**Glycemic Patterns of Male Professional Athletes With Type 1 Diabetes During Exercise, Recovery and Sleep: Retrospective, Observational Study Over an Entire Competitive Season**

**Short title:** Glycemia in Athletes with Type 1 Diabetes

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**Article highlights**

* The objective of this study was to analyze the glycemic control of 12 male professional cyclists with type 1 diabetes over a competitive season.
* Despite the substantial physical demands of professional endurance exercise, the athletes showed remarkably good glycemic control.
* Glycemic patterns were characterized by hyperglycemia during competitive exercise and nocturnal hypoglycemia after non-competitive exercise.
* Particular emphasis should be put on intensified monitoring during competitive exercise, but also during nights after non-competitive exercise, particularly after endurance training. This may optimize glycemic control, competitive performance, and recovery.

**Twitter summary**

Glycemic control of 12 professional male cyclists was analyzed over a competitive season. Despite the enormous demands of endurance exercise, glycemic control was considerable, albeit characterized by increased frequencies of hyperglycemia during races and nocturnal hypoglycemia after non-competitive exercise.

**ABSTRACT**

**Objective:** To analyze glycemic control of professional athletes with type 1 diabetes during a competitive season.

**Research Design and Methods:** We analyzed continuous glucose monitoring data of 12 professional male cyclists with type 1 diabetes during exercise, recovery, and sleep on days with competitive exercise (CE) and non-competitive exercise (NCE). We compared results with consensus targets, assessed differences between CE and NCE days, and analyzed associations between exercise and dysglycemia.

**Results:** Mean HbA1c was 6.7±0.5% (50±5 mmol/mol). There were 280.8±28.1 days of cycling. Overall, time in range (70–180 mg/dL) was 70.0±13.7%, time in hypoglycemia (<70 mg/dL) was 6.4±4.7%, and time in hyperglycemia (>180 mg/dL) was 23.6±12.5%. During NCE days, time in range was 71.0±13.8%, time in hyperglycemia was 22.2±12.1%, while time in hypoglycemia was 6.9±5.0%. This was related to an increased time in hypoglycemia overnight (10.1±7.4%), exceeding consensus targets (*p*=0.008). The probability of hypoglycemia during sleep was particularly increased after exercise in the endurance zones (*p*<0.05). CE days revealed a time in range of 70.1±14.1%, a time in hypoglycemia of 4.7±4.5%, but an increased time in hyperglycemia (25.2±12.5% vs. 25%, *p*=0.012). Time in hyperglycemia during CE was higher than during NCE (38.5±12.9% vs. 21.9±13.9%, *p*<0.001), exceeding consensus targets (*p*=0.003). The probability of hyperglycemia during exercise was particularly increased with longer duration, higher intensity, and higher variability of exercise (*p*<0.001).

**Conclusions:** While overall glycemic control of these professional athletes is generally within range, further improvements could be achieved by reducing hyperglycemia during competitions and nocturnal hypoglycemia after non-competitive exercise.

**INTRODUCTION**

Regular physical exercise is a key component in the management of type 1 diabetes (1-2), associated with increased longevity and improved cardio-metabolic health (3). Despite significant advances in supportive technology and guidance (1,4), exercise poses considerable challenges for people with type 1 diabetes, with fear of hypoglycemia and inadequate knowledge around exercise management being major barriers (5-6). Nevertheless, there are examples of people living with type 1 diabetes that undertake ultra-endurance exercise on a regular basis (7-12), some of them even competing at a professional level (e.g., 7-8).

Current guidelines to optimize exercise-related glycemia suggest insulin dose adaptation and ingestion of additional carbohydrates in the context of physical exercise (4,13). These recommendations are largely based on findings from laboratory-based studies and clinical experience of moderately trained individuals undertaking exercise of limited duration. Yet, the applicability to prolonged endurance exercise is less well known. This is partly due to the fact that reports on endurance exercise of prolonged duration in people with type 1 diabetes have only been provided over a short period of time, such as an individual race or training period (7-12), meaning that there is little research describing the associated glycemic control in endurance athletes living with type 1 diabetes beyond a single training camp or competitive event.

Developments in technology to monitor glucose (e.g., continuous glucose monitoring (CGM)) and physical exercise (e.g., power meters and heart rate monitors) enable readily available measurement of factors related to glycemic management and exercise behavior, regardless of the training or competition setting and environment. Combined monitoring of both glucose and exercise performance for a prolonged study period may allow for novel and expanded insights into the effects of training and competition on glycemia and the various challenges athletes may face in combining high-performance exercise with type 1 diabetes.

In this retrospective observational study, we assessed the glycemic control of professional cyclists with type 1 diabetes over an entire competitive season (Oct 2018 – Oct 2019). The objective of this study is twofold: (i) to assess glycemic control during phases of competitive exercise (CE) and non-competitive exercise (NCE), recovery, and sleep; and (ii) to analyze the association of specific exercise-related factors with the occurrence of hypo- and hyperglycemia during these phases.

**RESEARCH DESIGN AND METHODS**

**Participants**

This study included data from 12 male professional road cyclists with type 1 diabetes from Team Novo Nordisk over a competitive season (Oct 2018 – Oct 2019). The study was performed in accordance with the Declaration of Helsinki. Ethical approval was obtained from the Canton of Bern ethics committee (Project ID: 2019-01988). All participants provided both verbal and written informed consent. Participant eligibility criteria are reported in Supplemental Figure 1.

**Data collection**

Continuous glucose monitoring (CGM) was performed using Dexcom G6 (Dexcom, San Diego, CA) throughout the study period. HbA1c was measured quarterly (central measurement).

During cycling sessions, participants were equipped with a mobile power meter (SGYPM900H90, Pioneer, Aliso Viejo, CA), a cycling computer (Wahoo Fitness, Atlanta, GA), and a chest strap heart rate monitor (Wahoo Fitness, Atlanta, GA). The following data were monitored at a 1Hz frequency: power output [W], cadence [rpm], heart rate (HR) [bpm], temperature [°C], latitude [°N], longitude [°E], distance [km], and altitude [m]. Data collected from these sensors were exported daily to a central database (TrainingPeaks, Peaksware LLC, Louisville, CO).

Data collected from cycling sessions were aggregated by day to capture causes of physiological effects of exercise: duration [min], intensity factor, and variability index, as well as time in heart rate- and power zones [min] based on individualized Coggan heart rate- and power zones (14) (see Supplemental Table 1 for definitions and further details). If more than one exercise session had taken place during a day, data from the exercise sessions were aggregated cumulatively (i.e., through concatenation). To obtain ventilatory and metabolic thresholds, incremental cardiopulmonary exercise (CPX) tests were performed biannually on the athlete’s personal bike (Colnago C60, Colnago, Italy) attached to a cycle trainer (KICKR, Wahoo, Atlanta, GA). Together with collected anthropometric data, such as height [cm], body mass [kg], and fat mass [%], these tests yielded the maximum rate of oxygen consumption (V̇O2max) [mL/min/kg], maximum heart rate (HRmax) [bpm], lactate threshold heart rate (LTHR) [bpm], and functional threshold power (FTP) [W/kg].

Additionally, individual travel and competition calendars were collected to differentiate between CE and NCE sessions and adjust for relevant modifying factors.

**Analysis of glycemia**

CGM data were analyzed in accordance with the international consensus statement (15). For each participant, we calculated the mean glucose [mg/dL], glycemic variability expressed as coefficient of variation (CV) [%], time in hypoglycemia (L1: 54–69 mg/dL, L2: <54 mg/dL), time in range (70–180 mg/dL), and time in hyperglycemia (L1: 181–250 mg/dL, L2: >250 mg/dL). These statistics were calculated for five phases of the day: (i) the entire day (06:00–06:00h); (ii) wake (06:00–00:00h); (iii) exercise (corresponding to the times of the day that participants recorded cycling exercise; for further analyses, exercise is separated into competitive and non-competitive exercise); (iv) recovery (corresponding to the 4 hours after exercise, excluding times during subsequent exercise); and (v) sleep (00:00–06:00h). Phases (i), (ii), and (v) correspond to the time frames defined in the international consensus statement (15), but may not correspond to the actual wake and sleep times of participants. Statistics on glycemia were calculated for all days that met the inclusion criteria in Supplemental Figure 1, thus also comprising days without exercise.

Summary statistics of glycemic metrics across patients are reported as mean ± SD and compared with internationally accepted target values (16) using one-sided one-sample *t*-tests. Glycemic metrics are compared between CE and NCE days using paired *t*-tests.

**Association of exercise-related factors with dysglycemia**

To explore the potential association of specific exercise-related factors (i.e., duration, intensity factor, variability index, and time in different heart rate/power zones) with the occurrence of hypo- and hyperglycemia (<70 mg/dL and >180 mg/dL, resp. for duration of at least 15 minutes (15)), we applied multilevel, multivariate logistic regression models (see Supplemental Methods) for exercise, recovery, and sleep phases, respectively. To account for participant-specific variation in physiology and glycemia, random intercepts and random slopes were incorporated in the models. The models were fitted for each exercise variable separately, adjusting for the effects of environmental variables. Environmental variables were average temperature [°C] during exercise, average altitude [m] during exercise, day in the competitive season (counting from the start of the competitive season and accounting for a trend over the competitive season), and whether travel was undertaken in the current or the previous two days. Further details on the independent variables in this analysis are provided in Supplemental Table 1.

Prior to analysis, we standardized independent variables to enable comparisons of the strength of the associations. Variables with more than 30% missing values were excluded. Additionally, time in heart rate- and power zones were excluded if more than 80% of their values were zero. Missing values in all remaining variables were imputed with their mean. Estimated standardized associations are reported as the odds ratio (OR) [95% confidence interval (CI)].

**Data and resource availability**

The datasets generated during and/or analyzed in the current study are not publicly available to protect privacy of the participants, but are available in limited form from the corresponding author upon reasonable request. Code for the analysis of the current study in Python (version 3.7.5) and R (version 3.4.4) is available after publication under <https://github.com/im-ethz/TNN-analysis>.

**RESULTS**

**Participants**

Participant characteristics are summarized in Table 1. Mean±SD age was 25.6±4.4 years, duration of type 1 diabetes was 10.4±4.9 years, and HbA1c was 6.7±0.5% (50±5 mmol/mol). All participants were using a basal-bolus insulin regimen administered via multiple daily injections (MDI). On average, the participants cycled on 280.8±28.1 days during the study period, of which 34.7±15.3 days were during a competitive event.

**Analysis of glycemia**

Continuous glucose monitoring was analyzed for a total of 2,115 days, where we distinguished between NCE (1,536 days) and CE (256 days). Table 2 gives an overview of glycemic metrics, including the statistical comparisons against consensus targets and between CE and NCE days. Figure 1 shows the glucose concentrations over time during the exercise, recovery and sleep phases for NCE and CE days.

Overall, over the entire day (06:00-06:00h), the percentage of time in range (70–180 mg/dL) was 70.0±13.7%, time in hypoglycemia (<70 mg/dL) was 6.4±4.7%, time in hyperglycemia (>180 mg/dL) was 23.6±12.5%, and glycemic variability expressed as coefficient of variation (CV) was 38.9±8.5% (Table 2). Glycemic control did not differ significantly from the consensus targets as suggested by general guidelines (16). However, when looking at individual data, the target for time in range (i.e., >70%) was met by 5 out of 12 participants, the target for time in hypoglycemia (i.e., <4%) was met by 4 out of 12 participants, and the target for time in hyperglycemia (i.e., <25%) was met by 6 out of 12 participants (Supplemental Table 2).

During NCE days, time in range was 71.0±13.8%, time in hyperglycemia was 22.2±12.1%, but time in hypoglycemia was higher than suggested by consensus targets (6.9±5.0% vs. 4%, *p*=0.035). This related to a higher time in hypoglycemia during sleep after NCE, exceeding consensus targets significantly (10.1±7.4% vs. 4%, *p*=0.008). CE days were characterized by a time in range of 70.1±14.1%, a trend towards lower time in hypoglycemia when compared with NCE days (4.7±4.5% vs. 6.9±5.0%, *p*=0.073), and a higher time in hyperglycemia (25.2±12.5% vs. 22.2±12.1%, *p*=0.012). This was essentially driven by CE itself, where time in range dropped to 60.4±13.0%, which was below targets (*p*=0.017), and time in hyperglycemia was significantly elevated and above target (38.5±12.9% vs. 25%, *p*=0.003), while there were virtually no hypoglycemias during CE (1.1±1.4%). Accordingly, this translated into a significantly higher mean glucose concentration during CE than during NCE (172.3±16.3 mg/dL vs. 140.8±20.1 mg/dL, *p*<0.001).

The 4-hour post-exercise recovery phase was characterized by comparably high mean glucose (153.1±23.4 mg/dL). Of note, on CE days, mean glucose concentrations were significantly higher in recovery compared to NCE days (161.0±22.6 mg/dL vs. 151.3±23.8 mg/dL, *p*=0.041), especially in the first hours after exercise (Figure 1).

The sleep phase showed a time in range of 69.5±17.3%. During sleep, time in hypoglycemia was higher than consensus targets (9.6±7.2% vs. 4%, *p*=0.010). This pattern was mainly seen on NCE days, where time in nocturnal hypoglycemia exceeded guidelines significantly (10.1±7.4% vs. 4%, *p*=0.008). Still, targets for time in range overall during sleep were met by 7 out of 12 participants.

**Association of exercise-related factors with dysglycemia**

The associations of exercise-related factors with the occurrence of hypo- and hyperglycemia during exercise, recovery, and sleep phases are shown in Figure 2. Overall, competitive exercise, higher variability index, and time in the highest power zones were associated with a decreased probability of hypoglycemia (Figure 2a). Conversely, exercise of longer duration, higher intensity factor, and higher variability index was associated with increased probability of hyperglycemia during exercise (*p*<0.001 for all comparisons, Figure 2a). Time in higher heart rate zones, and in higher power zones were positively associated with the probability of hyperglycemia during exercise, and hyperglycemia during exercise was significantly more probable during competitions (*p*<0.001, Figure 2a).

During the recovery phase, the probability of hyperglycemia were reduced with longer duration of previous exercise (*p*=0.002, Figure 2b), while variability index of exercise was associated with higher probability of hyperglycemia (*p*=0.034, Figure 2b). The association between time in higher power zones and probability of hyperglycemia was non-linear, revealing decreased probability for power zones 2 and 3 (endurance and tempo). This pattern was confirmed for heart rate zones, where zones 2 and 3 were also negatively associated with the probability of hyperglycemia.

The probability of hypoglycemia during sleep were significantly increased after exercise in the endurance zones (both for power and heart rate, Figure 2c).

**CONCLUSIONS**

This is the first study to report on the glycemic control of professional athletes with type 1 diabetes over a prolonged study period of an entire competitive season. The most important findings of this analysis are four-fold. First, despite the high physical demands and related challenges of professional high-performance endurance exercise, the athletes revealed a considerable management of glycemic control, achieving an overall time in target range of 70%, which is the general consensus target for individuals with type 1 diabetes. Second, competitive exercise (CE) was associated with a significantly higher time in hyperglycemia compared to non-competitive exercise (NCE). Of note, hyperglycemia both during competitive- and non-competitive exercise was significantly associated with longer exercise duration, higher intensity factor, and higher variability index. Third, the post-exercise recovery period was characterized by comparably high glucose concentrations, particularly in the first hour after CE. Fourth, sleep phases revealed a significant percentage of time spent in hypoglycemia, particularly after NCE days. Nocturnal hypoglycemia was more frequent after exercise performed in the endurance zones.

Given the enormous challenges associated with the life of a professional cyclist, the average time in range (70.0±13.7%) and HbA1c (6.7±0.5%) of these athletes is considerable. This may reflect the fact that the study participants have generally developed strategies to manage glycemia during different situations (i.e., training, competition, recovery, etc.). On the other hand, the present findings may indicate that there is still room for improvement. Furthermore, our results may give information on specific aspects that athletes, team staff, and caregivers may want to focus their attention upon.

During CE, we observed significantly elevated glucose concentrations compared to NCE, along with more time spent in hyperglycemia. These findings may be attributed to several factors. First, during competition, strict avoidance of hypoglycemia is vital to avoid direct withdrawal from the race. Moreover, hypoglycemia during a competition is associated with an imminent risk of decreased performance along with a significantly increased risk of accidents (4). As a consequence, the athletes may deliberately aim for safety margins by increasing glucose values to higher levels before and during a race. Second, during competition, the athletes are generally advised to follow a diet that is higher in carbohydrate content than during training (17), because energy demands are high and it is considered to be metabolically more efficient when competing under high carbohydrate oxidation to optimize performance (18-19). While data on nutrition and insulin doses were unavailable for this study, previous reports of the Team Novo Nordisk athletes revealed an average consumption of 76±23 g/h carbohydrates during a 7-day World Tour stage race (7). In training camp conditions, a group of athletes from the same team consumed on average 41.9±6.8 g/h of carbohydrates (12). Third, the competitive aspect of a race is known to cause stress-induced changes in glucose metabolism, mainly mediated by an elevation in counter-regulatory stress hormones (20), resulting in substantial hyperglycemia.

During the 4-hour post-exercise recovery window, there was a distinct pattern of elevated glucose concentrations, particularly in the first hour. Values were significantly higher on CE days compared to NCE days. This may reflect the intake of substantial amounts of carbohydrates post-exercise, and in particular after competitions, to replenish glycogen stores, along with cautious application of insulin due to increased insulin sensitivity. The consistent pattern of post-exercise hyperglycemia suggests that athletes may benefit from implementing a more structured post-exercise recovery routine to not only help manage glycemia but optimize training adaptation and recovery (21).

Our data suggests that hypoglycemia is a substantial danger in the overnight periods, in particular after NCE. Given that these athletes spend the majority of the season in individual training, this merits particular consideration and calls for measures to improve monitoring and to reduce risks. The percentage of time spent in hypoglycemia during the nocturnal period is even more concerning, considering the dangerous repercussions of hypoglycemia (22-23) and the increased risk of subsequent hypoglycemia due to blunted counterregulatory response the following day (24), increasing risks for accidents. In addition, sleep is critical for athletic performance, particularly regarding the regenerative processes and adaptations following training and competition (25-28). As sleep deficiency has been associated with a range of negative health outcomes (29-30) and impaired athletic performance (28), greater attention should be given to improving overnight glycemic control (especially focused on reducing hypoglycemia), to optimize sleep in athletes with type 1 diabetes.

The challenges highlighted by this analysis, particularly in the post-exercise period, raise the question as to whether changing insulin therapy from MDI to an automated insulin delivery (AID) system or other hybrid insulin regimen may optimize glycemic control in athletes with diabetes. Hybrid closed-loop systems are generally known to increase time in range and quality of life of users (31-33). While exercise continues to be a challenge for hybrid closed-loop systems due to the rapid increase in insulin sensitivity and changes in contraction-mediated glucose uptake into the skeletal muscles, there is growing evidence on the efficacy of AID systems during and after exercise (34-36). Studies demonstrate improved time in range overnight and during exercise with AID systems (36-37), even under demanding environmental conditions and without pre-planning of exercise (38-39). An alternative to AID systems has been suggested by an “untethered” approach with a hybrid regimen of injected insulin and continuous subcutaneous insulin infusion, and with pump removal during exercise (40). While a recent study by Aronson et al. (40) provides evidence that a hybrid “untethered” approach may represent a safe and effective insulin delivery regimen, it has yet to be formally tested in athletes with type 1 diabetes in a competitive setting.

The key strength of the present study is the fact that we report on a unique group of professional athletes with type 1 diabetes who engaged in highly competitive endurance exercise over an entire competitive season. Our large dataset provides direct insights into the glucose control of professional endurance athletes with type 1 diabetes during a significant number of competitive and non-competitive events. Taking into account specific factors related to exercise and analyzing results according to specific phases (exercise, recovery, and sleep) further allowed identifying situations or settings with negative impact on glycemic control. Nevertheless, we acknowledge certain limitations. First, our findings are based on observational data and are not interventional assessments. For this reason, we emphasize that we only report associations, and not causal effects, of individual exercise variables with dysglycemia. Second, while the amount of data was large, the sample size was limited and exclusively consisted of young males, thus limiting the generalizability of our findings. Third, we did not have access to insulin and nutritional data in our study. Therefore, we cannot conclude whether the underlying causes of the observed associations of exercise variables with dysglycemia were insulin-, nutrition-, or exercise-related changes in metabolism, or other unknown factors. Future research may investigate other characteristics of dysglycemia, such as length or severity of dysglycemic episodes.

In conclusion, we provide insights into the glycemic control of a group of professional athletes with type 1 diabetes over an entire competitive season. While overall glycemic control was within range, the substantial challenges associated with the life of a professional athlete with type 1 diabetes may explain that consensus targets were not always reached. Specific emphasis should be put on intensified monitoring and treatment during competitions and overnight periods, not only to further improve training adaptation, but to reduce short-term and long-term risks for athletes with type 1 diabetes.

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**Conflict of Interest**

None

**Author Contributions and Guarantor Statement**

EW, FW, SNS and CS contributed to the design of the study. EW, FF, KS, CH and SNS contributed to data collection. EW, NB, SFö, MK, FF, SFe and FW contributed to data analysis. EW, FF, TZ, SFe, VL, SNS, FW, CS contributed to the interpretation of study results. EW and SNS prepared the first draft of the manuscript and all authors reviewed and approved the manuscript. CS is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

**Prior publication**

A part of this work will be presented at the conference on Advanced Technologies & Treatments for Diabetes (ATTD 2023), 22-25 February 2023, Berlin, Germany.

**Patient and Public Involvement Statement**

The cyclists participating in this study, and the rest of the Team Novo Nordisk staff, have been aware of our research team and its aims since January 2019. As the current study was an observation of current practices, every effort was made to avoid burdening the cyclists through researcher activity.

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**FIGURE LEGENDS**

**FIGURE 1. Glucose Concentrations over Time**

Glucose concentrations over time during exercise, recovery (4-hours post-exercise), and sleep (00:00–06:00h). We distinguish between non-competitive exercise (NCE) days (1,536 days), and competitive exercise (CE) days (256 days). Glucose concentrations are shown with 50th percentile (median), 25th-75th percentile (interquartile range) and 5th-95th percentile. The distribution of glucose concentrations is shown on the right-hand side of each panel.

**FIGURE 2. Association of Exercise-Related Factors with Dysglycemia**

Estimated associations of the exercise variables with the odds ratio [95% CI] of hypo- and hyperglycemia during (a) exercise, (b) recovery, and (c) sleep. Listed are *p*-values. Associations are presented on a logarithmic scale, with red and blue color indicating an increase and decrease in odds ratio, respectively, and color intensity indicating the strength of the association. Note that “Time in HR zone 5 (VO2max)” was excluded from the analysis due to the large number of zero values (i.e., 82%). HR, heart rate; CI, confidence interval.

**TABLE 1. Participant Characteristics**

|  |  |  |  |
| --- | --- | --- | --- |
| **Demography and anthropometry** | | | |
| Sex [male/female] | 12/0 | [100/0%] | |
| Age [yr] | 25.6 | ± | 4.4 |
| Weight [kg] | 67.4 | ± | 7.7 |
| Fat mass [%] | 7.6 | ± | 1.7 |
| Height [cm] | 177.6 | ± | 5.8 |
| **Diabetes and glycemia** | | | |
| Diabetes duration [yr] | 10.4 | ± | 4.9 |
| HbA1c [%] | 6.7 | ± | 0.5 |
| Days with CGM coverage ≥70% | 176.2 | ± | 88.3 |
| Insulin therapy [MDI/CSII] | 12/0 | [100/0%] | |
| **Physical exercise** | | | |
| Functional threshold power (FTP) [W/kg] | 5.0 | ± | 0.4 |
| Maximum heart rate (HRmax) [bpm] | 188.5 | ± | 7.2 |
| Lactate threshold heart rate (LTHR) [bpm] | 168.9 | ± | 15.1 |
| Maximum rate of oxygen consumption (VO2max) [mL/min/kg] | 70.6 | ± | 4.0 |
| *Competitive season* | | | |
| Cycling [days/yr] | 280.8 | ± | 28.1 |
| Competition [days/yr] | 34.7 | ± | 15.3 |
| Distance cycled [km/yr] | 26,171.6 | ± | 4,841.2 |
| *Average cycling day* | | | |
| Mean time cycled [h/day] | 3.1 | ± | 0.2 |
| Mean distance cycled [km/day] | 96.6 | ± | 10.6 |
| Mean elevation gain [m/day] | 1,235.0 | ± | 235.5 |

Demographic, anthropometric, diabetes and physical exercise characteristics of (n=12) professional cyclists. Data are mean ± SD or n [%] unless otherwise indicated. CGM, continuous glucose monitoring; MDI, multiple daily injections; CSII, continuous subcutaneous insulin infusion.

**TABLE 2. Summary Statistics of Glycemic Control**

|  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
|  | **Entire day**  (06:00–06:00h) | | | | | **Wake** (06:00–00:00h) | | | | | **Exercise** | | | | | **Recovery** (4h post-exercise) | | | | | | **Sleep**  (00:00–06:00h) | | | | |
| **Overall (2,115 days)** | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Mean glucose [mg/dL] | 145.2 | ± | 18.2 |  |  | 147.3 | ± | 18.1 |  |  | 145.6 | ± | 19.9 |  |  | 153.1 | ± | 23.4 |  |  | 138.7 | | ± | 24.5 |  |  |
| Coefficient of variation (CV) [%] | 38.9 | ± | 8.5 |  |  | 38.0 | ± | 8.3 |  |  | 39.7 | ± | 9.8 |  |  | 36.5 | ± | 7.1 |  |  | 40.3 | | ± | 9.6 |  |  |
| *CGM readings [%] in …* | | | | | | | | | | | | | | | | | | | | | | | | | | |
| … hyperglycemia (>180 mg/dL) | 23.6 | ± | 12.5 |  |  | 24.5 | ± | 12.5 |  |  | 24.3 | ± | 13.8 |  |  | 27.5 | ± | 14.3 |  |  | 20.8 | | ± | 15.7 |  |  |
| … L2 (>250 mg/dL) | 6.7 | ± | 5.6 |  |  | 6.8 | ± | 5.5 |  |  | 7.3 | ± | 5.7 |  |  | 7.7 | ± | 6.4 |  |  | 6.6 | | ± | 7.7 |  |  |
| … L1 (>180–250 mg/dL) | 16.9 | ± | 7.5 |  |  | 17.7 | ± | 7.9 |  |  | 17.0 | ± | 9.1 |  |  | 19.8 | ± | 9.2 |  |  | 14.2 | | ± | 8.3 |  |  |
| … target range (70–180 mg/dL) | 70.0 | ± | 13.7 |  |  | 70.1 | ± | 13.3 |  |  | 70.4 | ± | 14.8 |  |  | 68.1 | ± | 13.0 |  |  | 69.5 | | ± | 17.3 |  |  |
| … hypoglycemia (<70 mg/dL) | 6.4 | ± | 4.7 |  |  | 5.4 | ± | 4.4 |  |  | 5.3 | ± | 5.5 |  |  | 4.4 | ± | 4.6 |  |  | 9.6 | | ± | 7.2 | \*\* |  |
| … L1 (54–<70 mg/dL) | 4.5 | ± | 3.0 |  |  | 3.8 | ± | 2.7 |  |  | 3.9 | ± | 3.2 |  |  | 3.2 | ± | 3.1 |  |  | 6.5 | | ± | 4.8 | \* |  |
| … L2 (<54 mg/dL) | 2.0 | ± | 2.3 |  |  | 1.6 | ± | 2.2 |  |  | 1.4 | ± | 2.7 |  |  | 1.1 | ± | 1.5 |  |  | 3.2 | | ± | 3.1 | \* |  |
| **Non-Competitive Exercise (NCE; 1,536 days)** | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Mean glucose [mg/dL] | 142.4 | ± | 17.9 |  |  | 144.1 | ± | 17.7 |  |  | 140.8 | ± | 20.1 |  |  | 151.3 | ± | 23.8 |  |  | 137.2 | | ± | 24.9 |  |  |
| Coefficient of variation (CV) [%] | 38.9 | ± | 8.6 |  |  | 38.0 | ± | 8.6 |  |  | 39.7 | ± | 10.5 |  |  | 36.1 | ± | 7.1 |  |  | 40.0 | | ± | 9.8 |  |  |
| *CGM readings [%] in …* | | | | | | | | | | | | | | | | | | | | | | | | | | |
| … hyperglycemia (>180 mg/dL) | 22.2 | ± | 12.1 |  |  | 22.8 | ± | 12.1 |  |  | 21.9 | ± | 13.9 |  |  | 26.8 | ± | 14.6 |  |  | 20.2 | | ± | 16.1 |  |  |
| … L2 (>250 mg/dL) | 6.1 | ± | 5.3 |  |  | 6.1 | ± | 5.1 |  |  | 6.5 | ± | 5.4 |  |  | 7.3 | ± | 6.1 |  |  | 6.2 | | ± | 7.3 |  |  |
| … L1 (>180–250 mg/dL) | 16.0 | ± | 7.6 |  |  | 16.7 | ± | 7.8 |  |  | 15.4 | ± | 9.2 |  |  | 19.5 | ± | 9.5 |  |  | 14.0 | | ± | 9.3 |  |  |
| … target range (70–180 mg/dL) | 71.0 | ± | 13.8 |  |  | 71.3 | ± | 13.4 |  |  | 72.0 | ± | 15.3 |  |  | 68.7 | ± | 13.4 |  |  | 69.8 | | ± | 17.6 |  |  |
| … hypoglycemia (<70 mg/dL) | 6.9 | ± | 5.0 | \* |  | 5.9 | ± | 4.9 |  |  | 6.1 | ± | 6.4 |  |  | 4.5 | ± | 4.9 |  |  | 10.1 | | ± | 7.4 | \*\* |  |
| … L1 (54–<70 mg/dL) | 4.8 | ± | 3.2 | \* |  | 4.1 | ± | 2.9 |  |  | 4.5 | ± | 3.6 |  |  | 3.3 | ± | 3.4 |  |  | 6.9 | | ± | 5.2 | \* |  |
| … L2 (<54 mg/dL) | 2.1 | ± | 2.4 |  |  | 1.7 | ± | 2.5 |  |  | 1.6 | ± | 3.2 |  |  | 1.1 | ± | 1.6 |  |  | 3.2 | | ± | 2.8 | \* |  |
| **Competitive Exercise (CE; 256 days)** | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Mean glucose [mg/dL] | 150.6 | ± | 16.8 |  | † | 154.0 | ± | 14.8 |  | †† | 172.3 | ± | 16.3 |  | ††† | 161.0 | ± | 22.6 |  | † | 140.7 | | ± | 30.8 |  |  |
| Coefficient of variation (CV) [%] | 37.2 | ± | 9.8 |  |  | 35.9 | ± | 8.7 |  |  | 33.3 | ± | 8.8 |  | †† | 35.8 | ± | 8.7 |  |  | 36.8 | | ± | 14.5 |  |  |
| *CGM readings [%] in …* | | | | | | | | | | | | | | | | | | | | | | | | | | |
| … hyperglycemia (>180 mg/dL) | 25.2 | ± | 12.5 |  | † | 27.0 | ± | 11.3 |  | †† | 38.5 | ± | 12.9 | \*\* | ††† | 29.5 | ± | 13.7 |  |  | 19.9 | | ± | 19.6 |  |  |
| … L2 (>250 mg/dL) | 7.1 | ± | 6.1 |  |  | 6.9 | ± | 5.7 |  |  | 11.9 | ± | 7.7 | \*\* | †† | 9.1 | ± | 8.4 |  |  | 7.5 | | ± | 10.4 |  |  |
| … L1 (>180–250 mg/dL) | 18.1 | ± | 7.3 |  | † | 20.1 | ± | 7.1 |  | †† | 26.5 | ± | 7.8 | \*\* | ††† | 20.4 | ± | 8.6 |  |  | 12.3 | | ± | 9.7 |  |  |
| … target range (70–180 mg/dL) | 70.1 | ± | 14.1 |  |  | 69.3 | ± | 12.0 |  |  | 60.4 | ± | 13.0 | \* | †† | 67.2 | ± | 12.8 |  |  | 72.2 | | ± | 22.2 |  |  |
| … hypoglycemia (<70 mg/dL) | 4.7 | ± | 4.5 |  |  | 3.6 | ± | 3.1 |  |  | 1.1 | ± | 1.4 |  | † | 3.3 | ± | 3.5 |  |  | 7.9 | | ± | 9.8 |  |  |
| … L1 (54–<70 mg/dL) | 3.2 | ± | 2.5 |  |  | 2.6 | ± | 1.9 |  |  | 0.9 | ± | 1.0 |  | †† | 2.6 | ± | 2.5 |  |  | 5.0 | | ± | 5.7 |  |  |
| … L2 (<54 mg/dL) | 1.5 | ± | 2.4 |  |  | 1.1 | ± | 1.4 |  |  | 0.2 | ± | 0.4 |  |  | 0.8 | ± | 1.3 |  |  | 2.9 | | ± | 5.2 |  |  |

Summary statistics of continuous glucose monitoring (CGM) of (n=12) professional cyclists over a competitive season. We distinguish between the entire study period (overall; 2,115 days), non-competitive exercise (NCE) days (1,536 days), and competitive exercise (CE) days (256 days). Statistics were calculated for five phases of the day: entire day (06:00–06:00h), wake (06:00–00:00h), exercise, recovery (4-hours post-exercise), and sleep (00:00–06:00h). Data are mean ± SD calculated over all participants. Glycemic control is reported following the international consensus statement on CGM (15). The comparisons of glycemic values with consensus targets (16) through one-sided one-sample *t*-tests are reported with \*\*\* (*p*<0.001), \*\* (*p*<0.01), and \* (*p*<0.05). The comparison of glycemic values between NCE and CE days through paired *t*-tests are reported with ††† (*p*<0.001), ††(*p*<0.01), and † (*p*<0.05).