International Journal of Sports Physiology and Performance, 2013, 8, 582-592
© 2013 Human Kinetics, Inc.

INTERNATIONAL JOURNAL OF
SPORTS PHYSIOLOGY
AND PERFORMANCE
www.IJSPP-Journal.com
CASE STUDY

Natural Bodybuilding Competition Preparation and Recovery: A 12-Month Case Study

Lindy M. Rossow, David H. Fukuda, Christopher A. Fahs, Jeremy P. Loenneke, and Jeffrey R. Stout

Bodybuilding is a sport in which competitors are judged on muscular appearance. This case study tracked a drug-free male bodybuilder (age 26–27 y) for the 6 mo before and after a competition. ***Purpose:*** The aim of this study was to provide the most comprehensive physiological profile of bodybuilding competition preparation and recovery ever compiled. ***Methods:*** Cardiovascular parameters, body composition, strength, aerobic capacity, critical power, mood state, resting energy expenditure, and hormonal and other blood parameters were evaluated. ***Results:*** Heart rate decreased from 53 to 27 beats/min during preparation and increased to 46 beats/min within 1 mo after competition. Brachial blood pressure dropped from 132/69 to 104/56 mmHg during preparation and returned to 116/64 mmHg at 6 mo after competition. Percent body fat declined from 14.8% to 4.5% during preparation and returned to 14.6% during recovery. Strength decreased during preparation and did not fully recover during 6 months of recovery. Testosterone declined from 9.22 to 2.27 ng/mL during preparation and returned back to the baseline level, 9.91 ng/mL, after competition. Total mood disturbance increased from 6 to 43 units during preparation and recovered to 4 units 6 mo after competition. ***Conclusions:*** This case study provides a thorough documentation of the physiological changes that occurred during natural bodybuilding competition and recovery.

Keywords: bodybuilders, contest preparation, dieting, energy restriction, weight lifting, strength training, resistance training

Bodybuilding is a sport in which competitors are judged on muscular appearance. Natural bodybuilders are drug-tested and are banned from the sport if caught using illegal substances. Appropriate preparation for a natural bodybuilding contest generally involves years of strength training followed by a "contest prep" in which the athlete focuses on dramatically reducing body fat to enhance muscular appearance. Thus, changes seen during competition preparation are not due to sudden dramatic elevations in volume, intensity, or frequency of resistance training but, rather, to a self-induced reduction in energy intake and increase in aerobic activity.[1] While other sports may involve short-term (eg, 7–21 d) weight-cutting strategies before competition, bodybuilding is unique in that prolonged (12+ wk) caloric restriction and increases in physical activity with physique-oriented goals are placed above fitness and physical-performance goals.

Previous research on bodybuilders has mostly focused on the nutritional and body-compositional changes of the athletes.[2–5] A few studies have examined other aspects of contest prep such as hormonal changes[6,7] and strength changes.[8] They provide valuable contributions to the bodybuilding literature, but much speculation and misinformation still exists. Most currently published case studies on bodybuilders (excepting the work by Steen[5]) focus on the well-known negative effects of anabolic steroid use or oil injections, creating an image of all bodybuilders as diseased, obsessed, steroid-injecting individuals.[9–14] We believe there is more to bodybuilding than these profiles suggest.

In addition to the physical changes accompanying bodybuilding preparation, little is known about how such a rigorous regimen may affect mood states. Changes in mood states have been observed after short-term intense exercise training,[15] but the effects of long-term bodybuilding preparation on mood states have not been characterized.

A comprehensive profile of the physiological changes that occur during bodybuilding competition preparation has yet to be compiled. Also unknown are the changes that occur during recovery from competition preparation. To fill this void, we chose to use a battery of assessments on a single subject over an entire year. The

Rossow and Fahs are with the Exercise and Sports Science Dept, Fitchburg State University, Fitchburg, MA. Fukuda and Stout are with the Sport and Exercise Science Program, University of Central Florida, Orlando, FL. Loenneke is with the Dept of Health and Exercise Science, University of Oklahoma, Norman, OK.

purpose of our study was to provide a comprehensive profile of a young, male, natural bodybuilder during 6 months of competition preparation and 6 months after competition.

Methods

Subject and Design

The subject was a young (26–27 y), male, white, natural, professional bodybuilder. The status of professional was awarded 2 years before this competition when the subject won an amateur competition. This was a case study. This study was approved by the University of Oklahoma institutional review board (protocol #13315). The subject was informed of all aspects of the study and signed an informed-consent form.

Diet and Exercise

The subject tracked his diet and exercise training throughout the study. During the preparation period he was under the guidance of a contest-preparation coach who monitored his progress and provided nutritional advice. As an experienced bodybuilding competitor, the subject was adept at weighing and measuring his food. During competition preparation, he was very diligent about tracking his diet. In the 6 months after the competition, he was less motivated to track his diet, so there are gaps in the data during this time frame. The subject also tracked his own exercise training. The only supplement he consumed was 5g/d of creatine monohydrate for the duration of the study. Whey protein was also consumed; this quantity was considered part of his daily protein intake.

Laboratory Assessments

All nonexercise laboratory assessments were performed in the same visit, in the same order (as listed herein), and at the same time of day (beginning at ~6 AM). The subject was overnight fasted for all blood draws and cardiovascular and body-composition assessments except for during ultrasound assessments. As the measurements together took several hours to perform and the subject was in a chronically calorically deprived state, it was deemed necessary to allow him to consume a meal before the ultrasound measurements. The Profile of Mood States (POMS) questionnaire was administered after the ultrasound assessments (and the meal) to minimize the acute effect of fasting on mood. This sequence of events was the same for every assessment.

Cardiovascular Measures. All cardiovascular measurements were taken with the subject supine. The first measurement, brachial blood pressure, was taken after 10 minutes of quiet rest. After this, the other cardiovascular measurements were taken in the order in which they are described.

Brachial systolic and diastolic blood pressure (BP) were measured with an automatic BP-measuring device (Omron Healthcare Inc, Vernon Hills, IL). Two BP measurements were taken, and if these measurements were within 5 mmHg of each other the average of the measurements was reported. If the first 2 brachial systolic blood pressure measurements were not within 5 mmHg, the BP measurement was repeated.

Pulse-wave velocities (PWVs) were measured with a high-fidelity strain-gauge transducer (Millar Instruments, Houston, TX) that obtained pressure waveforms from the right common carotid artery and right femoral artery (aortic PWV), from the right femoral artery and the posterior tibial artery (femoral PWV), and from the right common carotid artery and the radial artery (brachial PWV). PWV was calculated using specialized software (SphygmoCor, AtCor Medical, Sydney, Australia) from the distances between measurement points and the measured time delay between proximal- and distal-foot waveforms as previously described.[16]

Pulse-wave analysis was also performed. Pressure waveforms were obtained from the right radial artery using a high-fidelity strain-gauge transducer (Millar Instruments, Houston, TX). Aortic BP waveforms were derived from radial BP waveforms using a generalized validated transfer function (SphygmoCor, AtCor Medical, Sydney, Australia). Heart rate was also obtained during this measurement.

Calf and forearm blood flow were measured on the right side using strain-gauge plethysmography (EC-6, Hokanson, Bellevue, WA) as described previously.[17]

Cardiac output, stroke volume (SV), end diastolic volume (EDV), end systolic volume (ESV), and ejection fraction were assessed by 2-dimensional echocardiography using a Fukuda Denshi UF-750XT (Tokyo, Japan) ultrasound unit. Measurements were obtained in the parasternal long-axis view. The Teicholz method was used for volume determination. SV was calculated as EDV – ESV. Cardiac output was calculated as heart rate multiplied by SV. Three beats were measured and the average of the measurements was reported. All volumes were normalized for body surface area and are expressed as an index. These are standard procedures.[18] Day-to-day coefficients of variation were as follows: EDV = 0.95%, ESV = 2.54%, SV = 3.01%.

Body-Composition Measures. Before all body-composition testing, proper hydration status via specific gravity (1.000–1.030) was determined from a urine sample. Bioimpedance spectroscopy was used to estimate total body water following the procedures recommended by the manufacturer (Imp SFB7; ImpediMed Ltd, Queensland, Australia). Dual-energy x-ray absorptiometry (DXA; software version 10.50.086, Lunar Prodigy Advance, Madison, WI) was used to estimate total bone mineral content, and this value was then converted to total body bone mineral.[19] Air-displacement plethysmography (Bod Pod, Life Measurement, Inc, Concord, CA) was used to estimate body volume with an adjustment for thoracic gas volume as outlined by the manufacturer.

Fat mass was estimated using the 4-compartment model described by Wang et al.[20] The equation includes measurements of body volume from air-displacement plethysmography, total body water from bioimpedance spectroscopy, total body bone mineral from DXA, and body weight. The equations for fat mass (FM), percentage body fat (FAT%), and fat-free mass (FFM) are as follows:

$$\text{FM (kg)} = 2.748(\text{BV}) - 0.699(\text{TBW}) + 1.129(\text{Mo}) - 2.051(\text{BW})$$

$$\text{FAT\%} = \text{FM}/(\text{BW} \times 100)$$

$$\text{FFM} = \text{BW} - \text{FM}$$

where BV = body volume, TBW = total body water, Mo = total body bone mineral, and BW = body weight.

Skinfolds were assessed at 3 sites (chest, abdomen, and thigh). Body density was calculated with the Jackson-Pollock equation: Body density = 1.10938 – 0.0008267(sum of 3 skinfolds) + 0.00000016(sum of 3 skinfolds)^2 – 0.0002574(age). Percentage body fat from skinfolds was calculated as (495/body density) – 450.[21]

Fat thickness was assessed at 11 sites (abdomen, forearm, chest, biceps, triceps, subscapular, quadriceps, hamstrings, gastrocnemius, tibialis posterior, and visceral) using a 5-MHz probe and Fukuda Denshi UF-750XT (Tokyo, Japan) ultrasound unit in B mode. The operator's day-to-day coefficient of variation across all sites was 4.27%.

Exercise Performance

Exercise assessments occurred on a different day during the same week as the resting laboratory assessments. These assessments were performed every other month to minimize interference with the subject's established training schedule. The subject did not perform any resistance exercise on the days of the cycle-exercise assessments.

Strength. Strength was assessed with 1-repetition maximum determination for 3 multijoint exercises: squat, bench press, and deadlift.

Maximal Oxygen Consumption and Ventilatory Threshold. The subject completed a continuous graded exercise test on an electronically braked cycle ergometer (Lode, Corival 400, Groningen, The Netherlands) to determine maximal oxygen consumption (VO_{2peak}) and the peak power output in watts at VO_{2peak}. He began pedaling at a cadence of 60 to 80 rpm at a workload of 20 W. The workload increased 1 W every 3 seconds (20 W/min) until he was unable to maintain 60 to 80 rpm or until volitional fatigue. Open-circuit spirometry was used to estimate VO_{2peak} (ml · kg^{-1} · min^{-1}) with a metabolic cart (True One 2400 metabolic measurement system, Parvo-Medics Inc, Sandy, UT) by sampling and analyzing the breath-by-breath expired gases. The metabolic-cart software calculated VO_{2peak} as the highest 30-second VO_2 value during the test. Ventilatory threshold was also determined by plotting ventilation against VO_2 as described previously.[22]

Critical Power and Anaerobic Working Capacity. Three-minute all-out cycling tests were performed on a cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands) using a protocol similar to that developed by Vanhatalo et al.[23] After a self-selected warm-up, each trial began with 60 seconds of unloaded cycling at 90 rpm, followed by an all-out 3-minute effort with resistance being set as a function of pedaling rate. The resistance was adjusted during the all-out effort using the linear mode on the cycle ergometer, which set the power output at 50% of the difference between the ventilatory threshold and peak power output during the graded exercise test when the subject pedaled at his preferred cadence (80 rpm). The average power output during the final 30 seconds of the test was termed critical power, and anaerobic working capacity (AWC or W′) was calculated as the power output–time integral above the critical power.

Blood Parameters

Blood samples were obtained by venipuncture from the antecubital vein with the subject seated and relaxed. Two samples were sent to Diagnostic Laboratory of Oklahoma, Oklahoma City, for immediate analysis (comprehensive metabolic panel and complete blood count), and 1 sample was frozen at –80°C for later analysis of testosterone, leptin, ghrelin, cortisol, insulin, thi-iodothyronine (T3), and thyroxine (T4) by Vanderbilt Pathology Laboratory Services, Nashville, TN.

Other

POMS. The athlete's mood was evaluated with the POMS standard form (MHS, North Tonawanda, NY) consisting of 65 adjectives that the subject rated on a 5-point Likert scale. The POMS is designed to assess 6 mood states: tension–anxiety, depression–dejection, anger–hostility, vigor–activity, fatigue–inertia, and confusion–bewilderment. The vigor–activity score is then subtracted from the sum of all other mood-state scores to determine a total mood-disturbance score. Thus, a higher total mood-disturbance score indicates greater total mood disturbance.

Metabolic Rate (Resting Energy Expenditure). Resting energy expenditure was estimated using indirect calorimetry with a metabolic cart (True One 2400 metabolic measurement system, Parvo-Medics Inc, Sandy, UT). The subject rested in a supine position for 15 to 20 minutes under a ventilated hood (Vacu Med, Ventura, CA). Expired air was collected at 30-second intervals and analyzed to determine resting energy expenditure. The procedure was completed immediately after the cardiovascular measurements, during which the subject was supine for approximately 60 minutes. An intraclass correlation coefficient of .893 using this system was previously reported.[24]

Results

Results are presented with zero representing the measurement taken the month of competition (1 week before competition). A negative number indicates a month before competition and a positive number indicates a month after competition. For example, –5 indicates 5 months before competition.

Throughout the entire study the subject consumed 5 meals per day, equally spaced out 4 to 4.5 hours apart. During the preparation period, the meals before and after the training session together contained ~50% of the subject's daily carbohydrate intake (~25% pretraining and ~25% posttraining). Initially during the preparation period, the subject's daily macronutrient breakdown was ~36% protein (PRO), ~36% carbohydrate (CHO), and ~28% fat for 5 days per week and ~30% PRO, ~48% CHO, and ~22% fat for 2 days of the week. During the preparation period, overall caloric intake did not change dramatically, but modest (5–10 g) weekly reductions in CHO and/or fat were made based on the progression of weight loss. Just before competition, the daily macronutrient breakdown was ~46% PRO, ~29% CHO, and 25% fat During the recovery period, the daily macronutrient breakdown was more variable but generally consisted of ~25% to 30% PRO, 35% to 40% CHO, and 30% to 35% fat.

During competition preparation, per week on average, the subject performed 4 days of resistance training (total 5 h/wk), 1 day of high-intensity interval training (total 40 min/wk), and 1 day of low-intensity, steady-state, aerobic exercise (30 min/wk). This resistance-training split enabled the athlete to train each major muscle group twice per week. In addition, the subject performed "posing practice" during the preparation period. This involved repeated sustained (~30–60 s) isometric contractions of major muscle groups while the subject held his limbs and torso in a position intended to make the muscles appear as large and defined as possible. These practice sessions lasted 15 to 30 minutes and varied in frequency from once per week during the initial weeks (weeks 1–6) of preparation up to 3 or 4 times per week during the final weeks (weeks 20–26) of preparation. In the 6 months after the competition, per week on average, the subject performed 4 days of resistance training (total 5 h/wk) and 1 day of high-intensity interval training (total 20 min/wk) only. The resistance-training frequency for each major muscle group remained at twice per week during this period.

Energy intake and resting energy expenditure are shown in Figure 1. Cardiovascular and body-composition parameters, exercise performance, blood parameters, and POMS results are presented in Figure 2 and Tables 1–5.

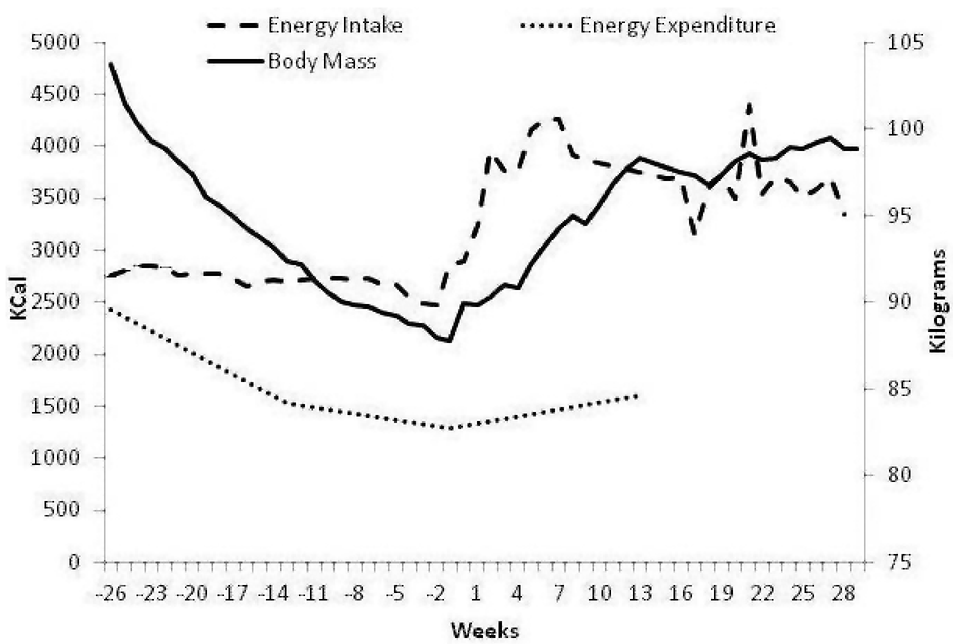

Figure 1 — Average weekly food intake (energy intake) and body mass (body mass) along with resting energy expenditure (resting energy expenditure measured at –26, –13, 0, and 13 weeks.

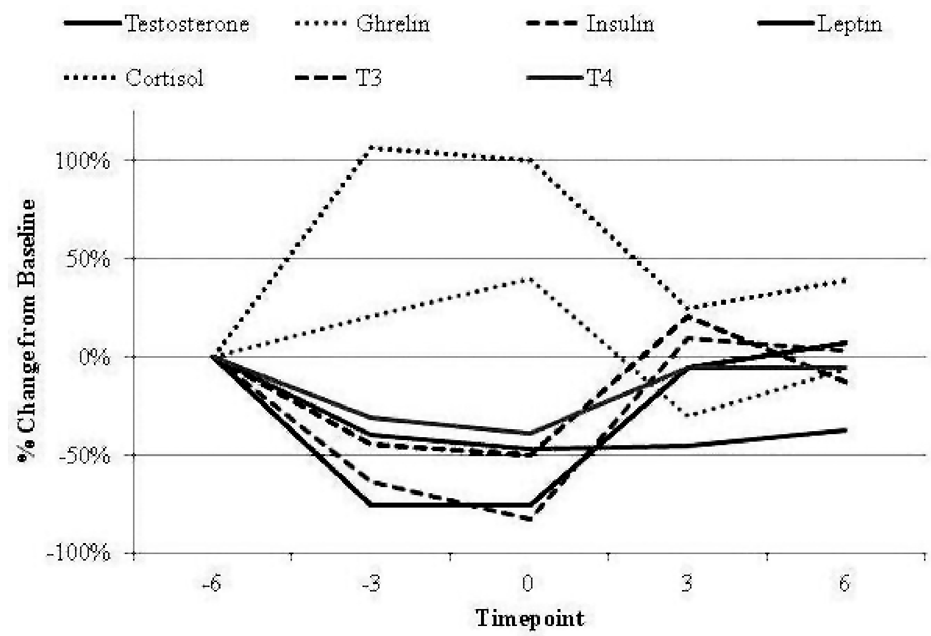

Figure 2 — Percent changes from baseline for hormone variables.

Table 1 Cardiovascular Variables

	Time Point												
Measure	**–6**	**–5**	**–4**	**–3**	**–2**	**–1**	**0**	**1**	**2**	**3**	**4**	**5**	**6**
Blood pressure (mmHg)													
brachial systolic	132	132	116	103	102	103	104	115	122	129	119	112	116
brachial diastolic	69	81	67	63	67	59	56	60	64	66	62	61	64
aortic systolic	105	112	99	88	88			93	99	103	98	94	99
aortic diastolic	69	81	67	63	67			60	64	66	62	61	64
aortic mean arterial	81	94	79	72	75			74	78	79	77	74	78
Limb blood flow (mL · 100 mL^{-1} · min^{-1})													
forearm	2.38	1.55	1.93	1.59	1.50	1.53	1.59	1.60	1.80	2.84	1.98	2.10	1.88
calf	1.71	2.14	0.90	1.42	1.70	1.48	1.61	1.50	1.18	2.08	0.83	2.23	1.95
Pulse-wave velocity (m/s)													
aortic	5.8	6.2	5.4	6.2	5.5	4.6	3.8	5.3	4.6	5.4	5.5	5.5	5.9
femoral	9.2	9.4	7.4	9.7	9.2	7.9	8.4	5.0	10.4	8.5	8.3	9.5	10.1
brachial	7.8	9.2	9.7	6.6	6.8	7.4		6.1	9.2	7.9	9.5	9.9	9.8
Cardiac measures													
heart rate (beats/min)	53	46	40	34	36	27	27	46	48	56	47	49	47
EDVi	85.5	87.9	89.2	102.3	100.8	112.4	113.2	75.4	84.4	83.0	80.2	77.1	89.7
ESVi	41.2	41.7	39.4	45.6	46.0	48.6	44.1	29.6	36.9	34.6	31.2	33.9	39.2
SVi	44.4	46.3	50.0	56.6	54.9	64.2	69.3	45.8	47.5	48.4	49.1	43.2	50.5
ejection fraction (%)	51.7	52.7	56.0	55.3	54.7	56.7	61.7	60.0	56.0	58.3	61.3	56.0	56.3
COi	2.4	2.1	2.0	1.9	2.0	1.7	1.9	2.1	2.3	2.7	2.3	2.1	2.4

Abbreviations: EDV, end-diastolic volume; ESV, end-systolic volume; SV, stroke volume; CO, cardiac output; i = indexed to body surface area.

Table 2 Body-Composition Variables

Measure	Time Point												
	–6	–5	–4	–3	–2	–1	0	1	2	3	4	5	6
Body mass (kg)	102.85	99.36	96.46	92.27	90.76	90.23	88.87	91.06	94.56	98.03	98.10	99.55	98.99
4-C model													
bone mineral content (kg)	3.91	3.91	3.89	3.92	3.91	3.94	3.93	3.89	3.86	3.82	3.85	3.91	3.87
total body water (L)	62.60	61.79	61.19	60.25	61.99	60.48	62.12	60.84	63.71	63.30	58.89	60.83	60.31
body density (g/mL)	1.0626	1.0716	1.0741	1.0821	1.0851	1.0886	1.0887	1.0807	1.0762	1.0673	1.0694	1.0683	1.0707
fat-free mass (kg)	87.65	86.01	84.82	84.09	84.97	84.25	84.84	83.42	86.29	85.68	82.88	84.66	84.59
% body fat	14.8	13.4	12.1	8.9	6.4	6.6	4.5	8.4	8.8	12.6	15.5	15.0	14.6
Skinfolds % body fat	8.9	8.2	6.1	4.6	5.0	3.5	3.1	7.0	7.2	6.7	6.7	8.2	6.3
Fat thickness													
visceral fat area (cm^2)	1.03	0.70	0.30	0.10	0.10				0.25	0.20	0.35	0.50	0.50
sum of all fat-thickness values (cm)	3.92	2.80	2.40	1.21	1.38	0.85	0.68	0.90	1.21	2.07	1.82	1.98	2.10

Table 3 Performance Variables

Measure	Time Point												
	–6	–5	–4	–3	–2	–1	0	1	2	3	4	5	6
Absolute 1-repetition maximum (kg)													
squat	210.9		201.9		195.5		181.9		195.5		210.0		212.3
bench press	161.0		154.2		145.2		147.4		150.1		152.4		150.1
Deadlift	259.0		249.9		245.4		240.9		249.9		259.9		259.9
Relative 1-repetition maximum													
squat	2.1		2.1		2.2		2.0		2.1		2.1		2.1
bench press	1.6		1.6		1.6		1.7		1.6		1.6		1.5
deadlift	2.5		2.6		2.7		2.7		2.6		2.6		2.6
Aerobic capacity													
absolute VO_{2peak} (L/min)	4.46	4.62		4.26		3.97		4.32		4.74		4.51	
relative VO_{2peak} ($mL \cdot kg^{-1} \cdot min^{-1}$)	42.2	46.7		45.7		44.6		47.6		48.3		46.2	
maximum heart rate (beats/min)	177			160				162				169	
3-minute all-out cycling test													
critical power (W)	223.3		266.3		228.8		199.3		259.2		243.0		225.0
anaerobic working capacity (kJ)	25.0		19.1		14.9		9.6		14.1		19.7		19.4
peak power (W)	1360		1517		1258		1020		1168		1451		1447
relative critical power (W/kg)	2.2		2.8		2.5		2.2		2.7		2.5		2.3
relative AWC (kJ/kg)	0.24		0.20		0.16		0.11		0.15		0.20		0.20
relative peak power (W/kg)	13.2		15.7		13.9		11.5		12.4		14.8		14.6

Table 4 Blood Variables

Measure		Time Point					
		Reference range	–6	–3	0	3	6
Hormones	testosterone (ng/mL)	2.9–13.0	9.22	2.25*	2.27*	8.7	9.91
	ghrelin (pq/mL)	300–800	633.17	762.20	882.95†	439.66	593.18
	insulin (µU/mL)	4.9–24.3	6.08	2.21*	1.06*	6.67	6.26
	leptin (ng/mL)	3.7–11.1	2.58*	1.56*	1.36*	1.41*	1.62*
	cortisol (µg/dL)	5–25	10.52	21.76	21.07	13.10	14.58
	T3 (ng/mL)	0.6–1.75	1.34	0.74	0.67	1.62	1.17
	T4 (ng/mL)	40–110	78.49	54.38	47.67	73.79	73.94
Lipid panel	total cholesterol (mg/dL)	125–200	207†	288†	239†	221†	182
	high-density lipoprotein (mg/dL)	>40	49	73	79	61	53
	triglycerides (mg/dL)	<150	62	66	63	72	69
	low-density lipoprotein (mg/dL)	<130	146†	203†	147†	146†	115
	total/HDL ratio	≤5.0	4.2	3.9	3.0	3.6	3.4
Metabolic panel	glucose (mg/dL)	65–99	83	52*	72	78	75
	urea nitrogen (mg/dL)	7–25	22	29†	34†	21	22
	creatinine (mg/dL)	0.8–1.3	1.36†	1.4†	1.54†	1.11	1.20
	estimated GFR (mL · min^{-1} · 1.73 m^{-2})	>60	>60		61	91	82
	creatine kinase (U/L)	29–196	582†	913†	495†	458†	517†
Complete blood count	white blood cells (1000/µL)	3.8–10.8	6.0	4.4	3.8	5.3	4.9
	red blood cells (1,000,000/µL)	4.20–5.10	5.07	4.69	4.02*	4.53	4.71
	hemoglobin (g/dL)	13.2–15.5	16.2†	15.0	13.3	15.0	15.1
	hematocrit (%)	38.5–45.0	47.6†	45.0	38.7	43.3	44.1

*Value below reference range. †Value above reference range.

Table 5 Profile of Mood States

Subscale	Time Point												
	–6	–5	–4	–3	–2	–1	0	1	2	3	4	5	6
Tension–anxiety	12	7	3	1	5	6	13	9	10	10	9	15	5
Depression–dejection	1	1	0	0	0	1	1	0	2	1	0	0	0
Anger–hostility	6	0	1	1	1	1	4	5	2	0	0	0	0
Vigor–activity	19	17	16	12	10	7	5	10	7	3	3	5	7
Fatigue–inertia	4	2	5	3	5	14	22	6	6	2	3	6	2
Confusion–bewilderment	2	2	2	2	3	5	8	4	8	8	6	6	4
Total mood disturbance	6	–5	–5	–5	4	20	43	14	21	18	15	22	4

Discussion

This study provided a comprehensive physiological profile of a young, male, natural bodybuilder. Immense physiological changes occurred during bodybuilding competition preparation. During the 6 months after competition, cardiovascular and blood parameters recovered quickly while body composition and strength recovered more slowly.

Competition preparation for this subject primarily involved reducing energy intake while increasing aerobic exercise (including both steady-state and high-intensity interval training). Thus, physiological changes during competition preparation were driven primarily by reduced energy intake and the addition of aerobic exercise. Similar to a previous report noting a high protein intake in bodybuilders,[25] this subject consumed a relatively high proportion of energy from protein in an effort to maintain muscle mass. It is a common misconception that bodybuilding competition preparation involves the sudden onset of a muscle-building program. At the time of competition preparation, the athlete is already very muscular and must then focus on maintaining the current muscle (via maintaining a strength-training program) and losing subcutaneous fat (via diet and aerobic exercise) to enhance muscular appearance. The weekly changes in diet are subtle to induce slow weight loss in contrast to the rapid weight loss achieved by other athletes to make weight for competition.[26] In the 6 months after the competition, the subject's energy intake increased, aerobic exercise decreased, and resistance exercise remained constant.

Of note, many of the physiological changes observed including an elevation in cortisol, reduction in testosterone, reduction in immune function, alterations in mood status, and decreases in physical performance and maximal heart rate that occurred during the preparation period are consistent with markers of overtraining.[27–29] While these changes may be considered a negative outcome in many sports judged or scored on physical performance, these outcomes having little bearing on the subjective outcome of a bodybuilding competition. In fact, these alterations may be almost a prerequisite for achieving an optimal physique for bodybuilding. One month after competition, total mood disturbance improved while aerobic performance began to increase. Two months after competition, strength and anaerobic performance began to recover while the hormonal profile returned to prepreparation (baseline) levels within 3 months of the competition. In contrast to the physical changes that occurred before the bodybuilding competition, preparation for other competitions that do not cause such reductions in body fat may also not elicit changes in either strength or aerobic fitness and may actually enhance anaerobic capabilities.[30]

Body Composition

During competition preparation, fat-free mass did not decrease greatly (–3.9%). The loss in body weight was thus primarily due to loss of body fat as desired. The subject's total body water was relatively stable over the preparation and recovery period and is similar to values previously reported in bodybuilders.[31] Total body water has been shown to be elevated in bodybuilders compared with untrained individuals, and this is thought to be due to an increase in cytoplasmic volume.[32] In addition, the substantial drop in resting energy expenditure during competition preparation appeared driven more by a decrease in energy intake than by loss of fat-free mass. During recovery, percent body fat increased gradually, not returning to baseline values until 4 months after competition. The subject's diet was more irregular during recovery than during preparation; however, a stated (and achieved) goal of the subject was to not regain body fat too quickly.

Exercise Performance

Despite near maintenance of fat-free mass, absolute 1-repetition-maximum strength for the squat (–13.8%), bench press (–8.4%), and deadlift (–7.0%) decreased. This loss of strength coincided with an increase in fatigue and a decrease in vigor as detected by the POMS and is likely explained by the change in these factors induced by the energy deficit. Decreases in AWC (or W′; –61%) can likely also be explained by a decrease in glycogen availability due to the decreased energy intake and increased training volume.[33] As body mass decreased, absolute VO_{2peak} and critical power decreased as expected while relative values for both remained fairly constant. In contrast, short-term weight cutting (ie, 1 wk) in other athletes may not have such a negative effect on physical performance.[34]

After competition, exercise abilities recovered rather slowly. Squat and deadlift strength and critical power recovered by month 4, while bench-press strength and AWC remained below baseline at month 6. This lack of strength and critical power/AWC recovery was reflected in continually depressed vigor as fatigue had returned to baseline by month 4.

Cardiovascular

The decrease in heart rate from 53 beats/min to 27 beats/min during competition preparation appears extreme. Heart rates this low during periods of energy restriction are not unprecedented, however; a heart rate of 27 beats/min was observed in Ancel Keys' well-known Minnesota Starvation Study.[35] The subject's heart-rate drop allowed for increased cardiac filling time and thus increased end-diastolic volume and stroke volume. Cardiac output still decreased overall during competition preparation, as the increase in stroke volume was not enough to counteract the substantial decrease in heart rate. During preparation, brachial BP decreased from a prehypertensive value (132/69 mmHg) at baseline to a normotensive level (116/67 mmHg) after 2 months and further deceased after 3 months (103/63 mmHg), where it basically remained for the rest of the precompetition phase. One month into recovery, BP rebounded to 115/60 mmHg, where, with the exception of a jump at month 3 to 129/66 mmHg, it basically remained throughout recovery.

The rapid recovery of these cardiovascular responses suggests that energy intake, not weight loss or gain, drove these changes. Neither peripheral blood flow nor peripheral arterial stiffness exhibited much change during preparation or recovery. However, central arterial stiffness dropped the month of competition and increased during recovery to the baseline value. This change in central stiffness was likely driven by a combination of factors such as changing BP, heart rate, and catecholamine levels (not measured).

Blood Parameters

During competition preparation, the hormonal changes reflect the energy deficit. Even 2 to 3 weeks of weight loss can reduce testosterone levels,[36] suggesting that alterations in the subject's hormonal profile may have occurred even earlier in the precompetition period. Cortisol levels rose, likely indicating increased lipolysis rather than muscle-tissue proteolysis, because, as stated previously, fat-free mass did not greatly decrease during this time. Ghrelin levels rose as leptin levels fell, as expected during energy deficiency. A study of 7 dieting natural male bodybuilders over 11 weeks found ghrelin and leptin responses similar to those observed in our study.[7] In addition, testosterone dropped to levels below the reference range as the athlete entered a state similar to energy-deficit-induced female amenorrhea.[37] This severe drop was observed at the 3-month time point, and no further drop was observed at the month of competition. Red blood cell count and hematocrit also decreased during competition preparation and increased during recovery; these changes were likely influenced by the changes in testosterone. In contrast, bodybuilders taking anabolic steroids may have reduced high-density lipoprotein levels and elevated hematocrit.[38] Previous work examining natural male bodybuilders' testosterone levels after an 11-week diet found a much less extreme drop in testosterone from baseline (11 weeks out) to the month of competition.[6] Our subject's insulin level decreased greatly and similarly to what has been shown previously.[6] Thyroid function, as indicated by T3 and T4 levels, reflected the observed decrease in resting energy expenditure and decreased from baseline and throughout competition preparation. All hormones, with the exception of ghrelin and leptin, returned to near baseline levels by 3 months after competition as the energy deficit was eliminated. Hormone levels may have recovered even earlier, but 3 months was the first postcompetition measurement.

The high-density lipoprotein level improved during competition preparation and returned to near the baseline level 6 months after competition; this change was likely influenced by the addition of aerobic exercise during competition preparation and the decline in aerobic-exercise frequency during recovery. The low-density lipoprotein level followed no trend; the reason for the spike 6 months before competition and the drop 6 months after competition is unknown. The subject's glucose level was hypoglycemic 3 months before competition but was back in the normal range by the month of competition.

The subject's elevated creatine kinase levels throughout preparation and recovery simply reflect the acute response to the previous day's resistance-training session; levels of this magnitude, in the absence of symptoms, are in no way indicative of rhabdomyolysis.[39] Previous studies examining creatine kinase levels after heavy resistance exercise,[40] army basic training,[39] and marathon running[41] have reported similar or greater creatine kinase values.

POMS

POMS unexpectedly detected an improvement in total mood disturbance, a global measure of affective state, from baseline until 2 months before competition. This improvement was driven by decreases in the emotional subscales for tension–anxiety and anger–hostility and occurred despite a steady decrease in the vigor–activity subscale. These changes are in contrast to the increase in confusion–bewilderment observed during short-term weight cutting in wrestlers,[34] although the subject in this investigation did increase in confusion–bewilderment during the month of competition. Subjectively, the subject indicated during this time that he enjoyed the preparation and felt excited for the competition. One month before competition, however, total mood disturbance greatly increased; this was driven by increases in the fatigue–inertia and tension–anxiety subscales and a decrease in the vigor–activity subscale.

Strengths and Limitations

Although randomized controlled trials are the norm in scientific literature, it has been satirically and accurately demonstrated that they are not always the best source of information.[42] We believe that our sample size of 1 is thus a strength of our study, as it enabled us to frequently perform a plethora of measurements for a full year in a difficult-to-study subject population. We are of the opinion that not many bodybuilders preparing for a competition would agree to regular fasted testing, maximal cycling tests, and maximal-strength assessments.

Practical Applications

Much speculation exists in the popular media about the physiological changes that occur with bodybuilding. This case study provides some empirical evidence to fill this void.

Conclusions

This study provides a thorough documentation of the changes that occurred in 1 subject during natural bodybuilding competition preparation and recovery. The most notable changes observed were in heart rate, percent body fat, and hormone levels. Most of the changes observed were similar to what would be expected during a period of energy restriction followed by a period of energy surplus.

References

1. Lambert CP, Frank LL, Evans WJ. Macronutrient considerations for the sport of bodybuilding. *Sports Med*. 2004;34(5):317–327. PubMed doi:10.2165/00007256-200434050-00004
2. Heyward VH, Sandoval WM, Colville BC. Anthropometric, body composition and nutritional profiles of bodybuilders during training. *J Appl Sport Sci Res*. 1989;3(2):22–29.
3. Walberg-Rankin J, Edmonds CE, Gwazdauskas FC. Diet and weight changes of female bodybuilders before and after competition. *Int J Sport Nutr*. 1993;3(1):87–102. PubMed

4. Brill JB, Keane MW. Supplementation patterns of competitive male and female bodybuilders. *Int J Sport Nutr*. 1994;4(4):398–412. PubMed
5. Steen SN. Precontest strategies of a male bodybuilder. *Int J Sport Nutr*. 1991;1(1):69–78. PubMed
6. Maestu J, Eliakim A, Jurimae J, et al. Anabolic and catabolic hormones and energy balance of the male bodybuilders during the preparation for the competition. *J Strength Cond Res*. 2010;24(4):1074–1081. PubMed doi:10.1519/JSC.0b013e3181cb6fd3
7. Maestu J, Jurimae J, Valter I, et al. Increases in ghrelin and decreases in leptin without altering adiponectin during extreme weight loss in male competitive bodybuilders. *Metabolism*. 2008;57(2):221–225. PubMed doi:10.1016/j.metabol.2007.09.004
8. Bamman MM, Hunter GR, Newton LE, et al. Changes in body composition, diet, and strength of bodybuilders during the 12 weeks prior to competition. *J Sports Med Phys Fitness*. 1993;33(4):383–391. PubMed
9. Thorsteinsdottir B, Grande JP, Garovic VD. Acute renal failure in a young weight lifter taking multiple food supplements, including creatine monohydrate. *J Ren Nutr*. 2006;16(4):341–345. PubMed doi:10.1053/j.jrn.2006.04.025
10. Koopman M, Richter C, Parren RJ, et al. Bodybuilding, sesame oil and vasculitis. *Rheumatology (Oxford)*. 2005;44(9):1135. PubMed doi:10.1093/rheumatology/keh712
11. Voelcker V, Sticherling M, Bauerschmitz J. Severe ulcerated 'bodybuilding acne' caused by anabolic steroid use and exacerbated by isotretinoin. *Int Wound J*. 2010;7(3):199–201. PubMed doi:10.1111/j.1742-481X.2010.00676.x
12. Schafer CN, Guldager H, Jorgensen HL. Multi-organ dysfunction in bodybuilding possibly caused by prolonged hypercalcemia due to multi-substance abuse: case report and review of literature. *Int J Sports Med*. 2011;32(1):60–65. PubMed doi:10.1055/s-0030-1267200
13. Mayr FB, Domanovits H, Laggner AN. Hypokalemic paralysis in a professional bodybuilder. *Am J Emerg Med*. 2012;30(7):1324.e5–1324.e 8. PubMed doi:10.1016/j.ajem.2011.06.029
14. Banke IJ, Prodinger PM, Waldt S, et al. Irreversible muscle damage in bodybuilding due to long-term intramuscular oil injection. *Int J Sports Med*. 2012;33(10):829–834. PubMed doi:10.1055/s-0032-1311582
15. Faude O, Meyer T, Urhausen A, et al. Recovery training in cyclists: ergometric, hormonal and psychometric findings. *Scand J Med Sci Sports*. 2009;19(3):433–441. PubMed doi:10.1111/j.1600-0838.2008.00795.x
16. Van Bortel LM, Duprez D, Starmans-Kool MJ, et al. Clinical applications of arterial stiffness, Task Force III: recommendations for user procedures. *Am J Hypertens*. 2002;15(5):445–452. PubMed doi:10.1016/S0895-7061(01)02326-3
17. Greenfield AD, Whitney RJ, Mowbray JF. Methods for the investigation of peripheral blood flow. *Br Med Bull*. 1963;19:101–109.
18. Helak JW, Reichek N. Quantitation of human left ventricular mass and volume by two-dimensional echocardiography: in vitro anatomic validation. *Circulation*. 1981;63(6):1398–1407. PubMed doi:10.1161/01.CIR.63.6.1398
19. Heymsfield SB, Wang J, Heshka S, et al. Dual-photon absorptiometry: comparison of bone mineral and soft tissue mass measurements in vivo with established methods. *Am J Clin Nutr*. 1989;49(6):1283–1289. PubMed
20. Wang Z, Pi-Sunyer FX, Kotler DP, et al. Multicomponent methods: evaluation of new and traditional soft tissue mineral models by in vivo neutron activation analysis. *Am J Clin Nutr*. 2002;76(5):968–974. PubMed
21. American College of Sports Medicine. *ACSM's Guidelines for Exercise Testing and Prescription*. 8th ed. Baltimore, MD: Lippincott, Williams, & Williams; 2010.
22. Orr GW, Green HJ, Hughson RL, et al. A computer linear-regression model to determine ventilatory anaerobic threshold. *J Appl Physiol*. 1982;52(5):1349–1352. PubMed
23. Vanhatalo A, Doust JH, Burnley M. Determination of critical power using a 3-min all-out cycling test. *Med Sci Sports Exerc*. 2007;39(3):548–555. PubMed doi:10.1249/mss.0b013e31802dd3e6
24. Dalbo VJ, Roberts MD, Stout JR, et al. Effect of gender on the metabolic impact of a commercially available thermogenic drink. *J Strength Cond Res*. 2010;24(6):1633–1642. PubMed doi:10.1519/JSC.0b013e3181db9bbd
25. Manz F, Remur T, Decher-Spliethoff E, et al. Effects of a high protein intake on renal acid excretion in bodybuilders. *Z Ernahrungswiss*. 1995;34(1):10–15.
26. Lingor RJ, Olson A. Fluid and diet patterns associated with weight cycling and changes in body composition assessed by continuous monitoring throughout a college wrestling season. *J Strength Cond Res*. 2010;24(7):1763–1772. PubMed doi:10.1519/JSC.0b013e3181db22fb
27. Cunha G, Ribeiro J, Oliveira A. Overtraining: theories, diagnosis and markers. *Rev Bras Med Esporte*. 2006;12(5):297–302. doi:10.1590/S1517-86922006000500014
28. Fry AC, Kraemer WJ, Ramsey LT. Pituitary-adrenal-gonadal responses to high-intensity resistance exercise overtraining. *J Appl Physiol*. 1998;85(6):2352–2359. PubMed
29. Fry AC, Kraemer WJ. Resistance exercise overtraining and overreaching: neuroendocrine responses. *Sports Med*. 1997;23(2):106–129. PubMed doi:10.2165/00007256-199723020-00004
30. Utter AC, O'Bryant HS, Haff GG, et al. Physiological profile of an elite freestyle wrestler preparing for competition: a case study. *J Strength Cond Res*. 2002;16(2):308–315. PubMed
31. Piccoli A, Pastori G, Codognotto M, et al. Equivalence of information from single frequency v. bioimpedance spectroscopy in bodybuilders. *Br J Nutr*. 2007;97(1):182–192. PubMed doi:10.1017/S0007114507243077
32. MacDougall JD, Sale DG, Elder GC, et al. Muscle ultrastructural characteristics of elite powerlifters and bodybuilders. *Eur J Appl Physiol Occup Physiol*. 1982;48(1):117–126. PubMed doi:10.1007/BF00421171

33. Miura A, Sato H, Whipp BJ, et al. The effect of glycogen depletion on the curvature constant parameter of the power-duration curve for cycle ergometry. *Ergonomics*. 2000;43(1):133–141. PubMed doi:10.1080/001401300184693
34. Marttinen RH, Judelson DA, Wiersma LD, et al. Effects of self-selected mass loss on performance and mood in collegiate wrestlers. *J Strength Cond Res*. 2011;25(4):1010–1015. PubMed
35. Keys A. *The Biology of Human Starvation*. St. Paul, MN: North Central Publishing Co; 1950.
36. Karila TA, Sarkkinen P, Marttinen M, et al. Rapid weight loss decreases serum testosterone. *Int J Sports Med*. 2008;29(11):872–877. PubMed doi:10.1055/s-2008-1038604
37. Ireland M, Nattiv A. *The Female Athlete*. Philadelphia, PA: Saunders; 2003.
38. Lane HA, Grace F, Smith JC, et al. Impaired vasoreactivity in bodybuilders using androgenic anabolic steroids. *Eur J Clin Invest*. 2006;36(7):483–488. PubMed doi:10.1111/j.1365-2362.2006.01667.x
39. Kenney K, Landau ME, Gonzalez RS, et al. Serum creatine kinase after exercise: drawing the line between physiological response and exertional rhabdomyolysis. *Muscle Nerve*. 2012;45(3):356–362. PubMed doi:10.1002/mus.22317
40. Wolf MR, Fragala MS, Volek JS, et al. Sex differences in creatine kinase after acute heavy resistance exercise on circulating granulocyte estradiol receptors. *Eur J Appl Physiol*. 2012;112(9):3335–3340. PubMed doi:10.1007/s00421-012-2314-z
41. Rogers MA, Stull GA, Apple FS. Creatine kinase isoenzyme activities in men and women following a marathon race. *Med Sci Sports Exerc*. 1985;17(6):679–682. PubMed doi:10.1249/00005768-198512000-00010
42. Smith GC, Pell JP. Parachute use to prevent death and major trauma related to gravitational challenge: systematic review of randomised controlled trials. *BMJ*. 2003;327(7429):1459–1461. PubMed doi:10.1136/bmj.327.7429.1459