to 42-day DT period ($p = 0.02$; Table 3; Figure 3). Maximum heart rate ($p = 0.27$), respiratory quotient ($p = 0.8$), and perceived exertion ($p = 0.79$) did not differ between TR and DT. Time to

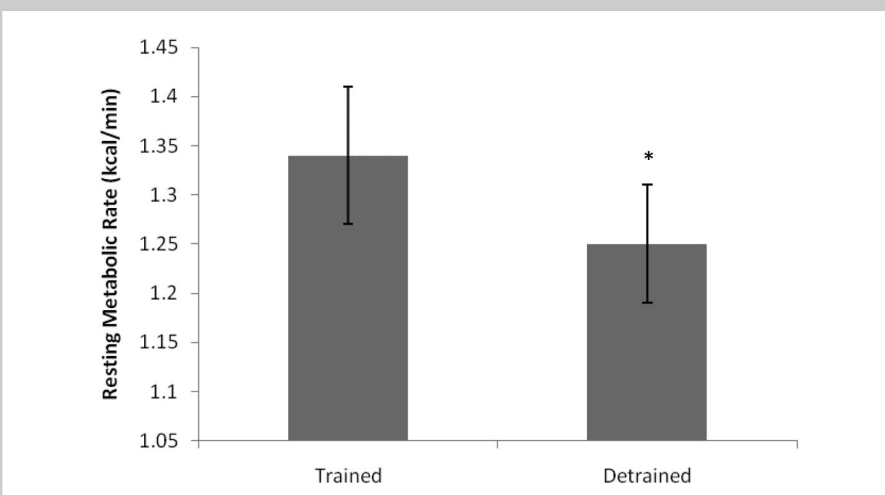

Figure 5. Resting metabolic rate when trained and after approximately 5 weeks of swim detraining. Values are mean ± *SD*. *Significantly different from trained bar, $p < 0.05$.

TABLE 4. Blood lipids in the TR state and after approximately 5 weeks of swim DT.*†

	TR	DT
Total cholesterol ($mg \cdot dl^{-1}$)	176.6 ± 40.4	176.4 ± 60.1
Exact *p* value	0.21	
Triglycerides ($mg \cdot dl^{-1}$)	83 ± 31	92.7 ± 39.7
Exact *p* value	0.065	
HDL-cholesterol ($mg \cdot dl^{-1}$)	52.2 ± 18.7	48.8 ± 9.0
Exact *p* value	0.42	
LDL-cholesterol ($mg \cdot dl^{-1}$)	105.8 ± 45.2	108 ± 53.6
Exact *p* value	0.39	

*TR = trained; DT = detraining; HDL = high-density lipoprotein; LDL = low-density lipoprotein.
†Values are mean ± *SD*.

exhaustion was significantly lower in DT than in TR ($p = 0.04$; Table 3; Figure 4).

Resting Metabolic Rate

The RMR was significantly lower (7%) in the DT compared with that in the TR state ($p = 0.008$; Figure 5).

Blood Lipids

Total cholesterol, HDL-C, and LDL-C remained unchanged, and TRGs showed a trend to increase after DT ($p = 0.06$; Table 4).

Mood State

Psychological mood state remained unchanged after the 5 weeks of DT (Table 5).

TABLE 5. Profile of mood states in the TR state and after approximately 5 weeks of swim DT.*†

	Trained	Detrained
Tension	4.3 ± 2.8	3.6 ± 2.1
Exact *p* value	0.32	
Depression	2.6 ± 5.2	1.0 ± 1.8
Exact *p* value	0.25	
Anger	2.6 ± 4.1	2.4 ± 3.5
Exact *p* value	0.45	
Vigor	7.1 ± 5.6	4.7 ± 3.4
Exact *p* value	0.16	
Fatigue	8.7 ± 4.4	10.3 ± 4.3
Exact *p* value	0.23	
Confusion	4.9 ± 1.7	4.7 ± 2.1
Exact *p* value	0.44	

*TR = trained; DT = detraining.
†Values are mean ± *SD*.

DISCUSSION

The primary goal of the current investigation was to examine the effects of 35–42 days of DT, after a competitive swim season, on body composition, resting metabolism, plasma lipid concentrations, peak oxygen uptake ($\dot{V}O_2$peak), and psychological mood state in collegiate swimmers. The main findings of this study were that 35–42 days of swim DT involving light-moderate physical exercise after a competitive swim season in healthy college-aged men and women resulted in a significant (a) 1.3% increase in body weight; (b) 12.2% increase in BF; (c) 7.7% decrease in peak oxygen consumption; (d) 7% decrease in RMR despite preservation of lean mass; and (e) no change in blood lipids or mood state. The implications from these findings strongly suggest that competitive collegiate swimmers and their coaches should devise transition, off-season periodization training programs that provide adequate rest and recovery from the demanding competitive season but that also allow these athletes to maintain healthy body composition and cardiovascular fitness during this time. Undoubtedly, competitive swimmers need time off after their season to recover and prevent overuse injuries. However, it is equally important that significant DT does not ensue during this off-season period that negatively impacts cardiovascular, metabolic and body composition health, making subsequent performance gains more difficult. Our findings stress the importance of devising specific transition phase training practices that enhance physiopsychological recovery after the intense swim season but that also provide opportunities to maintain overall health and well-being and promote future performance gains.

In only 35–42 days of nonstructured exercise training after a competitive swim season, the average body weight of our swimmers increased from 68.9 ± 9.7 to 69.8 ± 9.8 kg. This was entirely because of a gain in the FM as evidenced by a 2% increase (TR, 22.3 ± 11.5 vs. DT, 24.3 ± 11.0, $p < 0.001$) in %BF (Table 1). To the authors' knowledge, there has only been one other study that followed swimmers after a competitive season and measured changes in body composition. Almeras et al. (1) studied 6 female collegiate swimmers for 2 months while they detrained after an intensive 13-month training season. The fat gain in these women was 4.1 kg, an average fat gain of $0.51\ kg \cdot wk^{-1}$, an amount the authors hypothesized was largely attributable to the swimmers' previous energy cost of training. Our swimmers gained 0.9 kg over 5 weeks, averaging approximately 0.18-kg fat gain per week. This difference is likely because of the length of the in-season training period (13 vs. 5 months) and fitness levels of the swimmers. Almeras et al (1) reported a mean weekly training time of 18.8 $h \cdot wk^{-1}$ and a $\dot{V}O_2max$ of 54.9 $\pm$ 5.8 $ml \cdot kg^{-1} \cdot min^{-1}$, whereas swimmers in this study trained approximately 10 $h \cdot wk^{-1}$ and had a $\dot{V}O_2peak$ of 46.7 $\pm$ 10.8 $ml \cdot kg^{-1} \cdot min^{-1}$. Our results are also in agreement with those of previous work from our laboratory and others documenting significant increases in BF and other deleterious health outcomes after DT from endurance sports (5,16,28,36). In fact, we previously demonstrated that with just 7–10 days of inactivity there are significant decrements in glucose tolerance and RMR (4%) in highly trained endurance athletes (2). This study reported a 7% decrease in RMR. The decrease in RMR, despite maintenance of FFM and an increase in body mass is an interesting finding. As Fukagawa et al. have previously described, FFM is not the sole predictor for RMR (13). In fact, these authors report that alterations to the metabolic activity of FFM may change RMR, particularly as people age, indicating that RMR differences cannot only be attributed to FFM. More recently, Krems et al. (21) agreed with our findings in that differences in RMR between individuals may also be based upon the metabolic rate per unit of tissue mass. It is possible that the cessation of intense exercise training, as in this study, results in a lowering of the metabolic rate per unit of tissue mass and effectively decreases RMR, which may negatively impact body composition. Although we did not measure mitochondrial density changes or sympathetic nervous system activity, it is likely that reductions in these factors may have contributed to the decrease in metabolic rate after DT for 5 weeks as has been reported as a result of aging (13,21).

The rapidity and magnitude of fat gain after cessation of exercise suggests that athletes do not spontaneously reduce caloric intake in response to reduced training. Studies in which short-term energy expenditure is manipulated support this observation. In a recent study in which participants resided for 7 days in a whole-body indirect calorimeter, Stubbs et al. reported that the imposition of sedentary behavior (compared with moderate exercise) does not cause a compensatory decrease in ad libitum energy intake (32). Because the participants failed to decrease energy intake during the sedentary trial, by day 7, they had accrued an energy surplus of approximately 6,200 kcal, resulting in a 0.9-kg weight gain. Using a 2-day whole-body calorimeter protocol, Murgatroyd et al. have also shown that ad libitum food intake does not differ in participants with and without imposed physical activity, despite a caloric expenditure difference of approximately 700 kcals (24).

Because our measurements of body composition were only taken at baseline and follow-up, it is impossible for us to determine if FM steadily increased over the 6-week DT period or, if after an initial period of excess caloric intake, energy intake was reduced to levels similar to energy expenditure. Despite the consistent response among the participants studied in the short term to overeat during periods of reduced physical activity (24,32), it is unlikely that a large difference between energy intake and expenditure continues indefinitely. Although 3 long-term (15–20 years) follow-up studies of former elite athletes report that athletes who stop training have unfavorable body composition changes compared with those that continue training, the data also suggest that the amount of fat gain is similar to that of sedentary controls and is on the order of <0.5 $kg \cdot y^{-1}$ (22,30,33). Taken together, these data suggest that cessation of training may cause a short-term energy surplus and lead to weight gain, specifically FM, though in the long term, differences between energy intake and expenditure become smaller. We were unable to ascertain the types and amounts of foods consumed by the study participants throughout the DT phase that likely led to the unfavorable physiological alterations observed as a result of DT. The lack of a well-controlled nutritional intake measure during both the TR and DT periods is certainly a limitation, and future research in this area must account for the specific role of nutritional intake during a DT protocol. Just by analyzing the metabolic data, however, we estimate that the participants were in a positive energy balance by approximately 5,000 kcal over the 5-week period which would indicate approximately 0.64 kg of weight gain without accounting for any other component of total daily energy expenditure. This estimation is very close to the actual amount of weight (0.9 kg) gained by the participants. A limitation of note is that the menstrual status of our female participants was not accounted for and the ovarian cycle may influence some of our changes in body mass. Nevertheless, the body composition, blood, and performance data were all carefully regulated and physical activity was tightly controlled by the research team. Therefore, the results of this study clearly indicate that legitimate health perturbations occur within a very short period of time, whereas DT and effort should be focused on how to prevent these changes even during the off-season. This is not to say that competitive athletes should be in peak fitness and race-ready shape all year. Instead, our findings highlight the need to stress healthy nutritional intake patterns in combination with time-efficient, low-injury risk exercise regimens to enhance rest and recovery and maintain cardiometabolic and body composition health in competitive athletes during the off-season.

Body circumference measurements suggest that the fat accumulation during DT in our swimmers was predominantly in the abdominal region. Circumference measures of the arms, hips, and thighs were unchanged after DT (p = 0.63, 0.37, 0.13, respectively). In contrast, there was a significant increase in waist circumference ($p = 0.029$; Table 2). Because our swimmers were young and healthy and well within the healthy range of waist-hip ratios, even after DT, this slight increase in waist circumference may not have immediate health ramifications. However, given the association between abdominal fat accumulation and a variety of morbidities (29) athletes should be cognizant of their weight gain during DT and after their time as a competitive athlete.

From an exercise performance standpoint, it is quite logical that 35–42 days of DT would result in a decrement in performance capabilities. Indeed, from our data, we conclude that peak oxygen consumption (−7.7%) and TTE on a cycle ergometer (−9.0%) were significantly reduced after DT. These results agree with those of García-Pallarés et al. who reported a significant 11.3% reduction in maximal aerobic power (15) and a 10.1% decrease in maximal oxygen uptake ($\dot{V}O_2$max) and a significant 3.3% decrease in paddling speed at $\dot{V}O_2$max (14) after 5 weeks of training cessation in world-class kayakers. In general, a significant reduction in $\dot{V}O_2$peak is consistent in the literature after a cessation of exercise training for 3–12 weeks (10,14,16,28,35). This consistent decrement in aerobic performance is likely the reason for the decreased TTE in this study as has been reported previously (35). Given our findings and those reported by others, it is prudent to recommend that coaches and competitive swimmers incorporate off-season training workouts that include low-impact, high-intensity training sessions 1–2× per week to prevent the decline in $\dot{V}O_2$peak. This type of training will significantly reduce training volume to help aid in recovery and prevent overuse injury during the off-season transition phase.

Blood concentrations of TC, TRGs, HDL, and LDL in this study were unaffected by 35–42 days of DT. Alternatively, Giada et al. reported significant increases in TRGs, very low–density lipoproteins, and the LDL/HDL ratio with concurrent decreases in HDL cholesterol in both older and younger adult male cyclists after 8 weeks DT (16). These unfavorable alterations to the blood lipid profile have been reported by others in endurance athletes after a prolonged (52 weeks) DT (28). Taken together, these findings suggest an increase in the atherogenic profile in the detrained athlete in a relatively short period of time. Indeed, this study may not have had a long-enough cessation from training for the full blood lipid profile changes to take place.

Mood state was not affected by 35–42 days of DT (Table 5). This result was surprising because an increase in the volume of swim training (26) and other types of exercise (25) have been demonstrated to improve overall mood state. In addition, Koutedakis et al. (20) evaluated the psychological mood state of underperforming Olympic athletes after allowing for 3–5 weeks of physical rest and reported significant reductions in fatigue and an increase in vigor. Although we cannot explain why our mood results differ from those of others, it is possible that mood was unaffected because our athletes were likely not overtrained to begin with (~10 hours of swim training per week), therefore minimizing any decrease in fatigue, increase in vigor, or change to overall mood profile score that may have been expected. Another plausible explanation for the lack of change in mood state may have been because of timing of when the tests were administered. The TR state occurred at the end of the competitive swim season and in close proximity to the start of the spring academic semester, whereas the DT testing occurred during the midsemester examination period. The lack of difference in mood states may simply be a function of the similar influence that both increased physiological stress (TR state) and increased mental stress (DT state) have on psychological mood state.

In summary, 5 weeks of swim DT after a competitive swim season in healthy young collegiate athletes significantly increases body weight, BF, and waist circumference and decreases aerobic fitness and resting metabolism. Although an off-season is a useful and often beneficial time period for athletes to rejuvenate and recover from a strenuous season, many may experience deleterious physiological changes that may affect swim performance and overall health in the long term. Thus, it is important that coaches and athletes are cognizant of these pitfalls and incorporate nutritional and exercise training practices that are effective at maintaining cardiovascular, metabolic, and body composition health during the transition phase of the periodization schedule.

Practical Applications

Detraining for 35–42 days after a competitive swim season is not recommended for athletes. Athletes and coaches need to be aware of the rapid onset of negative consequences to DT and plan off-season training and nutritional practices accordingly so as not to suffer health and performance decrements. It seems logical to incorporate an appropriate off-season rest period and "active" rest periodization. However, specific guidelines regarding exercise frequency, intensity, and duration along with healthy nutritional strategies need to be followed by these athletes to maximize recovery, prevent overuse injuries, and maintain cardiovascular fitness and metabolic and body composition health during the transition period. These strategies may include performing low-volume/impact, high-intensity interval training 1–2× per week and frequent consumption of protein-rich, nutrient-dense foods, which have been shown to enhance cardiovascular fitness and body composition and metabolic health.

Acknowledgments

The authors would like to thank the members of the Skidmore College men's and women's varsity swim team for their dedication to the DT protocol. The authors are also grateful to Sara Sloper and Karissa Ercolani for their hard work with the subject recruitment, testing, and data analysis. The results of this study do not constitute endorsement by the authors or the National Strength and Conditioning Association.

References

1. Almeras, N, Lemieux, S, Bouchard, C, and Tremblay, A. Fat gain in female swimmers. *Physiol Behav* 61: 811–817, 1997.
2. Arciero, PJ, Gardner, AW, Benowitz, NL, and Poehlman, ET. Relationship of blood pressure, heart rate and behavioral mood state to norepinephrine kinetics in younger and older men following caffeine ingestion. *Eur J Clin Nutr* 52: 805–812, 1998.

3. Arciero, PJ, Gentile, CL, Martin-Pressman, R, Ormsbee, MJ, Everett, M, Zwicky, L, and Steele, CA. Increased dietary protein and combined high-intensity aerobic and resistance exercise improves body fat distribution and cardiovascular risk factors. *Int J Sport Nutr Exerc Metab* 16: 373–392, 2006.
4. Arciero, PJ, Gentile, CL, Pressman, R, Everett, M, Ormsbee, MJ, Martin, J, Santamore, J, Gorman, L, Fehling, PC, Vukovich, MD, and Nindl, BC. Moderate protein intake improves total and regional body composition and insulin sensitivity in overweight adults. *Metabolism* 57: 757–765, 2008.
5. Arciero, PJ, Smith, DL, and Calles-Escandon, J. Effects of short-term inactivity on glucose tolerance, energy expenditure, and blood flow in trained subjects. *J Appl Physiol* 84: 1365–1373, 1998.
6. Arciero, PJ, Vukovich, MD, Holloszy, JO, Racette, SB, and Kohrt, WM. Comparison of short-term diet and exercise on insulin action in individuals with abnormal glucose tolerance. *J Appl Physiol* 86: 1930–1935, 1999.
7. Brestoff, JR, Clippinger, B, Spinella, T, von Duvillard, SP, Nindl, BC, and Arciero, PJ. An acute bout of endurance exercise but not sprint interval exercise enhances insulin sensitivity. *Appl Physiol Nutr Metab* 34: 25–32, 2009.
8. Bruusgaard, JC, Johansen, IB, Egner, IM, Rana, ZA, and Gundersen, K. Myonuclei acquired by overload exercise precede hypertrophy and are not lost on detraining. *Proc Natl Acad Sci U.S.A* 107: 15111–15116, 2010.
9. Costill, DL, Fink, WJ, Hargreaves, M, King, DS, Thomas, R, and Fielding, R. Metabolic characteristics of skeletal muscle during detraining from competitive swimming. Les caracteristiques metaboliques du muscle squelettique lors du desentrainement de la natation de competition. *Med Sci Sports Exerc* 17: 339–343, 1985.
10. Coyle, EF, Martin, WH, Sinacore, DR, Joyner, MJ, Hagberg, JM, and Holloszy, JO. Time course of loss of adaptations after stopping prolonged intense endurance exercise. *J Appl Physiol* 57: 1857–1864, 1984.
11. Fatouros, IG, Kambas, A, Katrabasas, I, Nikolaidis, K, Chatzinikolaou, A, Leontsini, D, and Taxildaris, K. Strength training and detraining effects on muscular strength, anaerobic power, and mobility of inactive older men are intensity dependent. *Br J Sports Med* 39: 776–780, 2005.
12. Franklin, BA, Whaley, MH, and Howley, ET. ACSM's guidelines for exercise testing and prescription (8th ed.). Baltimore, MD: In: *Anonymous American College of Sports Medicine*, 2010.
13. Fukagawa, NK, Bandini, LG, and Young, JB. Effect of age on body composition and resting metabolic rate. *Am J Physiol* 259: E233–E238, 1990.
14. García-Pallarés, J, Carrasco, L, Diaz, A, and Sánchez-Medina, L. Post-season detraining effects on physiological and performance parameters in top-level kayakers: Comparison of two recovery strategies. *J Sport Sci Med* 8: 622–628, 2009.
15. García-Pallarés, J, Sánchez-Medina, L, Pérez, CE, Izquierdo-Gabarren, M, and Izquierdo, M. Physiological effects of tapering and detraining in world-class kayakers. *Med Sci Sports Exerc* 42: 1209–1214, 2010.
16. Giada, F, Vigna, GB, Vitale, E, Baldo-Enzi, G, Bertaglia, M, Crecca, R, and Fellin, R. Effect of age on the response of blood lipids, body composition, and aerobic power to physical conditioning and deconditioning. *Metabolism* 44: 161–165, 1995.
17. Harris, C, DeBeliso, M, Adams, KJ, Irmischer, BS, and Gibson, TAS. Detraining in the older adult: Effects of prior training intensity on strength retention. *J Strength Cond Res* 21: 813–818, 2007.
18. Issurin, VB. New horizons for the methodology and physiology of training periodization. *Sports Med* 40: 189–206, 2010.
19. Izquierdo, M, Ibañez, J, González-Badillo, JJ, Ratamess, NA, Kraemer, WJ, Häkkinen, K, Bonnabau, H, Granados, C, French, DN, and Gorostiaga, EM. Detraining and tapering effects on hormonal responses and strength performance. *J Strength Cond Res* 21: 768–775, 2007.
20. Koutedakis, Y, Budgett, R, and Faulmann, L. Rest in under-performing elite competitors. *Br J Sports Med* 24: 248–252, 1990.
21. Krems, C, Luhrmann, PM, Strassburg, A, Hartmann, B, and Neuhauser-Berthold, M. Lower resting metabolic rate in the elderly may not be entirely due to changes in body composition. *Eur J Clin Nutr* 59: 255–262, 2005.
22. Marti, B, Knobloch, M, Riesen, WF, and Howald, H. Fifteen-year changes in exercise, aerobic power, abdominal fat, and serum lipids in runners and controls. *Med Sci Sports Exerc* 23: 115–122, 1991.
23. McNair, DM, Lorr, M, and Droppleman, LF. *Manual for the Profile of Mood States.* San Diego, CA: Educational and Industrial Testing Services, 1971.
24. Murgatroyd, PR, Goldberg, GR, Leahy, FE, Gilsenan, MB, and Prentice, AM. Effects of inactivity and diet composition on human energy balance. *Int J Obes Relat Metab Disord* 23: 1269–1275, 1999.
25. O'Connor, PJ, Herring, MP, and Caravalho, A. Mental health benefits of strength training in adults. *Am J Lifestyle Med* 4: 1–20, 2010.
26. O'Connor, PJ, Morgan, WP, and Raglin, JS. Psychobiologic effects of 3 d of increased training in female and male swimmers. *Med Sci Sports Exerc* 23: 1055–1061, 1991.
27. Ormsbee, MJ, Thyfault, JP, Johnson, EA, Kraus, RM, Myung, DC, and Hickner, RC. Fat metabolism and acute resistance exercise in trained men. *J Appl Physiol* 102: 1767–1772, 2007.
28. Petibois, C, Cassaigne, A, Gin, H, and Deleris, G. Lipid profile disorders induced by long-term cessation of physical activity in previously highly endurance-trained subjects. *J Clin Endocrinol Metab* 89: 3377–3384, 2004.
29. Pi-Sunyer, FX. The epidemiology of central fat distribution in relation to disease. *Nutr Rev* 62: S120–S126, 2004.
30. Pollock, ML, Mengelkoch, LJ, Graves, JE, Lowenthal, DT, Limacher, MC, Foster, C, and Wilmore, JH. Twenty-year follow-up of aerobic power and body composition of older track athletes. *J Appl Physiol* 82: 1508–1516, 1997.
31. Staron, RS, Leonardi, MJ, Karapondo, DL, Malicky, ES, Falkel, JE, Hagerman, FC, and Hikida, RS. Strength and skeletal muscle adaptations in heavy-resistance-trained women after detraining and retraining. *J Appl Physiol* 70: 631–640, 1991.
32. Stubbs, RJ, Hughes, DA, Johnstone, AM, Horgan, GW, King, N, and Blundell, JE. A decrease in physical activity affects appetite, energy, and nutrient balance in lean men feeding ad libitum. *Am J Clin Nutr* 79: 62–69, 2004.
33. Trappe, SW, Costill, DL, Vukovich, MD, Jones, J, and Melham, T. Aging among elite distance runners: A 22-yr longitudinal study. *J Appl Physiol* 80: 285–290, 1996.
34. Vetter, RE and Symonds, ML. Correlations between injury, training intensity, and physical and mental exhaustion among college athletes. *J Strength Cond Res* 24: 587–596, 2010.
35. Volaklis, KA, Douda, HT, Kokkinos, PF, and Tokmakidis, SP. Physiological alterations to detraining following prolonged combined strength and aerobic training in cardiac patients. *Eur J Cardiovasc Prev Rehabil* 13: 375–380, 2006.
36. Vukovich, MD, Arciero, PJ, Kohrt, WM, Racette, SB, Hansen, PA, and Holloszy, JO. Changes in insulin action and GLUT-4 with 6 days of inactivity in endurance runners. *J Appl Physiol* 80: 240–244, 1996.
37. Weir, JB. New methods for calculating metabolic rate with special reference to protein metabolism. *J Physiol* 109: 1–9, 1949.