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ORIGINAL

INFLAMMATORY AND ANTINFLAMMATORY RESPONSE AFTER ACUTE SWIMMING EXERCISE

RESPUESTA INFLAMATORIA Y ANTIINFLAMATORIA TRAS EL ESFUERZO AGUDO EN NATACIÓN

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ABSTRACT

The aim of the study was to analyze inflammatory and anti-inflammatory responses to an intense and acute swimming effort during the preparatory phase for the Brazilian National Adult Championship. Twenty swimmers participated. Plasma concentrations of IL-6, TNF-α, sTNFR1, IP-10 and MCP-1 were determined before and 40 minutes after 2 sets of 4 repetitions of 50 meters swam at maximum intensity with 3 minutes of recovery between each repetition and an active recovery swimming 1500m at low intensity after each of the two series. In response to acute exercise only the sTNFR1 concentration was reduced, with no changes in IP-10, IL-6 or TNF-α. Thus, acute exercise in

swimming in well-trained people does not seem to produce an inflammatory response.

KEY WORDS: swimming, cytokines, chemokines.

RESUMEN

El objetivo del estudio fue analizar la respuesta inflamatoria y antiinflamatoria a un esfuerzo agudo e intenso de natación, durante la fase preparatoria para los campeonatos nacionales absolutos Brasileños. Participaron 20 nadadores. Se determinaron las concentraciones plasmáticas de IL-6, TNF-α, sTNFR1, IP-10 y MCP-1, antes y 40 minutos después de 2 series de 4 repeticiones de 50 metros nadados a la máxima intensidad con 3 minutos de recuperación entre cada repetición y una recuperación activa nadando 1500m a intensidad suave después de cada una de las dos series. En respuesta al ejercicio agudo únicamente la concentración sTNFR1 se redujo, no presentando cambios en IP-10, IL-6 ni TNF-α. Por tanto el ejercicio agudo en natación en personas bien entrenadas no parece producir una respuesta inflamatoria.

PALABRAS CLAVE: natación, citoquinas, quimiocinas.

INTRODUCTION

Exercise has been traditionally prescribed to improve health, but there is evidence showing that the volume and intensity of training are determinant factors for both positive and negative effects (WALSH et al., 2011b). Regular moderate intensity exercise has been commonly associated to decreased sensibility to infections, but intense acute exercise has also been described as temporary immunologic decrease which can facilitate opportunist infections, phenomena known as the “immunological window” (Walsh et al., 2011a; Gleeson and Walsh, 2012).

Intense acute exercise may induce muscular damage producing the release of cytokines along with other factors in local tissue related to the inflammatory phenomena (Smith, 2000). The cytokines family is diverse, as well as its function which can act as a facilitator (proinflammatory) or inhibitor (antiinflamatory) for inflammation, as well as a facilitator for the production of muscular damage (Smith, 2000; Robson, 2003; Ahima and Park, 2015; Allen, Sun and Woods, 2015).

The tendency for infection as wel as muscular damage are related in a clearer way with high volumen exercise, high intensity with insufficient intervals, tipically associated with overtraining syndrome.

The aim of this study was to analyze the inflammatory response to intense acute swimming exercise, during the previous stages to national championships for these athletes.

MATERIAL AND METHODS

Participants

The simple was composed by 20 male swimmers in preparatory training stages for the Brazilian national champsionship which included 10 weekly sessions. All athletes had over five years experience in competitive swimming.

Study protocol

Participants signed a consent form voluntarily in order to participate in the study, mandatory by the National Counsil of Health (Brazil), nº 466/12, base don the Helsinki declaration (1964 and later resolutions). The study was approved by the Ethical Committee in Human Reserach of the Federal University of Minas Gerais, Brazil. All athletes had medical authorization to undergo the proposed activities.

The acute exercise was performed after a week without any competitions and after a rest day to avoid interference with analytical data. After a warm up of 1500m of light swim, two series of fours repetitions of 50m (2x[4x50]) were performed at the highest intensity starting at the starting block. A three minute break was done between each repetition, and an active break between the series with 1500m light swim. After the two series, another 1500m for recovery.

Assessment of cytokines and chemokines

Immediately before the exercise and 40 minutes after the end of the protocol, 5ml of blood were drawn through venipuncture of the antecubital vein. Immediately centrifugated at 2500 rpm for 15 minutes and the supernatant separated in aliquots of 300µl frozen at -80ºC until analytical determinations.

The cytokines and chemokines were measures through the ummunoabsorption analysis connected to enzymes (ELISA) following manufacturer's procedures (R&D Systems, Minneapolis, MN). Polystyrene plates with 96 slots of 100 µl were filled with antibody solution in destilled water of sodium bicarbonate (NaHCO3) with 8.4 mg/ml and sodium chloride (NaCl) with 5.8 mg/mL, at a 9.6 pH. For each plate, 10ml of antibody was used, incubating at 4ºC during 10 hours and after that period plates were washed four times with saline phosphate buffer (PBS) 1X which contains 0.1% Tween 20 (PBS-T20). Later is was stabilized with 200 µl bovine serum albumine solution (BSA), 200 mg/plate at 0.1% and distilled water for one hour at room temperature; following, samples were put in the plates for immunoenzymatic reaction. Again, plates were washed four times with the same types of solutions and 60 µL of the samples were dilluted 1:50 with the PBS 1X solution, 500µl of distilled water in each of the six microtubes.

In each of the slots 100µl were added being the first two with only PBS 1X. The reading plate held the samples in pairs and incubation was performed for 10 hours at 4°C; after this period the plates were washed again for four times with

the same solutions used previously. 100µl of solution with antibodies were added in each slot, except for cytokine IL-6 because for its determination 10µl of ram serum was added per plate. Plates were covered and put on a rack for two hours at room temperature for cytokine TNF-α and receptor sTNFRI and one hour for cytokine IL-6. Then, after washing the plates for four times with the same solutions, another 100µl was added to each slot of a 2.5µl streptavidin solution for every 10ml solvent and shook during 25 minutes. Again the plats were washed four times with the same solutions. Next, the reaction added 50µl per slot of sulphuric acid (H2SO4) 1M. The optical density Reading was conducted by an ELISA detector.

Statistical Analysis

Statistical analyses were perfomed with the statistical package Graphic Prisma 4.0. Descriptive statistics was used as mean and standard deviation.

To verify normality, Shapiro-Wilk test was used for all variables. The statistical significance between the distinct parameters with normal distribution were showed through a t-test with significance level set at $p<0.05$.

RESULTS

On Table 1, decriptive data on age, body weight, and height of the study subjects.

Table 1. General characteristics of swimmers

		mean	± standard deviation
Age	(years)	18,7	± 1,1
Body weight	(kg)	72,4	± 4,0
Height	(cm)	179,0	± 7,0

On table 2 are the results of determinations of cytokines and chemokines. It is seen that in response to acute exercise there were no significant changes in IL-6, TGN-a, MCP-1 and IP-20, but there was a decrease in the levels of sTNFR1.

Table 2. Values of chemokines and cytokines (mean ± standard deviation)

	Pre	Post	p-value
IL-6 (pg/mL)	4.3 ± 3.7	4.5 ± 5	0.949
TNF-a (pg/mL)	230.4 ± 299.5	241.5 ± 416.9	0.925
sTNFR1 (pg/mL)	143.2 ± 34.7	83.4 ± 31.6	0.000
MCP-1 (pg/mL)	9.2 ± 12.9	7.759 ± 12.64	0.722
IP-10 (pg/mL)	909.4 ± 618.2	956.8 ± 700.6	0.826

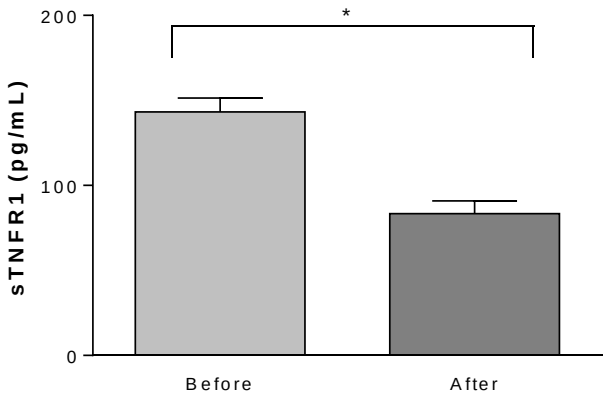

Figure 1: Concentration of cytokine inhibitor sTNFR1 in swimmers
Statistically significant (*)

DISCUSSION

Inflammatory response in this study was assessed by the response to intense acute exercise. We focused on cytokines and chemokines response to acute exercise, even though there is difficulty in interpreting the determinations of relative parameters to the immune system since it is a complex system where celular and hormonal factors also play a role. Nonetheless, it is interesting to study it since this system is affected by acute exercise importantly, in function of the intensity and duration (Walsh et al., 2011b).

In this study, the response in proinflammatory cytokine TNF-α on acute swimming exercise showed no changes, contrary to Nemet et al., (2003) where there was a decrease. In our study, levels of IL-6 were also the same likely because, as Pedersen et al., (2003) stated, TNF-α, IL-6 and cytokine are closely related and one of the functions of TNF-α is to estimulate the production of IL-6 during exercise and the IL-6 produced during exercise probably inhibits the effect TNF-α has to facilitate the resistance to insulin in periferic tissue (Pedersen et al., 2003). Similar results have been described in a study by Morgado et al., (2012), where no chages were found in inflammatory responses.

Another posible reason there were no modifications in the concentrations for IL-6 as a response to exercise in swimmers is what Morgado et al., (2012) suggest, the antiinflamatory protection that chronic exercise tends to produce, protecting over the physiological stress caused by exercise. It has been described that IL-6 facilitates the maintenance of the metabolic homeostasis of the carbón hidrates and lipids during long duration exercises (Pedersen et al., 2003). Perhaps it could be assumed that the no variation of IL-6 in response to the exercise performed in this study is due to the protocol of acute exercise causing an important reduction in muscular glycogen levels.

In a study by Morici et al., (2005) in which ta supramaxim effort was performed, the levels of IL-6 did not increase immediately, as well as in a study by Li and Gleeson (2005). However, the last study had a significant increase four days

after acute exercise. It could be expected a cummulative effect of the levels of IL-6 as a consequence for the work developed throughout the days, but our study was conducted after a rest day after a week of training of high volumen but low intensity.

The subjects in our study had low baseline levels of proinflammatory cytokines IL-1β and TNF-α, what could be due to the inhibitor effects of other cytokines of short half life, moreover, the low levels of such cytokines could contribute to preventing a great activation of systemic inflammation as suggested by Nieman et al., (2001). Lastly, the lack of response from the cytokines to exercise proposed by this study could be that, in order to produce a proinflammatory response, according to Suzuki et al., (2006), it is necessary a greater muscular damage, same for the increase of IL-1β, where our athletes did not have an apparent muscle damage. In studies such as Harnish and Sabo (2016), an elevation of TNF-α was found after exercise, maintaining it high for 24 hours after exercise.

In this study, the response of sTNFR1 to exercise consisted in a significant decrease. Out results are not similar to those in the literature, Pussieldi et al., (2014), after acute exercise in mountain biking in which an increase in antiinflammtory response was found and of this cytokine in particular.

The sTNFR1, joined to the TNF-α, presentes a longer half life than TNF-α in circulation, as described by Pussieldi et al. (2014), who found a return in the baseline levels of this cytokine after 24 hours of the end of the exercise. In swimmers, the sTNFR1 presented a decrease after 40 minutes of the end of the exercise, even though it was at a high level.

As a response to the exercise, there were also no differences in the plasmatic levels of MCP-1, similarly observed with levels of IP-10. Even though swim events are performed at a maximum intensity because athletes are looking to reach their best times, there were no increases in levels of MCP-1 as it would be expected. Therefore, the suggestion that levels of MCP-1 do not change after a maximum intensity exercise in swimming due in part to the duration of the effort and moreover the lack of mechanical impact with a clear predominance of concentric components to the muscular actions in this sport, where muscular damage would be small. As we have indicated, muscular damage is correlated to the systemic inflammatory (Pyne and Gleeson, 1998; Fehrenbach and Schneider, 2006). In a study by Willoughby et al. (2003), in which the effects of excentric and concentric contractions in muscular function and inflammatory response were studied, they found excentric contractions induce to higher muscular damage when compared to concentric, and higher increments in levels of IL-6.

The plasmatic concentrations of chemokine IP-10 did not produce changes in response to acute exercise in swimmers, probably due to the low accumulation of fatigue during previous training once Walsh et al. (2011) suggest that the intensity of exercise is an important factor for the response of the immune system to exercise so intense exercise would seem to have a cumulative effect in immune response. On the other hand, it is not clear the response of

chemokine IP-10 to acute exercise in the data reflected in the literature, which adds to the difficulty to compare these data. In a study by Deyhle et al. (2016) a significant increase in IP-10 was observed after a series of maximum excentric exercises, opposite to what was found in our study.

In our study these seems to be no increase in antiinflammatory response since there was no increase of proinflammatory cytokines even though there was a decrease in the concentration of na inhibitor cytokine, sTNFR-1, similar to Fischer et al. (2004) where after a ten week training, adaptations were reflected in the reduction of the levels of inflammatory cytokines as a response to acute exercise. Similarly, King et al. (2003) also found significant decrease in the response of inflammatory markers in participants of diverse sports, including swimming, but their results were inconclusive since duration and intensity of exercise were not controlled.

CONCLUSION

In trained athletes, acute exercise in swimming does not produce a generalized inflammatory response, significant muscular damage, nor compromisos immunitary response.

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