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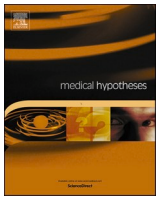
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Clinically-relevant reductions in oxygen partial pressure as possible contributor to cardiovascular benefits of sauna practice

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ABSTRACT

The practice of sauna has been found to have both acute and long-term cardiovascular benefits, which are generally postulated to be a result of thermoregulatory physiological adaptations. Another element of sauna conditions which has been overlooked is that the extremely high absolute water content of air at sauna temperature, even at low relative humidity, results in significantly decreased partial pressure of oxygen. Using the Arden-Buck equation for water-carrying capacity of air along with the barometric formula, it is shown in this hypothesis that typical sauna conditions have an oxygen partial pressure reduction that may be equivalent to significant elevations above sea level. This effect may also be enhanced by lower air density further reducing available oxygen relative to respiratory volume.

This paper presents the hypothesis that altitude adaptation may be a contributing factor in the cardiovascular benefits of sauna treatments, suggesting that sauna should be considered as an alternative in instances where intermittent hypoxic training is indicated but not available, and that clinical research into sauna treatment is merited for conditions in which intermittent hypoxic training is known to have applications. The hypothesis could be investigated through pulse oximetry of subjects under sauna conditions and by tracking blood markers of altitude adaptation compared to a control group using steam rooms.

Introduction

The practice of hot-air dry bathing, or sauna, refers to spending short periods in an enclosed room with temperature elevated to supra-physiologic temperatures. Typically, sauna conditions are characterized by temperatures of 80 °C to 90 °C with relative humidity between 5 % and 20 %, and duration may be as short as 5 min but in some studies has ranged as high as 30 – 40 min with short breaks. One study used a temperature of 100 °C (+- 10 °C) and relative humidity of 34 %-45 % [1], representing the extreme upper end of temperature as well as an unusually high humidity. Regular use of a sauna has been found to be safe and well-tolerated, and to confer a range of modest health benefits [2,3]. The physiological effects of sauna are attributed to thermoregulatory adaptations that occur in response to extreme temperatures drastically exceeding the range of conditions that are survivable on an ongoing basis. A sauna differs from a steam room mainly in its temperature and humidity ranges. A steam room will typically have a temperature between 35 °C and 45 °C, with relative humidity at or very near to 100 %.

Despite the much higher temperature of a sauna, both of these practices represent conditions that are not physiologically sustainable, as the human body, with a core temperature around 37 °C, is not able to effectively discharge heat in either of these sets of conditions.

In the case of a steam room, the driving forces for heat transfer are small, approaching zero. This is because the temperature being close to physiologic conditions means that there is a minimal driving force for conduction and convection of heat, and having relative humidity at 100 % at that temperature also precludes evaporation as a mechanism for heat transfer. Conversely, in a sauna there are still mechanisms by which heat transfer can take place, albeit in both directions. The extreme temperatures of the sauna allow for conduction of heat into the body from the surrounding air but the relative humidity being below 100 % does allow for heat loss through evaporation. Hence, it is not readily apparent which of the two practices imposes a larger thermoregulatory stress and, in fact, this may depend on physiological parameters of the user such as sweating and respiration rates, which will influence thermoregulation differently in the two practices.

There is evidence that regular saunas can result in improvements in

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athletic performance [3]. One study examining competitive athletes who underwent saunas at (89.9 ± 2.0 ∘C) following endurance training found that running performance was significantly increased in the experimental group compared to the control, with 32 % increase in time to exhaustion on a treadmill test at current best 5 km pace [4]. That study found statistically significant increases in total blood volume and plasma volume, with an observed increase in red blood cell volume but with a correlation of low statistical confidence.

The increased blood volumes were not accompanied by significant changes in haematocrit or Haemoglobin concentration. The resulting net increase in Haemoglobin mass and blood oxygen carrying capacity is likely a significant contributing factor in the noted improvements of athletic endurance/performance.

A 5 week trial of Heat Stress Training (HST) on elite cyclists performing light exercise under considerably less extreme temperature conditions (37.8 ± 0.5 °C; 65.4 ± 1.8 % RH) [5] reported increased haemoglobin mass, but no significant proportional changes in aerobic endurance measures compared to the control group. Another 3 week trial of sauna, 28 ± 2 min post-exercise (101–108 °C, 5–10 % RH) 3 ± 1 times per week, on a more mixed cohort [6]. This study, involving ten apparently healthy male volunteers aged 19 to 31 years, was aimed at establishing possible effects of a three-week course of normobaric intermittent hypoxic training (IHT) on the state of endothelial function (EF).

IHTs were performed on a daily basis with each training session lasting 60 min and the FIO2 operating level equal to 9 % in the 5-min hypoxia/5-min normoxia cyclic mode. EF state and pulse wave velocity (PWV, ΔPWV) were evaluated using a Tonocard device by a noninvasive method based on the capacity of the endothelium to release nitric oxide (NO) during reactive hyperemia. The adaptation to intermittent hypoxia was accompanied by a significant ($p < 0.05$) 34.3 % increase in the erythropoietin (Epo) concentration, a larger than twofold increase in the reticulocyte count, a 6.4 % increase in the erythrocyte count, and a 4.1 % increase in the hemoglobin content. After the course of IHT, the value of EF increased by 38.9 % ($p < 0.05$), which could be caused by a higher level of endothelium-dependent relaxation in muscular arteries. At the same time, the PWV and ΔPWV values reflecting the elastic–viscous characteristics of vascular wall remained at the prior-to-the study level. The data obtained in the study are discussed in this article from the position of possible trigger effects of the hypoxia-inducible transcription factors (HIF-1 and HIF-2), which create a broad molecular basis for the activation of endothelial cells and the increase in the NO production. Apart from increasing the erythrocyte production, which is greatly important for maintaining oxygen homeostasis under reduced PO2, Epo can participate in the complicated mechanisms of ventilatory response, activation of NO production by endothelial cells, and angiogenesis during the adaptation of the body to hypoxia. A study by Katunsev et al [7] found no significant changes in EPO and VEGF between trial and control groups, and were not able to report changes in RBC or plasma volumes [6]. However, improved aerobic capacity, typical of the expected haemodynamic changes seen in other studies, was noted, in the form of ~ 12 % improvement in a TTE test, as well as a 8 ± 12 % in V_{O2max} in comparison to 2 ± 8 % in the control group. Furthermore, repetitive exposure to passive heat stress (40 °C; ~40 % RH) for 40–50 min 3x/wk over 6 wks, when compared to exercise of the same duration, was observed to increase: capillarization by 21 % vs 12 %; Endothelial NO synthase 8 % vs 12 %; and VO2peak 5 % vs 7 % [8].

This body of research, therefore, highlights the observed clinical benefits of HST, most notably increased aerobic endurance.

The expression of Hypoxia inducible factor 1α (HIF-1 α) has been shown to be necessary for the process of heat adaptation [9,10]. Its expression upregulates NO synthase (NOS), Erythropoietin (EPO) and Vascular Endothelial Growth Factor (VEGF), which may explain a portion of the clinical benefits observed. An overlap in the response to heat and hypoxic stresses makes it difficult to isolate the independent contributions of the observed clinical benefits from each other.

Reduced V_{O2max} under heat stress is a well-known phenomenon. Blood flow to the skin is increased such to increase the driving force of heat lost to the environment and prevent overheating. It has been proposed that this shunting of the blood to the periphery results in reduced cardiac filling volumes, decreasing cardiac output, and subsequently resulting in a reduction in VO2max [11]. Impaired oxygen uptake (VO2max) and delivery to tissue may result in an acute hypoxia, explaining the reduced muscle power output, fatigue and occasional fainting observed during exercise under hot conditions, highlighting another mechanism of heat induced hypoxia independent of reduced oxygen content in the inhaled air. This is especially important for those studies considering heat therapy during exercise.

Additionally, the use of sauna bathing has been observed to have acute, mild endocrine effects – I.e. frequently reported elevations in norepinephrine and endorphins, as well as growth hormone and prolactin, with variable effects in response of Antidiuretic and related hormones along the same axis, and cortisol [12] – which should not be neglected when considering potential mechanisms of health benefit.

Intermittent hypoxic training (IHT) is a means of achieving adaptations similar to those that result from living at high altitude through short-term exposures to hypoxic conditions [13,14]. Various permutations of this approach exist, they may entail normobaric conditions with reduced oxygen concentration (I-NBHT), or hypobaric conditions with normal oxygen concentrations (I-HBHT) [13,15]. IHT can be carried out either during rest or during exercise [13,16].

Hypoxic training (HT) has been shown to increase erythrocyte mass and consequently improve the aerobic capacity and athletic performance of athletes [17–19].

A study involving 10 healthy male non-athletes, ages 19–31, found significant improvements in: ‘endothelial function (EF)’, blood oxygen carrying capacity and ‘respiratory endurance’, following a 3 week trial of mild short duration normobaric intermittent hypoxic trainings (IHTs) administered daily [7].

EF, defined as the capacity of the endothelium to release nitric oxide (NO) during reactive hyperaemia, saw an increase of 38.9 %, exceeding the normal range of values pre training. A 34.3 % increase in the Epo concentration was also observed, resulting in a twofold plus increase in the count of immature red blood cells (reticulocytes), a significant 6.4 % increase in the erythrocyte count and a 4.1 % increase in the hemoglobin content. Respiratory endurance, determined by maximum voluntary inhalation and exhalation times, also increased 23.3 % and 24.4 % respectively. All statistical tests indicated significance at the $p < 0.05$ level.

A study on cross acclimation between heat and hypoxic stressors found that HST can improve athletic performance under conditions of NBH. (3:16 ± 3:10 min:s; $p = 0.0006$) and (2:02 ± 1:02 min:s; $p = 0.005$) reductions in an NBH timed trial in IHT and HST trained individuals respectively as compared to control [20]. Furthermore, “HST induced a greater adaptive stimulus at lower levels of metabolic strain, and in a shorter time frame compared to hypoxic acclimation. This occurred despite the IHT group completing sessions at a higher relative intensity.”.

Thus, IHT has been shown to induce physiological adaptations that result in improved athletic performance, illustrating the value of short-duration hypoxic exposure as a training tool [19,21–23]. IHT has also been found to have cardiovascular health benefits in non-athletes, resulting in improvements in conditions such as hypertension and coronary heart disease [24]. IHT has also been found to decrease arterial stiffness and increase peak diameter of the popliteal artery [25]. IHT has also been explored as an intervention in other conditions including mountain sickness, sleep apnoea, hypertension and cardiovascular disease, spinal cord injuries, neurodegenerative disorders, Covid-19 and respiratory disease, depression, obesity and metabolic disease, diabetes and ischemia of the brain, heart and kidneys. IHT has also been been explored as an intervention in other miscellaneous diseases – Mountain sickness [26], Sleep apnoea [27], Hypertension and cardiovascular

disease [28–32], spinal cord injuries [33,34], COVID-19 and respiratory illness [35], obesity or metabolic disease [36], diabetes [37];ischemia of the: brain [38], heart [28] and kidneys [39].

This body of research indicates that IHT may have considerable value for athletes and non-athletes alike and could be a valuable tool for improving performance and general cardiovascular health, if widely available. However, effective IHT requires access to comparatively specialized equipment and facilities, which are not commonplace and not available at commercial gyms, whereas saunas are more widely available.

Hypothesis

The hypothesis is that the sauna conditions exhibit oxygen partial pressures sufficiently reduced to induce a physiological response equivalent to that of exposure to high altitude, and that this physiological response is a likely contributing mechanism in the cardiovascular benefits associated with the practice of sauna.

An under-reported aspect of sauna conditions is that the partial pressure of oxygen is significantly decreased as a result of dilution by virtue of the extremely high water carrying capacity of air at sauna temperatures. This dilution can occur because the vapour pressure of water increases exponentially with temperature, which means that at the high temperatures typical of sauna conditions, the absolute water content can reach values an order of magnitude higher than can occur at normal ambient temperatures, even at high values of relative humidity.

The plausibility of this hypothesis will hinge on determining whether the oxygen depleting characteristics are indeed comparable to those involved in high altitude or intermittent hypoxic training, and whether the reduced oxygen environment should therefore be considered as a likely contributing factor in physiological responses to sauna conditions, distinct from the purely thermoregulatory effects considered in the existing literature.

Evolution of the hypothesis

The Arden Buck equation (Equation (1) can be used to determine the vapour pressure of water at a given temperature within the range 0 °C to 100 °C.

$$P = 6.1121 * e^{\left(18.678 - \frac{T}{234.5}\right) * \left(\frac{T}{257.14+T}\right)} \quad (1)$$

where P represents the saturation vapor pressure in units of Hectopascals (hPa), and T denotes temperature in degrees Celsius.

Vapour pressure multiplied by relative humidity gives the partial pressure of water in equilibrium, by the definition of relative humidity. Hence, for a particular temperature and relative humidity, partial pressure of water can be calculated. By Raoult's Law, this can be used to determine the molar fraction of water vapour in the air. By the Ideal Gas Law, the molar fraction is equal to the volumetric fraction. Thus, assuming that dry air consists of 79 % Nitrogen and 21 % Oxygen, the volumetric fraction of Oxygen in the sauna atmosphere, assuming sea level, can be determined as follows:

$$P_{O_2} = 1atm * 0.21 * (1 - P_{water}) \quad (2)$$

At sea level, the partial pressure of Oxygen is 1 atm multiplied by the volume fraction, and so a partial pressure can be readily calculated. From this, the barometric equation, with a reference altitude of 0 m above sea level and assuming a temperature lapse rate of zero, can be used to determine the altitude that would have the same oxygen partial pressure under normal conditions, hereafter referred to as 'equivalent altitude.'

$$P = P_b * e^{(-g * M(h-h_b)/(R * T_b)} \quad (3)$$

where:

- P_b is reference pressure (1 atm)
- T_b is reference temperature (298 K)
- h is altitude (m)
- h_b is the reference altitude (m)
- R is the universal gas constant (8.314 J/mol.K)
- g is gravitational acceleration (9.807 m/s^2)
- M is the molar mass of gas

Using equations (1) to (3), with conditions of 1 atm pressure and 25 °C as the reference basis, it is possible to input any values for temperature and humidity and determine the altitude which would have the same partial pressure of oxygen at the same temperature. Of course, it is not the oxygen partial pressure outside the body that governs the oxygenation of blood, but rather the oxygen partial pressure inside the lungs. Some amount of condensation would occur during the air's cooling passage through the airways, thereby reducing the diluting effect. Conversely, however, the lower density of the air being breathed in would also reduce the mass of oxygen relative to inspired volume, which would result in less oxygen being available for the same respiratory rate, potentially resulting in greater depletion and therefore reduced concentration of oxygen in the lungs. The precise dynamics of this process would have to be determined experimentally, as a patient's respiration rate may also be altered in response to sauna conditions.

Moreover, hemoglobin's binding affinity for oxygen is strongly affected by temperature and so, the increases in core temperature that occur during sauna [40] could disrupt the normal patterns of oxygen uptake and delivery. This effect could enhance oxygen delivery to certain tissues, particularly those that experience the largest temperature increases, but impede delivery to others due, firstly, to the blood's reduced total capacity for oxygen uptake and secondly due to higher delivery in some tissues conversely resulting in less oxygen remaining for delivery elsewhere.

Hence, the partial pressure of oxygen and, with it, the equivalent altitude, will not correlate exactly with hypoxic stress, but do offer a good means of estimating the potential for induced hypoxia. Whether or not that degree of hypoxia occurs in practice would have to be determined experimentally.

Fig. 1 shows the resulting relationship between humidity and equivalent altitude for the range of typical sauna conditions.

It can be noted that, at the most extreme reported conditions from Pawlak et al (2012) [1], namely 100 °C and 45 % relative humidity, the equivalent altitude calculated through this method is 5700 m, far in excess of what is normally considered a high altitude exposure. However, those conditions are an extreme outlier and not representative of typical sauna use or of the main body of research. More standard conditions of 90 °C and 15 % humidity result in an equivalent altitude of 1000 m, an altitude adequate to result in modest adaptations in chronic exposure and therefore potentially suitable for Intermittent Hypoxic Training. Moreover, an altitude equivalent in excess of 2000 m can be achieved without resorting to extreme conditions, which suggests that slightly modified sauna conditions could offer significantly enhanced hypoxia-related adaptations. A temperature of 90 °C and humidity of 30 % would be quite achievable, and results in an equivalent altitude of 2100 m.

Fig. 2 shows the equivalent altitudes achieved in typical conditions for a steam room.

While an equivalent altitude slightly in excess of 1000 m is achieved at the high end of reported conditions, namely 45 °C and 100 % relative humidity, it is apparent that sauna conditions tend to result in a considerably larger reduction in oxygen partial pressure than steam room conditions do. This observation, coupled with the fact that cardiovascular benefits are reported for saunas and not for steam rooms, supports the hypothesis.

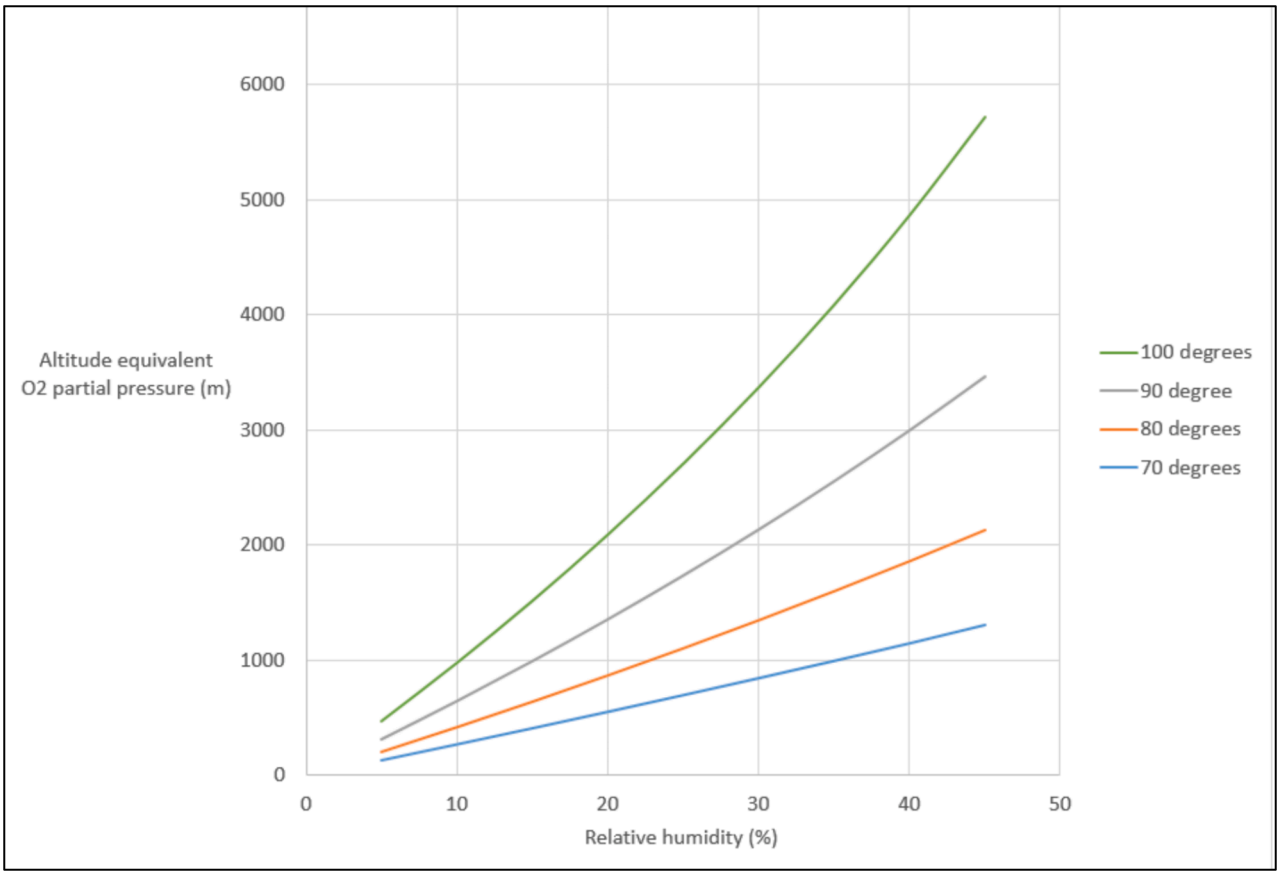


Fig. 1. relationship between relative humidity and equivalent altitude of O2 partial pressure, for different temperature isotherms in reported sauna conditions.

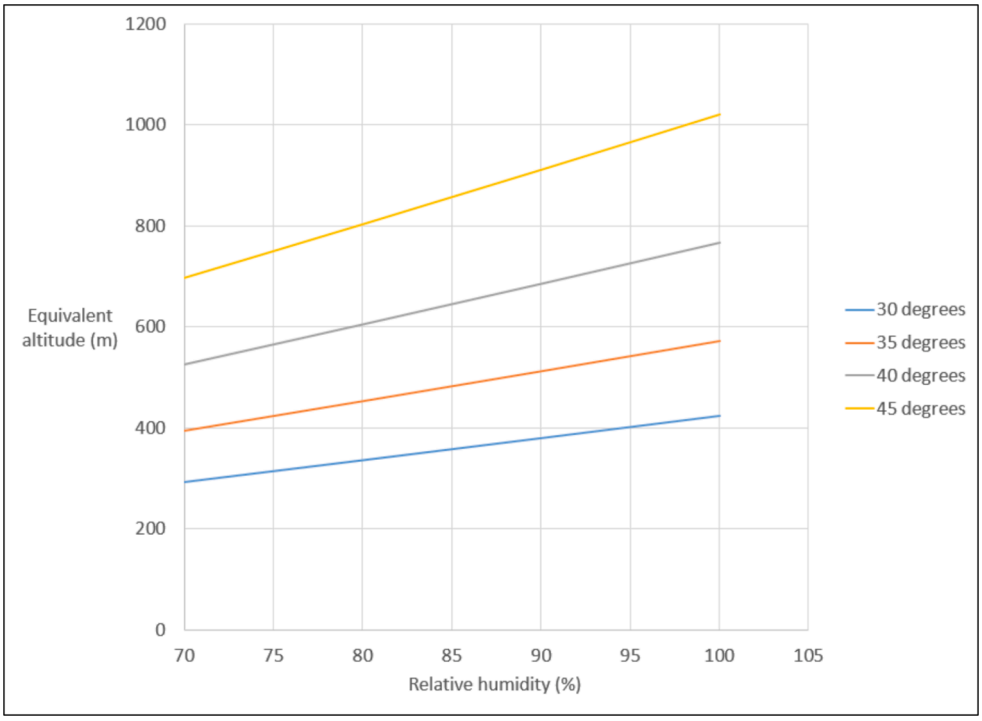


Fig. 2. relationship between relative humidity and equivalent altitude of O2 partial pressure, for different temperature isotherms in reported steam room conditions.

Hence it can be hypothesized that sauna use may induce mild hypoxia thereby triggering associated physiological adaptations resembling those of Intermittent Hypoxic Training. It can be further hypothesized that adjustments to sauna conditions, particularly increased humidity, would optimally induce these adaptations. It may also be desirable to increase sauna conditions beyond the usual ranges in order to enhance the hypoxic challenge. While this would likely make the thermoregulatory stress intolerable, this could be mitigated through simple means of cooling the body, such as a towel moistened with ice water draped over the user's shoulders. This approach could potentially offer a means of achieving conditions suitable for intense altitude adjustment for athletes and coaches who lack access to the conventional means of doing so.

Modifying sauna conditions in these ways could potentially exacerbate some forms of thermoregulatory distress and would increase the temperatures to which individual tissues are exposed and might, thereby, increase the risk of health complications. Increasing the magnitude of the reduction in oxygen partial pressure could, in and of itself, pose safety risks and hence, without prior safety testing it cannot be assumed that such modifications are without risk even if the overall thermoregulatory distress is managed.

Testing of hypothesis

The inherent plausibility of the hypothesis has been demonstrated by the fact that sauna conditions do exhibit oxygen partial pressures comparable to altitudes known to result in physiological adaptations, and comparable to the oxygen partial pressures used in intermittent hypoxic training. However, as discussed previously, other facets of the dynamics of oxygen delivery may modify this effect and so, the altitude-equivalent effects of sauna exposure would need to be verified experimentally to confirm or deny this altitude equivalence.

The most straightforward means of testing whether hypoxia is present are blood-gas analysis or pulse oximetry. However, these are both highly problematic in extreme temperatures. Hence, conducting blood-gas analysis or pulse oximetry on sauna users would require that a hatch be constructed with an insulated silicon seal through which a user's arm can be extended while their main body mass remains inside the sauna, allowing oximetry to be conducted, or blood samples to be extracted. Identifying short-term reductions in SpO_2 and comparing them to those observed during IHT would be just one facet involved in evaluating the hypothesis.

A more comprehensive approach would be to clinically test the adaptive responses, over an appropriate time period, in an experimental group exposed to saunas, as compared to those in a control group exposed to other thermoregulatory stresses but without the commensurate oxygen partial pressure reduction. A readily-available control would be steam rooms, which were shown by calculation in the evaluation of hypothesis section to not achieve equivalent oxygen reduction. A promising alternative control would be infrared saunas, which heat tissues directly without greatly elevating ambient temperature or humidity.

If typical sauna or modified sauna conditions do induce hypoxic effects, this would be detectable through measurements of blood markers that are altered by intermittent hypoxic training. These include acute and long-term increases in circulating erythropoietin, which in turn induces an increase in circulating red blood cell mass in the long term. The effects could also be tested by measuring changes in running performance, as an improvement would be expected when compared to a control group. Because steam rooms exhibit much smaller reductions in oxygen partial pressure, steam room usage would offer a useful placebo intervention with similar thermoregulatory stresses for control groups in experimentation. Any observed differences in physiological adaptations or performance improvements between an experimental sauna group and a control steam room group would corroborate the hypothesis that the reduced oxygen partial pressure is a causative mechanism for sauna adaptations rather than simple thermoregulatory distress. In particular, if it is observed that adaptations that typically occur in response to IHT are present in the sauna group but not in the control, that would strongly indicate a role played by reduced oxygen partial pressure.

Implications of hypothesis

If the reduced oxygen partial pressure and density in sauna conditions does generate hypoxic stimulus comparable to that of conventional Intermittent Hypoxic Training, then saunas would offer a more affordable means of accessing altitude training effects to many who would otherwise not have access to suitable facilities.

If the hypothesis is correct then the health benefits of saunas could be enhanced by increasing the temperature and/or humidity of saunas beyond typical conditions while mitigating the increased thermoregulatory stress through cooling of the body to reduce thermoregulatory stress or delay its onset. Modified sauna conditions such as these do not have a known safety profile, and could therefore pose safety risks not currently considered in the practice of sauna. Hence, it is inadvisable to attempt these modifications except in controlled conditions intended to establish their safety. In light of the potential clinical benefits and athletic performance improvements that have been hypothesized may arise from such modifications, such safety profile testing would be of value, for two reasons. Firstly, to determine whether commercial sauna protocols could be modified to optimize health benefits and, secondly, to determine whether sauna users attempting to enhance benefits might place themselves at risk.

If confirmed to be correct, the hypothesis would offer an accessible means of performing intermittent hypoxic training as well as offering a means of enhancing the health benefits of saunas, and would advance the understanding of the mechanisms by which saunas improve cardiovascular health. The range of clinical and athletic benefits established for IHT is far larger than for sauna practice and so, if it can be confirmed that saunas induce the same effects as IHT, the range of applications for saunas would be immediately expanded in its scope to include those known for IHT in addition to those specific to the practice of sauna.

Conclusions

In this paper, it has been shown that the diluting effect of water vapour in sauna conditions represents a reduction in oxygen partial pressure that is comparable to those that occur at high altitude. This suggests that the practice of sauna may have previously unexplored utility as a more commonly-available means of supplying the health and performance benefits associated with exposure to high altitude or the simulated high altitude conditions present in IHT.

This hypothesis is not yet adequately supported by experimental evidence, and merits further study to explore whether the expected reduction in SpO_2 does occur during sauna, and subsequently whether the long-term adaptations match those associated with high altitude exposure.

If confirmed, this hypothesis would suggest that sauna could be a viable alternative in instances where IHT is indicated but not available, and the other adaptations specific to saunas may offer synergistic effects and enhance those benefits.

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CRediT authorship contribution statement

Neil Stacey: Methodology, Investigation, Formal analysis, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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