Cardiac troponin I release after a basketball match in adult elite, adult amateur and junior elite players

Abstract

The impact of intermittent exercise on the release of cardiac troponins is controversial. This study had two objectives; 1) to examine the individual release of cardiac troponin I (cTnI) to a basketball match, and 2) to establish the influence of athlete status as well as biological age on cTnI release. Thirty-six basketball players (12 adult elite (PBA): 27.3 ± 4.1 yr, 12 adult amateur (ABA): 29.6 ± 2.9 yr, and 12 junior elite (JBA): 16.6 ± 0.9 yr) participated in a simulated basketball match with serial assessment of cTnI at rest, immediately post- and at 1, 3, 6, 12, and 24 h post-exercise. The basketball match increased cTnI levels (pre: 0.008 ± 0.006, peak post: 0.041 ± 0.057 µg/L; $P = 0.000$), with substantial individual variability in peak values. PBA and JBA players showed higher baseline and post-exercise cTnI values than ABA (all $P < 0.05$). Peak cTnI exceeded the upper reference limit (URL) in the 26% of players (3 PBA; 6 JBA). The current results suggest that intermittent exercise can promote the appearance of cTnI and that this is potentially mediated by athlete status with no apparent impact of biological age.

Keywords: cTnI, intermittent exercise, athlete status, biological age

Introduction

The release of biomarkers of cardiamyocyte insult during and after exercise, such as cardiac troponins (cTn), is the topic of intense scientific enquiry and debate (Shave and Oxborough 2012). Evidence to date suggests a substantial number of athletes exceed the upper reference limit (URL) of cTn after long- (Scott et al., 2009; Serrano-Ostáriz et al., 2009) and short-duration endurance exercise (Shave et al., 2010), as well as after moderate- (Serrano-Ostáriz et al., 2011) and low-intensity continuous exercise (Eijsvogels et al., 2010).

Beyond description, several recent studies have attempted to determine what personal and exercise-related factors may promote an increase in cTn after exercise. An elevation in cardiac troponin I (cTnI) has been associated with increased exercise duration and intensity (Serrano-Ostáriz et al., 2011), exercise mode (Shave et al., 2007); training level (Neilan et al., 2006) and biological age (Tian et al., 2012). In many of these areas data is contradictory.

For example, available scientific data related to cTn release after intermittent exercise such as soccer (Carranza-García et al., 2011; Rahnama et al., 2011), rugby (George et al., 2004), basketball (Nie et al., 2008) or floorball (Wedin and Henriksson, 2014) is limited, often poorly controlled (George et al., 2004) and with limited sampling times post-exercise (George et al., 2004; Rahnama et al., 2011; Wedin and Henriksson, 2014). Consequently this has produced contradictory data. Further it has been proposed that less experienced endurance athletes are more likely to exhibit detectable cTn levels than more experienced athletes after exercise (Neilan et al., 2006; Tian et al., 2006; Fortescue et al., 2007; Mingels et al., 2009; Nie et al., 2011; Mehta et al., 2012). These data are largely derived from field-base studies with limited post-exercise sample points in amateur runners that may underestimate peak cTn release (Middleton et al., 2008).

The cTn release in professional of elite athletes is less well understood with a few, older data sets also limited by sampling frequency (Bonneti et al., 1996; König et al., 2003). Evidence to date also suggests increased cTn appearance after exercise in adolescent athletes, possibly due to their immature cardiac muscle, (Tian et al., 2006; Fu et al., 2010; Nie et al., 2011a; Nie et al., 2011b; Tian et al., 2012) although only Tian et al. (2012) directly compared adult and adolescent athletes in a controlled study. Currently it is unknown if the cTn response to intermittent exercise is similar in adults and adolescents.

In an attempt to resolve these issues we examined, employing multiple sampling points during 24 h recovery from exertion, the influence of a simulated basketball match on the cTnI appearance in players with different status/training level (elite and amateur) as well as adult and adolescent basketball players. We hypothesise that cTnI appearance during recovery would be increased in adolescent and amateur players.

Materials and Methods

Subjects and design

A total of 36 male basketball players [12 adult, elite, professional players from Spanish ACB League; PBA), 12 adult, amateur players from a local league (ABA), and 12 elite junior players (Spanish Junior Top-Division; JBA] were recruited and gave personal (and parental in JBA) written informed consent to participate in a simulated basketball match with serial assessment of cTnI during the first 24 h of recovery. All players and their parents were informed of the purpose, nature, testing procedures, possible risks, and their right to terminate participation at will. The study was approved by the Research Ethics Committee of the Government of Aragón (Spain) and was performed

according to the Declaration of Helsinki. The characteristics of the study population are shown in Table 1. PBA had higher training history and current training volume than both ABA and JBA (all $P < 0.05$). By design JBA were younger but there was no difference in age between PBA and ABA.

All subjects attended the laboratory on three occasions. At a preliminary testing session, 1 week before the main study, body height was measured to the nearest 0.1 cm (SECA 225, SECA, Hamburg, Germany) and body mass was determined to the nearest 0.05 kg (SECA 861, SECA, Hamburg, Germany). A questionnaire was completed to obtain personal data, training history, and history of cardiac symptoms (if any). A 12-lead ECG was performed and exclusion criteria were significant cardiac history or pathological electrocardiograph. All players then performed the 20 m shuttle run test (Léger and Gadoury, 1989) with the objective to determine maximal heart rate (HR) using a Polar Team 2 system (1.4.1, Polar Electro Oy, Kempele, Finland).

Prior to the initial laboratory visit and the main train all players were instructed to avoid strenuous training for 48 h before the test and to maintain their normal diet, hydration, and sleep routine. Moreover, subjects ate their last meal at least 2 hours before the test and were asked not to ingest any potentially ergogenic product (i.e., caffeine).

For the second laboratory visit every group was divided in two teams to perform the match, and the players played in their usual position. The matches followed the regulations established by the International Basketball Federation (FIBA). Every team made a change every 4 min of actual game time, so all players participated for the same amount of time (32 of the 40 minutes actual playing time). All players performed a similar warm-up and treated the match as a normal competitive game. Strong and standardized verbal encouragement was offered during the matches to achieve and intensity of play similar to competition. HR was recorded continuously during the

basketball match using a Polar Team 2 system (1.4.1, Polar Electro Oy, Kempele, Finland). The matches began at 11.30 a.m. and took place in an indoor basketball field at a temperature of 20 ºC and relative humidity of 60%. Venous blood samples were taken before, immediately after (5 min), and 1, 3, 6, 12 h after exercise to assess serum cTnI. A final laboratory visit occurred the following day to collect a 24 h post-exercise blood sample.

Blood samples were drawn from an antecubital vein and quickly centrifuged. Serum and plasma were drawn off and stored at −80 °C for later analysis. cTnI was analyzed from samples of EDTA plasma with the Access AccuTnI assay (Beckman Coulter, Fullerton, California). The imprecision profile of 839 duplicate samples showed 10% and 20% coefficient of variation values of 0.014 and 0.008 μg/L, respectively. The URL for cTnI, defined as the 99th percentile of healthy participants, was 0.04 μg/L (Eggers et al., 2007).

Statistical analysis

Statistical analyses were performed using the IBM Statistical Package of Social Sciences (IBM SPSS Statistics, v. 20.0 for WINDOWS). Data are expressed as the mean ± SD unless otherwise stated. Kolmogorov-Smirnov tests were used to analyze for normal distribution and consequently cTnI data were log-transformed prior to inferential statistical testing. To measure the impact of sampling time during recovery (pre, 5 min, 1, 3, 6, 12, and 24 h post-exercise) and athletes status (PBA vs. ABA vs. JBA) upon cTnI, a mixed model 2-way ANOVA were performed with post-hoc Bonferroni tests employed when appropriate. The association between an increase in cTnI (the difference between baseline and peak post-exercise value) and other relevant variables (e.g. baseline cTnI, mean and max exercise HR during simulated basketball play) were

assessed using bivariate Pearson's product moment correlation coefficients. The values were considered to be significant if $P < 0.05$.

Results

The HR_{max} attained during the 20 m shuttle run was not different between groups. The mean HR and % HRmax during the match was lower in PBA compared to ABA and JBA (Table 2).

In the junior basketball match one player was injured and replaced. This subject was excluded from final data analysis. A significant main effect of sampling time was observed for cTnI with increases, compared to pre-exercise, at 1-, 3-, 6-, 12-, and 24-h post-exercise ($P = 0.000$) (Table 3, Fig. 1). A significant main effect of group was noted with both baseline and recovery cTnI lower in ABA when compared to both PBA and JBA. There was no significant group by time interaction term.

Compared with the basal levels, increased post-effort cTnI values were observed in all individuals. Despite this, individual variability in peak cTnI was noted whit 26% of players (3 PBA and 6 JBA) exceeding the URL of cTnI (Fig. 1). The maximum post-effort value was observed at 1 h in 2 individuals, 3 h in 2 individuals, 6 h in 23 individuals, 12 h in 5 individuals, and 24 h in 2 individuals suggesting a degree of heterogeneity in cTnI appearance "kinetics". It is also pertinent to note one JBA had an elevated cTnI at 24 h post-exercise.

Peak post-exercise values of cTnI was not associated with player position, mean HR or $\%HR_{max}$, but was associated with baseline values ($r = 0.54$, $P = 0.001$).

Discussion

This is the first study to study post-exercise cTnI "kinetics" after intermittent exercise performed in a controlled environment in distinct groups of athletes differentiated by athlete status and biological age. The key findings from our study are; 1) intermittent exercise resulted in a cTnI change in all athletes although the magnitude of response was heterogeneous, 2) the baseline and post-exercise cTnI was lowest in ABA compared to PBA and JBA, and 3) the cTnI response post-exercise was similar in elite adult and junior basketball players.

Previous studies documenting cTn release during intermittent exercise have reported contradictory results. A plausible explanation for this contradictory evidence has been the limited and varied number of biomarker sampling points employed in past studies. Whilst the current study observed an increased in cTnI post-exercise in all participants this runs contrary to both George et al., (2004) and Rahnama et al., (2011) who did not observe any cTnI increase. The likely explanation for this discrepancy was the use of a simple pre-post exercise sampling regime in previous work. The current data supports Nie et al., (2008), in basketball players, and Carranza-García et al., (2011), in futsal players, who reported an increase in cTn several hours after simulated match. Consequently, our findings show, in line with continuous endurance exercise studies (Middleton et al., 2008; Shave et al., 2010), the importance of obtained multiple blood samples during recovery to observe the maximum post-effort value.

Individual heterogeneity of peak cTnI was observed with 26% of the players presenting with one cTnI value over the URL. This percentage is lower than reported after endurance events such as marathon (Shave et al., 2007) or cycling (Serrano-Ostáriz et al., 2009) and the more modest changes in the current study likely reflect the low total exercise duration and volume compared to many previous endurance and ultra-endurance activities (Shave et al., 2007). Previous studies have noted that the increase in

cTnI or cTnT is mainly mediated by the exercise duration when the intensity is controlled (Fu et al., 2009; Serrano-Ostáriz et al., 2011).

Our results support heterogeneity in peak cTnI appearance and analysis of time to peak cTnI suggest some degree of variance in the pattern or "kinetics" of cTnI appearance, although it is noted that all participants, bar one JBA, demonstrated a return to (almost) baseline levels at the 24 h assessment point. The one JBA with and elevated (above URL) cTnI at 24 h post-exercise was sign and symptom free and unremarkable in personal or exercise details. Several studies have suggested a single blood sample 3-4 h post-exercise will reflect cTn peak post-exercise (Fu et al., 2010; Nie et al., 2011; Tian et al., 2014). Our results are somewhat different with the most common time for cTnI peak at 6 h post-exercise. These data suggest that time when the peak post-exercise value may be dependent upon exercise mode, duration and/or intensity and further work is required.

The pattern of cTn appearance and clearance post-exercise runs contrary to changes in cTn observed in acute coronary syndromes. This suggests that post-exercise cTn levels may be related to a physiological rather than a pathological response after the exercise stimulus. The hypothesis arises that endurance exercise causes an increase in membrane permeability due to the physiological stress placed on the cell, inducing a transient cytosolic leakage due to membrane damage, rather than cardiomyocyte necrosis (Shave and Oxborough, 2012).

Recently, some studies have suggested that cTn release after exercise may be more pronounced in less trained subjects (Neilan et al., 2006; Tian et al., 2006; Fortescue et al., 2007; Mingels et al., 2009; Nie et al., 2011; Mehta et al., 2012) as a consequence of a lower myocardial work efficiency. Thus, cTn release during exercise in those with lower training levels or athlete status could be one consequence of the adaptive process

in myocardial cells, similarly to the process observed in skeletal muscle, which protects them against future bouts of strenuous exercise (Fortescue et al., 2007; Mehta et al., 2012). Our results did not support this theory with PBA and JBA demonstrating greater post-exercise values than ABA, even when PBA achieved a slightly lower mean HR and $\%HR_{max}$ during the simulated game.

The higher post-exercise cTnI values in PBA and JBA than ABA were associated with differences in baseline cTnI. The association between baseline and post-exercise cTnI values have been previously reported (Legaz-Arrese et al., 2011; Serrano-Ostáriz et al., 2011; Klinkenberg et al., 2012). In healthy population little attention has been focused to the variability of baseline cTn values. Mingels et al. (2009) obtained significantly higher hs-cTnT in males than in females. The authors justified these differences as a consequence of the heart size of men is higher than women. The same explanation could be used to justify the higher baseline and post-exercise cTnI values our PBA players (adults), because the size of heart is greater in athletes with higher performance (Legaz-Arrese et al., 2005; Legaz-Arrese et al. 2006), although it is not clear if heart size is different in JBA when compared to older ABA. Further research into the factors associated with the inter-subject variability in the baseline and exercise-related values of cTn are required.

The lack of differences between PBA and JBA players indicates that biological age likely doesn't mediate significant differences in the cTn response to intermittent exercise in elite athletes. Some caution should be expressed in this comment based on the small, but statistically significant, differences in CV work undertaken during exercise in PBA when compared to JBA.

Compared with samples of adolescents of similar age to our adolescents (16.2 ± 0.6 - 16.5 ± 1.6 years), several studies suggest that the cTn release after endurance exercise

might be greater among adolescent athletes compared with adults (Tian et al., 2006; Fu et al., 2010; Nie et al., 2011a; Nie et al., 2011b). Our data do not extend this concept to intermittent exercise in elite adult and adolescent athletes. If anything the PBA post-exercise cTnI might be blunted by the slightly lower absolute and relative exercise intensity during the basketball match. Previous studies have suggested that increased cTn in adolescent may reflect greater relative oxidative stress and lower myocardial work efficiency in an immature heart (Nie et al., 2011; Tian et al., 2012). It is possible, given the age of our JBA, that they are in the final stages of physical maturation although no biomarker of maturational status was assayed.

Clinicians should be aware that the release of cTnI is not exclusive to long-term strenuous efforts. cTnI values above the URL can be observed after intermittent exercise, such as basketball match, with heterogeneity noted in peak and kinetic data. From a clinical perspective, there is a limited rationale for full cardiovascular examination all athletes with positive cTn concentrations in the absence of other clinical signs and symptoms. When evaluating cTn in an emergency setting, detailed information regarding any recent exercise activities should be obtained, especially in the first 24 h post-exercise.

In addition to comments above we note the inherent limitation of cross-sectional studies in that the observed differences in the values of cTnI between PBA and ABA may have resulted from differences other personal factors (e.g. genetic differences) as well as exposure to different training volumes. To resolve this issue it would be interesting to observe the impact of a training program on exercise-induced cardiac biomarker release.

In conclusion, our results show that intermittent exercise, in this case a simulated basketball match, resulted in an increase in post-exercise cTnI in all athletes with a varied peak and time-to-peak cTnI. The second key finding was that athlete status

mediated cTn appearance with elite athletes presenting with higher values than amateur athletes. The impact of biological age between adult and adolescent elite basketball players was negligible.

Perspectives

In this study, we highlighted that intermittent high-intensity exercise, as basketball match, resulted in a cTnI change in all athletes although the magnitude of response was heterogeneous. In addition, our results did not support the hypothesis of previous studies suggesting that cTnI release after exercise may be more pronounced in less trained subjects (Neilant et al., 2006) and in adolescents (Tian et al., 2012) as a consequence of a lower myocardial work efficency. We also observed that the pattern of cTnI appearance and clearance post-exercise runs contrary to changes in cTn observed in acute coronary syndromes. This suggests that post-exercise cTn levels may be related to a physiological rather than a pathological response after the exercise stimulus. Finally this investigation prompts further research into the factors associated with the inter-subject variability in the baseline and exercise-related values of cTnI.

References

Bonetti A, Tirelli F, Albertini R, Monica C, Monica M, Tredici G. Serum cardiac troponin T after repeated endurance exercise events. Int J Sports Med 1996: 17: 259–262.

Carranza-García LE, George K, Serrano-Ostáriz E, Casado-Arroyo R, Caballero-Navarro AL, Legaz-Arrese A. Cardiac biomarker response to intermittent exercise bouts. Int J Sports Med 2011: 32: 327–331.

Eggers KM, Lagerqvist B, Venge P, Wallentin L, Lindahl B. Persistent cardiac troponin I elevation in stabilized patients after an episode of acute coronary syndrome predicts long-term mortality. Circulation 2007: 116: 1907–1914.

Eijsvogels T, George K, Shave R, Gaze D, Levine BD, Hopman MT, Thijssen DH. Effect of prolonged walking on cardiac troponin levels. Am J Cardiol 2010: 105: 267–272.

Fortescue EB, Shin AY, Greenes DS, Mannix RC, Agarwal S, Feldman BJ, Shah MI, Rifai N, Landzberg MJ, Newburger JW, Almond CS. Cardiac troponin increases among runners in the Boston Marathon. Ann Emerg Med 2007: 49: 137–143.

Fu F, Nie J, George K, Tom TK, Lin H, Shi Q. Impact of 21 –km run on cardiac biomarkers in adolescent runners. J Exerc Sci Fit 2010: 8: 61–66.

Fu F, Nie J, Tong TK. Serum cardiac troponin T in adolescent runners: effects of exercise intensity and duration. Int J Sports Med 2009: 30: 168–172.

George KP, Dawson E, Shave RE, Whyte G, Jones M, Hare E, Gaze D, Collinson P. Left ventricular systolic function and diastolic filling after intermittent high intensity team sports. Br J Sports Med 2004: 38: 452–456.

Klinkenberg LJ, Res PT, van Loon LJ, van Dieijen-Visser MP, Meex SJ. Strong link between basal and exercise-induced cardiac troponin T levels: Do both reflect risk? Int J Cardiol 2012: 158: 129–131.

König D, Schumacher YO, Heinrich L, Schmid A, Berg A, Dickhuth HH. Myocardial stress after competitive exercise in professional road cyclists. Med Sci Sports Exerc 2003: 35: 1679–1683.

Legaz-Arrese A, George K, Carranza-García LE, Munguía-Izquierdo D, Moros-García T, Serrano-Ostáriz E. The impact of exercise intensity on the release of cardiac biomarkers in marathon runners. Eur J Appl Physiol 2011: 111: 2961–2967.

Legaz Arrese A, Serrano Ostáriz E, González Carretero M, Lacambra Blasco I. Echocardiography to measure fitness of elite runners. J Am Soc Echocardiogr 2005: 18: 419–426.

Legaz-Arrese A, González-Carretero M, Lacambra-Blasco I. Adaptation of left ventricular morphology to long-term training in sprint- and endurance-trained elite runners. Eur J Appl Physiol 2006: 96: 740–746.

Léger L, Gadoury C. Validity of the 20 m shuttle run test with 1 min stages to predict VO2max in adults. Can J Sport Sci 1989: 14: 21–26.

Mehta R, Gaze D, Mohan S, Williams KL, Sprung V, George K, Jeffries R, Hudson Z, Perry M, Shave R. Post-exercise cardiac troponin release is related to exercise training history. Int J Sports Med 2012: 33: 333–337.

Middleton N, George K, Whyte G, Gaze D, Collinson P, Shave R. Cardiac troponin T release is stimulated by endurance exercise in healthy humans. J Am Coll Cardiol 2008: 52: 1813–1814.

Mingels A, Jacobs L, Michielsen E, Swaanenburg J, Wodzig W, van Dieijen-Visser M. Reference population and marathon runner sera assessed by highly sensitive cardiac troponin T and commercial cardiac troponin T and I assays. Clin Chem 2009: 55: 101–108.

Neilan TG, Januzzi JL, Lee-Lewandrowski E, Ton-Nu TT, Yoerger DM, Jassal DS, Lewandrowski KB, Siegel AJ, Marshall JE, Douglas PS, Lawlor D, Picard MH, Wood MJ. Myocardial injury and ventricular dysfunction related to training levels among nonelite participants in the Boston marathon. Circulation 2006: 114: 2325–2333.

Nie J, Tong TK, Shi Q, Lin H, Zhao J, Tian Y. Serum cardiac troponin response in adolescents playing basketball. Int J Sports Med 2008: 29: 449–452.

Nie J, George KP, Tong TK, Gaze D, Tian Y, Lin H, Shi Q. The influence of a half-marathon race upon cardiac troponin T release in adolescent runners. Curr Med Chem 2011a: 18: 3452–3456.

Nie J, Tong TK, George K, Fu FH, Lin H, Shi Q. Resting and post-exercise serum biomarkers of cardiac and skeletal muscle damage in adolescent runners. Scand J Med Sci Sports 2011b: 21: 625–629.

Rahnama N, Faramarzi M, Gaeini AA. Effects of Intermittent Exercise on Cardiac Troponin I and Creatine Kinase-MB. Int J Prev Med 2011: 2: 20–23.

Scott JM, Esch BT, Shave R, Warburton DE, Gaze D, George K. Cardiovascular consequences of completing a 160-km ultramarathon. Med Sci Sports Exerc 2009: 41: 26–34.

Serrano-Ostáriz E, Terreros-Blanco JL, Legaz-Arrese A, George K, Shave R, Bocos-Terraz P, Izquierdo-Álvarez S, Bancalero JL, Echavarri JM, Quilez J, Aragonés MT, Carranza-García LE. The impact of exercise duration and intensity on the release of cardiac biomarkers. Scand J Med Sci Sports 2011: 21: 244–249.

Serrano-Ostáriz E, Legaz-Arrese A, Terreros-Blanco JL, López-Ramón M, Cremades-Arroyos D, Carranza-García LE, Izquierdo-Alvarez S, Bocos-Terraz P. Cardiac biomarkers and exercise duration and intensity during a cycle-touring event. Clin J Sport Med 2009: 19: 293–299.

Shave R, Oxborough D. Exercise-induced cardiac injury: evidence from novel imaging techniques and highly sensitive cardiac troponin assays. Prog Cardiovasc Dis 2012: 54: 407–415.

Shave R, Ross P, Low D, George K, Gaze D. Cardiac troponin I is released following high-intensity short-duration exercise in healthy humans. Int J Cardiol 2010: 145: 337–339.

Shave R, George KP, Atkinson G, Hart E, Middleton N, Whyte G, Gaze D, Collinson PO. Exercise-induced cardiac troponin T release: a meta-analysis. Med Sci Sports Exerc 2007: 39: 2099–2106.

Tian Y, Nie J, Huang C, George KP. The kinetics of highly sensitive cardiac troponin T release after prolonged treadmill exercise in adolescent and adult athletes. J Appl Physiol (1985) 2012: 113: 418–425.

Tian Y, Nie J, Tong TK, Cao J, Gao Q, Man J, Shi Q, Liu W. Changes in serum cardiac troponins following a 21-km run in junior male runners. J Sports Med Phys Fitness 2006: 46: 481–488.

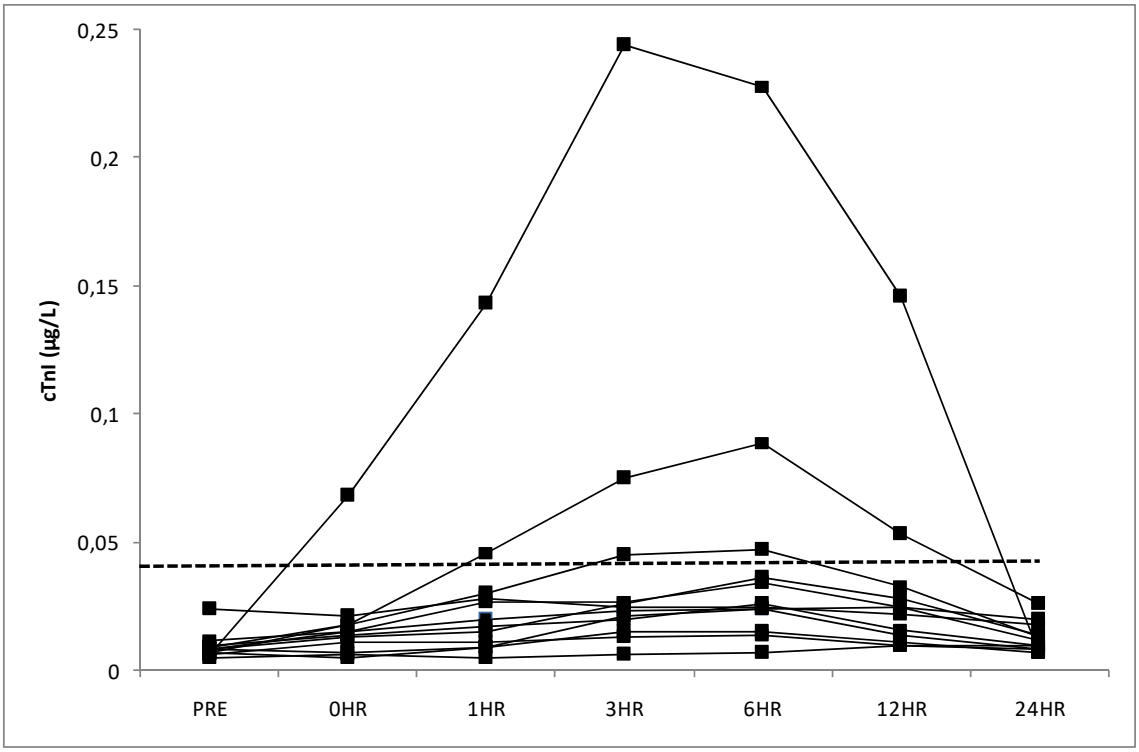
Wedin JO, Henriksson AE. Postgame elevation of cardiac markers among elite floorball players. Scand J Med Sci Sports 2014 Aug 11. doi: 10.1111/sms.12304.

Figue Legends

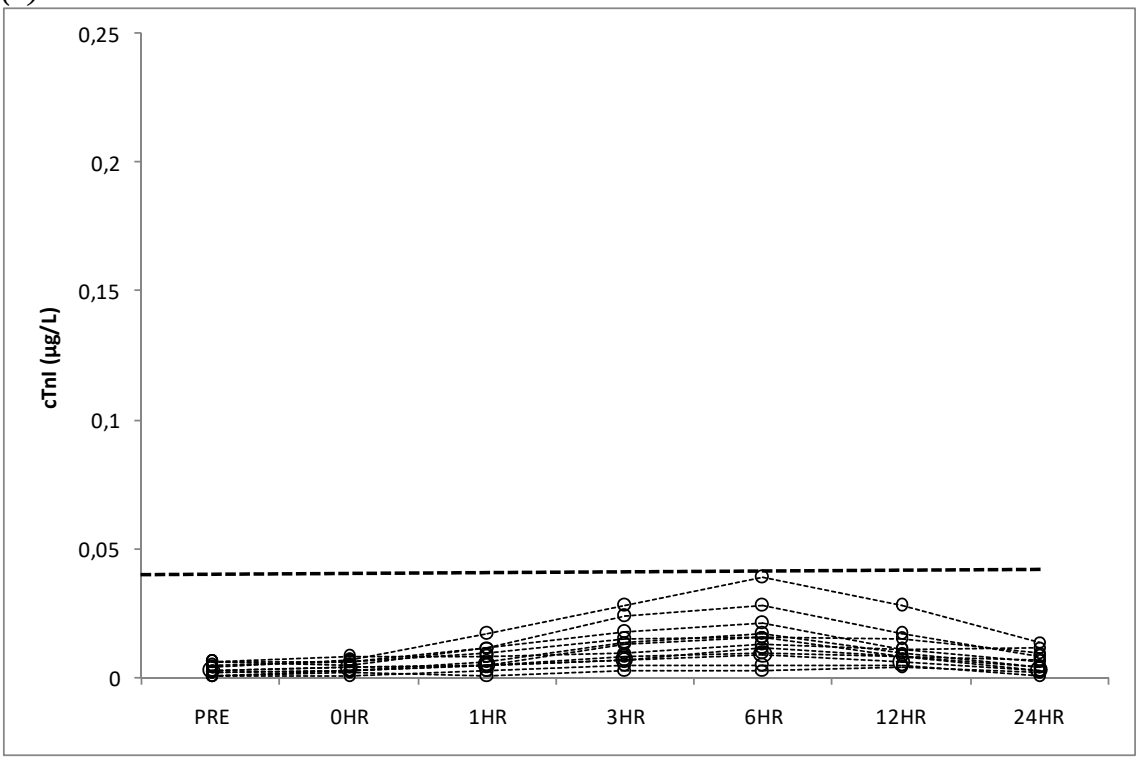
Fig. 1. Individual data points for cTnI (μg/L) in adult elite (PBA; n = 12) (a), adult amateur (ABA; n = 12) (b) and junior elite (JBA; n = 11) (c) basketball players at pre-exercise (PRE), as well as 5 min, 1, 3, 6, 12, and 24 h (0HR, 1HR, 3HR, 6HR, 12HR, 24HR, respectively). The horizontal dotted line is the upper reference limit (99th percentile) at 0.04 μg/L.

Figura 1. Cinética individual de cTnI (μg/L) en jugadores de baloncesto profesionales (*n* = 12) *(a)*, aficionados (*n* = 12) *(b)* y adolescentes (*n* = 11) *(c)*. La línea horizontal es el límite máximo de referencia.

(a)



(b)



(c)

Table 1. Characteristics of the basketball cohorts. Data are mean ± SD.							
	Age	Body	Heigh	VO_{2max}	Basketba	Basketball	Basketball

	(years)	mass (kg)	t (cm)	(ml/kg/min)	ll training history (years)	training frequency (sessions/week)	training volume (hours/week)
Adult Elite (PBA; *n* = 12)	27.3 ± 4.1$	98.3 ± 12.9*$	199 ± 7*$	58 ± 3	17 ± 5*$	6 ± 0*$	16 ± 0*$
Adult Amateur (ABA; *n* = 12)	29.6 ± 2.9#	83.8 ± 12.9	184 ± 6#	56 ± 7	13 ± 3#	4 ± 1	8 ± 4
Junior Elite (JBA; *n* = 11)	16.6 ± 0.9	82.8 ± 10.3	192 ± 8	58 ± 3	8 ± 4	4 ± 0	8 ± 0

* Significant difference between PBA and ABA basketball players.

Significant difference between ABA and JBA basketball players.

$ Significant difference between PBA and JBA basketball players.

Table 2. Heart rate during the basketball match. Data are mean ± SD.

	Mean HR (beats/min)	%HRmax (%)
PBA (*n* = 12)	150 ± 11*$	79 ± 4*$
ABA (*n* = 12)	168 ± 9	87 ± 3
JBA (*n* = 11)	167 ± 10	84 ± 4

* Significant difference between PBA and ABA basketball players.

$ Significant difference between PBA and JBA basketball players.

Table 3. cTnI (μg/L) before and after the basketball match in each cohort. Data are mean ± SD with the percentage of subjects with cTnI values exceeding the URL are shown in parentheses.

	Pre-exercise	5 min post	1 h post	3 h post	6 h post	12 h post	24 h post	p value		
								Time	Group	Time x Group
PBA (*n* = 12)	0.009 ± 0.005 (0)	0.018 ± 0.017 (8)	0.030 ± 0.037 (17)	0.045 ± 0.065 (25)	0.047 ± 0.061 (25)	0.033 ± 0.038 (17)	0.013 ± 0.006 (0)	0.000	0.001	0.174
ABA (*n* = 12)	0.003 ± 0.002 (0)	0.004 ± 0.002 (0)	0.007 ± 0.005 (0)	0.013 ± 0.008 (0)	0.016 ± 0.010 (0)	0.011 ± 0.007 (0)	0.006 ± 0.004 (0)			
JBA (*n* = 11)	0.011 ± 0.007 (0)	0.012 ± 0.008 (0)	0.023 ± 0.017 (9)	0.039 ± 0.039 (27)	0.052 ± 0.065 (36)	0.045 ± 0.060 (18)	0.029 ± 0.043 (27)			