

## Effect of sprint duration (6 s or 30 s) on plasma glucose regulation in untrained male subjects

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**Aim.** We have explored in the following study the glucoregulatory responses (glycemia, insulinemia, catecholamines) at the end of 2 supramaximal tests of different durations.

**Methods.** Seven untrained male subjects ( $21.9 \pm 0.3$  y) performed an isolated exercise of 6 s (T6) and a Wingate-test of 30 s. To determine the levels of lactate (La), plasma concentrations of glucose, insulin, adrenaline (A) and noradrenaline (NA), blood samples have been collected successively at rest, after a warm-up period of 15 min, immediately after T6 and T30, and after 5, 10, 20, and 30 min of recovery.

**Results.** Whether expressed as absolute or relative values, the peak power recorded during the 2 tests is statistically the same in T6 and T30. The maximal value of lactate ( $La_{max}$ ) measured 5 min after the end of the 2 exercises is significantly greater after T30 ( $12.3 \pm 0.9$  mmol  $\cdot$  L<sup>-1</sup>) than after T6 ( $5.4 \pm 0.4$  mmol  $\cdot$  L<sup>-1</sup>) and T30 ( $4.2 \pm 0.2$  mmol  $\cdot$  L<sup>-1</sup>). No significant difference is observed between the plasma glucose concentrations recorded after the 2 tests until the first 10 min of recovery. However the plasma glucose values recorded after 20 and 30 min of recovery are significantly higher after T6 than after T30. Whatever the duration of the test, the insulinemia level remains unchanged at the end of the exercise and during the 30 min of recovery. On the other hand, the values of adrenaline and noradrenaline after T6 and T30 become considerably higher than those recorded at rest. However, the increase remains significantly higher after T30 ( $13.5 \pm 1.8$  nmol  $\cdot$  L<sup>-1</sup> for NA and  $2.7 \pm 0.7$  nmol  $\cdot$  L<sup>-1</sup> for A) than after T6 ( $4.9 \pm 0.3$  nmol  $\cdot$  L<sup>-1</sup> for NA and  $1.2 \pm 0.2$  nmol  $\cdot$  L<sup>-1</sup> for A).

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**Conclusion.** These results suggest that the mechanism responsible for increasing blood glucose surpass those which decrease it during supramaximal exercise. However, plasma glucose concentrations is affected by the duration of supramaximal exercise. The lower increase of plasma glucose concentration after T30 than after T6 might be explained by the resting of muscle glycogen stores which are more used during T30 than after T6, but in the absence of muscle glycogen content measurement we cannot conclude.

**KEY WORDS:** Wingate-test - Glucose, blood - Exercise.

The assumption of the literature concerning the glycemic response after brief and intense exercises remain limited and non-univocal. While most of these works <sup>1-7</sup> conclude that the interruption of brief and intensive exercises induces a state of hyperglycemia, of which Kjaer <sup>8</sup> has explained the principal responsible mechanisms, the recent data provided by Wouassi *et al.* <sup>9</sup> shows that a decrease in glycemia can also be observed. Methodological factors certainly contribute to explain these discrepancies because the nature of exercises, their duration, intensity and number

vary according to studies. However, such discrepancies also indicated that the mechanisms controlling the glycemic response at the end of sprint exercises are yet to be completely clarified.<sup>5, 10</sup>

In this study, we have chosen to isolate the effect of the sprint duration on the glycemic response. Therefore, we have studied the evolution of both glycemia and the principal glucoregulatory hormones, namely adrenaline, noradrenaline and insulin at the end of 2 sprint exercises of respectively 6 s (T6) and 30 sec (T30), performed by untrained male subjects on a cycle-like ergometer against the same load.

## Materials and methods

### Methods

The experimental protocol has been carried out with the consent of the Consultative Committee of Rennes 1 for the Protection of Subjects Engaged in Biomedical Researches (CCPPRB).

### Subjects

Seven untrained male subjects (20-25 y), with no past history of pathological, cardiac, or pulmonary conditions, took part in this study. After the conditions of the experiment had been explained to them, they gave their written consent, and joined the laboratory for 4 different days (D1, D2, D3, D4) separated by a maximal period of 15 d.

### Protocol

On D1, a medical examination was performed and an electrocardiogram was recorded at rest to eliminate any eventual contraindications to stressful exercises. The subjects' anthropometric characteristics were also determined, including weight, height, and the percentage of body fat (%BF). The %BF was estimated from 4 skinfold thicknesses (biceps, triceps, sub-scapular and supra-iliac) according to the method of Durnin and Rahaman.<sup>11</sup> The lean body mass (LBM) was calculated by subtracting the total body fat from the body weight. According to the procedure of Flandrois *et al.*,<sup>12</sup> the maximal oxygen uptake ( $\dot{V}O_{2max}$ ) was measured during an incremental exercise-test adapted by Delamarche *et al.*<sup>13</sup> and Gratas-Delamarche *et al.*<sup>14</sup> using a breath by breath

automated exercise metabolic system (CPX, Medical graphics, St-Paul, Minnesota).

On the 2<sup>nd</sup> day (D2), the force-velocity test (F/V test) was performed using the technique of Vandewalle *et al.*<sup>15</sup> and adapted by Zouhal *et al.*<sup>16</sup> This test, using an Ergomeca bicycle, consists of a succession of several supramaximal bouts of roughly 6 s against loads increased by 1 kg after each bout until inability to pursue the test. A complete recovery (5 min) was allowed between successive bouts. The velocity was recorded every 2 s using a photoelectric cell fixed on the wheel of the cycle and connected to a revolution-counter (MEV 2000). Only the highest speed value (V) was recorded for each load. By multiplying load and speed values measured, a power curve was then compiled to each bout up to the maximum. The optimal load (F) corresponding to this maximal power was then used for the isolated exercise of 6 s (T6), as well as for the Wingate-test (T30).

Each subject returned to the laboratory on D3 and D4 to undergo the isolated test of 6 s (T6) or the Wingate test (T30). All subjects were required to avoid intense exhausting physical activity.

The experiment started in the morning (at about 9 a. m.) 2 h after a standardized breakfast (10 kcal/kg, 55% of which came from carbohydrates, 33% from lipids and 12% from proteins). On arrival each subject was asked to lie down after which a catheter was inserted into an antecubital vein to regularly extract blood samples. After a 30 min rest the subject sat on the bicycle and the 1<sup>st</sup> blood sample was drawn (5 ml) to determine rest values. A warm-up was allowed for 15 min at a supramaximal power about 50-60 W at a very low velocity subsequently each subject performed the following in random order:

- either an isolated exercise of 6 s (T6) which consists of pedalling at maximum speed against the load (F), previously determined during the Force/Velocity test;

- either a Wingate-test of 30 s (T30), performed according to the procedure of Jacobs *et al.*<sup>17</sup> During this exercise the subject was asked to cycle for 30 s as fast as possible on the same machine and against the previously same determined load (F). The velocity was again recorded throughout the test. The power produced could be calculated at any time with the highest value chosen for maximal power ( $\dot{W}_{max}$ ). The average of all measured power values during the 30 s was also kept as the mean power ( $\bar{W}$ ).

Both tests were performed on the same cycle-like ergometer, and using the same recording system. The heart rate was recorded continuously during the warm-up, during the 2 exercises (T6 and T30) and during the recovery.

### Blood extractions

Blood samples were extracted on (D3) and (D4) only, that is, before and after T6 and T30.

Ten ml of venous blood were extracted at rest, both when the subject was lying down during the insertion of the catheter and sitting on the bicycle (this time represents the rest value), as well as after the warm-up period, once the test was over, and 5, 10, 20, and 30 min after recovery. At each extraction, the blood was collected in a vacutainer tube containing Ethylene Diamine Tetra Acetic Acid (EDTA) (the EDTA is used to eliminate inhibition of enzyme catalyzed reactions due to tracers of heavy metals).

Three min after the exercise was over, an "arterialized" capillary blood microextraction was performed by pricking the tip of a finger, in order to measure the maximal value of blood lactate. In fact, the highest levels of lactic acid are obtained 3 to 5 min after the end of this type of exercise.<sup>18</sup>

### Blood analysis

The collected samples of venous blood are immediately placed in an icy environment. Packed cell volume (PCV) was immediately measured in duplicate with microcentrifuge. Then plasma was separated by centrifugation for 10 min at 3 000 g and stored at -80°C for subsequent chemical analysis. The lactate concentration in whole blood, and plasma concentrations of glucose, insulin, adrenaline (A) and noradrenaline (NA) were measured in each venous blood sample.

Only the plasma collected with the use of EDTA was used to determine the concentrations of glucose and insulin.

While plasma glucose was measured by enzymatic fluorometric methods, the concentration of insulin was determined with radioimmunoassays, according to the "competition" method, and using an insulin-marked antigen called "tracer" (I125).

Catecholamines were evaluated by high-performance liquid chromatography, and using a HPLC-THI column

(Electrochemical detection), according to the method of Koubi *et al.*:<sup>19</sup> before the HPLC run the catecholamines were extracted by selective adsorption to aluminum oxide (Chromosystems-HPLC-kit, Waters, Mildford, Mass., USA). The aluminum oxide was shaken up briefly in extraction buffer (50 µL) and then 1 ml of plasma was added with 50 µL internal standard solution (600 pg dihydroxybenzylamine). The aluminum oxide was then washed 3 times, with a brief centrifugation between washes. The catecholamines were extracted with 120 µL elution buffer by shaking briefly and subjecting into a final centrifugation at 1 500 g for 1 min. Then 50 µL of the sample eluant was injected into HPLC column (Resolve TM 5 µL Spherical C 18, HPLC column, Waters) and eluted with a mobile phase. The flow rate was 1 ml · min<sup>-1</sup> at 13.8 mPa and a potential of 0.60 V. The chromatogram was analyzed by computer integration (Baseline 815, Waters).

Finally, the concentration of lactate was determined using both arterialized capillary blood and venous blood, by a microenzymatic method that used the "microzym" L. Cétrix.

All concentrations thus measured (except the lactate ones) were corrected by taking into account the variations of the plasmatic volume, which were estimated according to the hematocrit (Ht) variation between rest and the specific moment in question, by applying the formula provided by Van Beaumont *et al.*:<sup>20</sup>

$$X_c = X_m \times \left[ \frac{Ht_1}{Ht_2} \times \frac{1 - Ht_1}{1 - Ht_2} \right]$$

with the % change expressed in decimal form.

X<sub>c</sub>: corrected concentration; X<sub>m</sub>: measured concentration; Ht<sub>1</sub>: rest hematocrit; Ht<sub>2</sub>: hematocrit corresponding to X<sub>m</sub>.

### Statistics

Data were summarized with means ( $\bar{x}$ ) and standard deviation (SD). In order to affirm the significance of eventual differences, a 2-way (duration × time) ANOVA for repeated measures was performed by using Sigma-Stat 2.0 (Jandel San Raphael, CA). Where appropriate, significant differences identified by ANOVA were isolated using Newman-Keuls's significant difference tests. Correlation analysis was done

TABLE I.—*Morphological and physiological characteristics of the subjects.*

Subjects	Age (y)	Height (cm)	Weight (kg)	Fat (%)	LBM (kg)	$\dot{V}O_{2\max}$ (ml · kg <sup>-1</sup> min. <sup>-1</sup> )
Untrained (n=7)	21.7±0.6	178.5±2.8	70.2±2.1	14.6±1.3	59.8±1.4	45±2.3

Data are means (±SD). LBM: lean body mass.

TABLE II.—*Performances of untrained subjects recorded after 6 s sprints (T6) and after the Wingate-test (T30).*

$\dot{W}_{\max}$	T6	T30
W	1075±41	1016±44
W · kg <sup>-1</sup>	15.3±0.4	14.50±0.7
W · kg <sup>-1</sup> LBM	17.9±0.4	17.03±0.7

Data are means (±SD).  $\dot{W}_{\max}$ : maximum power. Power values are also referred to body mass (W · kg<sup>-1</sup>) and lean body mass (W · kg<sup>-1</sup> LBM).

TABLE III.—*Venous blood lactate (mmol · L<sup>-1</sup>) concentrations of the subjects determined during T6 and T30.*

Test	Rest	Warm-up exercise	End of recovery	5 min recovery	10 min recovery	20 min recovery	30 min
T6	1.5±0.2	2.6±0.5	3.0±0.4	5.4±0.9*	3.3±0.5	2.3±0.3	1.7±0.2
T30	1.7±0.4	4.5±0.5	7.8±0.7*°	12.3±0.9*°	10.7±0.8*°	8.1±1.4*°	5.9±0.9*°

\*: significantly different from rest values; °: significantly different between T6 and T30.

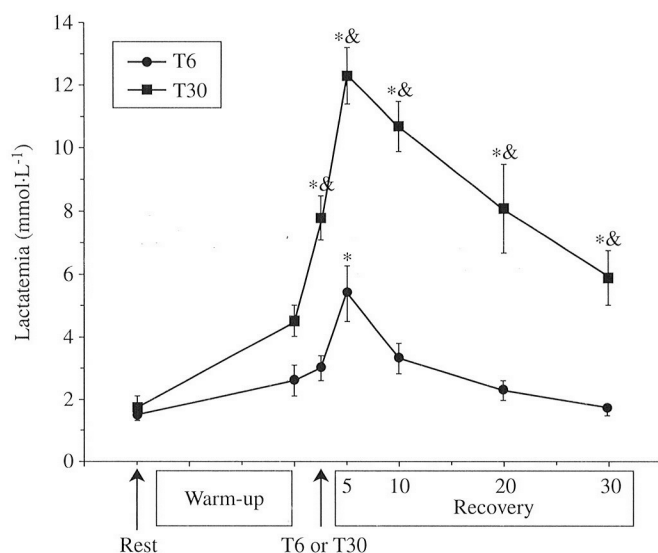


Figure 1.—Venous blood lactate concentrations of the subjects determined during T6 and T30. \*Significantly different from rest values; &amp; significantly different between the values of T6 and T30.

by the Spearman rank test. A value of  $p < 0.05$  was accepted as the minimal level of statistical significance.

## Results

### *Morphological characteristics, maximal oxygen uptake ( $\dot{V}O_{2\max}$ ), and performance during T6 and T30*

The anthropometric and physiological characteristics of the subjects are listed in Table I. The performances of the subjects during T6 and T30 are found in Table II. Whether expressed in absolute or relative values, the maximal power ( $\dot{W}_{\max}$ ) of the subjects were statistically the same during T6 and T30.

### *Metabolic and hormonal parameters*

#### *The concentrations of blood lactate*

The 2 supramaximal tests, T6 and T30, were associated with a very significant increase in lactatemia at the end of the exercise (Table III). In fact, the lactatemia reaches its peak 5 min after the end of the exercise, during T6 and T30. It is important to mention that this increase was significantly higher after T30 than after T6. The maximal recorded values ( $-La_{\max}$ ) measured on arterialized capillary blood were respectively ( $5.4 \pm 0.9$  mmol · L<sup>-1</sup>) in T6 and ( $12.3 \pm 0.9$  mmol · L<sup>-1</sup>) in T30.

TABLE IV.—Plasma glucose ( $\text{mmol} \cdot \text{L}^{-1}$ ) concentrations of the subjects determined during T6 and T30.

Test	Rest	Warm-up exercise	End of recovery	5 min recovery	10 min recovery	20 min recovery	30 min
T6	$3.91 \pm 0.40$	$4.10 \pm 0.17$	$4.25 \pm 0.42$	$4.45 \pm 0.33$	$4.46 \pm 0.30$	$5.42 \pm 0.45^{* \circ}$	$5.01 \pm 0.53^{* \circ}$
T30	$3.27 \pm 0.60$	$3.66 \pm 0.40$	$3.62 \pm 0.20$	$3.97 \pm 0.35^{*}$	$4.26 \pm 0.20^{*}$	$4.09 \pm 0.12$	$4.27 \pm 0.26$

\*: significantly different from rest values; °: significantly different between T6 and T30.

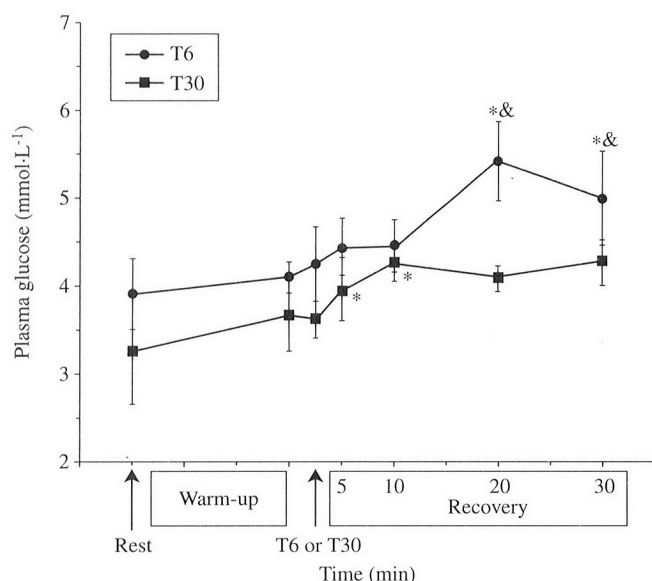


Figure 2.—Plasma glucose concentrations of the subjects determined during T6 and T30.\*Significantly different from rest values; & significantly different between the values of T6 and T30.

We could also notice that the venous blood lactate dropped to its “rest” value in 30 min in the case of T6 and remained high in the case of T30 (Figure 1).

### Plasma glucose

At rest, the values of plasma glucose were respectively  $3.9 \pm 0.4 \text{ mmol} \cdot \text{L}^{-1}$  before T6 and  $3.2 \pm 0.6 \text{ mmol} \cdot \text{L}^{-1}$  before T30 (Table IV).

The 15 min warm-up at about 50% of the ( $\dot{V}\text{O}_{2\text{max}}$ ) had no effect on plasma glucose concentrations whatever type of exercise was performed (Figure 2).

After T6 and T30, the plasma glucose concentrations increased significantly, and reached a maximal value of  $5.42 \pm 0.4 \text{ mmol} \cdot \text{L}^{-1}$  in T6 at the 20<sup>th</sup> min of recovery and  $4.26 \pm 0.2 \text{ mmol} \cdot \text{L}^{-1}$  in T30 at the 10<sup>th</sup> min that follows the end of the test. How-

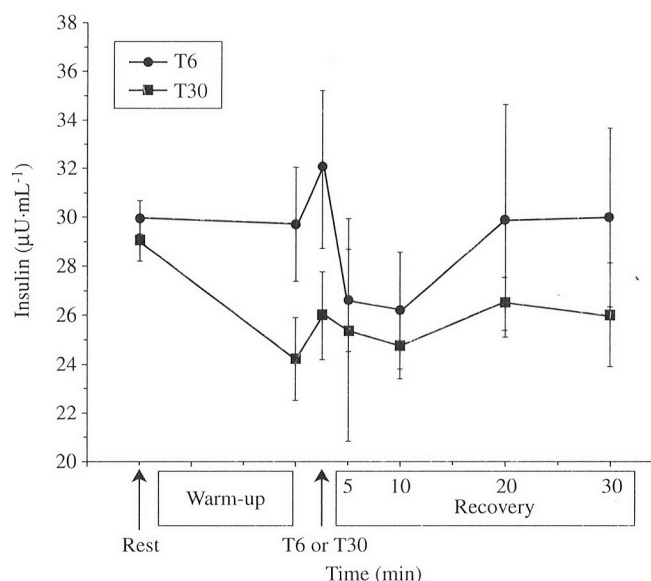


Figure 3.—Plasma insulin concentrations of the subjects determined during T6 and T30.

ever, the glycemia was significantly higher in T6 than T30 only after 20 and 30 min of recovery.

But when these glycemia levels were compared to the resting values, the differences thus determined were always lower after T30 than after T6 (Figure 3).

### Insulinemia

At rest, the measured values were respectively  $30 \pm 2.3 \text{ μU} \cdot \text{mL}^{-1}$  for T6 and  $29 \pm 0.8 \text{ μU} \cdot \text{mL}^{-1}$  for T30. These values were within the limits of the theoretical norms determined during the postprandial period <sup>5, 9, 21</sup> (Table V).

The concentrations of insulin was affected neither by the warm-up nor by duration of the supramaximal exercise. In fact, the insulinemia remained unchanged once T6 and T30 were over, and throughout the 30 min of recovery which followed both tests.

TABLE V.—Plasma insulin concentrations ( $\mu\text{U} \cdot \text{mL}^{-1}$ ) of the subjects determined during T6 and T30.

Test	Rest	Warm-up exercise	End of recovery	5 min recovery	10 min recovery	20 min recovery	30 min
T6	30±2.3	29.7±2.3	32±3.3	26.6±2.1	26.2±2.4	29.9±4.8	30±3.7
T30	29±0.8	24.2±1.7	26±1.8	25.4±4.6	24.7±1.3	26.5±1.1	26±2.1

TABLE VI.—Plasma glucose ( $\text{nmol} \cdot \text{L}^{-1}$ ) concentrations of the subjects determined during T6 and T30.

Test	Rest	Warm-up exercise	End of recovery	5 min recovery	10 min recovery	20 min recovery	30 min
T6	0.57±0.03	0.7±0.12	1.23±0.25*	0.64±0.06	0.61±0.03	0.72±0.08	0.50±0.09
T30	0.48±0.07	0.98±0.12	2.70±0.74*°	1.00±0.3*°	0.61±0.09	0.64±0.1	0.68±0.1

\*: significantly different from rest values; °: significantly different between T6 and T30.

TABLE VII.—Plasma noradrenaline concentrations ( $\text{nmol} \cdot \text{L}^{-1}$ ) of the subjects determined during T6 and T30.

Test	Rest	Warm-up exercise	End of recovery	5 min recovery	10 min recovery	20 min recovery	30 min
T6	2.25±0.27	3.75±0.5	4.93±0.3*	4.41±0.7	2.60±0.5	2.43±0.3	2.23±0.3
T30	2.65±0.27	5.6±0.7	13.5±1.8*°	8.0±1.7*°	5.0±0.6*°	5.15±1.9*°	3.91±0.3

\*: significantly different from rest values; °: significantly different between T6 and T30.

### Plasma concentrations of catecholamines

At rest, no significant difference was observed between T6 and T30 concerning the values of adrenaline (A) and noradrenaline (NA), (Tables VI, VII). However, our results showed that the duration of the supramaximal exercise clearly affects catecholamine response. The values of A and NA after T6 and T30 became significantly higher than those recorded at rest. However, the increase measured immediately after the 2 tests remained significantly higher after T30 ( $13.5 \pm 1.8 \text{ nmol} \cdot \text{L}^{-1}$  for NA and  $2.7 \pm 0.7 \text{ nmol} \cdot \text{L}^{-1}$  for A) than after T6 ( $4.9 \pm 0.3 \text{ nmol} \cdot \text{L}^{-1}$  for NA and  $1.2 \pm 0.2 \text{ nmol} \cdot \text{L}^{-1}$  for A). Catecholamines disappeared very quickly during recovery. The averages of A and NA dropped to their basic values at the end of our experimentation (Figures 4, 5).

### Discussion

The study shows that the interruption of a supramaximal exercise induces a significant increase of plasma glucose concentration compared to the rest values, which are affected by the duration of the sprint. Indeed, glycemia increase is less mani-

fest after the long sprint T30 than after the short sprint T6.

Each subject performed these 2 exercises in random order, with the same load applied in both cases. If we consider the similar values of the maximal power ( $\dot{W}_{\text{max}}$ ) which fall under the previously published norms for untrained male adults,<sup>16, 22</sup> we can estimate that each subject has done the test to the best of his ability. Therefore, the duration of the test constituted the only factor of methodological variation between T6 and T30. When the exercise is prolonged, it becomes a lot more exhaustive, and the effort exerted, evaluated according to the value of W, is therefore more important. On the biological scale, the exhaustion of the subject, the heart rate (183 bpm after T30 and 140 bpm after T6) and the lactatemia measured in arterialized capillary blood were significantly higher at the end of T30 than at the end of T6.

After the 2 exercises we observed a significant decrease of the plasma volume (7.9% after T30 and 5.8% after T6), so all the plasma concentrations measured were corrected by taking into account these variations.<sup>20</sup>

The plasma glucose concentration climbs at the end of both tests, but increases less after T30 than after

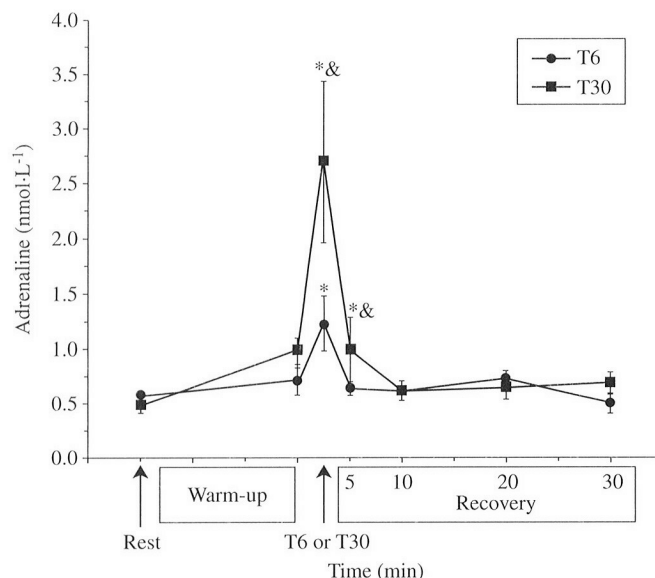


Figure 4.—Plasma adrenaline concentrations of the subjects determined during T6 and T30. \*Significantly different from rest values; & significantly different between the values of T6 and T30.

T6, although the 1<sup>st</sup> exercise was more stressful than the 2<sup>nd</sup>. If we analyze all the factors involved in the increase of the hepatic production of glucose during the exercise, as described by Kjaer,<sup>8</sup> we can conclude that these factors must be potentially stronger after T30 than after T6. According to Kjaer,<sup>8</sup> the essential, and yet unknown factor responsible for the initial increase of the hepatic production of glucose during exercise is the activation of the superior, probably hypothalamic nervous centers. Not to mention the possible interference of reflex phenomena linked to the appearance, in the circulating space, of different metabolites produced by muscles in activity, and more adjunctly, the stimulation of nervous fibers of muscular origin. We can assume that these factors must be stronger after T30 than after T6. Concerning the hormonal factors, (A) and (NA) are undoubtedly the first to climb as shown in this study. The increase of (A) and (NA) was a lot more obvious after T30 than after T6, thus emphasizing the exerting aspect of the 30-s sprint. By relying on the results obtained in the hepatically transplanted people, Kjaer<sup>8</sup> concludes that only (A) and not (NA) contributes to the hepatic production of glucose during exercises, and that its role remains always overestimated. Concerning the other glucoregulatory hormones, they probably play a negligible role in sprint exercise. The insulinemia measured in this study remains unchanged before and after the 2 tests. The glucagon,

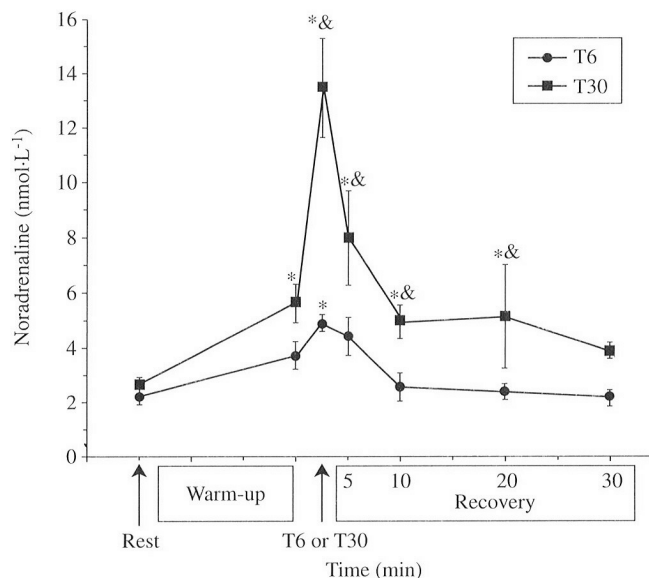


Figure 5.—Plasma noradrenaline concentrations of the subjects determined during T6 and T30. \*Significantly different from rest values; & significantly different between the values of T6 and T30.

the growth hormone and the cortisol have not been measured in this case, but numerous studies have shown that their increase was nul or negligible.<sup>7</sup> Consequently, it was impossible to explain the lower levels of glycemia after T30 by a lower hepatic production of glucose in the same exercise.

Thus the only hypothesis left to mention is that of a higher muscular intake of plasma glucose after T30 than after T6. This assumption is supported by previous results which demonstrated that very short sprint exercises, of less than 6 s to not significantly decrease the muscle glycogen stores,<sup>6</sup> whereas longer sprint exercises of 30 s are associated with a significant drops of these fuels<sup>5, 10</sup> and thus by a simultaneous decrease in intramuscular concentrations of glucose-6-phosphate (G6P) and glucose-1-phosphate (G1P).<sup>23</sup> Therefore the large use of muscle glycogen during T30 increases the gradient of glucose between plasma and muscle, which is enough to activate the facilitated transport of glucose through the sarcolemma, and then limits the increase of glycemia induced by recovery.

## Conclusions

In conclusion, this work helps to better understand the mechanisms of glucoregulation during supramax-

imal exercise. It demonstrates that the duration of the sprint affects the plasma glucose levels measured during recovery. The increase in plasma glucose concentration is less pronounced after 30 s exercise than after 6 s sprint exercise. These results suggests that plasma glucose may be used to restore the previously reduced muscle glycogen stores, but in the absence of muscle glycogen content measurement we cannot conclude.

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