Thermal strain and fluid balance during a 72-km military route march in a field setting

Beng Hoong Poon1,2, MBBS, MMed, Suriya Prakaash1,3, MBBS, MMed, Ya Shi Teo1, BSc, Priscilla Weiping Fan4, BSc, Jason Kai Wei Lee4,5,6,7,8,9,10, PhD, FACSM
1Singapore Armed Forces, HQ Medical Corps, 2Chairman Medical Board Office, 3Department of Sports and Exercise Medicine, Changi General Hospital, 4Defence Medical and Environmental Research Institute, DSO National Laboratories, 5Human Potential Translational Research Programme, 6Department of Physiology, Yong Loo Lin School of Medicine, 7N.1 Institute for Health, 8Global Asia Institute, National University of Singapore, 9Institute for Digital Medicine, Yong Loo Lin School of Medicine, National University of Singapore, 10Singapore Institute for Clinical Sciences, Agency for Science, Technology and Research (A*STAR), Singapore

Abstract

Introduction: A physiological profiling study was conducted to evaluate thermal strain as well as fluid and electrolyte balances on heat-acclimatised men performing a 72-km route march in a field setting.

Methods: 38 male soldiers (age range 18–23 years) participated in the study, as part of a cohort that marched for 72 km, with loads for about 26 hours. Core temperature and heart rate sensors were used for the duration of the march. Fluid and food intake and output were monitored for the duration of the march. Blood samples were taken one day before the march (pre-march), immediately after the march (Post 1) and on the 15th day after the march to ascertain recovery (Post 2) to assess fluid and electrolyte profiles.

Results: Mean core temperature was within safe limits, ranging from 37.1 to 38.1°C throughout the march. There was an overall decrease in serum sodium levels, a decline in serum sodium concentration in 28 participants and three instances of hyponatraemia (serum sodium concentration <135 mmol/L).

Conclusion: Our study found low thermal strain heat-acclimatised individuals during a 72-km route march. However, there was an overall decrease in serum sodium levels, even when the participants were allowed to drink *ad libitum*. Challenges of exercise-associated hyponatraemia during prolonged activities remain to be addressed.

Keywords: Endurance, exercise, hydration, prolonged physical activity, soldiers

INTRODUCTION

In military operations, exertional heat injuries as well as fluid and electrolyte imbalances continue to be salient challenges.[1] The physiological profiles of different activities vary and understanding these profiles is crucial to risk assessment and subsequent management. Since prolonged exercise is one of the key risk factors for incurring significant dehydration or exercise-associated hyponatraemia (EAH), accurate assessment of fluid balance will allow safe hydration recommendations during extreme events to prevent incidences of hyper- and hyponatraemia. Research on understanding the physiological determinants during extreme events is limited. Furthermore, there is limited data concerning the physiological responses to extreme events collected under field conditions.[2]

It is therefore important to profile the actual demands of extreme events in a field setting to gain critical insights into physiological responses and thereby enhance both safety and performance, as recommendations derived from laboratory studies can, at times, misrepresent actual demands (either due to restrictions or the complexities of replicating the actual activity in a laboratory setting).

Correspondence: Prof Jason Kai Wei Lee, Human Potential Translational Research Programme, Yong Loo Lin School of Medicine, National University of Singapore, Singapore, 2 Medical Drive, 117593, Singapore. E-mail: phsjkwl@nus.edu.sg

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We profiled an extreme event of a 72-km military route march, as it provided an excellent field setting to study an array of military-related stressors. To the best of our knowledge, this was the first study to systemically profile the heat strain and fluid balance of participants during a 72-km route march in a field setting.

**METHODS**

During the 72-km route march, soldiers were required to march for a day without sleep while carrying a load of approximately 20 kg throughout. Ethics approval was obtained from the Institutional Review Board of the Defence Medical and Environmental Research Institute, DSO National Laboratories, Singapore.

This activity was conducted at the final stage of a course and the participants were heat acclimatised. The study recruited 38 healthy young soldiers to participate and informed consent was obtained in writing after reading a document describing the nature, benefits and risks of the study. The unit medical officer medically reviewed and cleared the participants for their participation in the study. A health declaration form was administered prior to the commencement of each sampling point.

The mean 2.4-km run timing of participants was 9.8 ± 0.5 minutes, and accordingly, the mean VO\(_{2\text{max}}\) was estimated to be 53 ± 3 mL/minute/kg based on the ACSM metabolic equation.\(^{(1)}\) Anthropometry measurements for height and body composition were obtained five days before the 72-km route march and the physical characteristics of participants were also recorded.

To estimate thermal and cardiovascular strain, core temperatures and heart rates were monitored. On the evening before the march, each participant ingested the VitalSense® temperature sensor for continuous core temperature monitoring. An ambulatory core temperature data recording system (Mini Mitter Co, Bend, OR, USA) was placed in two sealed waterproof bags and carried in the uniform pockets throughout the route march. Heart rate was monitored throughout the route march through the strapping of a heart rate sensor and wearing of a heart rate monitoring watch (Polar Vantage, Polar Electro Oy, Kempele, Finland).

Measurements were taken one day before the march (pre-march), immediately after the march before the participants had any opportunity to recover (Post 1) and on the 15th day post march to ascertain recovery (Post 2). Nude body mass and blood biochemistry were measured at these time points.

Blood samples were obtained via venipuncture of a forearm from each participant at the pre-march, Post 1 and Post 2 time points, for which blood electrolytes, glucose and urea concentrations were determined immediately using a handheld analyser (i-STAT®1 Analyser; Abbott Point of Care Inc, Princeton, NJ, USA); aldosterone and arginine-vasopressin (AVP) were determined via radioimmunoassay (Coat-A-Count Aldosterone assay; Siemens Healthcare Diagnostics Inc., Los Angeles, CA, USA and BÜHLMANN Laboratories AG, Schönenbuch, Basel-Land, Switzerland, respectively). Serum osmolality was calculated using the formula:

\[
\text{Serum osmolality (mOsm/kg)} = 2 \times [\text{Na}^+] + [\text{BUN}] / 2.8 + \frac{[\text{Glucose}]}{18}, \text{where Na}^+ \text{ was serum sodium and BUN denoted blood urea nitrogen.}
\]

We measured fluid intake and urine output, if any, at every rest point (total of 18) of the march in addition to the above mentioned measurements at the pre-march, Post 1 and Post 2 time points. Fluid intake was monitored by recording the mass of the participants’ water canteen or water bags at each rest stop, taking into account the top-up made. Fluids consumed from canned drinks during meals were also recorded and included in the computation of fluid intake. Participants were allowed to consume their own food and fluids, with intake accounted for through collection of food wrappers and left over food at the end of the activity for recording and analysis using the Foodworks Professional 2007 software version 5 (Xyris, Brisbane, Queensland, Australia).

The 72-km route march commenced at 0825 hours. Participants marched at a common pace, as a cohort, and carried a 17.1 ± 0.8 kg load. Eighteen rest stops at 4-km intervals were made along the 72-km route and participants rested for 15–84 minutes at each rest stop. Three meals (i.e. lunch, dinner and supper) were provided during the march. Fluids were consumed ad libitum and plain water was provided for refilling at every rest stop.

Environmental conditions were monitored throughout the march: wet bulb, dry bulb and globe temperatures, relative humidity and wind velocity were measured using a portable climate data logger (Squirrel 2020 Universal Input Data Logger; MicroDAQ.com Ltd, Contoocook, NH, USA) at each rest stop. The wet bulb globe temperature (WBGT) index was calculated as 0.1 (T\(_{\text{dry bulb}}\)) + 0.7 (T\(_{\text{wet bulb}}\)) + 0.2 (T\(_{\text{glob}}\)).

SPSS for Windows version 15.0 (SPSS Inc, Chicago, IL, USA) was used for statistical analysis. Paired t-test and one-way analysis of variance followed by post hoc Tukey’s test, whenever necessary, were used to analyse differences between measurements. Relationships between parameters were analysed using Pearson’s product moment correlation coefficient (r). Normality of data was assessed using the Shapiro-Wilk test. Normally distributed data was presented as mean ± standard deviation, whereas non-normal data was presented as median (range). Statistical significance was accepted as P < 0.05. Graphs and data were presented as mean ± standard deviation, unless otherwise indicated.

**RESULTS**

To cover the 72 km, the participants walked for 16 hours and 27 minutes with a total of 9 hours and 29 minutes of
rest (inclusive of the lunch, dinner and supper breaks). The average pace of the route march was between 3.6–5.3 km/hour. Participants carried a total load of approximately 17.1 ± 0.8 kg, made up of their full pack, load-bearing vest (10.1 ± 0.7 kg), water bottle (1.2 ± 0.04 kg), water bag (3.0 ± 0.3 kg) and weapon (2.8 kg). Based on the marching pace of about 3.6–5.3 km/hour, with a load of 17.1 kg, metabolic equivalent of task (MET) was estimated to be 5 during marching. Resting MET was estimated to be 1.5.

The physical characteristics of our participants are presented in Table 1.

Throughout the route march, the median WBGT was 26.6°C, with a range of 24.2°C to 30.2°C. This level of heat stress would be denoted as mild-to-moderate based on international guidelines by the American College of Sports Medicine. Relative humidity averaged 78% and was in the range 55%–95%, and these conditions are common in Singapore. Thus, the environmental conditions during the march were considered moderate and would not have induced high levels of thermal strain in the participants.

Throughout the march, the mean core temperature of participants ranged from 37.1°C to 38.1°C [Figure 1a]. Hypothermia and hyperthermia did not occur at any point along the route march, as minimum and maximum core temperature were 36.7°C and 38.7°C, respectively. Computed from the corresponding core temperature and heart rate, the mean Physiological Strain Index (PSI) [3] was 3 ± 1 [Figure 1b] for 21 participants with complete data.

The participants were instructed to drink fluids *ad libitum* and the average fluid consumption was 9.9 ± 2.0 L over 26 hours, inclusive of fluid intake from food or other sources. A few participants drank between 1.5–2.0 L of fluids between rest stops (within approximately 1.5 hours).

Mean sweat loss was 10.95 ± 1.58 L, and the rate of sweat loss was approximately 0.42 ± 0.06 L/hour. Sweat loss correlated negatively with the change in serum sodium concentration (R = −0.36, P < 0.05). All participants lost body mass, with an average of 4% of body mass lost immediately after the route march at Post 1. [Figure 2].

At the end of the route march, 34 of 38 participants provided usable blood samples (Post 1) and a significant decrease of 0.7% in mean serum sodium concentrations (pre-march vs. Post 1: 139 mmol/L vs. 138 mmol/L) was seen between the pre-march and Post 1 samples [Figure 3]. Three participants were hyponatraemic at the end of the route march (serum sodium level <135 mmol/L) and there was no incidence of hypernatraemia (serum sodium level >145 mmol/L). Among 34 participants, 28 (82.4%) participants had a decline in serum sodium concentration. Among the hyponatraemic participants, two participants were asymptomatic (serum sodium levels 133 mmol/L and 134 mmol/L) and one participant was symptomatic (130 mmol/L), but subsequently recovered well without a need for medical intervention.

Among participants who provided blood samples, there was a 220% increase in aldosterone (n = 29) and a 200% increase of AVP (n = 28) levels, on average, immediately after the 72-km route march. For one participant with EAH, AVP levels increased slightly (pre-march vs. Post 1: 2.7 pg/mL vs. 5.5 pg/mL) even though serum sodium levels and osmolality both decreased. AVP levels for the other two participants with hyponatraemia could not be obtained.

### Table 1. Participants’ physical characteristics (n=38).

| Variable            | Mean ± standard deviation (range) |
|---------------------|----------------------------------|
| Age (yr)            | 20.9 ± 1.0 (18.0-23.0)           |
| Body mass (kg)      | 66.5 ± 5.6 (53.7-80.1)           |
| Height (m)          | 1.74 ± 0.06 (1.61-1.83)          |
| Body mass index (kg/m²) | 21.4 ± 1.7 (18.7-26.0)       |
| Body fat (%)        | 10.7 ± 3.2 (6.3-18.0)            |

**Figure 1:** Graphs show (a) body core temperatures (n = 30) and (b) corresponding PSIs (n = 21) during the 72-km military route march. PSI: Physiological Strain Index, Tc: core temperature.
DISCUSSION

The military is well attuned to the dangers of heat stress and has established robust systems based on evidence to mitigate the risk. Meta‑analysis of published studies have concluded that heat adaptation regimes induce adaptations to high ambient temperature, exercise performance and capacity in the heat.[4] Cardiovascular fitness is also an important contributor to exercise‑heat acclimatisation that acts to minimise physiological strain, reduce risk of heat injury and sustain exercise capacity.[5] Our participants had undergone systematic heat‑acclimatisation regimes and also progressively improved their cardiovascular fitness prior to taking part in the 72‑km route march. The pace of the march was also calibrated over the years in line with the changes in training safety regulations.

This physiological profile study of heat‑acclimatised participants revealed that the observed core temperatures were within safe thermal thresholds, as the core temperature for which the individual was at the threshold of fatigue and risk of heat injury was often near or beyond 40°C. Furthermore, it is not uncommon for core temperatures to reach 40°C–42°C during exercise.[6,7] Similarly, physiological strain was relatively mild throughout the route march; the core temperature and heart rate profiles of our participants were within the limits of core temperature and heart rate upper constraints of 39.5°C and 180 beats/minute, respectively, for the purpose of PSI. The mild thermal strain, for this activity, was due to the low intensity of exercise relative to the degree of heat acclimatisation and cardiovascular fitness of the participants. The participants marched at a MET of 5 and, taking oxygen consumption to be 3.5 mL/minute for 1 MET/kg body mass, the mean oxygen consumption for the march (VO₂) would be approximately 17.5 mL/kg/minute. With a mean VO₂ max of 53 mL/minute/kg among our participants, they were exercising on approximately an average of 33% VO₂ max during the march.

In contrast to heat stress, the quality of information available on EAH to the public is poor. It was noted in a 2016 review that only 7.3% of 110 websites discussed that fluid intake should be based on thirst and only half of them highlighted the risk of hyponatraemia from overhydration.[8] In the early 2000s, awareness of hyponatraemia in marathon runners was raised through a series of reports,[9‑11] and even the deaths of two female charity marathon runners.[12] The Third International EAH Consensus Development Conference held in 2015 concluded that the primary aetiology and mechanism leading to EAH was the overconsumption of hypotonic fluids, likely in combination with non‑osmotic stimulation of AVP secretion.[13] In addition to over‑drinking, other risk factors for EAH included prolonged low‑intensity exercise, and warm and humid environmental temperatures.

Over‑drinking is a key risk factor in the development of EAH. It is known that prolonged low‑intensity activities, such as the present study, carries a significant risk of EAH, as the long duration allowed sodium losses to accumulate while the low intensity of exercise permitted participants to consume fluids more frequently.

Compared against other studies, participants in our study consumed 9.9 ± 2.0 L of fluids over 26 hours versus a study profiling a 110‑km military route march, wherein participants consumed 14 L over 24 hours.[14] This may have accounted for the greater decline in serum sodium levels of 5.6% recorded in the latter study when compared to 0.7% in our study. That our
participants’ sweat loss correlated negatively with the change in serum sodium indicated that sodium depletion was a probable additional contribution to the fall in sodium concentration. In contrast, studies of a shorter route march of 25 km over four hours found that there was no decrease in serum sodium concentration after the activity.\(^{[15]}\)

Body mass gain was seen in a majority of, but not all, athletes who became symptomatic with EAH\(^{[16]}\) and it was observed in the 2002 Boston Marathon that the strongest single predictor of hyponatraemia was considerable body mass gain during the event.\(^{[17]}\) This study demonstrated the limitations of using body mass as an indicator for events that last more than several hours. And even though all participants lost body mass (average loss of 4%) at the end of the route march, this could not be directly used to correlate with their hydration status, as body mass change was a combined effect of fluid and energy intake, substrate oxidation, water stored with muscle glycogen made available to the body water pool, as well as both fluid and energy losses (including fat mass losses).

The 220% increase in aldosterone and 200% increase of AVP levels, on average, immediately after the 72-km route march in our study were not uncommon and similar increases have been recorded in other studies. In two ultramarathon events (of 161 km and 56 km), the aldosterone increases recorded were 700%\(^{[18]}\) and 660%\(^{[18]}\) respectively. Studies measuring AVP levels following prolonged endurance exercise have also shown similar increases of 200%.\(^{[19,20]}\) Our study was not able to provide further information on the non-osmotic stimulation of AVP secretion as a contribution to EAH, as we were only able to obtain AVP levels from one of three participants with EAH.

While there are considerable risk management practices for the mitigation of dehydration and exertional heat injury in the military, challenges confronting the prevention of EAH in the field setting are not fully addressed for a myriad of reasons.

Fluid and electrolyte imbalances are common among military personnel performing demanding physical activities over long durations. The emphasis on adequate hydration, a robust fluid resupply chain at all levels and hydration policies to prevent dehydration are deeply entrenched in the consciousness of commanders and soldiers alike. Consequently, there is a subconscious downplaying of the risk of over-consumption of fluids and EAH. From the perspective of training safety policies, it is pragmatic and sensible to set a minimum fluid consumption guideline and ensure fluid availability.

The primary EAH risk mitigation approach continues to be the implementation of educational programmes to a wide audience, including commanders, soldiers and medical staff, to correct misconceptions regarding hydration. The mistaken, but oft-heard, ‘drinking beyond the point of thirst’ must be corrected to an emphasis on application of ad libitum drinking. Also, recognising the signs and symptoms of EAH and its medical treatment would need to be emphasised, similar to that of exertional heat injuries.

Further research should seek to identify parameters that are reliable predictors of levels of hydration and, more importantly, include the development of a simple field-usable tool to evaluate hydration status and predict the risk of EAH for individuals to supplement the use of body mass gain as a surrogate measure of hydration status. Training safety policies could then be updated to mitigate the risk of EAH.

Our study was not without limitations. Being a field study, the measurements could not be too demanding and sophisticated, as that would affect participants’ performance on the route march. Changes in the blood profile post exercise could be masked by the interval between the end of the route march and sampling time. Restoration, for at least some of these parameters, might have occurred rapidly after the march. Finally, it was difficult to ascertain the precise contributions of the singular effects of physical exertion, sleep deprivation and psychological stress on the outcome variables in this study.

In conclusion, there is generally limited information on the thermal and fluid demands of extreme events, especially in a military context. The data from this study revealed that there was low thermal strain during a 72-km route march for heat-acclimatised individuals with good cardiovascular fitness. However, it was noted that there was an average overall decrease in serum sodium levels and three participants had hyponatraemia even when the participants were allowed to drink ad libitum. Challenges confronting the prevention of EAH in the field setting warrant more research.

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Conflicts of interest

There are no conflicts of interest.

Supplementary material

The Appendix is available online.
REFERENCES

1. Carter R 3rd. Exertional heat illness and hyponatremia: An epidemiological prospective. Curr Sports Med Rep 2008;7:S20-7.
2. DeGroot DW, Kenney WL. Thermal response to running across the Sahara desert: Data for three men. Aviat Space Environ Med 2008;79:909-13.
3. Glass S, Gregory B. ACSM’s Metabolic Calculations Handbook. Baltimore: Lippincott Williams and Wilkins; 2007:25-74.
4. Tyler CJ, Reeve T, Hodges GJ, Cheung SS. The effects of heat adaptation on physiology, perception and exercise performance in the heat: A meta-analysis. Sports Med 2016;46:1699-724.
5. Périard JD, Travers GJS, Racinais S, Sawka MN. Cardiovascular adaptations supporting human exercise-heat acclimation. Auton Neurosci 2016;196:52-62.
6. Dill DB, Soholt LF, McLean DC, Drost TF Jr, Loughran MT. Capacity of young males and females for running in desert heat. Med Sci Sports 1977;9:137-42.
7. Nadel ER, Wenger CB, Roberts MF, Stolwijk JA, Cafarelli E. Physiological defenses against hyperthermia of exercise. Ann N Y Acad Sci 1977;301:98-109.
8. Hoffman MD, Bross TL 3rd, Hamilton RT. Are we being drowned by overhydration advice on the Internet? Phy Sportsmed 2016;44:343-8.
9. Davis DP, Videen JS, Marino A, Vilke GM, Dunford JV, Van Camp SP, et al. Exercise-associated hyponatremia in marathon runners: A two-year experience. J Emerg Med 2001;21:47-57.
10. Hew TD, Chorley JN, Cianca JC, Divine JG. The incidence, risk factors, and clinical manifestations of hyponatremia in marathon runners. Clin J Sport Med 2003;13:41-7.
11. Hsieh M, Roth R, Davis DL, Larrabee H, Callaway CW. Hyponatremia in runners requiring on-site medical treatment at a single marathon. Med Sci Sports Exerc 2002;34:185-9.
12. Siegel AJ, Verbalis JG, Clement S, Mendelson JH, Mello NK, Adner M, et al. Hyponatremia in marathon runners due to inappropriate arginine vasopressin secretion. Am J Med 2007;120:461.e11-7.
13. Hew-Butler T, Rosner MH, Fowkes-Godek S, Dugas JP, Hoffman MD, Lewis DP, et al. Statement of the Third International Exercise-Associated Hyponatremia Consensus Development Conference, Carlsbad, California, 2015. Clin J Sport Med 2015;25:303-20.
14. Ashkenazi I, Epshtein Y. Alternations in plasma volume and protein during and after a continuous 110-kilometer march with 20-kilogram backpack load. Mil Med 1998;163:687-91.
15. Nolte HW, Noakes TD, Van Vuuren B. Trained humans can exercise safely in extreme dry heat when drinking water ad libitum. J Sports Sci 2011;29:1233-41.
16. Noakes TD, Sharwood K, Speedy D, Hew T, Reid S, Dugas J, et al. Three independent biological mechanisms cause exercise-associated hyponatremia: Evidence from 2,135 weighted competitive athletic performances. Proc Natl Acad Sci U S A 2005;102:18550-5.
17. Almond CS, Shin AY, Fortescue EB, Mannix RC, Wypij D, Binstadt BA, et al. Hyponatremia among runners in the Boston Marathon. N Engl J Med 2005;352:1550-6.
18. Stumpfle KJ, Lehmann DR, Case HS, Bailey S, Hughes SL, McKenzie J, et al. Hyponatremia in a cold weather ultraendurance race. Alaska Med 2002;44:51-5.
19. Hew-Butler T, Hoffman MD, Stumpfle KJ, Rogers IR, Morgenthal NG, Verbalis JG. Changes in copeptin and bioactive vasopressin in runners with and without hyponatremia. Clin J Sport Med 2011;21:211-7.
20. Hew-Butler T, Dugas JP, Noakes TD, Verbalis JG. Changes in plasma arginine vasopressin concentrations in cyclists participating in a 109-km cycle race. Br J Sports Med 2010;44:594-7.
APPENDIX

Deriving effective sweat loss

The effective sweat loss in our participants was calculated based on the following formulae first put forth by Maughan et al in 2007\(^{(1)}\) and subsequently adjusted by King et al in 2008.\(^{(2)}\)

\[
SL = BML + IF + FI - UL - RWL - SO + MW, \text{ where}
\]

- \(SL\) = sweat loss
- \(BML\) = body mass loss (measured)
- \(IF\) = ingested fluid (measured)
- \(FI\) = real food intake (measured)
- \(UL\) = urine output (measured)
- \(RWL\) = respiratory water loss (calculated based on formula below)
- \(SO\) = substrate oxidation (measured with assumption below)
- \(MW\) = metabolic water (calculated based on assumption below)

\(RWL\) was calculated using the following equation:

\[
m_e = 0.019 \times VO_2 (44 - P_a), \text{ where}
\]

- \(m_e\) = rate of evaporative water loss from the respiratory tract (g/min)
- \(P_a\) = ambient water vapour pressure (mmHg)

\[
= \left[ \text{relative humidity} \times \text{saturated vapour pressure} \right] / 100
\]

SO refers to the loss of mass from carbohydrates and fats consumed throughout the route march. These were assumed to be expended entirely during the course of the route march as all participants lost body mass after the march. MW loss refers to the water released during oxidation of substrates (carbohydrates and fats) as well as glycogen stores. The method used
in this study follows that stated in the study by Maughan et al and assumes each gram of carbohydrate oxidised liberates one gram of water.

The figure below illustrates the mean sweat loss rate (L/hour) of the 38 participants.

References

1. Maughan RJ, Shirreffs SM, Leiper JB. Errors in the estimation of hydration status from changes in body mass. J Sports Sci 2007; 25:797-804.

2. King RF, Cooke C, Carroll S, O’Hara J. Estimating changes in hydration status from changes in body mass: considerations regarding metabolic water and glycogen storage. J Sports Sci 2008; 26:1361-3.