*Review*

# The Impact of Ultraviolet Radiation on Energy Metabolism and Metabolic Disorders in Mice: Mechanisms and Implications

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**Abstract:** Ultraviolet (UV) radiation has both harmful and beneficial effects on human health. It can cause skin damage and cancer, but also provides the primary source of vitamin D. Additionally, UV radiation affects energy metabolism in mice with metabolic diseases and has protective effects on hypertension and cardiovascular diseases.

Metabolic Syndrome is a collection of metabolic dysfunctions such as dyslipidemia, hypertension, hyperglycemia, abdominal/central obesity, and insulin resistance. Chronic UVR exposure suppressed obesity and type 2 diabetes in high-fat diet-fed mice, while vitamin D supplementation did not replicate these effects. On the other hand, topical application of nitric oxide (NO) donors replicated UV effects on mice, and NO scavengers prevented the positive impact of UV. UVR may prevent the development of obesity and metabolic syndrome through mechanisms that depend on UVR-induced NO, not vitamin D. Exposure to sunshine has a protective impact on blood pressure and cardiovascular disease. Although lower vitamin D concentration is linked to increased hypertension and the incidence of CVD, oral vitamin D supplementation does not decrease blood pressure or the risk of CVD, indicating that vitamin D may not mediate the beneficial effects of sun exposure. Instead, NO plays a crucial role as an endogenous vasodilator, produced by the vascular endothelium. Solar UVA may release nitrogen oxides from skin storage into the bloodstream, lowering blood pressure and cardiovascular mortality.

In conclusion, UVR exposure could be a promising non-pharmacological intervention for metabolic syndrome, hypertension, and CVD prevention, and NO may play a crucial role in mediating these effects. Further research is needed to explore the precise mechanisms and identify the optimal doses and exposure times of UVR.

**Keywords:** metabolic disorders; nitric oxide; obesity; ultraviolet; vitamin D

## 1. Introduction

As one of the essential environmental variables, sunshine is inextricably linked to people's daily lives. According to wavelength, sunlight that reaches the surface of the Earth can be separated into three types of radiation: visible light, ultraviolet, and infrared light. Even though UV can be divided into UVA (320-400nm), UVB (290-320nm), and UVC (100-290nm), the whole of UVC and most of UVB are absorbed by the stratospheric ozone layer, resulting in UVA accounting for more than 95 percent [1]. Furthermore, the UVB/UVA ratio is not constant because UVB strength varies with latitude, season, and time of day. Since the relationship between the radiated energy and wavelength is inverse,

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UVA radiation penetrates the dermis, whereas UVB radiation primarily penetrates the epidermis and scarcely enters the dermis [2]. UVR impacts on the skin could be classified as acute or chronic: tanning (increased melanogenesis), immunosuppression, and sunburn (erythema) are acute UV radiation reactions, whereas photoaging, immunosuppression, and skin cancer are chronic effects [1]. In addition to the harmful consequences mentioned above, skin biosynthesis, which is based on UVB exposure (not UVA), is the significant sources of vitamin D in the human body. It's challenging to determine how much sunlight we need to get enough vitamin D because it depends on many factors, including age, latitude, season, and time of day [3]. Apart from skin, acute UVR ocular exposure can promote photokeratitis or snow blindness, and chronic overexposure can increase the risk of cataracts [4]. Several reviews focus on the effects of UVR on other organs rather than simply on the skin [5-7]. This minireview will highlight how UV affects energy metabolism in mice with metabolic diseases and its underlying mechanisms.

## 2. Metabolic syndrome

Metabolic Syndrome is a collection of metabolic dysfunctions such as dyslipidemia, hypertension, hyperglycemia, abdominal/central obesity, and insulin resistance. Summer has a lower prevalence of metabolic syndrome than winter, which is the reverse of UV intensity. A recent human study showed that lower serum 25(OH)D was associated with an increased risk of metabolic syndrome [8]. Although 10-20 g of vitamin D per day can reduce all-cause and cancer mortality in middle-aged and older persons, there was no evidence that supplementation or vitamin D could affect cardiovascular disease, obesity, or glucose metabolism [9]. A recent mouse experiment discovered chronic UVR suppressed obesity and type 2 diabetes in high-fat diet-fed mice, characterized by weight gain, impaired glucose and insulin tolerance, fatty livers, and gonadal fat deposition [10]. Still, vitamin D supplementation did not replicate these effects. Further, topical nitric oxide (NO) donors to the dorsal skin replicated UV effects on mice, and NO scavengers prevented the positive impact of the UV [10]. Based on these findings, UVR (sunlight exposure) may effectively prevent the development of obesity and metabolic syndrome through mechanisms that depend on UVR-induced NO rather than vitamin D.

## 3. Hypertension and cardiovascular diseases

Hypertension is a significant risk factor for cardiovascular and cerebrovascular diseases, which are major causes of death and disability. The prevalence of hypertension and cardiovascular disease (CVD) increases with latitude [11] and is higher in the winter than in summer [12], which is the opposite of UV intensity. The intensity of UVR from the sun diminishes over the winter and with increasing latitude. A growing amount of research indicates that exposure to sunshine has a protective impact on blood pressure and cardiovascular diseases [13]. Inadequate exposure to sun also increases the risk of acquiring heart disease [14]. Although lower 25-hydroxyvitamin D concentration as an indicator of vitamin D status is linked to increased hypertension and the incidence of cardiovascular diseases [15], oral vitamin D supplementation does not decrease blood pressure or the risk of cardiovascular disease [16], indicating that vitamin D may not be a mediator of beneficial effects of sun exposure.

NO is a significant endogenous vasodilator produced by the vascular endothelium [17], produced when one of the three nitric oxide synthase (NOS) enzymes in the NOS family reacts with L-arginine [18]. It is generally acknowledged that NO-related compounds such as nitrate, nitrite, and RSNOs serve as significant NO storage forms [19, 20]. Large amounts of nitrogen oxides, mainly nitrate, are stored in the skin [21]. Inducible nitric oxide synthase (iNOS), an enzyme that converts L-arginine into NO, is increased in human skin 8–10 hours after UV exposure [22]. However, Paunel *et al.* [23] showed that the synthesis of NO due to photodecomposition of nitrite and S-nitrosothiols (RSNOs) was at its peak in 20 minutes after UVA exposure to *ex vivo* full-thickness human skin, which coincides with results from the randomized controlled trial that UVA exposure to

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the skin of normotensive human volunteers decreased blood pressure and expanded blood vessels in a NOS-independent manner [24]. According to Feelisch et al., Solar UVA may release nitrogen oxides from skin storage into the bloodstream, lowering blood pressure and cardiovascular mortality [5]. Liu *et al.* [24] also showed that circulating nitrite increased as blood pressure dropped and plasma nitrate decreased after UVA exposure, indicating a possible mechanism for the skin's regulation of systemic NO bioactivity.

Even earlier, the photoactivity of RSNOs, N-nitrosamines (RNNOs), and nitrite in rat vascular tissue *in vitro* was demonstrated [25]. Rodriguez *et al.* [25] determined the NO release action spectra from RSNOs (310–340 nm) and nitrite (310–350 nm), which is overwhelmingly the UVA wavelength range. Ferguson and colleagues confirmed that chronic solar UV could reduce the number of atheromata in atherosclerosis-prone mice fed a diet high in sugar and fat [26]. Similar results have been shown that UVB prevents atherosclerosis in apolipoprotein E–deficient (Apoe−/−) mice with the mechanism of activating regulatory T cells [27]. UVB exposure upregulates IL-33 expression in keratinocytes and dermal fibroblasts [28], which has been demonstrated to protect against atherosclerosis [29], might be another possibility for atherosclerosis.

**4. Type 2 diabetes mellitus (T2DM)**

Lindqvist *et al.* demonstrated a dose-dependent inverse relationship between increased sun exposure and the frequency of T2DM [30]. A recent human study showed that lower serum 25(OH)D was associated with an increased risk of type 2 diabetes [31]. However, treatment with 4000 U vitamin D did not reduce the incidence of T2DM [32]. Meta-analysis research also discovered contradictory relationships between biochemically measured and genetically predicted changes in blood 25(OH)D with T2D risk [33]. A study on mice fed a high-fat diet together with long-term sub-erythemal UVR showed an increase in glucose tolerance and a decrease in insulin resistance, as well as a suppression of weight gain [10], but vitamin D supplementation did not replicate these effects. Meanwhile, insulin resistance was predisposed in inducible nitric oxide synthase (iNOS) knockout mice [34]. It has been shown that increased endothelial NOS (eNOS) activity protects the obesogenic consequences of a high-fat diet without increasing systemic insulin resistance, partly through promoting metabolic activity in adipose tissue in mice overexpressing eNOS mice [35]. In particular, NO may regulate insulin sensitivity and blood flow with needed nutrients to insulin-sensitive tissues, while NO suppression may induce insulin resistance [36]. Based on these findings, UVR may effectively prevent the development of T2DM through mechanisms that depend on UVR-induced NO.

**5. Obesity**

Obesity is a chronic disease caused by an imbalanced energy metabolic balance, excessive energy acquisition, and insufficient energy consumption. Brown adipose tissue (BAT), which has a high number of mitochondria, can enhance energy expenditure and produce heat [37], and boosting its amount and function could be a safe and effective obesity therapy [35]. Previous research has demonstrated that eNOS-derived NO promotes mitochondrial biogenesis, as eNOS-deficient mice have fewer mitochondria in skeletal muscle and adipose tissue [38, 39]. In contrast to WT mice, Kanuri *et al.* [34] discovered that iNOS KO mice had vastly increased body weight and fat content but reduced respiratory exchange ratio (RER), the volume of carbon dioxide (VCO2), and heat generation. Similarly, UV irradiation prevented obesity-induced whitening in interscapular BAT in a nitric oxide-dependent manner. [40]. Ferguson *et al.* [26] also found that chronic UV reduced weight gain and fat mass while increasing UCP-1 in BAT with no changes in food consumption, distance traveled, or energy expenditure. Allemann *et al.*[41] reported that UVR (alone) did not significantly affect food intake and adiposity. However, Parikh *et al.* [42] found that prolonged UVB exposure (50 mJ/cm$^2$) increased food-seeking behavior and food intake exclusively in mice and human men via p53-mediated ghrelin. This process is inhibited in females due to estrogen interfering with UV-induced p53. Through a skin-

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brain-gonadal axis, the sex differences in UV effects are also found in UV-induced sexual behavior [43].

**6. Nonalcoholic fatty liver disease (NAFLD)**

NAFLD is a set of liver disorders that impact people who drink little to no alcohol and have an excess of fat stored in liver cells. Obesity, metabolic syndrome, and other fat-related diseases are all linked to NAFLD. Recent studies have revealed that UV reduced lipid accumulation in the liver in high-fat fed mice [26, 44] and topical S-nitroso-N-acetyl-penicillamine (SNAP, nitric oxide donor) suppressed liver pathology, and 2-(4-carboxy-phenyl)-4,4,5,5-tetramethylimidazoline-1-oxyl-3-oxide (cPTIO) antagonized the effects of UVR [10], suggesting that UVR-induced NO may have a significant impact on the progression of NAFLD. Altered glucose and lipid homeostasis in liver and adipose tissue also are found in inducible NOS knockout [34]. The deficiency of eNOS with NO levels reduced liver steatosis by controlling the blood flow to the hepatic tissue in eNOS-knockout mice [45]. Sheldon *et al.* also demonstrated that in obese rats, chemical eNOS inhibitors reduced hepatic mitochondrial respiration and increased hepatic triacylglycerol buildup [46]. Supplementing with vitamin D also reduced the severity of NAFLD and suppressed circulating TNF-$\alpha$ levels, a critical factor in the development of NAFLD, in mice fed a high-fat diet [10]. Since UVR did not reduce serum TNF-$\alpha$ levels, dietary vitamin D and UVR may have different mechanisms for working to reduce NAFLD.

**7. Discussion**

UV is widely known to be hazardous to skin health, like photoaging and skin cancer, and be beneficial to synthesize vitamins D. Numerous recent studies have demonstrated that UV exposure benefits in the suppression of metabolic disorders like metabolic syndrome, hypertension, diabetes, and obesity as well as the decrease of all-cause mortality. In addition, it has been shown that low serum 25(OH)D levels could increase the risk of these metabolic diseases. Because UV light is responsible for more than 80% of the body's vitamin D production and serum 25(OH)D indicates vitamin D levels in the whole body, vitamin D is considered the mediator through which UV affects metabolic diseases. However, interventional increases in serum 25(OH)D could not regenerate the effects of UV in human and mice experiments, suggesting that UV effects are independent of vitamin D . However, low serum vitamin D itself might be a sign of insufficient sunlight exposure and unhealthiness, and by monitoring its level, it is more beneficial to prevent the development of chronic metabolic diseases. Currently, the systemic effects of NO are widely investigated rather than vitamin D. The skin has abundant NO storage forms that UVA can quickly photolyze. In the skin, erythema, edema, and melanogenesis are primarily influenced by NO with UV exposure [21]. Interventional increase or decrease of NO in mice experiments reproduce or reduce the effect of UV on metabolic disease, indicating the mechanism of UV effect on NO.

It is well-recognized that vitamin D contributes to calcium homeostasis and good bone health [47]. In addition, UV-induced vitamin D is also essential for colorectal cancer [48] and respiratory tract infections [49]. Karapiperis *et al.* found that UV radiation was more significantly related to incidence rates than mobility in COVID-19 [50]. Low 25(OH)D serum levels are associated with rising mortality in both the general population and in chronic kidney disease (CKD) and end-stage kidney disease (ESKD) patients [51]. Krause *et al.* reported that UVB irradiation of the skin was preferable to oral treatment for maintaining their vitamin D level [52]. Additionally, chronic exposure to UV light activates p53 in keratinocytes, which then boosts β-endorphin signaling at opioid receptors and results in sun-addiction behavior [53]

In conclusion, although UV causes skin cancer, its advantages for chronic metabolic diseases are not negligible. For the maintenance of a healthy balance, adequate UV exposure is crucial. A better comprehension of the systemic mechanisms governing UV impact

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will contribute to lowering the risk of chronic metabolic disorders, extending life, and even improving quality of life.

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