Heat Therapy With Relevance to the Reversal of NAFLD and Diabetes

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Editorial

The use of heat therapy in individuals with obesity and Type 2 diabetes mellitus has become an important treatment for metabolic and cardiovascular diseases [1-5] for individuals in the developing and developed world. The cellular response to heat therapy includes the transcriptional up-regulation of genes encoding Heat Shock Proteins (HSPs) as part of the cell’s internal repair mechanism [6,7]. These stress-proteins respond to heat, cold and oxygen deprivation by activating several cascade pathways that may be relevant to survival and apoptosis of mitochondria in cells [8]. Heat therapy has been used with the plasma analysis of adiponectin, AMP-Activated Protein Kinase (AMPK), Heat Shock Factor 1 (HSF1), Heat Shock Protein (HSP) 27, HSP70, and HSP90 important as markers for heat stress therapy [9]. Diabetes that previously has involved pancreatic disease in Type 2 diabetic individuals [10] now involves global Non Alcoholic Fatty Liver Disease (NAFLD) with heat therapy critical to improvement in hepatic insulin resistance [11] in obese/obese diabetic individuals. Heat therapy in individuals with NAFLD has now become important to increase hepatic fat metabolism and to improve insulin resistance in these individuals [12]. Plasma bacterial Lipopolysaccharides (LPS) have risen markedly [13] in individuals in the developing world and the relationship between LPS and the repression of the heat shock gene Sirtuin 1 (Sirt 1) has been reported with relevance to NAFLD and diabetes [14-17]. LPS induces NAFLD and the relevance of dietary fat such as virgin coconut oil/palm oil consumption [14] should be carefully controlled to prevent insulin resistance and accelerated NAFLD. Individuals in the developing world are more susceptible to NAFLD and diabetes and the heat shock response by LPS is involved in the transformation of liver cells (NAFLD) [13] and associated with defective heat stress response that involves the Sirt 1/HSF1 interaction [18]. Sirt 1 (NAD+ dependent class III histone deacetylase) is important to the deacetylation of HSF1 [19-23] and hepatic HSP metabolism [12,15] with relevance to in heat therapy in NAFLD and diabetes (Figure 1). Sirt 1 is involved with the circadian regulation of HSP 60, 70 and 90 with temperature regulation closely associated with Sirt 1 activity/HSP levels in cells [24-26] and may be relevant to the heat shock response in Type 2 diabetes. The metabolism of HSP is relevant to insulin resistance with HSP linked to amyloid beta metabolism [10] with relevance to insulin receptor interactions [27-37]. Sirt 1 and transcriptional dysregulation [16] has become of major concern with p53 regulation of HSF1 and microRNA-34a (mir-34a) relevant to the heat shock response (Figure 1) [38,39]. Sirt 1 is involved in the deacetylation of the transcription factor p53 [16] with heat therapy linked to the induction of p53 related cell apoptosis [40-41]. Sirt 1 is involved with various markers of heat stress such as adiponectin transcription [42], AMPK connections [43], HSF1 regulation and HSP metabolism. AMPK-Sirt 1 (Figure 1) [43] is involved with Nitric Oxide (NO)/HSP crosstalk [44-46] with relevance to natural killer cell activity and the induction of NAFLD [47,48]. LPS has been shown to induce HSPs in various cells [49-51] and LPS in various species has been shown to induce thermo regulatory dysfunction [52,53]. Heat therapy and the involvement of Sirt 1 has become of importance with relevance to the treatment of heart disease [4,5] and NAFLD in diabetes. LPS is involved in the induction of cardiovascular disease [54] and its effects on the repression of Sirt 1 have raised concern on the use of heat treatment in diabetic individuals in the developing world. LPS is involved in the interference in Sirt 1’s role in the deacetylation of p53/mir-34a/ HSF1 (Figure 1) [55] with the effects of heat therapy relevant to the immune response [56,57] and accelerated apoptosis in various cells and tissues. The role of LPS in thermodynamics involved Sirt 1 dysregulation [16] with temperature regulation by Sirt 1 now relevant to the regulation of p53 with heat shock protein associated with p53 accumulation [58].

Conclusion

The understanding of cellular gene response to heat therapy has accelerated with the global epidemic in obesity and diabetes that is related to chronic diseases such as cardiovascular disease and NAFLD. Heat therapy that involves Sirt 1 should be carefully assessed with relevance to Sirt 1’s transcriptional regulation of HSF1/HSP interactions and with excessive heat therapy may accelerate Sirt 1 mediated HSP induced cell apoptosis. Heat therapy to maintain glucose homeostasis

Keywords: Bacterial lipopolysaccharides; Cardiovascular disease; Heat shock factor 1; Heat therapy; Heat shock gene; Heat shock protein; NAFLD; Sirtuin 1; Transcriptional dysregulation

Abbreviations

Sirtuin 1 (Sirt 1), Heat Shock Proteins (HSP), Heat Shock Factor 1 (HSF1), Non Alcoholic Fatty Liver Disease (NAFLD), Bacterial Lipopolysaccharides (LPS), AMP-Activated Protein Kinase (AMPK)

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Figure 1: Heat therapy has become important to the treatment of global NAFLD and cardiovascular disease in individuals with diabetes. Heat therapy now involves the heat shock gene Sirt 1 that is involved in the metabolism of Heat Shock Proteins (HSPs), deacetylation of p53 (heat shock factor 1 response) and nitric oxide/HSP homeostasis involved with the immune response and programmed cell death.

in diabetic individuals in the developed world may differ from developing world diabetic individuals that lack the heat shock gene Sirt 1. Heat therapy intervals that involve sauna versus hot tub temperatures should be carefully reassessed for safety with relevance to time limits for heat therapy that may last for weeks/months. Healthy diets that do not contain saturated fats with heat therapy may prevent global NAFLD but heat therapy in these individuals may be more successful with the consumption of Sirt 1 activators versus Sirt 1 inhibitors with relevance to the reversal of global NAFLD and diabetes.

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References


