Factors Associated with Energy Expenditure and Energy Balance in Acute Sport-Related Concussion.

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Factors Associated with Energy Expenditure and Energy Balance in Acute Sport-Related Concussion.

Context: Sport-related concussion (SRC) is characterized by a pathological neurometabolic cascade that results in an increased intracranial energy demand with decreased energy supply. Little is known about the whole-body energy-related effects of SRC.

Objective: To examine factors associated with whole-body resting metabolic rate (RMR), total energy expenditure (TEE), energy consumption (EC), and energy balance (EBal) in student-athletes acutely after SRC and healthy-matched controls.

Design: Case-Control.

Setting: University Research Laboratory.

Patients or Other Participants: Student-athletes diagnosed with SRC (n=28, 50% female, aged 18.4±1.83 years, BMI=20.3±4.13 kg·(m²)⁻¹) assessed ≤72 hours of injury and matched-controls (n=28, 50% female, aged 19.4±2.90 years, BMI=18.8±4.79 kg·(m²)⁻¹).

Main Outcome Measures: RMR was measured via indirect calorimetry. Participants reported physical activity and dietary intake for three days, which were used to estimate TEE and EC, respectively, and to calculate EBal (EC:TEE ratio). RMR, TEE, and EC were normalized to body mass. Group and group-by-sex comparisons were made for RMR·kg⁻¹, TEE·kg⁻¹, EC·kg⁻¹, and EBal using independent t-tests with a-priori α=0.05.
Associations of age, sex, concussion history, BMI, and symptom burden with RMR·kg\(^{-1}\) and EBal were explored with linear regression models.

**Results:** TEE·kg\(^{-1}\) was lower \((p<0.01; \text{mean difference}\[SD]=-5.31[1.41]\text{kcal·kg}^{-1})\) and EBal was higher \((p<0.01; 0.28[0.10])\) in SRC participants than controls. Both sexes \((p's<0.04)\) with SRC had lower TEE·kg\(^{-1}\) compared to controls; females with SRC had higher EBal than controls \((p=0.01)\), but male groups did not differ. Higher RMR·kg\(^{-1}\) was associated with concussion history \((\text{adjusted-}R^2=0.10; \beta=0.65)\). Younger age \((\beta =-0.35)\), fewer concussions \((\beta =-0.35)\), lower BMI \((\beta =-0.32)\), greater symptom duration \((\beta =1.50)\), and lower symptom severity \((\beta =-1.59)\) were associated with higher EBal \((\text{adjusted-}R^2=0.54)\).

**Conclusions:** TEE·kg\(^{-1}\) and EBal appeared to be affected by acute SRC, despite no differences in RMR·kg\(^{-1}\). Concussion history, sex, BMI, and symptoms were associated with acute energy-related outcomes.

**Abstract Word Count:** 285

**Key Words:** Mild Traumatic Brain Injury (mTBI), Resting Metabolic Rate, Concussion History, Sex Differences, Symptom Burden

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**Key Points:**
• Student-athletes with acute SRC reported increased energy consumption relative to their estimated energy expenditure (presenting with a relative energy surplus) – this was associated with sex, age, concussion history, and symptom burden.

• Females with SRC had a greater acute (within 72hrs) energy surplus following their injury compared to controls while their male counterparts did not differ between SRC and controls.

• Student-athletes with multiple prior concussions exhibited greater whole-body resting metabolic rate than those with fewer prior concussions – suggesting either a beneficial adaptation (facilitative) or maladaptive response to a new SRC.
The pathophysiologic response to sport-related concussion (SRC) has been described as a neurometabolic cascade of events that disrupt typical neurobiological functioning and maintenance of ionic homeostasis. Immediately following SRC, the brain typically undergoes a transient (minutes to hours) hypermetabolic phase followed by a subsequent hypometabolic phase within a period of minutes to days. Presently, there is little research investigating whether altered metabolism in the brain after injury affects whole-body energy expenditure. Resting metabolic rate (RMR) is the amount of energy required by the whole body to perform life-sustaining functions in an awake but resting state. The healthy human brain demands approximately 20% of the body’s total RMR, and studies of patients with moderate to severe TBI (defined by a Glasgow Coma Score less than 13) have reported up to a two-fold increase in whole-body RMR above predicted values acutely after injury and throughout recovery. Early evidence in collegiate student-athletes with SRC suggests that RMR may not be altered acutely following injury, and energy-related changes following SRC did not appear to linger beyond the resolution of symptoms or return to play. However, total daily energy expenditure, the sum of both RMR and energy expended through physical activity, and the proportion of energy consumption to energy expenditure may change throughout recovery from SRC. At this point, factors that may influence energy-related outcomes in patients with SRC have not been studied.

The metabolic response to SRC may be influenced by intrinsic factors like age, concussion history, and sex. Specifically, patient age and level of sport participation appear to associate with clinically measured neurocognitive and balance performance, as well as SRC-related symptom burden. Age is also a known modifier in the estimation
of energy expenditure in healthy individuals.\textsuperscript{16,17} Previous experience of concussion has been associated with increased self-reported symptom severity, which is a known modifier of clinical recovery.\textsuperscript{18} Recent studies have demonstrated that patients with a higher initial total symptom severity had a longer times to clinical recovery (in days) than those with a lower total symptom severity.\textsuperscript{15,19,20} Further, specific pathophysiologic responses to SRC in the brain, such as the metabolic “crisis” of increased energy demand with concurrently decreased supply, may predispose individuals to future injury.\textsuperscript{1} Taken together, patients with a history of one or more concussions may experience exacerbated physiological and clinically measurable responses to a new SRC. In terms of biological sex, females tend to report greater symptom burden than males following SRC, and a recent literature review suggests that this phenomenon exists also during the pre-injury (baseline) assessment.\textsuperscript{21} Notwithstanding symptom reports, whole-body metabolism differs between healthy males and females,\textsuperscript{16,17} but the sex-specific metabolic response to SRC is unknown. Presently, the interactions of these intrinsic factors with whole-body metabolism in the context of SRC and other mTBI are unknown.

Therefore, we examined the acute (within 72 hours of injury) whole-body metabolic response to SRC in physically active high school and collegiate students as compared to similarly matched healthy controls. We also examined whether energy consumption (EC) and relative energy balance (EBal; ratio of energy consumed compared to energy expended) were different between concussed and matched-control participants. We then sought to examine whether age, concussion history, sex, and measures of symptom burden (i.e., symptom severity and duration) were associated with RMR and EBal acutely following SRC. Based on studies with moderate and severe TBI,
we hypothesized that participants with SRC would have greater energy expenditure than control participants.\textsuperscript{11,12} We also hypothesized that younger athletes would have greater energy expenditure than older athletes. We further hypothesized that more prior concussions would not exhibit a statistically significant association with RMR or EBal outcomes.

\textbf{METHODS}

\textit{Participants}

The university’s institutional review board approved the current study. All adult participants provided written informed consent prior to their participation in our study. Participants under 18 years of age provided assent and their parent or legal guardian provided consent prior to participation. Concussed participants were recruited from a large public university and its surrounding high schools. Data were collected between the Spring of 2015 to the Spring of 2019. The diagnosis of SRC was made by a certified athletic trainer or sports medicine physician according to the current definition from the Concussion in Sport Group at the time of diagnosis.\textsuperscript{13,22} Specific criteria for the diagnosis of SRC differed between recruitment sites but included, at a minimum, a multidimensional assessment with symptoms and standardized clinical assessments of function (e.g., SCAT and/or computerized neurocognitive testing).

Following the diagnosis of a SRC, participants reported to our laboratory for their initial assessment within 72 hours of their injury as part of a larger study.\textsuperscript{6,7} Healthy control participants were matched to injured participants based on sex, age, height, mass, and sport when possible. Participants were excluded if they reported treatment for an
acute musculoskeletal injury (e.g., fracture), had a diagnosis of any pathology known to
affect metabolism (e.g., thyroid dysfunction), or if they had sustained another concussion
within six months prior to their initial assessment. There were no data available regarding
menstrual cycle phase or use of hormone-regulating birth control in female participants,
nor was it a criterion for inclusion or exclusion.

Energy Expenditure, Consumption, and Balance Measures
A detailed description of the outcome measures and procedures utilized in the
current study has been reported elsewhere. In brief, indirect calorimetry (VMax
Metabolic Cart; Carefusion, Yorba Linda, CA) was used to measure RMR (kcal·day⁻¹). Total daily energy expenditure (TEE; kcal·day⁻¹) was calculated as the product of RMR multiplied by a corresponding physical activity correction factor. The physical activity correction factor was based on physical activity volume and biological sex. Physical activity was monitored via step-count with a Fitbit (Fitbit, Inc.) and self-reported exercise in which wearing the Fitbit was not permissible (e.g., swimming). Step-counts were binned into five physical activity level categories: Sedentary (< 5,000 steps per day); Low Active (5,000-7,499 steps per day); Somewhat Active (7,500-9,999 steps per day); Active (10,000-12,499 steps per day); and, Highly Active (> 12,500 steps per day). Self-reported physical activity when the Fitbit was not worn resulted in a one category increase in estimated physical activity level and the resultant modifier was applied to RMR accordingly. The Fitbit was not used to collect any data other than step-count.
RMR (RMR·kg⁻¹) and TEE (TEE·kg⁻¹) were each normalized to body mass to be included in our analyses.
Energy consumption (EC; kcal·day\(^{-1}\)) was estimated based on a self-reported food and beverage intake record. This paper-based record was provided by the research team, and was to be completed on the day of the in-person assessment and on each of the following two days. Participants were provided verbal and written instructions, including examples, in order to optimize the information provided on these intake records.

Completed records were entered into the MyFitnessPal online portal (MyFitnessPal, Inc., Baltimore, MD) by a member of the research team (SRW). MyFitnessPal has been shown to yield similar results to traditional paper-based measures when estimating EC.\(^{25}\) EC was averaged across all three days for analyses and normalized to body mass (EC·kg\(^{-1}\)). Due to the novelty of this dietary recall instrument, we assessed the test-retest reliability as part of a larger study in our reference (matched controls) sample and found that all ICC values between time points (separated by 3-7 days) were $\geq 0.80$, indicating an acceptable level of stability.\(^{6}\) Energy balance (EBal) was calculated as the ratio between EC and TEE (EC/TEE). A value $> 1.0$ suggests that the participant reported consuming more energy than was expended (energy surplus) and vice versa (energy deficit). Missing data were not imputed for any variable in the present study.

**Procedures**

Participants were instructed to fast after midnight (12am) prior to reporting to the research laboratory between 6:00am and 9:00am, although water was allowed. A detailed health history form including demographic, concussion-related, and pertinent medical history questions was completed by each participant. Participants then completed the Revised Head Injury Scale (HIS-r), an inventory that consists of a 22-item symptom
checklist to indicate the presence of a concussion-related symptom. The HIS-r also contains Likert scales of symptom severity (0-6) and duration (1-6). The Likert scale for HIS-r symptom duration refers to the length of time (i.e., from “0” = 15 minutes to “6” = 24 hours) a participant experienced a concussion-related symptom during the 24 hours leading up to their assessment. Severity refers to the worst experience of a concussion-related symptom in that same 24-hour period. Participants self-reported only symptoms that were not typical for them in a normal day. Participants were then instructed to lay supine on a treatment table for 30 minutes while covered with a ventilated canopy to measure RMR. They were continually monitored by a member of the research team to ensure that they did not fall asleep during this assessment. The last five minutes of steady-state data from each session were used for our analyses.

Afterward, participants were provided a Fitbit, physical activity journal, and dietary intake record along with instructions for measuring and recording physical activity and dietary intake over three consecutive days, starting with the day of assessment. The decision to collect data for three days was made in order to account for daily variation while not placing undue burden on individuals with acute SRC. All data collection materials were returned to the PI at the end of their study participation. After returning these materials, participants were compensated with a $10 iTunes or Amazon gift card.

Statistical Analyses

Independent t-tests and Cohen’s d effect sizes were used to compare RMR·kg⁻¹, TEE·kg⁻¹, EC·kg⁻¹, and EBal between the full SRC and control groups, and then again separately for each sex. In the case that TEE·kg⁻¹ was different between groups,
exploratory Chi-squared ($\chi^2$) analyses of physical activity levels were performed. Within each group, Pearson ($r$) or Spearman ($\rho$) bivariate correlation analyses (depending on normality) were used to relate RMR·kg$^{-1}$, TEE·kg$^{-1}$, EC·kg$^{-1}$, and EBal with age, total quantity of symptoms (0-22), total symptom severity (the sum of all individually endorsed severities), and total symptom duration (the sum of all individually endorsed durations). To relate RMR·kg$^{-1}$, TEE·kg$^{-1}$, EC·kg$^{-1}$, and EBal to concussion history, Kruskal-Wallis tests were used to compare concussion history categories (0, 1, 2, 3+).

For exploratory purposes, we also performed separate multivariable linear regression analyses with backwards removal in each group. Lean body mass is a strong predictor of RMR, but was not measured in our participants. Instead, we included body mass index (BMI) in our correlation and regression models as a surrogate measure for body composition to help address any mediating effects body composition may have had. In a subset of collegiate participants with measured lean body mass data in our sample (combined from the SRC and control groups; $n = 36$), BMI and lean body mass were significantly correlated ($\rho = 0.80; p < 0.01$). There were two separate models fit with each group: the first model was with RMR·kg$^{-1}$ as the response variable; the second model was with EBal as the response variable. Age, number of prior concussions, sex, BMI, and the previously mentioned symptom outcomes each served as predictors in all models.

Criterion for stepwise removal of predictors from the exploratory regression models was a $p$-value greater than 0.10. These regression analyses were performed separately for the SRC and control groups.

Prior to correlation and regression analyses, we assessed the normality of each response and predictor variable of interest using Shapiro-Wilk’s test. Total symptom
duration was not normally distributed (Shapiro-Wilk = 0.891, \( p < 0.01 \)) and therefore Spearman (\( \rho \)) correlations were used to interpret its relationship with other outcomes. Total symptom duration was subsequently transformed by calculating its square root (Shapiro-Wilk = 0.953, \( p = 0.26 \)) for inclusion in the regression analyses. All analyses were performed in SPSS version 25 (Armonk, NY) with statistical significance set a-priori at \( \alpha \leq 0.05 \).

RESULTS

Demographics and Symptoms

A total of 56 participants were allocated to the SRC (\( n = 28 \)) and matched control (\( n = 28 \)) groups. Participants with SRC reported for their assessment (mean ± standard deviation) 2.0 ± 0.85 days after their injury. Demographics, concussion history, and HIS-r outcomes are presented by group, sex, and level of sport participation (college versus high school) in Table 1. The proportions of specific symptoms reported by participants with SRC are presented in Figure 1 for the full group and separately for each sex.

Group Comparisons

Overall, the SRC group had lower TEE·kg\(^{-1}\) (\( p < 0.01 \), \( d \) [95% confidence interval] = 1.01 [0.46, 1.01]) and higher EBal (\( p < 0.01 \), \( d = 0.72 \) [0.18,1.27]) than the control group (Table 2). Similarly, the SRC group was less physically active than the control group (\( \chi^2(5) = 19.847, p < 0.01 \); Figure 2). There were no group differences regarding RMR·kg\(^{-1}\) or EC. When stratified by biological sex, we observed that females with SRC had lower TEE·kg\(^{-1}\) (\( p < 0.01 \), \( d = 1.67 \) [0.81, 2.53]), higher EBal (\( p < 0.01 \), \( d \)
= 1.33 [0.51, 2.15]), and were less physically active than their control counterparts ($\chi^2(5) = 17.738, p < 0.01$). Similar to the full group, females with SRC did not differ from controls regarding $\text{RMR} \cdot \text{kg}^{-1}$ and EC. Unlike our female participants, males with SRC differed from their control counterparts only with regard to TEE $\cdot \text{kg}^{-1}$ ($p < 0.01, d [95\%$ confidence interval] = 0.90 [0.11, 1.69]) but not for the remaining outcomes of interest (all $p$’s > 0.31). Group averages for energy expenditure, EC, and EBal outcomes are presented in Table 2.

**Correlation Analyses**

In the SRC group, $\text{RMR} \cdot \text{kg}^{-1}$ was significantly correlated with TEE $\cdot \text{kg}^{-1}$ ($r = 0.56$, $r^2 = 0.31, p < 0.01$); and EBal was significantly correlated with TEE $\cdot \text{kg}^{-1}$ ($r = -0.46$, $r^2 = 0.21, p = 0.02$), EC $\cdot \text{kg}^{-1}$ ($r = 0.78$, $r^2 = 0.61, p < 0.01$), and age ($r = -0.42$, $r^2 = 0.18, p = 0.03$). Additionally, BMI was significantly correlated with age ($r = 0.39$, $r^2 = 0.15, p = 0.04$). Symptom outcomes were not significantly correlated with any other outcomes (all $p$’s > 0.10).

In the control group, $\text{RMR} \cdot \text{kg}^{-1}$ was significantly correlated with TEE $\cdot \text{kg}^{-1}$ ($r = 0.78$, $r^2 = 0.61, p < 0.01$) and BMI ($r = -0.50$, $r^2 = 0.25, p < 0.01$); and EBal was significantly correlated with TEE $\cdot \text{kg}^{-1}$ ($r = -0.65$, $r^2 = 0.42, p < 0.01$) and EC $\cdot \text{kg}^{-1}$ ($r = 0.77$, $r^2 = 0.59, p < 0.01$). Additionally, BMI was significantly correlated with age ($r = 0.57$, $r^2 = 0.32, p < 0.01$). Symptom outcomes were not included in correlation analyses for the control group as almost all participants did not endorse symptoms.

$\text{RMR} \cdot \text{kg}^{-1}$, TEE $\cdot \text{kg}^{-1}$, EC $\cdot \text{kg}^{-1}$, and EBal were not different across concussion history categories for either the SRC or control group ($p$’s > 0.05).
Exploratory Regression Analyses

In the SRC group, backward removal of predictors from the model for RMR·kg⁻¹ resulted in the removal of all predictors except for concussion history (final model adjusted $R^2 = 0.10$; Table 3). Regression of these same initial predictors onto EBal resulted in the inclusion of age, concussion history, BMI, total symptom severity, and total symptom duration in the final model (final model adjusted $R^2 = 0.54$; Table 3).

Biological sex was not significantly associated with either response variable in the SRC group. In the control group, RMR·kg⁻¹ was associated with both sex and BMI (adjusted $R^2 = 0.43$; Table 4); and there were no significant associations between EBal and any of the predictors of interest.

DISCUSSION

The key findings of this study were that RMR·kg⁻¹ was not different between the SRC and Control groups, although TEE·kg⁻¹ and EBal were. These findings are consistent with our related work with an overlapping sample.⁶ The current group differences in EBal appeared to be driven by lower TEE·kg⁻¹, and particularly physical activity levels, in SRC participants. Moreover, females with SRC had significantly higher EBal and lower TEE·kg⁻¹ when compared with their healthy counterparts, whereas males with SRC differed from their control group counterparts only regarding TEE·kg⁻¹, but not EBal.

Energy Expenditure
The lack of group differences in RMR·kg⁻¹ between groups was contrary to our hypotheses. We expected that RMR would be elevated in this sample given the extant evidence of a similar phenomenon in patients with moderate and severe TBI.¹¹,¹² In the Control group only, sex and BMI were associated with RMR·kg⁻¹, which is in keeping with known factors that influence RMR in healthy individuals.¹⁶ However, another unanticipated exploratory finding from this study was that history of concussion was not removed from the regression model for RMR·kg⁻¹ in the SRC group, while it was in the control group. More specifically, we observed a relationship between an increased number of prior concussions and greater energy expenditure (higher RMR·kg⁻¹) within 72 hours of sustaining an SRC; this relationship was not observed in the matched control sample without current SRC. This finding is suggestive of a more exaggerated resting-state metabolic response to a new SRC injury in those with one or more prior concussions.

It is presently unknown whether this exaggerated metabolic response is a marker of a residual pathophysiological consequence from previous injury,¹,²⁸ a protective adaptation to neurometabolic stress,²⁹,³⁰ or some combination thereof. If the response is a pathophysiological consequence, one plausible theory is that prior concussion(s) could physically damage glial cells or uncouple them from neurovascular junctions (e.g., blood-brain barrier) and synaptic clefts, where they are responsible for transportation of metabolites and neurotransmitters.²⁸ This could make restoring homeostasis following the neurometabolic cascade of events from acute SRC less energetically efficient, thereby deleteriously increasing energy demands. Inversely, if it is a protective adaptation, perhaps the elevated metabolic response to a new insult is in fact a facilitative response.
enabled through prior pathophysiological experience with an altered neurochemical environment.\textsuperscript{29,30} For instance, the body could have adapted from previous injury experiences after which more energy was required to restore homeostasis. Thus, the higher metabolic rate observed in our study in relation to a history of more prior concussions could have been a helpful response to the ionic change in the environment. Future studies should seek to identify whether metabolic changes following SRC are a maladaptive or facilitative response to new injury.

\textit{Energy Balance}

While our observation of lower physical activity levels after SRC is consistent with the clinical recommendations at the time these injuries occurred,\textsuperscript{13,22} our data also suggest that participants with SRC consumed a surplus of energy relative to their estimated energy expenditure needs. We observed that participants with SRC had EBal values that were approximately 26\% higher than those in the control group. This disparity between groups was higher in the female participants (37\%) than the male participants (17\%). In our related work, college-aged female student-athletes with SRC exhibited higher EBal initially following their injury compared to their male counterparts, and those females also reported symptom resolution and return to unrestricted sport activity multiple days sooner than males.\textsuperscript{6} In combination, these findings emphasize the need to understand the role of EBal in response to SRC and how sex may influence this relationship.

Importantly, if greater EBal (greater energy surplus) is associated with quicker recovery from SRC, then therapeutic dietary intervention should be investigated.
Presently, there are no published data regarding the utility or effectiveness of dietary interventions for SRC. The 2014 National Athletic Trainers’ Association position statement on the management of sport-related concussion recommended that athletes consume a well-balanced diet following injury, and there have been no further recommendations or empirical evidence to update the present recommendation to date.\textsuperscript{31} It is beyond the scope of the current study to provide specific recommendations to clinical practice; however, athletic trainers and other clinicians that manage patients with SRC should view the current data as sufficient enough to encourage monitoring of post-injury diet to ensure that it is at least in accordance with the recommendation put forth by the NATA position statements.

In the exploratory regression model for EBal in the SRC group, we observed that younger age, fewer prior concussions, lower BMI, lower total symptom severity, and higher total symptom duration were associated with higher EC in relation to TEE. This model was estimated to explain 54\% of the variance associated with EBal. The associations with age and BMI were not surprising as these factors are also interrelated with RMR, both in the current study and elsewhere.\textsuperscript{16} The association of concussion history with EBal was also anticipated given our findings that concussion history had a positive association with RMR·kg$^{-1}$. Regarding symptom burden, total symptom severity and total symptom duration had opposing relationships with EBal. Those experiencing nausea may have altered their eating behaviors which could have affected EC in this sample, though only 30\% ($n = 8/27$) of our sample endorsed this symptom within the 24 hours leading up to their assessment with varying self-reported degrees of severity. It is unknown whether the most frequently experienced symptoms in our sample (headache,
sensitivity to light, cognitive- and sleep-related symptoms; Figure 1) had any influence on appetite or eating behavior. The relationships between symptom burden and EBal could possibly have been moderated by symptom influences on participation in physical activity rather than on EC or RMR directly. It is important to also note that symptom severity and duration were rated based on the participant’s experience within the prior 24 hours leading up to their assessment, while EC was reported on the day of assessment and subsequent 2 days. It is possible that symptom experiences may have improved over the subsequent days, and this was not measured in the current study. In this light, the impact of specific symptoms and/or symptom clusters on EC and energy expenditure following SRC warrant further study.

Limitations

A notable limitation of the current study is the timing in which participants with SRC reported for assessment after their injury. Given the rapidly evolving nature of the pathophysiological response to SRC, participants assessed 24 hours after injury may have been in a different physiological state than those measured at 72 hours after injury. There were not enough participants at each time point (24, 48, and 72 hours after injury) to determine whether or not there was an effect of time since injury, but this should be considered in future studies. Further, there have been no prior studies investigating energy expenditure, energy intake, or energy balance after SRC. As these data are from the first study of its kind, we were unable to conduct a power analysis prior to planning the analyses for the current study. Therefore, findings from this study should be interpreted as preliminary evidence.
Measurement of physical activity in our study may have been under- or over-estimated. Many control participants did not wear the Fitbits during their normal athletic activities (e.g., lacrosse games) and the exact amount of energy expended during each of these sporting activities was not determined. However, we incorporated participant-reported activities into our estimates of TEE. EC was assessed according to self-reported dietary intake of specific foods and beverages. Self-reported food and beverage intake has inherent risks of poor compliance, biased reporting (typically under-reporting of energy consumption), and the potential to unintentionally influence eating behavior.\textsuperscript{32,33} Especially in concussed, young individuals, the effects of SRC symptoms on a subject’s ability to accurately report all foods consumed may also have affected our EC estimates. However, if subject’s symptoms affected reporting ability, the direction would have been towards underreporting. In this case, our results would have underestimated the amount of energy consumed, and subsequently, the EBal estimates. In alignment with clinical recommendations, we designed our study in a manner that would not be over burdensome for our injured participants. For example, visual disturbances are a common symptom following SRC. While using an electronic device such as a smartphone to either take a photo of each meal a participant may consume, or to record intake via a smartphone application, it may have placed undue stress on our injured participants resulting in the exacerbation of symptoms. Future studies should consider a more robust approach, such as measured plate-waste food intake protocols in combination with providing standardized meals to participants to better understand the influence of SRC on dietary intake behavior and the potential for a therapeutic intervention to optimize EBal. Nevertheless, to circumvent inaccurate reporting behaviors, we verbally encouraged all
participants to report their EC as accurately and consistently as possible and we provided specific instructions and examples on the dietary intake journal.

Conclusions

Our study was an important first step in the examination of the relationships between demographic, clinical, metabolic, and energy consumption-related outcomes following SRC. Student-athletes with acute SRC self-reported consumption of an energy surplus acutely following injury relative to their physical activity levels (energy expenditure), and this relationship was exaggerated in females compared to males. Within 72 hours of SRC, estimated EBal was associated with sex, age, concussion history, BMI, and symptom burden. Our results also suggest that more prior concussions may increase whole-body energy expenditure following a new SRC. Moving forward, future studies should investigate whether clinical interventions with diet can modify the physiologic response to SRC and improve patient outcomes by reducing symptom burden and/or lessening the amount of time removed from sport.
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LEGENDS TO FIGURES

Figure 1. Proportions of Endorsed Symptoms in the Sport-Related Concussion Group ($n = 27$). No participants endorsed vomiting and therefore it was not included in this figure. Additionally, one female and no males experienced numbness and tingling, and those symptoms were also not included in this figure. One participant did not complete the symptom assessment during their study visit. The five symptom domains are similar to those in a recent study by Howell et. al.$^{20}$

Figure 2. Physical Activity in Acutely Concussed ($\leq$ 72 hours after injury) and Matched Control Participants. Physical activity was self-reported for the day of assessment and the following 2 days by the participants according to step counts (via Fitbit) and activities performed when wearing the Fitbit was not permissible (e.g., football game). Physical activity in the SRC group ($n = 26$) was significantly less than in the Control group ($n = 28$) ($\chi^2(5) = 19.847, p < 0.01$). Two participants in the SRC group did not provide physical activity information.
Table 1. Demographics, Concussion History, and Self-Reported Symptoms. *Only two of the three high school females in the SRC group completed the symptom assessment; therefore median, minimum and maximum values are presented. SD = standard deviation; IQR = interquartile range.

|                        | Full group n = 28 | Males n = 14 | Females n = 14 | College males n = 9 | College females n = 11 | High school males n = 5 | High school females n = 3 | Full group n = 28 | Males n = 14 | Females n = 14 | College males n = 9 | College females n = 11 | High school males n = 5 | High school females n = 3 |
|-----------------------|------------------|--------------|---------------|--------------------|------------------------|------------------------|------------------------|------------------|--------------|---------------|----------------------|------------------------|------------------------|------------------------|
| **Age (years)**       |                  |              |               |                    |                        |                        |                        |                  |              |               |                      |                        |                        |                        |
| mean (sd)             | 18.4 (1.8)       | 18.6 (1.8)   | 18.2 (1.9)    | 19.7 (1.0)         | 19.0 (1.1)             | 16.8 (1.3)             | 15.3 (1.5)             | 19.6 (2.9)       | 19.9 (3.4)   | 19.0 (2.2)    | 21.8 (2.6)            | 19.9 (1.3)             | 16.4 (1.5)             | 15.7 (1.5)             |
| **Height (meters)**   |                  |              |               |                    |                        |                        |                        |                  |              |               |                      |                        |                        |                        |
| mean (sd)             | 1.76 (0.10)      | 1.82 (0.08)  | 1.70 (0.09)   | 1.85 (0.08)        | 1.70 (0.10)            | 1.76 (0.07)            | 1.69 (0.10)            | 1.76 (0.08)      | 1.80 (0.07)  | 1.72 (0.08)   | 1.82 (0.08)            | 1.72 (0.05)            | 1.77 (0.03)            | 1.73 (0.03)            |
| **Weight (kilograms)**|                  |              |               |                    |                        |                        |                        |                  |              |               |                      |                        |                        |                        |
| mean (sd)             | 75.6 (21.0)      | 86.6 (23.7)  | 64.5 (9.3)    | 96.5 (24.1)        | 65.7 (10.2)            | 69.0 (7.6)             | 60.0 (2.1)             | 77.5 (21.2)      | 87.7 (24.8)  | 67.4 (9.6)    | 97.6 (26.1)            | 69.1 (9.7)             | 69.7 (3.5)             | 61.1 (7.3)             |
| **Body mass index (kg·m⁻¹)** |             |              |               |                    |                        |                        |                        |                  |              |               |                      |                        |                        |                        |
| mean (sd)             | 24.1 (4.1)       | 25.9 (5.0)   | 22.2 (1.7)    | 28.0 (5.2)         | 22.5 (1.7)             | 22.2 (0.9)             | 21.1 (1.2)             | 24.7 (4.8)       | 26.7 (5.8)   | 22.7 (2.3)    | 29.1 (5.9)            | 23.3 (2.0)             | 22.3 (1.4)             | 20.5 (2.2)             |
| **Concussion history (#)** | 1 (0-6)   | 1 (0-6)     | 0.5 (0-3)    | 1 (0-6)            | 1 (0-3)                | 0 (0-2)                | 0 (0-2)                | 0 (0-3)          | 0.5 (0-2)    | 0 (0-3)       | 0 (0-2)               | 0 (0-3)                | 1 (0-2)                | 0 (0-0)                |
| **Total number of symptoms** | 8 (6-11) | 8 (5.5-11.5)| 7.5 (6-11)   | 10 (7.5-12)        | 8 (6-11)               | 5.5 (3.5-6.75)         | 7 (7-7)                | 0 (0-0)          | 0 (0-0)      | 0 (0-0)       | 0 (0-0)               | 0 (0-0)                | 0 (0-0)                | 0 (0-0)                |
| **Total Symptom Duration** | 22 (16-36) | 23 (15-34) | 22 (17.75-38.25) | 22 (15-36) | 18.5 (6.25-23.25) | 19 (18-20) | 0 (0-0) | 0 (0-0) | 0 (0-0) | 0 (0-0) | 0 (0-0) | 0 (0-0) | 0 (0-0) |
| **Total Symptom Severity** | 19 (12-26) | 15 (6.5-26) | 19 (14.75-17.25) | 19 (14.27-17.25) | 10 (3.5-18) | 15.5 (15-16) | 0 (0-0) | 0 (0-0) | 0 (0-0) | 0 (0-0) | 0 (0-0) | 0 (0-0) | 0 (0-0) |
Table 2. Energy Expenditure, Consumption, and Balance. All values are presented as means ± standard deviations. Values were normalized by dividing absolute measures by participant body mass. RMR = Resting Metabolic Rate; TEE = Total Energy Expenditure; EC = Energy Consumption; Energy Balance = EC/TEE.  

|                           | Sport-Related Concussion (SRC) Group | Matched Control Group |
|---------------------------|--------------------------------------|-----------------------|
|                           | Full group n = 28                     | Full group n = 28     |
|                           | Males n = 14                          | Males n = 14          |
|                           | Females n = 14                        | Females n = 14        |
|                           | Collegiate n = 20                     | Collegiate n = 20     |
|                           | High school n = 8                     | High school n = 8     |
| RMR (kilocalories per day)| 1076.6 (306.1)                        | 1311.1 (246.1)        |
| Normalized RMR (kilocalories per kilogram per day) | 14.3 (2.12) | 15.4 (2.45) |
| TEE (kilocalories per day)| 1683.1 (652.1)                        | 2087.4 (617.8)        |
| Normalized TEE (kilocalories per kilogram per day) | 21.9 (4.72) | 27.2 (5.69) |
| EC (kilocalories per day)| 2069.1 (693.7)                        | 2121.9 (810.4)        |
| Normalized EC (kilocalories per kilogram per day) | 28.2 (8.38) | 27.3 (6.62) |
| Energy balance (ratio)    | 1.33 (0.42)                           | 1.05 (0.35)           |

Concussed group significantly lower than controls (p < 0.02).  
Concussed group significantly higher than controls (p < 0.01).
Table 3. Final Regression Models for Normalized Resting Metabolic Rate (RMR·kg\(^{-1}\)) and Energy Balance (EBal) in the Sport-Related Concussion Group \((n = 27)\). One participant did not complete the symptom assessment (Revised Head Injury Scale [HIS-r]) during their study visit and they were therefore not included in these regression analyses.

| Predictor variable                  | Unstandardized beta | Standardized beta | p-value |
|-------------------------------------|---------------------|-------------------|---------|
| **Normalized Resting Metabolic Rate (RMR·kg\(^{-1}\))** |                     |                   |         |
| Constant                            | 13.45               |                   |         |
| Concussion history                  | 0.65                | 0.37              | 0.06    |
| **Energy Balance (EBal)**           |                     |                   |         |
| Constant                            | 2.07                |                   |         |
| Age                                 | -0.08               | -0.35             | 0.06    |
| Concussion history                  | -0.13               | -0.35             | 0.04    |
| Body mass index                     | -0.03               | -0.32             | 0.09    |
| Total symptom severity              | -0.06               | -1.59             | < 0.01  |
| Square root of total symptom duration | 0.54               | 1.50              | < 0.01  |
Table 4. Final Regression Model for Normalized Resting Metabolic Rate (RMR·kg^{-1}) in the Control Group (n = 28). There were no significant associations between any of the predictor variables and EBal in the control group. Symptom outcomes were not included in these analyses as only 3 control participants reported experiencing symptoms at the time of their study visit.

| Predictor variable                | Unstandardized beta | Standardized beta | p-value  |
|----------------------------------|---------------------|-------------------|----------|
| Normalized Resting Metabolic Rate (RMR·kg^{-1}) |                     |                   |          |
| Adjusted $R^2 = 0.43$            |                     |                   |          |
| Constant                         | 25.65               |                   |          |
| Sex (Male = 1, Female = 2)       | -2.10               | -0.53             | < 0.01   |
| Body mass index                  | -0.31               | -0.72             | < 0.01   |
Figure 2.

![Bar chart showing the level of physical activity among Concussed and Control groups.](http://meridian.allenpress.com/jat/article-pdf/doi/10.4085/359-20/2632270/10.4085_359-20.pdf)

**Level of Physical Activity**

(According to Heyward et al. [27])

- **Sedentary**
- **Lightly Active**
- **Moderately Active**
- **Very Active**
- **Exceptionally Active**

Legend:
- ■ Concussed
- □ Control