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Review Article

Significance of thermoregulatory homeostasis in sustaining human physiological equilibrium

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ABSTRACT

Physiological control is based on homeostasis, which is the outcome of intricate interactions and competition between several negative and positive feedback systems. The dynamic process of thermoregulatory homeostasis may modify internal circumstances to withstand shocks from the outside world. Homeostatic management of the internal environment is responsible for the organism's health and vitality; knowledge of this idea is essential to a comprehension of normal physiology. In contrast, sickness is caused by disruption of homeostatic mechanisms, and effective therapy must try to re-establish these homeostatic conditions, working with rather than against nature.

1. Introduction

Thermoregulatory homeostasis is a key concept in physiology that describes how organisms maintain internal stability while reacting to external stimuli. This dynamic mechanism is the consequence of various feedback systems, which allows for more precise control and adaptability. Homeostatic regulation is critical to an organism's health and vitality, and comprehending normal physiology necessitates respect for this concept. Disruption of homeostatic processes produces disease, and successful treatment must strive to reverse these abnormalities.

2. How Human Body Generates Heat?

Being endotherms, humans can control their body temperature through the production and dissipation of heat. Cellular reactions that sustain life depend on the ability to generate heat from food. Food molecules store energy in chemical bonds, which can be extracted and used to fuel metabolic operations. Energy metabolism is strictly regulated to meet the body's energy requirements in a range of situations. Through the electron transport chain and the tricarboxylic acid cycle (TCA cycle), the mitochondria generate the majority of the body's heat and regulate cellular metabolism. The TCA cycle begins with pyruvate-derived substrates such acetyl-CoA and oxaloacetate, followed by fat beta-oxidation.

These substrates undergo redox reactions, resulting in the formation of high-energy bonds like NADH and FADH2. These intermediates are oxidatively phosphorylated to form ATP molecules, which are subsequently used to power chemical reactions throughout the body, generating heat. Thermogenin, another name for the unique uncoupling protein (UCP-1) found in the inner mitochondrial membrane, controls heat production and membrane permeability. [1,2]

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3. Mechanisms of Heat Exchange between Human Body and Environment

The body maintains thermoregulatory homeostasis in non-thermo-neutral settings by convection, conduction, evaporation, and radiation. The air surrounding the skin is heated and cooled via convection. This causes hypothermia, with around 15% of the body's heat lost by convection. Conduction is the transfer of heat between two things in direct touch, such as holding an iced glass of water or wrapping your hands around a hot mug of coffee. Evaporation transmits heat by evaporating water, which extracts energy from the skin. Sweat is the major mechanism of cooling during exercise, and the evaporation rate is proportional to relative humidity. Radiation, which uses infrared waves to transfer heat between objects, accounts for around 60% of the body's heat loss. The rate at which these systems operate varies with temperature and environmental factors.

4. Discussion

A self-regulating mechanism called homeostasis aids biological systems in remaining stable and adjusting to shifting environmental factors. It is a central organizing tenet of physiology and is essential for understanding the body's function in health and disease. Disrupting homeostatic mechanisms leads to disease, and effective therapy should focus on re-establishing these conditions, working with nature rather than against it. Homeostasis is a crucial aspect of organisms, where their internal environment is maintained within a narrow range through self-adjusting mechanisms. Feedback and feedforward mechanisms are used to achieve thermoregulatory homeostasis, with feedback systems being closed-loop structures that control future actions based on past actions. There are two types of feedback systems: negative feedback, which seeks goals and responds to failure, and positive feedback, which generates growth processes. Higher levels of control over these systems enable modifications to support behavioural reactions to external inputs. Multiple feedback loops produce homeostatic regulation, which offers more control and flexibility.

The tightly regulated process of thermoregulation, which maintains body temperature, is driven by the hypothalamus, a master regulator that controls the body's heat intake or loss. Up to 60% of the heat generated during metabolic processes is used to regulate body temperature. Hypothermia or hyperthermia can thus be caused by dysregulation of thermoregulatory mechanisms.[3] When the body's core temperature drops below 35 degrees Celsius, or 95 degrees Fahrenheit, it is referred to as hypothermia. Shivering and behavioural abnormalities are signs of mild hypothermia, which can be mild, moderate, or severe. Dilation of the pupils, cardiac arrhythmias, disorientation, and unconsciousness are all symptoms of moderate hypothermia. Ventricular fibrillation and severe bradycardia can result from severe hypothermia, which is defined as below 28 degrees Celsius. The hypothalamus can raise the body's metabolic rate to combat hypothermia.

Shivering, an involuntary response to cold temperatures, can increase basal metabolic rate by 5 to 6 times. Studies show decreased shivering is linked to increased body fat. Peripheral vessels can undergo vasoconstriction, preventing heat loss to the environment and acting as a barrier. Treatment for mild to moderate hypothermia involves removing cold clothing and rapid rewarming with a hot bath. Severe hypothermia may require active internal rewarming. On the contrary, hyperthermia is a condition where the body's core temperature rises beyond 37.5-38.3°C, resulting from the body's inability to dissipate heat. Drugs, poisons, immunological responses, and malignant hyperthermia are among the causes. The hypothalamus controls thermoregulatory mechanisms like sweating, shivering, and vasoconstriction. Breakdowns can cause temperature elevations ranging from mild to dangerously high, potentially life-threatening.

5. Physiological Responses to Cold

People respond to cold in two primary ways. While metabolic responses aim to replenish heat lost to the environment, vasomotor reactions minimize dry heat loss.

5.1. Vasomotor reactions

One important physiological reaction in people exposed to cold is peripheral vasoconstriction, which lowers blood flow when water temperature rises. This enhances insulation and reduces convective heat transfer between the skin, skeletal muscle, and subcutaneous fat that make up the body's shell and core. Insulation increases as the skin temperature drops below 35 degrees Celsius, peaking at 31 degrees Celsius or lower. Whole-body cold exposure lowers skin temperature.[4] Cold-induced vasoconstriction[5] makes the hands and fingers particularly susceptible to cold damage and loss of manual dexterity.[6] This decrease in blood flow makes cold injuries worse.[7] Cold-induced vasodilation, which causes cyclical changes in skin temperature, modifies the effects of vasoconstriction. According to research on the forearm,[8,9] this reaction might be centrally mediated. It seems to be caused by vasodilation in the muscle vasculature rather than the epidermis.

5.2. Metabolic responses

Increasing metabolic heat production to replenish lost heat during cold exposure is one of the many ways humans safeguard their body temperature. The main source of the increased metabolic heat generation, which releases about 70% of the total energy used, is the muscles.

Exercise or work-related voluntary physical activity raises the amount of heat produced by metabolism. Shivering starts when there is no increase in voluntary muscular activity. Some animals respond to cold exposure with non-shivering thermogenesis, but there is no clear evidence that humans share this mechanism.[10] Shivering is an involuntary pattern of repetitive muscle contractions, often referred to as a "quasi-exercising" state.[11] Shivering thermogenesis is quantified by measuring the increase in whole-body oxygen uptake, which is associated with the onset of shivering in cold.[12] Moreover, exposure to cold raises cardiac output, mostly because of an increase in stroke volume.[13]

6. Effects of Exercise on Thermoregulation in the Cold

Whole-body oxygen uptake can exceed two litres per minute when engaging in voluntary physical activity, which can raise metabolic heat generation more than shivering. This effect depends on factors like exercise intensity, environmental conditions, and mode of activity. Exercise also facilitates heat loss from the body by increasing blood flow to the skin and active muscles, enhancing convective heat transfer from the central core to the peripheral shell. Because the arms have a greater surface area to mass ratio and a thinner subcutaneous fat layer than the legs, convective heat transmission is higher in the arms. Although metabolic heat generation can counteract increased heat loss in cold air, it might not be sufficient to maintain core temperature in cold water.[10]

Depending on the intensity of the exercise, whole-body oxygen consumption during submaximal exercise in cold weather may be more than or equal to that during temperate conditions.[14] At low intensities, whole-body oxygen uptake is higher in cold than in temperate conditions, as metabolic heat production is insufficient to maintain core and skin temperatures high enough to prevent shivering. As metabolic heat production rises with increasing exercise intensity, the afferent stimulus for shivering declines, and exercise metabolism is high enough to prevent shivering completely.

7. Use of Glycogen for Energy Production as Heat

Shivering, a muscular activity, relies on an adequate supply of substrate for metabolic processes producing energy for contractions. Metabolic rate can increase two- to fivefold depending on the intensity of shivering, which has nutritional implications for persons living and working in cold conditions. Accommodating clothing and shelter from the environment does not significantly affect nutritional requirements, while those who are not adequately protected from the cold will shiver and have greater nutritional energy requirements than in warmer climates. In 1989, Vallerand and Jacobs[15] employed indirect calorimetry to measure the proportional roles of fat and carbohydrate metabolism in the overall energy needs of sedentary men who shivered for two hours in cold air. They discovered that whereas shivering is maintained by both fat and carbohydrate metabolism, glucose is the primary energy source. Carbohydrate for shivering thermogenesis may come from muscle glycogen stores, blood glucose, or both.

Young et al.[14] tried to find out if shivering causes muscle glycogen stores to be depleted and if this restricts shivering or affects thermoregulation in cold weather. They discovered that although rest and a high-carb meal produced very high levels of muscle glycogen before immersion, exercise and a low-carb diet produced very low levels. However, there were no significant differences in metabolic rate or fall in core temperature during immersion. In a high-glycogen immersion study, muscle glycogen levels decreased, but not in a low-glycogen trial.[16] It is hard to explain why the two studies' findings were different. In contrast to Young et al.,[14] the subjects in Martineau and Jacobs's[16] study were incredibly thin; yet, they did not shiver more violently than the subjects who were bulkier. Similar metabolic rates, or roughly 25 to 30 percent of whole-body oxygen uptake maximum, were found in both experiments. Such a minimal level of steady-state exercise would not cause muscle glycogen to be depleted.

Muscle glycogen might be a crucial substrate for maintaining shivering in the cold at high elevations. Ascent to high altitude decreases whole-body oxygen uptake max, and muscle glycogenolysis during exercise is faster at high altitude than at sea level. Individual characteristics that modify human heat balance in the cold. Body size, configuration, and composition significantly influence an individual's ability to defend body temperature during cold exposure.[10] These body characteristics modify the stress of a given environmental condition, resulting in different responses to the same environment. The thermoregulatory reaction to cold is influenced by a number of factors, including acclimatization, gender, fitness, and age. Body shape and mass significantly influence an individual's tendency to lose heat in cold environments. A large surface area favours greater heat loss than a smaller surface area, while a large body mass maintains a constant temperature due to a greater heat content. Heat loss is influenced by the surface area to body mass ratio; those with a high surface area to mass ratio see larger drops in body temperature when exposed to cold.

Another significant factor that modifies the stress of cold exposure is body fat. Although fat has a higher thermal resistivity than skin or muscle, all bodily tissues offer thermal resistance to heat transmission from within the body.[10] Compared to lean people, overweight people shiver less and have lower drops in body temperature when exposed to cold temperatures because subcutaneous fat offers substantial insulation against heat loss in the cold. Preventing soldiers from losing body fat during cold

weather, particularly during extended operations, should be the goal of nutritional tactics.

8. Thermoregulatory Responses for Cold

8.1. Physical fitness

Physical fitness, particularly aerobic ability, has no significant effect on the thermoregulatory response to cold. Some cross-sectional investigations have discovered that fit people have warmer skin temperatures at rest in cold air, although this is related to decreased subcutaneous fat thickness and increased metabolic heat production. In cold air, Budd et al.[17] found no correlation between skin temperature at repose and whole-body oxygen uptake maximum. Longitudinal studies indicate that endurance training enhances the cutaneous vasoconstrictor response to cold, resulting in a faster skin temperature drop during exercise in cold water and resting in cold air. As a result, persons exposed to cold benefit from endurance exercise in terms of thermoregulation.

8.2. Age and gender

Aging is commonly thought to impair body temperature defense during cold exposure. The incidence of hypothermia on hospital admission is higher in elderly people (60 years or older) than in younger people,[18] although the overall incidence is low when compared to other illnesses resulting in hospital admission.[19] Epidemiological assessments of body temperature in older people conducted in their own homes do not show a high incidence of hypothermia.[20]

Controlled laboratory studies indicate that older men may be less capable of maintaining core temperature during cold exposures. Older males may have a slower cutaneous vasoconstrictor response to cold, and cold-induced vasodilation may be reduced.[21] Shivering thermogenesis may also be lower in older men than in younger men, probably due to muscle mass loss rather than an age-related influence on thermoregulation. These aging effects become obvious in men at roughly 45 years of age.[14]

Gender-related changes in body size, shape, and composition, as well as hormonal impacts linked with the menstrual cycle, all influence heat balance and thermoregulatory response to cold.[22] Most women have more fat content and subcutaneous fat thickness than men of similar age, resulting in better maximal tissue insulation and a lower critical water temperature. However, increased fat content may not give women a thermoregulatory advantage over men.

8.3. Acclimatization

Chronic cold exposure causes thermoregulation modifications, with habituation being the most typical one.[14] This entails less shivering and cold-induced vasoconstriction, which keeps the skin warm but results in heat loss and a considerable drop in core temperature. Cold acclimatization and acclimation can boost reactions to cold, with metabolic acclimatization-acclimation characterized by a more prominent thermogenic response, and insulative acclimatization-acclimation involves improved heat conservation mechanisms. Some people who are consistently exposed to cold may experience faster cutaneous vasoconstriction, which could be due to an increased sympathetic nerve reaction. Although physiological adaptations to protracted cold exposure are less practical, shivering response modifications may have nutritional consequences.

8.4. Cognizant reflexes

While it's generally accepted that being exposed to local cold is uncomfortable, the level of discomfort varies. Immersion of the hands in water at 5°C was more uncomfortable than that at 8°C, 10°C, or 15°C.[23] Thermal feeling was identical at all four water temperatures, possibly due to nociceptive rather than thermal stimulus.

Because of the increased surface area exposed and the higher survival risk from submersion and drowning, sudden whole-body cold-water immersion may be viewed as a more severe analogy to local cold exposure of the peripheries. Cold (2°C) water immersion or dousing is the safest first aid approach for recovery from exertional heat sickness.[24] However, a number of experiments on cold water immersion have clearly shown the significant impacts of anxiety and psychological interventions.[25] Greater acute anxiety, induced by tricking participants about approaching immersion into colder water than anticipated, dramatically exacerbated cold shock reactions of tachycardia and hyperventilation as compared to correct immersion temperature information.[26]

Reduced pain or subjective thermal discomfort is often the outcome of long-term adaptation to local cooling.[27] Subjective acclimatization usually takes precedence over adaptation reactions when physiologic adaptation is limited.[28] Cold-induced vasodilation adaptability may be influenced by changes in sympathetic outflow caused by a gradual reduction in psychological stress or worry, as repeated immersions should eventually result in a decrease in sympathetic outflow.

8.5. Hypoxia

Regulation of body temperature and Cold-Induced Altitude exposure and hypoxia were used to study visceral injury during climbing excursions. The high mean incidence rate of cold injuries indicates that altitude and cold are common stresses at moderate elevations. Exertional exhaustion and fuel depletion have been proposed and rejected

as variables in changing vaso-constrictory and shivering threshold temperatures or developing hypothermia.[29,30] Understanding the influence of hypoxia on thermal sensitivity and comfort under cold stress is vital because perceptual cues are required for optimal behavioural thermoregulatory responses to prevent cold damage.

Acute altitude exposure decreases cold-induced vasodilation response in lowland natives, whereas prolonged altitude exposure gradually improves extremities blood flow.[31] The impact of extended altitude living or experience on cold-induced vasodilation is unknown, as whole-body thermal status is an essential predictor of cold-induced vasodilation prevalence and intensity. Overall, the divergent data indicate that the effects of acute and short-term hypoxic exposure, as well as the mechanisms driving enhanced extremities blood flow in seasoned mountaineers or altitude natives, are unclear. Because of its propensity to change peripheral blood flow and presumably thermal perception, hypoxia has great promise for furthering cold-induced vasodilation research, with or without whole-body cold stress. Autonomic nervous system activity changes are most likely additional responses to cold stress and hypoxia. Hypoxia can be used alone or in combination with cold to investigate particular pathways of cold-induced vasodilation because it can result in particular changes in sympathetic neuronal drive that are simpler to regulate than whole-body cold exposure or pharmaceutical medications.

8.6. Muscle function

Survival in the cold requires important emergency actions such as controlling escape mechanisms and closing zippers. Even brief cold exposure immediately impairs the performance of these tasks,[32] and CIVD research is centered on its potential benefits for maintaining or increasing hand dexterity and muscle function during cold exposure.[33] However, findings from the last decade indicate that muscle function is only little impacted during the CIVD cycle. For CIVD to be advantageous for manual function, Chen[34] stated that there should be greater heat and blood flow outside of the immediate region of the fingers being assessed.

Additional information regarding the connection between CIVD and manual function can be obtained by examining the neuromuscular function of the entire hand with moderate to prolonged local cold exposure, with a focus on the first dorsal interosseous muscle. According to these findings, contractile parameters during induced twitches, such as delayed time to peak tension, delayed half-relaxation time, and lowered peak twitch force, were adversely affected when the first dorsal interosseous muscle was cooled with 30 minutes of local hand immersion in 8°C. Minimal improvements and even a decreased CIVD response resulted during two to three weeks of hand immersion acclimation at 8°C, which neither improved nor reversed these deficiencies.[35] Another key difficulty with manual performance in the cold is alterations in motor control caused by diminished or changed tactile sensitivity when digits cool.[36] In both males and females, cooling the hands and forearms led to a systematic underestimation of handgrip power,[37] possibly as a result of decreased proprioceptive activation. Although the anticipatory and temporal coupling pattern of grip and load forces remained in place despite the increased force requirement, temporary finger cooling with the topical anesthetic ethyl chloride led to noticeably larger grip forces.[38]

8.7. Neural mechanisms and mediators in cold responsiveness

Both neuronal and non-neural regulation play a complex role in the interaction between whole-body and local control over skin blood flow during cold and heat exposure.[39] Initial vasoconstriction seems to be encouraged by systemic sympathetic activity.[40] According to Janský,[41] frequent local acclimation can increase sympathetic activity and vasoconstrictor tone. Flouris and Cheung[42] suggested that CIVD originated from sympathetic withdrawal and may serve primarily as a whole-body heat loss mechanism. Brandstrom et al.[43] found decreased thermal responses in both the hand used for repeated cold immersion acclimation and the contralateral hand. These ind[44] irect investigations suggest that local cold exposure regulates peripheral blood flow and vasodilation. Direct examinations are not possible in humans, save for post-injury examinations. Rats with peripheral sympathectomy had mechanical hypersensitivity and cold intolerance, but their hind paw CIVD response was unaffected, suggesting that the sympathetic nervous system is not involved in the initiation or control of CIVD. Basal blood flow was elevated by neural blockage at the finger tip and significantly more by local skin anaesthesia.

Furthermore, nerve blocking at the base of the finger maintained vasoconstriction during local cooling, while local anaesthetic reduced it. Vasomotor tone can then be influenced by a range of non-adrenergic mediators that are generated either locally or systemically. Under cold stress, nitric oxide, a potent vasodilator, has been studied as a local regulator of epidermal blood flow.[45] To ascertain whether local NO activity in the extremities and the fast vasodilation linked to CIVD are directly related, more investigation is necessary. The vasoconstrictor peptide endothelin-1 is another hormone that responds to cold exposure. Endothelin-1 levels and the expression of receptors in the kidneys, heart, and mesenteric arteries were increased in a rat model after being exposed to cold for up to five weeks. Variations in neurotransmitter sensitivity or activity affect the extremities' cold reaction.

9. Conclusion

Humans have a temperature-regulating feedback system that promotes either heat loss or uptake. A group of brain cells called the "heat-loss centre" is activated when the brain's temperature control centre receives information from sensors that show the body's temperature has increased above the typical range. Although this stimulation causes the skin's blood vessels to expand, increasing the amount of blood that flows from the body's centre to the skin's surface and releasing heat into the surrounding environment, it has few significant benefits. Sweat glands are activated as the skin's blood supply increases, leading to an increase in output. As perspiration evaporates from the skin's surface into the surrounding air, it transports heat with it. Furthermore, the depth of respiration rises, and a person may breathe through the open mouth rather than the nasal airways. This promotes heat escape from the lungs. In contrast, exposure to cold triggers the brain's heat-gain centre, which restricts blood flow to the skin and redirects blood from the limbs into a system of deep veins. This design keeps heat closer to the body's core, reducing heat dissipation and increasing blood pressure. When there is a large loss of heat, the skeletal muscles tense and shiver due to increased random signals from the brain. Shivering muscular contractions use ATP to release heat.

The thyroid gland in the endocrine system is also stimulated by the brain to create thyroid hormone, which increases heat generation and metabolic activity in all the body's cells. Acute exposure to cold environments activates the sympathetic nervous system, resulting in system-wide catecholamine output (nor-epinephrine). It induces systemic arteriolar constriction, increased heart rate, and cardiac contractility. The heart works harder to get blood through the restricted blood arteries. Additionally, limited blood vessels in the limbs divert superficial blood flow to the centre of the body, reducing the amount of heat that is released into the surroundings. Vasoconstriction that results raises the blood flow barrier and raises blood pressure. A reduced pulse in the arteries of the hand, fingers, and skin is the result of vasoconstriction.

A person reacts physiologically to cold in two ways: shivering and peripheral vasoconstriction. The degree of environmental stress, the effectiveness of vasoconstriction, and the level of exercise all influence heat balance and the need for shivering. Increased calorie intake is required because the thermogenic response increases metabolic heat generation. Carbohydrate metabolism makes up a larger share of total energy metabolism in colder climates. Age, acclimatization, and gender all affect thermoregulatory responses, although they have little bearing on diet. When it comes to thermoregulatory tolerance, body composition matters.

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None.

11. Conflict of Interest

None.

References

1. Korla K, Mitra CK. Modelling the Krebs cycle and oxidative phosphorylation. *J Biomol Struct Dyn*. 2014;32(2):242–56.
2. Chouchani ET, Kazak L, Spiegelman BM. New Advances in Adaptive Thermogenesis: UCP1 and Beyond. *Cell Metab*. 2019;29(1):27–37.
3. Contreras C, Nogueiras R, Diéguez C, Medina-Gómez G, López M. Hypothalamus and thermogenesis: Heating the BAT, browning the WAT. *Mol Cell Endocrinol*. 2016;438:107–15.
4. Veicsteinas A, Ferretti G, Rennie DW. Superficial shell insulation in resting and exercising men in cold water. *J Appl Physiol Respir Environ Exerc Physiol*. 1982;52(6):1557–64.
5. Gaydos HF. Effect on complex manual performance of cooling the body while maintaining the hands at normal temperatures. *J Appl Physiol*. 1958;12(3):373–6.
6. Boswick JA, Thompson JD, Jonas RA. The epidemiology of cold injuries. *Surg Gynecol Obstet*. 1979;149(3):326–32.
7. Purdue GF, Hunt JL. Cold injury: a collective review. *J Burn Care Rehabil*. 1986;7(4):331–42.
8. Ducharme MB, Vanhelder WP, Radomski MW. Cyclic intramuscular temperature fluctuations in the human forearm during cold-water immersion. *Eur J Appl Physiol Occup Physiol*. 1991;63(3-4):188–93.
9. Lindblad LE, Ekenvall L, Klingstedt C. Neural regulation of vascular tone and cold induced vasoconstriction in human finger skin. *J Auton Nerv Syst*. 1990;30(2):169–73.
10. Toner MM, McArdle WD. Physiological adjustments of man to the cold. In: Pandolf KB, Sawka MN, Gonzalez RR, editors. Human Performance Physiology and Environmental Medicine at Terrestrial Extremes. Indianapolis, Ind: Benchmark Press; 1988.
11. Horvath SM. Exercise in a cold environment. *Exerc Sport Sci Rev*. 1981;9:221–63.
12. Mcardle WD, Katch FI, Katch VL. Measurement of human energy expenditure. In: Exercise Physiology. Philadelphia, Pa: Lea & Febiger; 1991. p. 145–57.
13. Muza SR, Young AJ, Sawka MN, Pandolf KB, Bogart JE. Power spectral analysis of the surface electromyogram during shivering. *Aviat Space Environ Med*. 1986;57(12 Pt 1):1150–3.
14. Young AJ, Sawka MN, Neufer PD, Muza SR, Askew EW, Pandolf KB. Thermoregulation during cold water immersion is unimpaired by low muscle glycogen levels. *J Appl Physiol (1985)*. 1989;66(4):1809–16.
15. Vallerand AL, Jacobs I. Rates of energy substrates utilization during human cold exposure. *Eur J Appl Physiol Occup Physiol*. 1989;58(8):873–8.
16. Martineau L, Jacobs I. Muscle glycogen availability and temperature regulation in humans. *J Appl Physiol*. 1989;66(1):72–8.
17. Budd GM, Brotherhood JR, Hendrie AL, Jeffery SE. Effects of fitness, fatness, and age on men's responses to whole body cooling in air. *J Appl Physiol (1985)*. 1991;71(6):2387–93.
18. Leblanc J, Cote J, Dulac S, Dulong-Turcot F. Effects of age, sex, and physical fitness on responses to local cooling. *J Appl Physiol Respir Environ Exerc Physiol*. 1978;44(5):813–7.
19. Coleshaw SRK, Easton JC, Keatinge WR, Floyer MA, Garrard J. Hypothermia in Emergency Admissions in Cold Weather. *Clin Sci*. 1986;70:93. doi:10.1042/cs070093pb.
20. Collins KJ, Dore C, Exton-Smith AN, Fox RH, Macdonald IC, Woodward PM. Accidental hypothermia and impaired temperature homeostasis in the elderly. *Br Med J*. 1977;1(6057):353–6.
21. Mathew L, Purkayastha SS, Singh R, Gupta JS. Influence of aging in the thermoregulatory efficiency of man. *Int J Biometeorol*. 1986;30:137–45.
22. Stephenson LA, Kolka MA. Thermoregulation in women. *Exerc Sport Sci Rev*. 1993;21:231–62.

23. Birnie KA, Parker JA, Chambers CT. Relevance of water temperature, apparatus, and age to children's pain during the cold pressor task. *Pain Pract*. 2014;16(1):46–56.
24. Proulx CI, Ducharme MB, Kenny GP. Effect of water temperature on cooling efficiency during hyperthermia in humans. *J Appl Physiol (1985)*. 2003;94(4):1317–23.
25. Barwood MJ, Dalzell J, Datta AK, Thelwell RC, Tipton MJ. Breath-hold performance during cold water immersion: effects of psychological skills training. *Aviat Space Environ Med*. 2006;77(11):1136–42.
26. Barwood MJ, Corbett J, Green R, Smith T, Tomlin P, Weir-Blankenstein L, et al. Acute anxiety increases the magnitude of the cold shock response before and after habituation. *Eur J Appl Physiol*. 2013;113:681–9.
27. Cheung SS, Daanen HAM. Dynamic adaptation of the peripheral circulation to cold exposure. *Microcirculation*. 2012;19(1):65–77.
28. Geurts CL, Sleivert GG, Cheung SS. Central and peripheral factors in thermal, neuromuscular, and perceptual adaptation of the hand to repeated cold exposures. *Appl Physiol Nutr Metab*. 2006;31(2):110–7.
29. Passias TC, Meneilly GS, Mekjavic IB. Effect of hypoglycemia on thermoregulatory responses. *J Appl Physiol (1985)*. 1996;80(3):1021–32.
30. Tikuisis P, Ducharme MB, Moroz D, Jacobs I. Physiological responses of exercised-fatigued individuals exposed to wet-cold conditions. *J Appl Physiol (1985)*. 1999;86(4):1319–28.
31. Daanen HA, Ruiten HJV. Cold-induced peripheral vasodilation at high altitudes–a field study. *High Alt Med Biol*. 2000;1(4):323–9.
32. Cheung SS, Montie DL, White MD, Behm D. Changes in manual dexterity following short-term hand and forearm immersion in 10 degrees C water. *Aviat Space Environ Med*. 2003;74(9):990–3.
33. Daanen HA. Manual performance deterioration in the cold estimated using the wind chill equivalent temperature. *Ind Health*. 2009;47(3):262–70.
34. Chen F, Liu ZY, Holmer I. Hand and finger skin temperatures in convective and contact cold exposure. *Eur J Appl Physiol Occup Physiol*. 1996;72:372–9.
35. Geurts CLM, Sleivert GG, Cheung SS. Local cold acclimation of the hand impairs thermal responses of the finger without improving hand neuromuscular function. *Acta Physiol Scand*. 2005;183(1):117–24.
36. Provins KA, Morton R. Tactile discrimination and skin temperature. *J Appl Physiol*. 1960;15:155–60.
37. Cheng CC, Shih YC, Tsai YJ, Chi CF. The influence of cooling forearm/hand and gender on estimation of handgrip strength. *Ergonomics*. 2014;57(10):1499–511.
38. Nowak DA, Hermsdorfer J. Digit cooling influences grasp efficiency during manipulative tasks. *Eur J Appl Physiol*. 2003;89(2):127–33.
39. Johnson JM, Minson CT, Kellogg DL. Cutaneous vasodilator and vasoconstrictor mechanisms in temperature regulation. *Compr Physiol*. 2014;4(1):33–89.
40. Johnson JM, Yen TC, Zhao K, Kosiba WA. Sympathetic, sensory, and nonneuronal contributions to the cutaneous vasoconstrictor response to local cooling. *Am J Physiol Heart Circ Physiol*. 2005;288(4):1573–9.
41. Janský L, Matousková E, Vávra V, Vybíral S, Janský P, Jandová D, et al. Thermal, cardiac and adrenergic responses to repeated local cooling. *Physiol Res*. 2006;55(5):543–9.
42. Flouris AD, Cheung SS. Influence of thermal balance on cold-induced vasodilation. *J Appl Physiol (1985)*. 2009;106(4):1264–71.
43. Brändström H, Wiklund U, Karlsson M, Ängquist KA, Grip H, Haney M. Autonomic nerve system responses for normal and slow rewarmers after hand cold provocation: effects of long-term cold climate training. *Int Arch Occup Environ Health*. 2013;86(3):357–65.
44. Smits ES, Duraku LS, Niehof SP, Daanen HAM, Hovius SER, Selles RW, et al. Cold-induced vasodilatation in cold-intolerant rats after nerve injury. *J Plast Reconstr Aesthet Surg*. 2013;66(9):1279–86.
45. Johnson JM, Kellogg DLJ. Local thermal control of the human cutaneous circulation. *J Appl Physiol (1985)*. 2010;109(4):1229–38.

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