QUALITY IN SPORT
eISSN 2450-3118 · Open Access · Peer-reviewed
apcz.umk.pl/QS Nicolaus Copernicus University in Toruń

Cite as: NAZIRI, Amir, CHYL, Julianna Rozalia, ZIELNIK, Martyna, STRÓŻNA, Katarzyna, BŁAŻOWSKA, Olga, NOWAK, Weronika, GUTOWSKA, Oliwia Barbara, KOSTUCH, Wiktoria Magdalena, JUREWICZ, Alicja and ZIMON, Agata. Effect of Finnish Sauna and other heat therapies on type 2 diabetes management and complications. Quality in Sport. 2026;60:72761. https://doi.org/10.12775/QS.2026.60.72761

ARTICLE TIMELINE
Received: 28.05.2026. Revised: 23.06.2026. Accepted: 23.06.2026. Published: 27.06.2026.

The journal has been awarded 20 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32553). Unique Journal Identifier: 201398. Scientific disciplines: Medical Sciences; Health Sciences.
Punkty Ministerialne z 2019 – aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Nauki medyczne; Nauki o zdrowiu.

Effect of Finnish Sauna and other heat therapies on type 2 diabetes management and complications

Authors:

1.Amir Naziri, ORCID https://orcid.org/0009-0009-4465-1685
University Clinical Centre, Gdańsk, Poland
ul. Dębinki 7, 80-952 Gdańsk
E-mail: amirnaziri@o2.pl

2.Julianna Rozalia Chyl, ORCID https://orcid.org/0009-0003-1115-1226
Medical University of Gdańsk, Poland
ul. Marii Skłodowskiej-Curie 3a, 80-210 Gdańsk
E-mail: julianna.chyl@gmail.com

3.Martyna Zielnik, ORCID https://orcid.org/0009-0007-9787-0625
The John Paul's II Western Hospital in Grodzisk Mazowiecki, Poland
ul. Daleka 11, 05-825 Grodzisk Mazowiecki
E-mail: martynazie@icloud.com

4.Katarzyna Stróżna, ORCID https://orcid.org/0009-0007-7614-1108
Medical Center of Postgraduate Education, Professor Witold Orłowski Independent Public Clinical Hospital, Warszawa, Poland
Czerniakowska 231, 00-416 Warszawa
E-mail: katarzyna.strozna1@gmail.com

5.Olga Błażowska, ORCID https://orcid.org/0009-0003-6989-3062
Wolski Hospital, Warsaw, Poland
Marcina Kasprzaka 17, 01-211 Warszawa
E-mail: olga.blazowska@gmail.com

6. Weronika Nowak ORCID **https://orcid.org/0009-0009-4325-4602**
Medical University of Gdańsk, Poland
ul. Marii Skłodowskiej-Curie 3a, 80-210 Gdańsk
E-mail: weronikanowak@gumed.edu.pl

7. Oliwia Barbara Gutowska ORCID **https://orcid.org/0009-0000-7378-7383**
Medical University of Gdańsk, Poland
ul. Marii Skłodowskiej-Curie 3a, 80-210 Gdańsk
E-mail: oliwia.gutowska.01@gmail.com

8. Wiktoria Magdalena Kostuch ORCID **https://orcid.org/0009-0003-8084-0551**
Medical University of Gdańsk, Poland
ul. Marii Skłodowskiej-Curie 3a, 80-210 Gdańsk
E-mail: wiktoriakostuch@gumed.edu.pl

9. Alicja Jurewicz ORCID **https://orcid.org/0009-0002-4834-1499**
Independent Public Health Care Institution of the Ministry of Interior and Administration in Gdańsk, Poland Kartuska 4/6, 80-104 Gdańsk
E-mail: alajurewicz@gmail.com

10. Agata Zimon ORCID https://orcid.org/0009-0000-6163-841X
Medical University of Gdańsk, Poland
ul. Marii Skłodowskiej-Curie 3a, 80-210 Gdańsk
E-mail: agatazimon1@gmail.com

Corresponding Author
Amir Naziri, E-mail: amirnaziri@o2.pl

Abstract

Background. Type 2 diabetes mellitus affects approximately 589 million adults worldwide and is projected to reach 900 million by 2050. Current management is limited by high treatment costs, low patient compliance, and disability caused by complications. Passive heat therapy has attracted growing scientific interest as an accessible complementary intervention.

Aim. To evaluate whether passive heat therapy offers benefit as a complementary treatment in patients with type 2 diabetes and its complications.

Material and Methods. Narrative literature review using PubMed, Google Scholar, and Scopus databases. Search terms included: "Finnish sauna", "passive heat therapy", "hot water immersion", "diabetes", and "diabetes complications". Only English-language publications were included with no time restrictions.

Results. Repeated heat therapy sessions improved fasting plasma glucose and HbA1c, while single exposures produced no measurable glycaemic benefit. Heat therapy was associated with increased insulin sensitivity and HSP-70 upregulation. Regular sauna use reduced cardiovascular mortality, improved endothelial function, and lowered blood pressure. Preliminary evidence suggests benefits in retinopathy, nephropathy, neuropathy, and diabetic foot perfusion.

Conclusions. Passive heat therapy shows promise as a complementary intervention in type 2 diabetes, particularly regarding cardiovascular outcomes and glycaemic control. Evidence is currently limited by small sample sizes and heterogeneous protocols. Patients on insulin therapy should be monitored for hypoglycaemia during heat exposure.

Key words: Finnish sauna, heat therapy, type 2 diabetes mellitus, HSP-70, glycaemic control **cardiovascular complications**

1. Introduction

Heat therapy is a common health practice around the globe. Thermal baths in ancient Greece and Rome, Turkish hammam, Japanese onsen, Russian banya and Finnish sauna are all examples of how throughout history different civilisations attributed beneficial health properties to passive heating of the whole body. In modern times, with rapid technological advancements in medicine and a focus on pharmacological treatment, traditional methods have been sidetracked. However, recently, with the publication of several articles on the benefits of heat therapy in patients with cardiometabolic [1], respiratory [2] and cognitive diseases [3], the interest of researchers is shifting back towards the potential benefits of heat therapy, especially Finnish sauna.

Diabetes is currently one of the fastest growing global epidemics [4], estimated in 2024 at around 589 million adults, constituting approximately 11% of the global adult population, and projected to reach 900 million by 2050 [5]. It poses a major challenge to healthcare professionals around the world. Current general treatment of type 2 diabetes is focused on reducing complications through management of body weight and glycaemia, achieved through lifestyle modifications such as dietary restrictions and exercise, along with pharmacological therapy [6]. The high financial burden of newer generation pharmaceuticals, low compliance with lifestyle changes, and the high prevalence of disability caused by complications preventing exercise [7, 8] attenuate the effects of standard treatment in individual patient groups. There is therefore a need for additional treatment methods. The purpose of this article is to assess the potential of passive heat therapy as a complementary tool in the management of type 2 diabetes and its most common complications.

Type 2 diabetes is a chronic metabolic disease characterised by insulin resistance and progressive hyperinsulinaemia, followed by insulin deficiency due to pancreatic beta cell failure [9]. Long-term hyperglycaemia caused by insulin resistance leads to both macro- and microvascular damage, resulting in life-threatening complications such as ischemic heart disease, cerebrovascular disease, retinopathy, neuropathy, and nephropathy.

2. Heat Shock Proteins

Heat acclimatisation leads to an increase in heat shock protein (HSP) expression [10]. HSPs are proteins whose expression is triggered in cells by exposure to stress. They act as molecular chaperones, protecting and repairing other proteins to prevent cellular damage [11]. HSP-70 is the most extensively researched HSP family, and one of its members — HSP-72 — has been found to be associated with the pathogenesis of type 2 diabetes. Reduced HSP-72 expression has been reported to increase insulin resistance through activation of JNK kinase, which leads to phosphorylation of IRS-1 (insulin receptor substrate 1), disrupting insulin receptor function [12].

However, HSP-70 may play a dual role in the pathophysiology of type 2 diabetes depending on its cellular localisation. The intracellular fraction (iHSP-70) is associated with an anti-inflammatory role, while the extracellular fraction (eHSP-70) promotes inflammation by inducing neutrophil activity, activating natural killer (NK) cells, and increasing production of pro-inflammatory cytokines [13]. HSP-70 levels have been found to be elevated in diabetic patients [14, 15].

Animal studies confirmed the beneficial effect of HSP-72 by demonstrating that increasing its expression reduced insulin resistance and hyperglycaemia [16]. Some of these studies used whole-body heating as the method to induce HSP-72 expression, suggesting heat therapy's potential as a tool to combat diabetes [17, 18, 19]. Research in humans has shown that heat exposure upregulates the HSPA1A gene, which encodes HSP-70-1 [20]. However, studies comparing HSP-70 levels before and after heat exposure in humans have yielded conflicting results, with some demonstrating an increase [21, 22] and others showing no significant difference [23, 24, 25].

Regardless, researchers broadly agree that while better standardised research is needed, passive heat exposure may be useful in the treatment of type 2 diabetes and other metabolic diseases. This potential extends beyond HSP-mediated pathways, with a growing body of evidence examining the direct effect of heat therapy on glycaemic markers and metabolic control, as discussed in the following section.

3. Effect on Glucose Metabolism

Diabetes is diagnosed when one of the following criteria is met: HbA1c ≥6.5%, fasting plasma glucose (FPG) ≥126 mg/dL, 2-hour plasma glucose ≥200 mg/dL (≥11.1 mmol/L) during an oral glucose tolerance test (OGTT), or a random plasma glucose ≥200 mg/dL accompanied by symptoms of hyperglycaemia or hyperglycaemic crisis. These markers are also used in the diagnosis of prediabetes [26], serve in monitoring disease progression and the effectiveness of introduced therapy, and have been used as predictors of complication prevalence and morbidity. Maintaining HbA1c below 7% reduces cardiovascular disease risk by 37%, and every percentage point above this level increases the risk of macrovascular complications by 18% [27, 28].

Given the significance of glycaemic markers in both diagnosis and long-term outcomes, identifying non-pharmacological strategies capable of improving them remains an important area of research. One such approach that has attracted scientific interest is passive heat therapy, with early evidence suggesting it may positively influence HbA1c and fasting plasma glucose in patients with type 2 diabetes.

A study by Hooper in 1998 showed promise of passive heat therapy in the regulation of HbA1c and glycaemia in diabetic patients. Over the three-week study period, participants' fasting plasma glucose decreased from 182±37 mg/dL (10.1±2.0 mmol/L) to 159±42 mg/dL (8.8±2.3 mmol/L) (P=0.02), and mean glycosylated haemoglobin decreased from 11.3±3.1% to 10.3±2.6% (P=0.004) [29]. These promising results incited scientists to further study the effect of heat therapy on glycaemic control, with conflicting results.

Hooper's study was included in a meta-analysis of 5 studies by Sebok et al., which showed only a slight, statistically insignificant decrease in HbA1c and fasting glucose. However, the authors note that while mean levels showed no overall improvement, two of the included studies individually demonstrated a significant change in HbA1c [29 ,30] and two showed a significant change in fasting glucose level [29, 31].

In a separate study, hot water immersion repeated 8–10 times over 14 days resulted in increased fasted insulin sensitivity and lowered fasted plasma insulin. However, postprandial insulin sensitivity (P=0.19), fasting plasma glucose (P=0.40), and postprandial plasma insulin (P=0.47) were not significantly different. These results are consistent with the broader pattern seen across the literature, in which chronic passive heating appears to improve fasting glucose levels [32] and HbA1c [33], while postprandial glucose levels remain largely unaffected.

A meta-analysis of 14 studies by Maley et al. involving a single heat exposure found that isolated sessions did not improve the glycaemic profile in diabetic individuals [34]. These findings suggest that any beneficial effect of passive heating on glycaemic control requires repeated exposure rather than isolated sessions. Interestingly, one included study also reported an increase in plasma leptin following heating — a hormone involved in satiety regulation — which may warrant further investigation in the context of obesity management in diabetic patients.

4. Role of Heat Therapy in Type 2 Diabetes Complications

The most common cause of death in patients with type 2 diabetes are events related to long-term complications. The highest morbidity is attributed to acute myocardial infarction, stroke, end-stage renal disease, and lower extremity amputation [35]. Risk factors associated with an increased likelihood of developing complications include male sex, smoking, presence of other complications, elevated HbA1c, and raised systolic blood pressure [36].

Complications can be divided into microvascular — retinopathy, nephropathy, and neuropathy — and macrovascular — cardiovascular, cerebrovascular, and peripheral artery disease.

Prolonged elevation of blood glucose causes dysregulation of cellular metabolic pathways throughout the body, contributing to the development of complications. Researchers propose several theories to explain this mechanism:

AGE theory — Advanced Glycation End-products (AGEs) are formed by the binding of sugars to proteins, causing structural damage and impairing various metabolic cascades, ultimately leading to tissue damage and complications. Experimental evidence suggests that heat therapy may reduce AGE accumulation by upregulating HSP expression, which helps protect proteins from glycation-related damage.

Aldose reductase theory — Under normal conditions, aldose reductase reduces harmful aldehydes to non-toxic compounds. However, in hyperglycaemia, glycolysis is insufficient to process excess glucose, and the enzyme begins reducing glucose to sorbitol and subsequently fructose, consuming the cofactor NADPH in the process. NADPH is critical to many other cellular reactions, and its depletion may impair normal cell function. Heat therapy has been proposed to partially mitigate this pathway through its anti-inflammatory and metabolic effects, though direct evidence remains limited.

ROS theory — This hypothesis proposes that the intensification of cellular glucose metabolism during hyperglycaemia leads to increased production of byproducts such as free radicals, which cause cellular damage. Heat therapy may attenuate ROS production through HSP-mediated cytoprotection and improvements in mitochondrial function.

PKC theory — Protein kinase C is activated during hyperglycaemia, modifying gene and protein expression, leading to activation of growth factors and aberrant angiogenesis. Reductions in hyperglycaemia achieved through repeated heat therapy sessions may indirectly reduce PKC activation.

All of these mechanisms contribute to the induction of inflammation, vascular damage, and pathological angiogenesis [37]. The following subsections examine the evidence for heat therapy's role in modulating both macrovascular and microvascular complications.

4.1 Macrovascular Complications

Macrovascular damage is mostly attributed to endothelial dysfunction caused by hyperglycaemia and insulin resistance. It leads to an increase in Reactive Oxygen Species (ROS) production, causing cellular damage and a reduction of endothelial nitric oxide synthase (eNOS) mediated NO secretion. NO is an important factor in vascular protection through its vasodilating effect, improving organ blood flow and preventing blood clot formation [38]. A meta-analysis of 39 studies demonstrated that acute hyperglycaemia impairs endothelial function in large vessels in both healthy individuals and those with cardiometabolic disease [39].

In the last decade, Finnish researchers have shown across multiple studies that sauna use reduces mortality from both coronary events [40] and cerebrovascular events [41]. A study using infrared sauna on 25 patients with cardiovascular risk factors, including type 2 diabetes, showed a significant decrease in both diastolic and systolic pressure, as well as an increase in flow-mediated, endothelium-dependent dilation [32]. Similar effects were observed in other studies [42, 43]. This beneficial effect is attributed to the similarity between the body's response to passive heating and physical exercise — namely an increase in heart rate, an increase in left ventricular ejection fraction, and a reduction in blood pressure with regular use [44].

4.2 Microvascular Complications

Microvascular damage represents one of the most burdensome consequences of long-term hyperglycaemia in type 2 diabetes. The following subsections examine the evidence for heat therapy's role across the main microvascular complication subtypes.

Retinopathy

Growth factors including VEGF have been found to play an important role in the pathogenesis of diabetic retinopathy [45]. Although currently no studies have examined the direct effect of whole-body heating on diabetic retinopathy, studies on in vitro human cells showed that increased HSP-70 expression is associated with suppression of VEGF and inflammatory cytokines [46], and in mice it protected retinal capillary cells from apoptosis [47]. These findings suggest potential benefit of whole-body heating, given its capacity to elevate HSP-70 levels. However, due to the absence of direct clinical studies, it is not currently possible to determine whether heating methods such as sauna provide a sufficient stimulus to replicate the effects observed in these experimental models.

Nephropathy

Acute exposure to high temperature forces the body to introduce adaptive mechanisms to reduce core temperature, inducing changes in water balance and heavy sweating. This may lead to dehydration and hypovolaemia, resulting in hypoperfusion, which has been identified as a risk factor for renal injury [48]. A prospective cohort study on 2,071 Finnish men aged 42–61 years with normal kidney function, comparing one sauna session per week with four to seven sessions per week, found no significant changes in baseline levels of estimated GFR, creatinine, or sodium.

Chronic exposure to ambient heat has been shown to reduce GFR in patients with chronic kidney disease and to promote acute kidney injury [49]. Studies in both mice [50] and humans [51] have shown that heat acclimatisation protects against heat-induced kidney injury. This research suggests that passive heating in a controlled environment not only does not promote kidney disease progression but may also play a protective role.

Neuropathy

Diabetic neuropathy is a common microvascular complication of type 2 diabetes and the most frequently diagnosed type of neuropathy worldwide. Its pathophysiology is linked to neuronal disruption caused by dyslipidaemia and insulin resistance [52]. Symptoms include paraesthesia such as numbness, tingling, loss of sensation, and pain,

typically localised to the distal extremities and often described in a “glove and stocking” distribution [53]. A study on 68 patients with type 2 diabetes showed improvement in sensation measured by vibration sensation testing following exposure to a heated water foot bath, although the mechanism underlying this improvement remained unclear [54]. Survey-based studies have also reported alleviation of neuropathic pain following sauna exposure [55, 56]. While the effect of heating on neuropathy remains under-researched, the studies presented here suggest potential benefits from heat exposure in neuropathy management.

Diabetic Foot

Diabetic foot remains a significant challenge for healthcare professionals and has a considerable negative impact on patients’ quality of life. The combination of neuropathy and microangiopathy contributing to its development creates difficulties in both diagnosis and management. Natural healing processes are disrupted by restricted blood flow, and reduced sensation leads to neglect of ulcers, which in many cases may result in amputation [57]. Beyond potential positive effects on glycaemic management and inflammation, research has shown that local heating improved circulation around ulcers and accelerated the healing process [58, 59]. Local heating methods have shown significant improvement in this regard; however, caution should be exercised with whole-body heating, as studies in mice showed that temperature elevation exceeding 43°C had negative effects on wound healing [60].

5. Conclusions

The evidence reviewed in this article suggests that passive heat therapy holds meaningful promise as a complementary tool in the management of type 2 diabetes. Findings span several domains: improvements in glycaemic markers and insulin sensitivity, potential anti-inflammatory and cytoprotective effects mediated by heat shock proteins, a well-evidenced cardiovascular benefit, and preliminary positive signals across microvascular complication subtypes including neuropathy and diabetic foot.

However, several limitations of existing research must be acknowledged. The majority of studies involve small sample sizes of typically 10–20 participants, and there is a lack of agreed standardisation of heating conditions or objective measurement of core temperature change, which contributes to conflicting results between otherwise comparable studies. Additionally, most studies do not differentiate between intracellular and extracellular HSP-70 fractions, limiting interpretation of their respective roles. There is also a methodological inconsistency across research areas — cardiovascular studies predominantly use sauna as the heating modality, while studies on glycaemic parameters and microvascular complications more commonly use hot water immersion — making direct comparison between modalities difficult.

An ongoing randomised controlled trial, “Effect of HEAT therapy in patients with type 2 diabetes mellitus (HEATED): protocol for a randomised controlled trial” [61], with approximately 65 participants per group, is currently evaluating the effect of heat therapy on glycaemia and complication control, and its results are likely to substantially advance the field. Future research should prioritise larger sample sizes, standardised protocols, and head-to-head comparisons between heating modalities.

Finally, a practical safety note: in patients receiving insulin therapy, caution is warranted, as studies have demonstrated accelerated insulin absorption during sauna use, which may increase the risk of hypoglycaemia [62, 63].

Declaration of the use of generative AI and AI-assisted technologies in the writing process.

In preparing this work, the author(s) used AI language model assistance (Claude, Anthropic) for the purpose of assistance with text translation, selection of appropriate English scientific terminology, and stylistic organisation of the text. After using this tool/service, the author(s) have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

Disclosure:

Author's Contribution:

Conceptualization: Olga Błażowska , Wiktoria Magdalena Kostuch
Methodology: Agata Zimon,Weronika Nowak
Investigation: Katarzyna Stróżna, Julianna Rozalia Chyl
Data curation: Olga Błażowska, Martyna Zielnik, Weronika Nowak
rough preparation: Amir Naziri, Agata Zimon, Katarzyna Stróżna, Julianna Rozalia Chyl
Writing –review and editing: Amir Naziri , Martyna Zielnik, Alicja Jurewicz
Visualisation: Alicja Jurewicz , Oliwia Barbara Gutowska
Supervision: Oliwia Barbara Gutowska
Project Administration: Wiktoria Magdalena Kostuch

Funding: The study did not receive external funding.
Institutional Review Board Statement: Not applicable.
Informed Consent Statement: Not applicable.
Data Availability Statement: Not applicable.
Acknowledgements: Not applicable.
Conflicts of Interest: The authors declare no conflicts of interest.
All authors have read and agreed with the published version of the manuscript.

REFERENCES

1. Laukkanen T, Khan H, Zaccardi F, Laukkanen JA. Association between sauna bathing and fatal cardiovascular and all-cause mortality events. JAMA Intern Med. 2015 Apr;175(4):542-8. doi: 10.1001/jamainternmed.2014.8187. PMID: 25705824.
2. Kunutsor SK, Laukkanen T, Laukkanen JA. Sauna bathing reduces the risk of respiratory diseases: a long-term prospective cohort study. Eur J Epidemiol. 2017 Dec;32(12):1107-1111. doi: 10.1007/s10654-017-0311-6. Epub 2017 Sep 13. PMID: 28905164.
3. Laukkanen T, Kunutsor S, Kauhanen J, Laukkanen JA. Sauna bathing is inversely associated with dementia and Alzheimer's disease in middle-aged Finnish men. Age Ageing. 2017 Mar 1;46(2):245-249. doi: 10.1093/ageing/afw212. PMID: 27932366.
4. Kunutsor SK, Laukkanen T, Laukkanen JA. Sauna bathing reduces the risk of respiratory diseases: a long-term prospective cohort study. Eur J Epidemiol. 2017 Dec;32(12):1107-1111. doi: 10.1007/s10654-017-0311-6. Epub 2017 Sep 13. PMID: 28905164.
5. Genitsaridi I, Salpea P, Salim A, Sajjadi SF, Tomic D, James S, Thirunavukkarasu S, Issaka A, Chen L, Basit A, Luk AOY, Ma RCW, Mbanya JC, Ramachandran A, Wild SH, Duncan BB, Boyko EJ, Magliano DJ. 11th edition of the IDF Diabetes Atlas: global, regional, and national diabetes prevalence estimates for 2024 and projections for 2050. Lancet Diabetes Endocrinol. 2026 Feb;14(2):149-156. doi: 10.1016/S2213-8587(25)00299-2. Epub 2025 Dec 15. PMID: 41412135.
6. Kalyani RR, Neumiller JJ, Maruthur NM, Wexler DJ. Diagnosis and Treatment of Type 2 Diabetes in Adults: A Review. JAMA. 2025 Sep 16;334(11):984-1002. doi: 10.1001/jama.2025.5956. PMID: 40549398; PMCID: PMC13014273.
7. Kirkman MS, Rowan-Martin MT, Levin R, Fonseca VA, Schmittdiel JA, Herman WH, Aubert RE. Determinants

of adherence to diabetes medications: findings from a large pharmacy claims database. Diabetes Care. 2015 Apr;38(4):604-9. doi: 10.2337/dc14-2098. Epub 2015 Jan 8. PMID: 25573883; PMCID: PMC4370331.
8. MacDonald CS, Ried-Larsen M, Soleimani J, Alsawas M, Lieberman DE, Ismail AS, Serafim LP, Yang T, Prokop L, Joyner M, Murad MH, Barwise A. A systematic review of adherence to physical activity interventions in individuals with type 2 diabetes. Diabetes Metab Res Rev. 2021 Nov;37(8):e3444. doi: 10.1002/dmrr.3444. Epub 2021 Mar 26. PMID: 33769660.
9. Ahmad E, Lim S, Lamptey R, Webb DR, Davies MJ. Type 2 diabetes. Lancet. 2022 Nov 19;400(10365):1803-1820. doi: 10.1016/S0140-6736(22)01655-5. Epub 2022 Nov 1. PMID: 36332637.
10. Nava R, Zuhl MN. Heat acclimation-induced intracellular HSP70 in humans: a meta-analysis. Cell Stress Chaperones. 2020 Jan;25(1):35-45. doi: 10.1007/s12192-019-01059-y. Epub 2019 Dec 10. PMID: 31823288; PMCID: PMC6985308.
11. Morimoto RI. Regulation of the heat shock transcriptional response: cross talk between a family of heat shock factors, molecular chaperones, and negative regulators. Genes Dev. 1998 Dec 15;12(24):3788-96. doi: 10.1101/gad.12.24.3788. PMID: 9869631.
12. Hotamisligil GS. Inflammation and metabolic disorders. Nature. 2006 Dec 14;444(7121):860-7. doi: 10.1038/nature05485. PMID: 17167474.
13. Rodrigues-Krause J, Krause M, O'Hagan C, De Vito G, Boreham C, Murphy C, Newsholme P, Colleran G. Divergence of intracellular and extracellular HSP72 in type 2 diabetes: does fat matter? Cell Stress Chaperones. 2012 May;17(3):293-302. doi: 10.1007/s12192-011-0319-x. Epub 2012 Jan 4. PMID: 22215518; PMCID: PMC3312959.
14. Al-Zuhaeri AA, Al-Shakour AA, Ali Mansour A. Serum Level of Heat Shock Protein 70 in Patients with Type 2 Diabetes Mellitus in Basrah, Iraq. Arch Razi Inst. 2022 Oct 31;77(5):1837-1844. doi: 10.22092/ARI.2022.358129.2155. PMID: 37123136; PMCID: PMC10133620.
15. de Lemos Muller CH, Schroeder HT, Rodrigues-Krause J, Krause M. Extra and intra cellular HSP70 levels in adults with and without metabolic disorders: a systematic review and meta-analysis. Cell Stress Chaperones. 2023 Nov;28(6):761-771. doi: 10.1007/s12192-023-01368-3. Epub 2023 Jul 26. PMID: 37495770; PMCID: PMC10746644.
16. Li Y, Lin S, Xu X, Jin W, Su Y, Yuan F, Zhang Y, Li Z, Zhou Y, Zhu L, Zhang L. Skeletal muscle HSF1 prevents insulin resistance by improving glucose utilization. FASEB J. 2022 Dec;36(12):e22667. doi: 10.1096/fj.202201160RR. Erratum in: FASEB J. 2023 Feb;37(2):e22756. doi: 10.1096/fsb2.22756. PMID: 36421020.
17. Chung J, Nguyen AK, Henstridge DC, Holmes AG, Chan MH, Mesa JL, Lancaster GI, Southgate RJ, Bruce CR, Duffy SJ, Horvath I, Mestril R, Watt MJ, Hooper PL, Kingwell BA, Vigh L, Hevener A, Febbraio MA. HSP72 protects against obesity-induced insulin resistance. Proc Natl Acad Sci U S A. 2008 Feb 5;105(5):1739-44. doi: 10.1073/pnas.0705799105. Epub 2008 Jan 25. PMID: 18223156; PMCID: PMC2234214.
18. Kokura S, Adachi S, Manabe E, Mizushima K, Hattori T, Okuda T, Nakabe N, Handa O, Takagi T, Naito Y, Yoshida N, Yoshikawa T. Whole body hyperthermia improves obesity-induced insulin resistance in diabetic mice. Int J Hyperthermia. 2007 May;23(3):259-65. doi: 10.1080/02656730601176824. PMID: 17523018.
19. Kondo T, Sasaki K, Matsuyama R, Morino-Koga S, Adachi H, Suico MA, Kawashima J, Motoshima H, Furukawa N, Kai H, Araki E. Hyperthermia with mild electrical stimulation protects pancreatic β-cells from cell stresses and apoptosis. Diabetes. 2012 Apr;61(4):838-47. doi: 10.2337/db11-1098. Epub 2012 Feb 23. PMID: 22362176; PMCID: PMC3314363.
20. Bouchama A, Aziz MA, Mahri SA, Gabere MN, Dlamy MA, Mohammad S, Abbad MA, Hussein M. A Model of Exposure to Extreme Environmental Heat Uncovers the Human Transcriptome to Heat Stress. Sci Rep. 2017 Aug 25;7(1):9429. doi: 10.1038/s41598-017-09819-5. Erratum in: Sci Rep. 2026 Mar 3;16(1):8052. doi: 10.1038/s41598-026-40575-7. PMID: 28842615; PMCID: PMC5573409.
21. James TJ, Corbett J, Cummings M, Allard S, Shute JK, Belcher H, Mayes H, Gould AAM, Piccolo DD, Tipton M, Perissiou M, Saynor ZL, Shepherd AI. The effect of repeated hot water immersion on insulin sensitivity, heat shock protein 70, and inflammation in individuals with type 2 diabetes mellitus. Am J Physiol Endocrinol Metab. 2023 Dec 1;325(6):E755-E763. doi: 10.1152/ajpendo.00222.2023. Epub 2023 Nov 8. PMID: 37938179.
22. Pilch W, Szarek M, Olga CL, Anna P, Żychowska M, Ewa SK, Andraščíková Š, Pałka T. The effects of a single and a series of Finnish sauna sessions on the immune response and HSP-70 levels in trained and untrained men. Int J Hyperthermia. 2023;40(1):2179672. doi: 10.1080/02656736.2023.2179672. PMID: 36813265.
23. Behzadi P, Ravanelli N, Gravel H, Barry H, Debray A, Chaseling GK, Jacquemet V, Neagoe PE, Nigam A, Carpentier AC, Sirois MG, Gagnon D. Acute effect of passive heat exposure on markers of cardiometabolic function in adults with type 2 diabetes mellitus. J Appl Physiol (1985). 2022 May 1;132(5):1154-1166. doi: 10.1152/japplphysiol.00800.2021. Epub 2022 Mar 24. PMID: 35323077.
24. Pallubinsky H, Phielix E, Dautzenberg B, Schaart G, Connell NJ, de Wit-Verheggen V, Havekes B, van Baak MA, Schrauwen P, van Marken Lichtenbelt WD. Passive exposure to heat improves glucose metabolism in overweight humans. Acta Physiol (Oxf). 2020 Aug;229(4):e13488. doi: 10.1111/apha.13488. Epub 2020 Jun 1.

PMID: 32359193; PMCID: PMC7379279.
25. Hoekstra SP, Bishop NC, Faulkner SH, Bailey SJ, Leicht CA. Acute and chronic effects of hot water immersion on inflammation and metabolism in sedentary, overweight adults. J Appl Physiol (1985). 2018 Dec 1;125(6):2008-2018. doi: 10.1152/japplphysiol.00407.2018. Epub 2018 Oct 18. PMID: 30335579.
26. American Diabetes Association Professional Practice Committee for Diabetes*. 2. Diagnosis and Classification of Diabetes: Standards of Care in Diabetes-2026. Diabetes Care. 2026 Jan 1;49(Supplement_1):S27-S49. doi: 10.2337/dc26-S002. PMID: 41358893; PMCID: PMC12690183.
27. Selvin E, Marinopoulos S, Berkenblit G, Rami T, Brancati FL, Powe NR, Golden SH. Meta-analysis: glycosylated hemoglobin and cardiovascular disease in diabetes mellitus. Ann Intern Med. 2004 Sep 21;141(6):421-31. doi: 10.7326/0003-4819-141-6-200409210-00007. PMID: 15381515.
28. Joseph JJ, Deedwania P, Acharya T, Aguilar D, Bhatt DL, Chyun DA, Di Palo KE, Golden SH, Sperling LS; American Heart Association Diabetes Committee of the Council on Lifestyle and Cardiometabolic Health; Council on Arteriosclerosis, Thrombosis and Vascular Biology; Council on Clinical Cardiology; and Council on Hypertension. Comprehensive Management of Cardiovascular Risk Factors for Adults With Type 2 Diabetes: A Scientific Statement From the American Heart Association. Circulation. 2022 Mar;145(9):e722-e759. doi: 10.1161/CIR.0000000000001040. Epub 2022 Jan 10. PMID: 35000404.
29. Hooper PL. Hot-tub therapy for type 2 diabetes mellitus. N Engl J Med. 1999 Sep 16;341(12):924-5. doi: 10.1056/NEJM199909163411216. PMID: 10498473.
30. Oláh M, Koncz Á, Fehér J, Kálmánczhey J, Oláh C, Nagy G, Bender T. The effect of balneotherapy on antioxidant, inflammatory, and metabolic indices in patients with cardiovascular risk factors (hypertension and obesity)--a randomised, controlled, follow-up study. Contemp Clin Trials. 2011 Nov;32(6):793-801. doi: 10.1016/j.cct.2011.06.003. Epub 2011 Jul 6. PMID: 21763463.
31. Koçak FA, Kurt EE, Milletli Sezgin F, Şaş S, Tuncay F, Erdem HR. The effect of balneotherapy on body mass index, adipokine levels, sleep disturbances, and quality of life of women with morbid obesity. Int J Biometeorol. 2020 Sep;64(9):1463-1472. doi: 10.1007/s00484-020-01924-x. Epub 2020 May 6. PMID: 32377931; PMCID: PMC7223765.
32. Imamura M, Biro S, Kihara T, Yoshifuku S, Takasaki K, Otsuji Y, Minagoe S, Toyama Y, Tei C. Repeated thermal therapy improves impaired vascular endothelial function in patients with coronary risk factors. J Am Coll Cardiol. 2001 Oct;38(4):1083-8. doi: 10.1016/s0735-1097(01)01467-x. PMID: 11583886.
33. Kamioka H, Mori Y, Horiuchi T, Hayashi T, Ohmura K, Yamaguchi S, Kato M. Association of Daily Home-Based Hot Water Bathing and Glycemic Control in Ambulatory Japanese Patients with Type 2 Diabetes Mellitus During the COVID-19 Pandemic: A Multicenter Cross-Sectional Study. Diabetes Metab Syndr Obes. 2020 Dec 22;13:5059-5069. doi: 10.2147/DMSO.S279270. PMID: 33376375; PMCID: PMC7764630.
34. Maley MJ, Hunt AP, Stewart IB, Faulkner SH, Minett GM. Passive heating and glycaemic control in non-diabetic and diabetic individuals: A systematic review and meta-analysis. PLoS One. 2019 Mar 22;14(3):e0214223. doi: 10.1371/journal.pone.0214223. PMID: 30901372; PMCID: PMC6430508.
35. Gregg EW, Li Y, Wang J, Burrows NR, Ali MK, Rolka D, Williams DE, Geiss L. Changes in diabetes-related complications in the United States, 1990-2010. N Engl J Med. 2014 Apr 17;370(16):1514-23. doi: 10.1056/NEJMoa1310799. PMID: 24738668.
36. Arnold SV, Khunti K, Tang F, Chen H, Cid-Ruzafa J, Cooper A, Fenici P, Gomes MB, Hammar N, Ji L, Saraiva GL, Medina J, Nicolucci A, Ramirez L, Rathmann W, Shestakova MV, Shimomura I, Surmont F, Vora J, Watada H, Kosiborod M. Incidence rates and predictors of microvascular and macrovascular complications in patients with type 2 diabetes: Results from the longitudinal global discover study. Am Heart J. 2022 Jan;243:232-239. doi: 10.1016/j.ahj.2021.10.181. Epub 2021 Oct 16. PMID: 34666013.
37. Sheetz MJ, King GL. Molecular understanding of hyperglycemia's adverse effects for diabetic complications. JAMA. 2002 Nov 27;288(20):2579-88. doi: 10.1001/jama.288.20.2579. PMID: 12444865.
38. Lüscher TF. Endothelium-derived nitric oxide: the endogenous nitrovasodilator in the human cardiovascular system. Eur Heart J. 1991 Nov;12 Suppl E:2-11. doi: 10.1093/eurheartj/12.suppl_e.2. PMID: 1790780.
39. Loader J, Montero D, Lorenzen C, Watts R, Méziat C, Reboul C, Stewart S, Walther G. Acute Hyperglycemia Impairs Vascular Function in Healthy and Cardiometabolic Diseased Subjects: Systematic Review and Meta-Analysis. Arterioscler Thromb Vasc Biol. 2015 Sep;35(9):2060-72. doi: 10.1161/ATVBAHA.115.305530. Epub 2015 Jun 25. PMID: 26112007.
40. Laukkanen T, Khan H, Zaccardi F, Laukkanen JA. Association between sauna bathing and fatal cardiovascular and all-cause mortality events. JAMA Intern Med. 2015 Apr;175(4):542-8. doi: 10.1001/jamainternmed.2014.8187. PMID: 25705824.
41. Kunutsor SK, Khan H, Zaccardi F, Laukkanen T, Willeit P, Laukkanen JA. Sauna bathing reduces the risk of stroke in Finnish men and women: A prospective cohort study. Neurology. 2018 May 29;90(22):e1937-e1944. doi: 10.1212/WNL.0000000000005606. Epub 2018 May 2. PMID: 29720543.
42. Brunt VE, Howard MJ, Francisco MA, Ely BR, Minson CT. Passive heat therapy improves endothelial function, arterial stiffness and blood pressure in sedentary humans. J Physiol. 2016 Sep 15;594(18):5329-42. doi:

10.1113/JP272453. Epub 2016 Jun 30. PMID: 27270841; PMCID: PMC5023696.
43. Greyling A, Schreuder TH, Landman T, Draijer R, Verheggen RJ, Hopman MT, Thijssen DH. Elevation in blood flow and shear rate prevents hyperglycemia-induced endothelial dysfunction in healthy subjects and those with type 2 diabetes. J Appl Physiol (1985). 2015 Mar 1;118(5):579-85. doi: 10.1152/japplphysiol.00936.2014. Epub 2015 Jan 15. PMID: 25593286.
44. Cullen T, Clarke ND, Hill M, Menzies C, Pugh CJA, Steward CJ, Thake CD. The health benefits of passive heating and aerobic exercise: To what extent do the mechanisms overlap? J Appl Physiol (1985). 2020 Dec 1;129(6):1304-1309. doi: 10.1152/japplphysiol.00608.2020. Epub 2020 Oct 1. PMID: 33002381.
45. Iwona BS. Growth Factors in the Pathogenesis of Retinal Neurodegeneration in Diabetes Mellitus. Curr Neuropharmacol. 2016;14(8):792-804. doi: 10.2174/1570159x14666160813182009. PMID: 27528260; PMCID: PMC5333593.
46. Fujino R, Totsuka K, Shiraya T, Ueta T, Araki F, Terao R. Combined thermal and mild electrical stimulations modulate heat shock protein and VEGF in retinal pigment epithelial cells under high glucose. BMC Res Notes. 2025 Oct 29;18(1):459. doi: 10.1186/s13104-025-07539-y. PMID: 41163032; PMCID: PMC12574004.
47. Kowluru RA, Mohammad G, dos Santos JM, Zhong Q. Abrogation of MMP-9 gene protects against the development of retinopathy in diabetic mice by preventing mitochondrial damage. Diabetes. 2011 Nov;60(11):3023-33. doi: 10.2337/db11-0816. Epub 2011 Sep 20. Erratum in: Diabetes. 2021 Jan;70(1):301. doi: 10.2337/db21-er01a. PMID: 21933988; PMCID: PMC3198054.
48. Juett LA, Funnell MP, Carroll HA, James LJ, Mears SA. 24 h severe fluid restriction increases a biomarker of renal injury in healthy males. Eur J Appl Physiol. 2025 Jul;125(7):1983-1995. doi: 10.1007/s00421-025-05749-7. Epub 2025 Mar 7. PMID: 40053075; PMCID: PMC12227480.
49. Zhang Z, Heerspink HJL, Chertow GM, Correa-Rotter R, Gasparrini A, Jongs N, Langkilde AM, McMurray JJV, Mistry MN, Rossing P, Toto RD, Vart P, Nitsch D, Wheeler DC, Caplin B. Ambient heat exposure and kidney function in patients with chronic kidney disease: a post-hoc analysis of the DAPA-CKD trial. Lancet Planet Health. 2024 Apr;8(4):e225-e233. doi: 10.1016/S2542-5196(24)00026-3. PMID: 38580424.
50. Goto H, Nakashima M, Nakashima H, Noguchi M, Imakiire T, Oshima N, Kinoshita M, Kumagai H. Heat acclimation ameliorated heat stress-induced acute kidney injury and prevented changes in kidney macrophages and fibrosis. Am J Physiol Renal Physiol. 2022 Sep 1;323(3):F243-F254. doi: 10.1152/ajprenal.00065.2022. Epub 2022 Jul 7. PMID: 35796461; PMCID: PMC9394728.
51. Ravanelli N, Barry H, Schlader ZJ, Gagnon D. Impact of passive heat acclimation on markers of kidney function during heat stress. Exp Physiol. 2021 Jan;106(1):269-281. doi: 10.1113/EP088637. Epub 2020 Jun 19. PMID: 32495481.
52. Vincent AM, Callaghan BC, Smith AL, Feldman EL. Diabetic neuropathy: cellular mechanisms as therapeutic targets. Nat Rev Neurol. 2011 Sep 13;7(10):573-83. doi: 10.1038/nrneurol.2011.137. PMID: 21912405.
53. Callaghan BC, Cheng HT, Stables CL, Smith AL, Feldman EL. Diabetic neuropathy: clinical manifestations and current treatments. Lancet Neurol. 2012 Jun;11(6):521-34. doi: 10.1016/S1474-4422(12)70065-0. Epub 2012 May 16. PMID: 22608666; PMCID: PMC4254767.
54. Strobel A, Laputsina V, Heinze V, Schulz S, Wienke A, Reer M, Schlitt A. Nonpharmaceutical treatment of distal sensorimotor polyneuropathy in diabetic patients: an unblinded randomized clinical trial. BMC Complement Med Ther. 2025 Mar 6;25(1):93. doi: 10.1186/s12906-025-04830-0. PMID: 40050870; PMCID: PMC11887202.
55. Masuda A, Koga Y, Hattanmaru M, Minagoe S, Tei C. The effects of repeated thermal therapy for patients with chronic pain. Psychother Psychosom. 2005;74(5):288-94. doi: 10.1159/000086319. PMID: 16088266.
56. Nurmikko T, Hietaharju A. Effect of exposure to sauna heat on neuropathic and rheumatoid pain. Pain. 1992 Apr;49(1):43-51. doi: 10.1016/0304-3959(92)90186-F. Erratum in: Pain 1992 Jun;49(3):419. PMID: 1375727.
57. Pecoraro RE, Reiber GE, Burgess EM. Pathways to diabetic limb amputation. Basis for prevention. Diabetes Care. 1990 May;13(5):513-21. doi: 10.2337/diacare.13.5.513. PMID: 2351029.
58. Petrofsky JS, Lawson D, Berk L, Suh H. Enhanced healing of diabetic foot ulcers using local heat and electrical stimulation for 30 min three times per week. J Diabetes. 2010 Mar;2(1):41-6. doi: 10.1111/j.1753-0407.2009.00058.x. Epub 2009 Oct 26. PMID: 20923474.
59. Petrofsky JS, Lawson D, Suh HJ, Rossi C, Zapata K, Broadwell E, Littleton L. The influence of local versus global heat on the healing of chronic wounds in patients with diabetes. Diabetes Technol Ther. 2007 Dec;9(6):535-44. doi: 10.1089/dia.2007.0231. PMID: 18034608.
60. Dos Santos-Silva MA, Trajano ET, Schanuel FS, Monte-Alto-Costa A. Heat delays skin wound healing in mice. Exp Biol Med (Maywood). 2017 Feb;242(3):258-266. doi: 10.1177/1535370216675066. Epub 2016 Oct 23. PMID: 27798118; PMCID: PMC5384502.
61. Sebők J, Édel Z, Dembrovszky F, Farkas N, Török Z, Balogh G, Péter M, Papp I, Balogi Z, Nusser N, Péter I, Hooper P, Geiger P, Erőss B, Wittmann I, Váncsa S, Vigh L, Hegyi P. Effect of HEAT therapy in patiEnts with type 2 Diabetes mellitus (HEATED): protocol for a randomised controlled trial. BMJ Open. 2022 Jul 12;12(7):e062122. doi: 10.1136/bmjopen-2022-062122. PMID: 35820741; PMCID: PMC9277369
62. Koivisto VA. Sauna-induced acceleration in insulin absorption from subcutaneous injection site. Br Med J.

1980 Jun 14;280(6229):1411-3. doi: 10.1136/bmj.280.6229.1411. PMID: 7000239; PMCID: PMC1601709.
63. Vanakoski J, Seppälä T. Heat exposure and drugs. A review of the effects of hyperthermia on pharmacokinetics. Clin Pharmacokinet. 1998 Apr;34(4):311-22. doi: 10.2165/00003088-199834040-00004. PMID: 9571303