©Journal of Sports Science and Medicine (2015) **14,** 702-707
http://www.jssm.org

Case report

Clinical and Laboratory Responses of Cross-Country Skiing for a 24-H World Record: Case Report

Markus Niemelä [1], Jukka Juvonen [2,3], Päivikki Kangastupa [4], Onni Niemelä [4]✉ and Tatu Juvonen [1]
[1] Department of Surgery, Oulu University Hospital, [2] Internal Medicine, Oulu University Hospital, [3] Kainuu Central Hospital, Kajaani, [4] Department of Laboratory Medicine and Medical Research Unit, Seinäjoki Central Hospital, and University of Tampere, Finland

Abstract
The physiological consequences of ultra-endurance cross-country skiing in cold conditions are poorly known. We report here clinical, echocardiographic and laboratory findings from a 41-y old male elite skier in a world record trial for 24-h skiing. The athlete completed a total of 406.8 km outdoors with the temperature ranging between -24°C and –5°C during the 24-h period. Post exercise, notable increases from baseline values were observed in myoglobin (50-fold), creatinine kinase (30-fold) and proBNP (6-fold), whereas troponin T or troponin I levels remained unchanged. At baseline, echocardiographic findings showed cardiac hypertrophy and after skiing, a 5% reduction of left-ventricular end-diastolic dimension. Increases in markers of kidney (creatinine) and liver function (alanine aminotransferase), serum uric acid, C-reactive protein and white blood cell counts were also noted. In addition, electrolyte disturbances including hyponatremia, hypophosphatemia and hypocalcaemia were noted during the follow-up. The data indicates that a prolonged period of high-intensity skiing leads to muscle, heart and kidney affection and activation of inflammation even in an experienced elite skier. The observed health effects underscore the need for strict medical surveillance of participants in extreme sports with long duration.

Key words: Ultra-endurance sports, organ function, muscle, kidney, heart.

Introduction

Physical activity in proper amounts is widely acknowledged to be a key part of a healthy lifestyle leading to a reduction in the risk of health problems (O'Donovan et al., 2010; Ruiz et al., 2011; Schnohr et al., 2015). The relationships between doses of physical activity and health have, however, remained as a subject of scientific debate (Schnohr et al., 2015). Over the past few decades, marathon running and long-distance skiing have gained increasing popularity such that even tens of thousands of individuals now participate in many of such events. While the potentially harmful effects of marathon and ultramarathon running to participants have already been addressed in a number of publications (Fortescue et al., 2007; Lippi et al., 2012; McCullough et al., 2011; Niemelä et al., 1984; Roberts 2007), less is known on the biological responses of uninterrupted long-distance skiing in cold weather.

Unlike in running, skiing involves most of the body for propulsion and therefore, different patterns of eccentric-muscle-activity induced damage may be expected to result from these two types of exercises. In the present work, we report data from a male elite skier who volunteered for detailed medical observations and laboratory measurements in association with his world record trial for 24-h cross-country skiing.

Case report

We examined a 41-y-old male elite skier in association with a world record trial for 24-h cross-country skiing. He completed a total of 406.8 kilometres on cross-country skis outdoors with the temperature ranging between –24°C and –5°C during the 24-h period. Venous blood samples were drawn for the determination of blood cell counts and biomarkers of muscle, heart, kidney and liver function, and inflammatory and metabolic parameters before and at various time points 3 to 48 hours after finishing the activity. In addition, serum electrolytes, cortisol and blood lactate levels were followed. Measurements values were determined using standard clinical chemical and haematological methods in an SFS-EN ISO/IEC 17025:2005 and SFS-EN ISO 15189:2007 accredited laboratory. Echocardiographic (ECHO) measurements were obtained before the experiment and at three hours after finishing. The study was carried out after informed consent from the participant after detailed instructions and discussions on the possible risks, benefits and alternatives of the experimental protocol. The study was approved by the institutional review board and conducted according to the provisions of the Declaration of Helsinki.

On the day of the experiment, the skier commenced his activity at noon with a rather steady speed of 20 km/h over the initial six hours. At that time, the sky was clear with an outdoor temperature of –7°C. Humidity ranged from 50% to 100% and the wind speed fell between 1 to 11 km/h during the 24-h period. In the evening the temperature dropped down markedly the lowest temperature (–24°C) being reached at 05:00 h the next morning. Due to the colder and tougher conditions the participant´s speed of skiing during the night slowed down to approximately 17 km/h. Signs of generalized fatigue, leg muscle pain and toe numbness developed gradually during the latter part of the experiment; however, the athlete was remarkably able to increase his speed of skiing to over 20 km/h for the last hour and he finished the experiment in a reasonable good mood and physical condition. The outdoor temperature at the time of finishing was

Received: 01 July 2015 / Accepted: 27 July 2015 / Published (online): 24 November 2015

Table 1. Main cardiac ECHO findings before (baseline) and 3 h after 24 h cross–country skiing.

	Unit	Baseline	After 3 h	Difference	Normal value
LVEDD	mm	55	52	–5.5%	46–52
LVESD	mm	34	35	2.9%	28–37
LVEF	%	67	59	–11.9%	≥ 55
IVS	mm	12	13	8.3%	7.1–11.1
LVPW	mm	13	13	0.0%	7.3–10.0
RVEDD	mm	31	30	–3.2%	10.0–26.0
Mitral e velocity	cm/s	7.4	6.3	–14.9%	
e'	cm/s	9	8	–11.1%	
e/e' ratio		8.6	7.9	–8.1%	

LVEDD, left ventricular end diastolic diameter; LVESD, left ventricular end-systolic dimension; LVEF, left ventricular ejection fraction; IVS, interventricular septum; LVPW, left ventricular posterior wall; RVEDD, right ventricular end-diastolic diameter; e, early diastolic velocity; e' mitral annular early diastolic velocity; e/e' ratio, diastolic function marker

–5°C. While skiing, liberal amounts of various fluids and food (mostly exogenous carbohydrates) were provided ad libitum. After the trial, the athlete was carefully monitored in a hospital setting while being hydrated (approximately 300 $mL \cdot h^{-1}$) for the next 20 h. The self-concept of athletic ability expressed by the participant and the medical and psychological assessments suggested a rapid recovery, which was completed within 7 d with no apparent signs of emotional or physiological exhaustion remaining.

The body weight was 79.1 kg before the trial and 76.7 kg after finishing thus decreasing by 3.0%. Clinical examinations during the hospital follow-up indicated pain observed in quadriceps areas whereas, no apparent other symptoms occurred. ECHO cardiographic data before and after the experiment is summarized in Table 1. At baseline, the athlete showed cardiac hypertrophy with a large left ventricular (55 mm) and right ventricular (31 mm) end diastolic diameters and increased interventricular septal thickness (12 mm). In comparison between the pre- and postrace values the left ventricular end-diastolic dimension (EDD) was reduced by 5.5%, whereas the end-systolic dimension (ESD) increased only slightly from 34 mm to 35 mm (2.9%). Ejection fraction and markers of diastolic function declined approximately by 10% when compared to baseline values.

In muscle and cardiac markers, there was a 50-fold increase in serum myoglobin and a 30-fold increase in creatinine kinase (CK) levels in a sample taken 7 h post skiing (Figure 1). There were also marked (15-fold) elevations in CK-MBm and pro-BNP (6-fold) levels, whereas troponin T and troponin I, markers of cardiomyocyte damage, even as assayed using ultrasensitive techniques, remained stable and within normal limits (Figure 1). Serum creatinine values also exceeded the upper normal limits and returned to normal within 48 h.

CRP, a marker of inflammation and of acute phase response, increased 10-fold (Figure 2). Blood leukocytes and serum uric acid values were also above normal briefly after finishing. Blood platelet counts decreased initially and returned to normal levels in a sample taken 48 h after skiing. Liver enzymes (AST and ALT) increased approximately 5-fold; however with different patterns of kinetics such that AST was an earlier responder. Serum sodium, phosphate and calcium concentrations decreased during the follow-up, whereas potassium remained within normal limits (Figure 2). Post blood gas analysis showed elevated pO_2 (6.4–8.3 $mmol \cdot L^{-1}$), pH (7.47–7.50) and a base excess ranging between 6.4 and 8.3 $mmol \cdot L^{-1}$ in samples taken between 7 and 20 h post exercise. Blood lactate levels remained normal in all samples (< 1.2 $mmol \cdot L^{-1}$). Serum cortisol level was highest in a sample taken 3 h after finishing.

Discussion

The data shows that a well-trained athlete is able to ski more than 400 km during 24 h in cold conditions without any apparent clinical signs of exhaustion, hypothermia or lactic acid build-up. ECHO measurements taken before the trial showed cardiac hypertrophy as a sign of long-term chronic effect of exercise, which has also previously been shown to be a typical finding among ultramarathon runners, the left-ventricular end-systolic dimensions (LVEDD and LVESD) serving even as a predictor of faster race times (Nagashima et al., 2006). In the present case, the prolonged period of skiing was found to result in a mild reduction in left ventricular function as an acute effect of exercise, although this occurred with no obvious clinically significant signs of cardiac fatigue. The ECHO cardiovascular consequences were comparable to those seen previously as the result of competitive running of a 160- km (Scott et al., 2009) or running for 24 h (Niemelä et al., 1984), suggesting a risk of impaired left ventricular performance upon extreme sports activity of long duration. Depressed left ventricular diastolic function has been observed previously even as a result of marathon running among non-elite race participants (Neilan et al., 2006).

Current data shows notable changes in those biochemical parameters reflecting skeletal muscle injury. Such biomarkers increased in a similar manner to that previously observed among marathon and ultramarathon runners although running and skiing are markedly different in terms of eccentric muscle activity how the body is used for creating force to the movement. The release of large quantities of myoglobin and creatinine kinase from muscles into circulation may increase the risk of kidney damage especially under conditions of dehydration when excess myoglobin can precipitate in renal tubules leading to tubular obstruction and tissue necrosis (Holt and Moore, 2001; Lappalainen et al., 2002). Creatinine kinase levels peaked here at nearly 8000 $U \cdot L^{-1}$ and previous research has indicated that levels over 5000 $U \cdot L^{-1}$ are

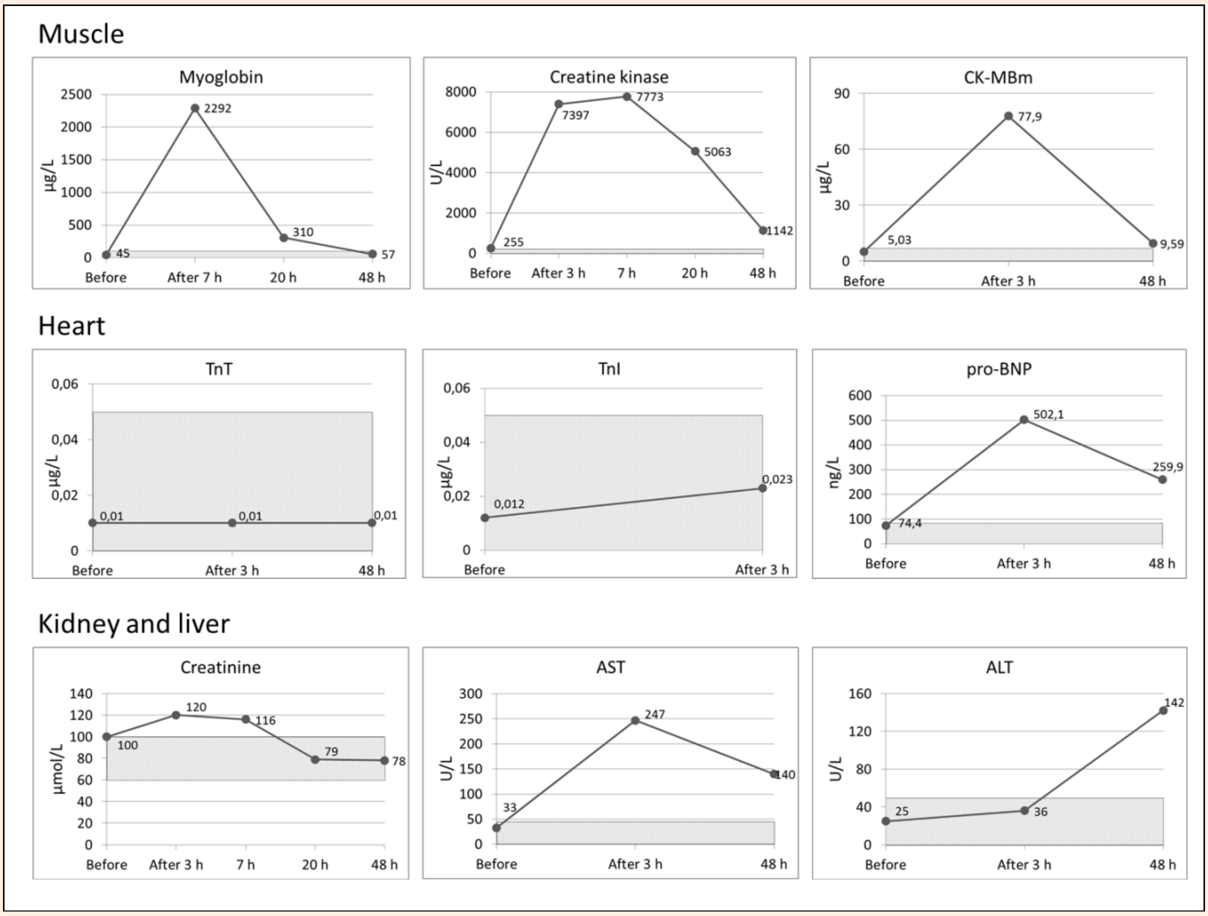

Figure 1. Changes in laboratory markers before and at various time points after finishing 24-hour skiing. The shaded area demonstrates the normal limits of each parameter. ALT, alanine aminotransaminase; AST, aspartate aminotransferase; BNP, brain natriuretic peptide; CK-MBm, creatine kinase isoenzyme MB mass.

associated with a 50% risk of acute renal failure (Huerta-Alardín et al., 2005). A slight increase was noted here in serum creatinine indicating kidney affection which was rapidly resolved suggesting only a transient change in renal filtration and perfusion. Similar observations were recently reported among 10 out of 25 individuals (40%) from a marathon running trial (McCullough et al., 2011). Kidney damage as a result of strenuous exercise may be associated with haem-protein injury, free radical generation and lipid peroxidation. In support of this view, the present data also shows elevation of uric acid concentrations, which, due to its free radical scavenging properties, may be considered a sign of an increased need of antioxidant capacity during the exercise (Kurra et al., 2009; Waring et al., 2003). White blood cells and C-reactive protein also increased suggesting an activation of inflammation, which may show a synergistic relationship between the generation of oxidative stress and production of reactive oxygen species (Devries et al., 2008).

Interestingly, the tests for cardiomyocyte damage, both troponin T and troponin I, remained normal whereas proBNP increased to levels commonly found in patients with myocardial infarction or unstable angina. The lack of relationship between proBNP and troponin levels suggests, however, that the present observations in cardiac status are primarily related to the mechanical load and myocardial wall stress. Previous trials among runners have frequently shown increases in serum troponin levels in marathon (Fortescue et al., 2007; Mingels et al., 2009) and even among a high percentage (53–65%) of half-marathon (Lippi et al., 2012) finishers with diverse running experience. While normal troponin levels in the present case may be explained by excellent cardiac health, long training history and associated metabolic adaptation, it should be noted that the frequently observed troponin increases as a result of marathon running may deserve further attention as possible indicators of cardiomyocyte damage in susceptible individuals. It is possible that during strenuous exercise increased myocardial wall oxidative stress and impaired endothelial function could lead to an increased risk of atherothrombotic events (Dawson et al., 2008).

Previous research has suggested that mechanical and thermal (heat) injury, ATP depletion and the generation of oxygen free radicals are important determinants of muscle damage in marathon runners (Roberts, 2007). Exposure to cold conditions could aggravate tissue ischemia by reducing muscle perfusion and by exacerbating hyponatremia through uncontrolled sweating and hemodynamically inappropriate antidiuretic hormone secretion.

It is also possible that the combination of prolonged exercise and cold exposure can create additive

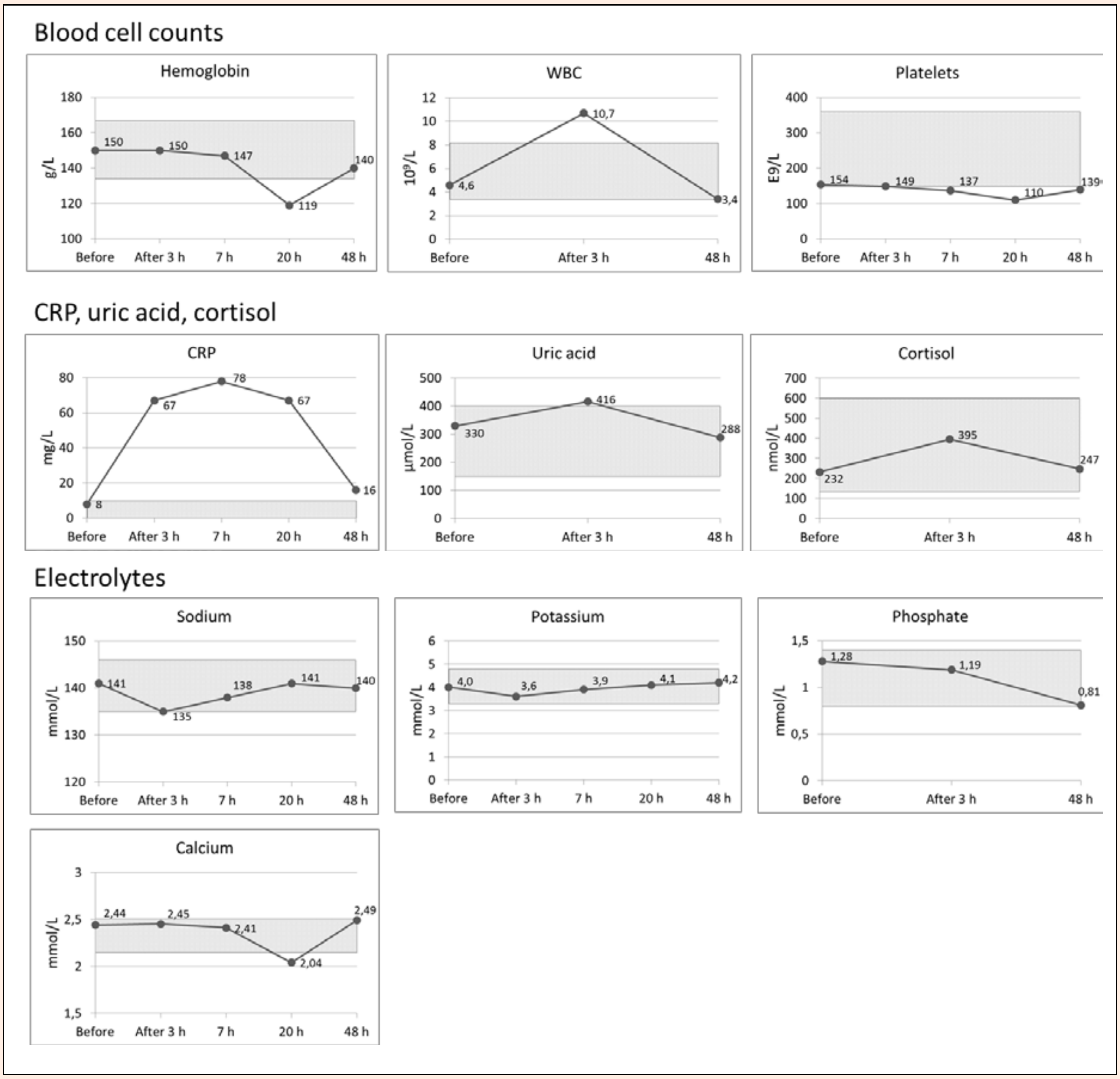

Figure 2. Changes in laboratory markers before and at various time points after finishing 24-hour skiing. The shaded area demonstrates the normal limits of each parameter. CRP, C-reactive protein.

effects on muscle and kidney status. Decreased plasma calcium in the present instance may indicate an increased flow to the intracellular space as also found in patients with rhabdomyolysis as a key feature of muscle destruction. Symptoms of a possible hypocalcaemia- related cardiovascular compromise were, however, not observed here. Obviously, the early aggressive re-hydration with saline after the trial may have facilitated a rapid recovery thereby preventing the development of myocyte necrosis and kidney injury.

Our data further indicates that a prolonged period of high intensity cross-country skiing may lead to changes in serum liver enzymes. The reactions between the transaminase enzymes (ALT, AST) were of similar magnitude but showed different patterns of kinetics and alterations in their ratios. While the mechanism behind such observations remain unclear it is likely that AST increases, which followed similar kinetics to those of the muscle-derived enzymes, originate from the muscle compartment. The response ALT response, which is considered more specific to the liver, however, similarly exceeded the upper normal limit by 5-fold suggesting also the possibility of a disturbed liver cell integrity. Since the physiological role of serum aminotransferases is to catalyse the transfer of amino groups to generate products in gluconeogenesis and amino acid metabolism, it is possible that the changes could also be related to the process of acute changes to maintain energy and nutrient homeostasis (Gerhart-Hines et al., 2007). Rapid changes in energy needs and availability can trigger a rearrangement of glucose and lipid metabolism in both skeletal muscle and liver. As glucose is the main cellular fuel substrate, peripheral tissues must undergo a shift from glucose to fatty acid oxidation and gluconeogenetic precursors to be delivered from muscle to the liver for glucose synthesis.

Conclusion

The present case demonstrates that cross-country skiing lasting for 24 h leads to marked abnormalities in biochemical parameters reflecting reduced organ health even in an experienced elite athlete. We suggest a careful follow-up and an effective hydration schedule during and after extreme sports of this nature in order to prevent the development of severe health threats, including rhabdomyolysis and kidney injury. The present data should also be considered in the medical surveillance of the continuously increasing number of individuals participating in long duration extreme sports events.

Acknowledgements
No conflicts of interest

References

Dawson, E.A., Whyte, G.P., Black, M.A., Jones, H., Hopkins, N., Oxborough, D., Gaze D., Shave, R.E., Wilson, M., George, K.P. and Green, D.J. (2008) Changes in vascular and cardiac function after prolonged strenuous exercise in humans. *Journal of Applied Physiology* **105**, 1562-1568.

Devries, M.C., Hamadeh, M.J., Glover, A.W., Raha, S., Samjoo, I.A. and Tarnopolsky, M.A. (2008) Endurance training without weight loss lowers systemic, but not muscle, oxidative stress with no effect on inflammation in lean and obese women. *Free Radical Biology and Medicine* **45**, 503-511.

Fortescue, E.B., Shin, A.Y., Greenes, D.S., Mannix, R.C., Agarwal, S., Feldman, B.J., Shah, M.I., Rifai, N., Landzberg, M.J., Newburger, J.W. and Almond, C.S. (2007) Cardiac troponin increases among runners in the Boston Marathon. *Annals of Emergency Medicine* **49,** 137-143.

Gerhart-Hines, Z., Rodgers, J.T., Bare, O., Lerin, C., Kim, S.H., Mostoslavsky, R., Alt, F.W., Wu, Z. and Puigserver, P. (2007) Metabolic control of muscle mitochondrial function and fatty acid oxidation through SIRT1/PGC-1alpha. *EMBO Journal* **26**, 1913-1923.

Holt, S.G. and Moore, K.P. (2001) Pathogenesis and treatment of renal dysfunction in rhabdomyolysis. *Intensive Care Medicine* **27**, 803-811.

Huerta-Alardín, A.L., Varon, J. and Marik, P.E. (2005) Bench-to-bedside review: Rhabdomyolysis -- an overview for clinicians. *Critical Care* **9**, 158-169.

Kurra, V., Eräranta, A., Jolma, P., Vehmas, T. I., Riutta, A., Moilanen, E., Tahvanainen, A., Kalliovalkama, J., Niemelä, O., Myllymäki, J., Mustonen, J. and Pörsti, I. (2009) Hyperuricemia, oxidative stress, and carotid artery tone in experimental renal insufficiency. *American Journal of Hypertension* **22**, 964-970.

Lappalainen, H., Tiula, E., Uotila, L. and Mänttäri, M. (2002) Elimination kinetics of myoglobin and creatine kinase in rhabdomyolysis: implications for follow-up. *Critical Care Medicine* **30**, 2212-2215.

Lippi, G., Schena, F., Dipalo, M., Montagnana, M., Salvagno, G.L., Aloe, R. and Guidi, G.C. (2012) Troponin I measured with a high sensitivity immunoassay is significantly increased after a half marathon run. *Scandinavian Journal of Clinical & Laboratory Investigation* **72**, 467-470.

McCullough, P.A., Chinnaiyan, K.M., Gallagher, M.J., Colar, J.M., Geddes, T., Gold, J.M. and Trivax, J.E. (2011) Changes in renal markers and acute kidney injury after marathon running. *Nephrology (Carlton)* **16**, 194-199.

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

Nagashima, J., Musha, H., Takada, H. and Murayama, M. (2006) Left ventricular chamber size predicts the race time of Japanese participants in a 100 km ultramarathon. *British Journal of Sports Medicine* **40**, 331-333.

Neilan, T.G., Januzzi, J.L., Lee-Lewandrowski, E., Ton-Nu, T.T., Yoerger, D.M., Jassal, D.S., Lewandrowski, K.B., Siegel, A.J., Marshall, J.E., Douglas, P.S., Lawlor, D., Picard, M.H. and Wood, M.J. (2006) Myocardial injury and ventricular dysfunction related to training levels among nonelite participants in the Boston marathon. *Circulation* **114**, 2325-2333.

Niemelä, K.O., Palatsi, I.J., Ikäheimo, M.J., Takkunen, J.T. and Vuori, J.J. (1984). Evidence of impaired left ventricular performance after an uninterrupted competitive 24 hour run. *Circulation* **70**, 350-356.

O'Donovan, G., Blazevich, A. J., Boreham, C., Cooper, A. R., Crank, H., Ekelund, U., Fox, K.R., Gately, P., Giles-Corti, B., Gill, J.M., Hamer, M., McDermott, I., Murphy, M., Mutrie, N., Reilly, J.J., Saxton, J.M. and Stamatakis, E. (2010) The ABC of Physical Activity for Health: a consensus statement from the British Association of Sport and Exercise Sciences. *Journal of Sports Sciences* **28**, 573-591.

Roberts, W.O. (2007) Heat and cold: what does the environment do to marathon injury? *Sports Med* **37**, 400-403.

Ruiz, J.R., Morán, M., Arenas, J. and Lucia, A. (2011) Strenuous endurance exercise improves life expectancy: it's in our genes. *British Journal of Sports Medicine* **45**, 159-161.

Schnohr, P., O'Keefe, J. H., Marott, J.L., Lange, P. and Jensen, G.B. (2015) Dose of jogging and long-term mortality: the Copenhagen city heart study. *Journal of the American College of Cardiology* **65**, 411-419.

Scott, J.M., Esch, B.T., Shave, R., Warburton, D.E., Gaze, D. and George, K. (2009) Cardiovascular consequences of completing a 160-km ultramarathon. *Medicine and Science in Sports and Exercise* **41**, 26-34.

Waring, W.S., Convery, A., Mishra, V., Shenkin, A., Webb, D.J. and Maxwell, S.R. (2003) Uric acid reduces exercise-induced oxidative stress in healthy adults. *Clinical Science (London)* **105**, 425-430.

Key points

- An elite athlete was able to ski over 400 km during 24 hours with an outdoor temperature ranging between –5 °C and –24 °C.
- Several postrace abnormalities occurred in biomarkers of muscle, heart, kidney, liver and inflammation status.
- Serum troponins, specific markers of myocardial cell damage, remained stable.
- The report supports careful medical surveillance of participants in extreme sports with long duration.

AUTHOR BIOGRAPHY

Markus NIEMELÄ
Employment
Medical Faculty, University of Oulu, Finland
Degree
Medical student
Research interest
Physical activity and health
E-mail: markus.v.niemela@student.oulu.fi

Jukka JUVONEN
Employment
Head of Internal Medicine, Kainuu Central Hospital, Kajaani, Finland
Degree
M.D., Ph.D.
Research interest
Cardiology
E-mail: jukka.juvonen@kainuu.fi

Päivikki KANGASTUPA
Employment
Department of Laboratory Medicine, Seinäjoki Central Hospital, Finland
Degree
Ph.D.
Research interest
Clinical chemistry
E-mail: paivikki.kangastupa@epshp.fi

Onni NIEMELÄ
Employment
Professor of Laboratory Medicine, Seinäjoki Central Hospital and University of Tampere, Finland
Degree
M.D., Ph.D.
Research interest
Biomarkers, lifestyle, liver disease
E-mail: onni.niemela@epshp.fi

Tatu JUVONEN
Employment
Professor, Department of Surgery, Oulu University Hospital, Finland
Degree
M.D., Ph.D.
Research interest
Cardiology, ischemia-reperfusion injury, neuroprotection
E-mail: tatu.juvonen@oulu.fi

✉ **Onni Niemelä, MD, PhD**
Department of Laboratory Medicine, Seinäjoki Central Hospital and University of Tampere, Finland