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Short Communication

Prospective Mechanism for the Inhibition of Skin Cancer in Experimentally Induced Type 1 Diabetes Mellitus

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Abstract

Background: A previous study examining the effects of ultraviolet radiation (UVR) on experimentally induced Type 1 diabetic skin demonstrated inhibition of skin cancer compared with the well-documented carcinogenic effects of ultraviolet light radiation (UVR) on normal skin. Using hairless SKH-hr2 mice, Type 1 diabetes (T1D) and Type 2 diabetes (T2D) were induced by administration of 30 and 20 mg/kg streptozocin, respectively. Mice were then exposed to UVR three times weekly for six months. Remarkably, T1D mice did not develop skin squamous cell carcinoma or pigmented nevi, in contrast to normal and T2D skin. **Methods:** Metabolic parameters were quantified at specific time intervals. Navigation dividers were used to measure differences relative to the published curves, and a proportional equation was applied based on standard x-axis values to calculate metabolic parameters and, consequently, their differences over time. **Results:** A significant increase (4.8-fold) in hyperglycemia was observed at month five, accompanied by a marked decrease in glutathione (indicating redox imbalance); a non-significant increase in oxidative stress (OS) in the stratum corneum, a non-living tissue, suggesting that the free-radical burden was predominantly of endogenous mitochondrial origin, a reduction in body weight of 23% at three months and 44% at six months, and complete inhibition of skin cancer in the T1D animals. **Conclusions:** Given the metabolic complexity of the diabetic phenotype and its established link to cancer, application of Occam's Razor suggests a parsimonious explanation. Extreme hyperglycemia leads to elevated levels of reduced oxidized nicotinamide adenine dinucleotide (NAD^+) and reduced flavin adenine dinucleotide ($FADH_2$) cofactors, increasing reactive oxygen species (ROS) generation. This, in turn, elevates OS due to glutathione depletion related to nicotinamide adenine dinucleotide (NADH)/NAD+ redox imbalance, and alters the proton motive force across the inner mitochondrial membrane, essentially uncoupling oxidation from phosphorylation. The resulting depletion of adenosine triphosphate (ATP) the energy available to sustain normal cellular growth and to support the cancer phenotype. Caloric restriction (CR) is known to inhibit neoplasia. The profound loss of body weight in T1D mice reflects a state metabolically equivalent to CR and is proposed as the mechanism underlying the observed inhibition of UVR-induced skin cancer in experimental T1D.

Keywords: Type 1 diabetes; Type 2 diabetes; UVR-induced skin cancer; caloric restriction; oxidative stress; reactive oxygen species; redox imbalance

1. Introduction

Globally, Diabetes Mellitus (DM), of which about 95% is Type 2 (T2D), is the sixth leading cause of death [1]. Cancer is the second leading cause of death in the U.S. [2] and globally, as determined for 36 cancers in 185 countries in the Global Burden of Disease Study [3]. The global economic and societal burden of cancer is great and accounted for 1.8% of the Gross Domestic Product (GDP) of the U.S. in 2017 and 1.0% of GDP in the European Union [4]. The global burden of skin cancer has dramatically increased from 1990–2017, with a 310% increase in squamous cell carcinoma (SCC), a 161% increase in melanoma, and a 77% increase in basal cell carcinoma (BCC) [5]. Globally, diabetes and cancer have emerged as serious threats to public health, although their associations have been less intensively studied. That is the intent of this report.

2. Materials and Methods

A previous study employed hairless SKH-hr2 mice [6]. Type 1 diabetes (T1D) and T2D were induced by administration of daily injections for five consecutive days of 30 and 20 mg/kg, streptozocin, respectively, and subjected to ultraviolet light radiation (UVR) three times weekly for six months. Solar-simulated UVR (289–499 nm) was administered from a 1000 W Xenon lamp and filtered through appropriate filters for these wavelengths. The dose, measured in Minimal Erythemal Dose (MED), was 0.75 MED (4.5 s) in the first week and increased by 25% a week until stabilization at 3 MED (18 s). Quantitation of metabolic parameters of that study, at specific time intervals, was achieved through use of navigation dividers to precisely measure differences on the published curves, and using the standard values on the X-axis, to employ a proportional equation to quantitatively determine metabolic parameters and, thus, differences in those parameters.

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3. Results and Discussion

The metabolic parameters that were closely inspected for the prospective mechanism described here were derived from the previously uncharted examination of the effects of UVR on diabetic skin, in which details of experimental design; microscopic and photographic evaluations; histological evaluations; and statistical methods, including hierarchical clustering and K-means with respect to treatment and time, may be found [6]. Briefly, comprehensive measurements of photoaging and photocarcinogenesis in diabetic skin, compared to normal mice, were assessed. These assessments included hydration, trans-epidermal water loss, elasticity, and the oxidative stress and hydrophilic antioxidants in the stratum corneum. Type 1 diabetic skin showed heightened dehydration, thinning, and accelerated photoaging. However, ultraviolet (UV)-radiated T1D skin did not develop skin cancer or pigmented nevi.

When experimentally induced T1D was found to totally suppress non-melanoma skin cancer (NMSC), several metabolic parameters provided insight into potential mechanisms. High dose streptozocin induced an extremely elevated hyperglycemic condition (Table 1). Glucose levels were elevated by nearly 3-fold at one month post treatment with high dose streptozocin and nearly 5-fold at 5 months.

Table 1. Elevated hyperglycemia.

Glucose (mg/dL)			
	Baseline	1 Month	5 Months
Control	121.14	---------	119.43
Grp T2D	120.17	154.00	156.50
Grp T1D	127.29	344.14	575.67

T2D, Type 2 diabetes; T1D, Type 1 diabetes.

Glutathione is a major endogenous antioxidant and marker for redox balance. Under normal euglycemic conditions, oxidized glutathione is re-reduced by glutathione reductase in the presence of nicotinamide adenine dinucleotide phosphate (NADPH). In the current study, glutathione was significantly reduced in T1D skin. Oxidative stress (OS), based on reactive oxygen species (ROS) levels in the non-living stratum corneum (SC), while showing a mild increase (25%), was not statistically significantly elevated. The SC is a non-living layer of desquamated cells and the ROS measured in the SC could only result from the UVR (Fig. 1).

Nevertheless, the dramatic reduction in glutathione levels at three (55%) and five months (70%) (Table 2) indicates a severe redox imbalance and that elevated ROS levels occur endogenously in the mitochondria.

Body weights of the T1D animals exhibited extreme weight loss during the study, averaging 23%, compared to controls, at month three (29.36 g vs 37.99 g and 44% (24.40 g vs 43.86 g)) at six months (Table 3).

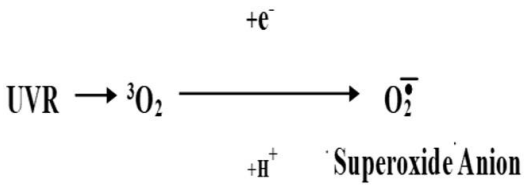


Fig. 1. Superoxide anion is the primary ROS formed by the univalent reduction of molecular oxygen (triplet state) via an energy exchange reaction upon absorption of energy from UVR. ROS, reactive oxygen species.

Table 2. Glutathione depletion in T1D skin.

Glutathione (nmol/mg)		
	3 Months	5 Months
Grp I, Control	2.86	2.62
Grp VI, T1D	1.28	0.80

Extreme glutathione depletion in T1D animals.

Table 3. Extreme body weight loss in T1D animals.

Body weight (g)			
	Baseline	3 Months	6 Months
Control	31.41	37.99	43.86
Group V, T2D	30.48	35.08	37.42
Group Vl, T1D	31.17	29.36	24.40

Extreme loss of body weight in T1D animals.

There are both exogenous and endogenous sources of ROS that provide the link between DM and Cancer [7], both of which are in play in this study showing suppression of experimental skin carcinogenesis in T1D skin [6]. UVR provides the exogenous source of ROS whereas the endogenous sources are related to metabolic dysfunction. An excess of ROS leads to OS that is characterized as an imbalance between the production and removal of these species (Redox imbalance). This imbalance results in insulin resistance/sensitivity, beta-cell dysfunction, and impaired glucose intolerance [8]. Furthermore, redox imbalance is an important event for cancer development, progression, and metastasis [9]. Endogenous excess ROS can arise from excessive calorie intake that may lead to a 5-10-fold increase in ROS resulting from normal respiratory chain regulation [10]. Nevertheless, ROS may act as a double-edged sword in the prevention and therapy of cancer [11].

Obesity and sedentary lifestyle are primary risk factors for T2D, as well as several types of cancer [12,13], including skin cancer [14,15]. These risk factors lead to hyperglycemia, insulin-resistant glucose metabolism, and, as noted, higher levels of ROS [16]. The latter, due to hyperglycemia, may cause mutations to mitochondrial DNA (mtDNA) that may result in increased levels of ROS and mitochondrial dysfunction, signal transduction, and dyslipidemia (Fig. 2). Dyslipidemia is a hall

mark of DM and may also affect signal transduction, beta-oxidation/Polyunsaturated fatty acids (PUFA), peroxidation, and chronic inflammation and immunity.

Obesity and lifestyle, hyperglycemia, Insulin resistance (T2D), insulin sensitivity (T1D), and dyslipidemia represent a cluster of metabolic high-risk factors for a number of metabolic disorders and have been given the term Metabolic Syndrome (MetS) [17]. Diabetes Mellitus is considered a metabolic disorder, and some cancers share the pathophysiology.

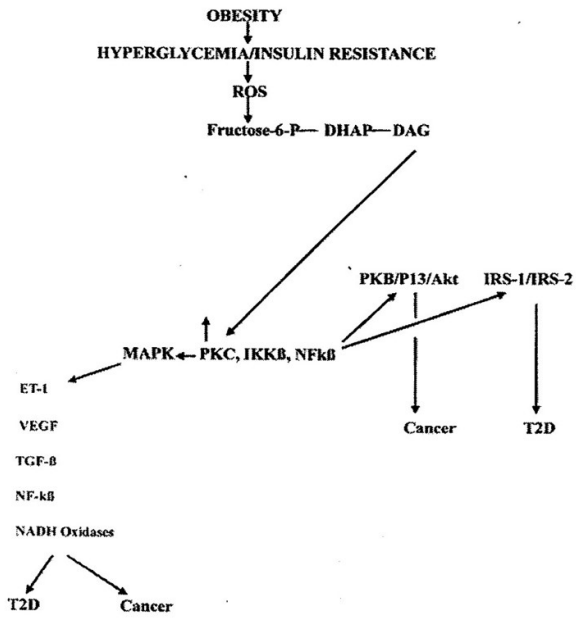


Fig. 2. Protein kinase C (PKC) signal transduction and the positive link of oxidative stress and ROS to T2D and Cancer. ROS induction of the Fructose-6-P shunt leads to the upregulation of dihydroxyacetone phosphate (DHAP) and diacylglycerol (DAG) that activates PKC, leading to a cascade of kinases, including Mitogen-Activated Protein Kinases (MAPK). Nicotinamide adenine dinucleotide (NADH) oxidase, a major source of ROS in T2D and redox imbalance. IKKβ, IκB kinase beta; NF-κB, Nuclear Factor-kappa-β; PKB/P13/Akt, protein kinase B/phosphoinositide 3/protein kinase B; IRS, insulin receptor substrate; ET-1, endothelin-1; VEGF, vascular endothelial growth factor; TGF-β, transforming growth factor beta.

Activation of the PKC pathway results in upregulation of Endothelin-1 (ET-1) a Vaso-constrictor; Vascular Endothelial Growth Factor (VEGF), a mediator of certain T2D conditions, and promotes lung metastasis; Transforming Growth Factor-β (TGF-β), a cytokine that has a pro-inflammatory action that is important in host immunity; Nuclear Factor-kappa-β (NF-kβ), a binding protein that is a central factor to the inflammatory response and, is thus, a major participant in innate and adaptive immune responses, and regulates the ability of both preneoplastic and malignant cells to avert apoptosis-mediated tumor surveillance; and nicotinamide adenine dinucleotide phosphate oxidase (NADPH Oxidase), a major source of ROS in T2D and redox imbalance. One isoform, NADPH oxidase 5 (NOX5), is over-expressed in melanoma and several other cancers [14]. The upregulation of these factors leads to expression of both the T2D and cancer phenotypes. In addition, the activation of the PKC signal transduction pathway results in upregulation of NF-kβ that suppresses downstream protein kinase B (PKB), phosphoinositide 3 (P13/Akt) and Insulin Receptor Substrate (IRS-1 and 2), the former IRS-1, decreases insulin stimulated glucose transport, glucose uptake, and subsequent signaling pathways that contribute to the development of T2D.

Mechanistically, PKC signal transduction must be considered in the low-dose Streptozocin induced T2D, as mice presented with high grade, well differentiated, invasive squamous cell carcinoma (SCC) and high grade, well differentiated non-invasive SCC, along with dysplasia. PKC signal transduction may be eliminated as a mechanism in T1D skin as tumor occurrence was totally suppressed. In the latter case, other pathways of glucose metabolism in hyperglycemia must be examined.

Some molecular markers have been shown to be associated with the development of SCC. The bark extract of *Pinus maritima* (PBE), rich in polyphenols, was examined for skin cancer prevention [18]. It was shown that when experimental animals were exposed to UVR, or in combination with 7,12-dimethylbenz[a]anthracene, a significant decrease in the number of tumor bearing animals and the number of tumors per animal was observed in the PBE treated group. In an effort to understand the events occurring that inhibited SCC induction, an immunochemical analysis revealed that the expression of P-glycoprotein, multi-drug resistance associated protein (MRP), and glucose (GLUT-1) transporters in SCC, SCC adjacent area, and normal skin tissues were expressed in all tissues, although expression of all transporters was notably higher in tumor and tumor adjacent areas [19]. Type 1 diabetic skin did not develop any SCC or pigmented nevi.

Early physicians recognized that food quantity could influence cancer and advocated a starvation diet ("*fames cura*") as treatment [20,21]. This treatment led to unacceptable results, and the end result was mortality equivalent to other treatment modalities. Nevertheless, many earlier studies examined the growth inhibiting effects of undernourishment on the development of new and existing cancers [22]. Tannenbaum [23] examined the dependence of the genesis of chemically induced skin cancers on the caloric intake during different stages of carcinogenesis. More recently, Colman and colleagues [24] found that a 30% caloric restriction lowered the incidence of neoplasia by 50% in rhesus monkeys, as compared to their non-calorically restricted cohorts.

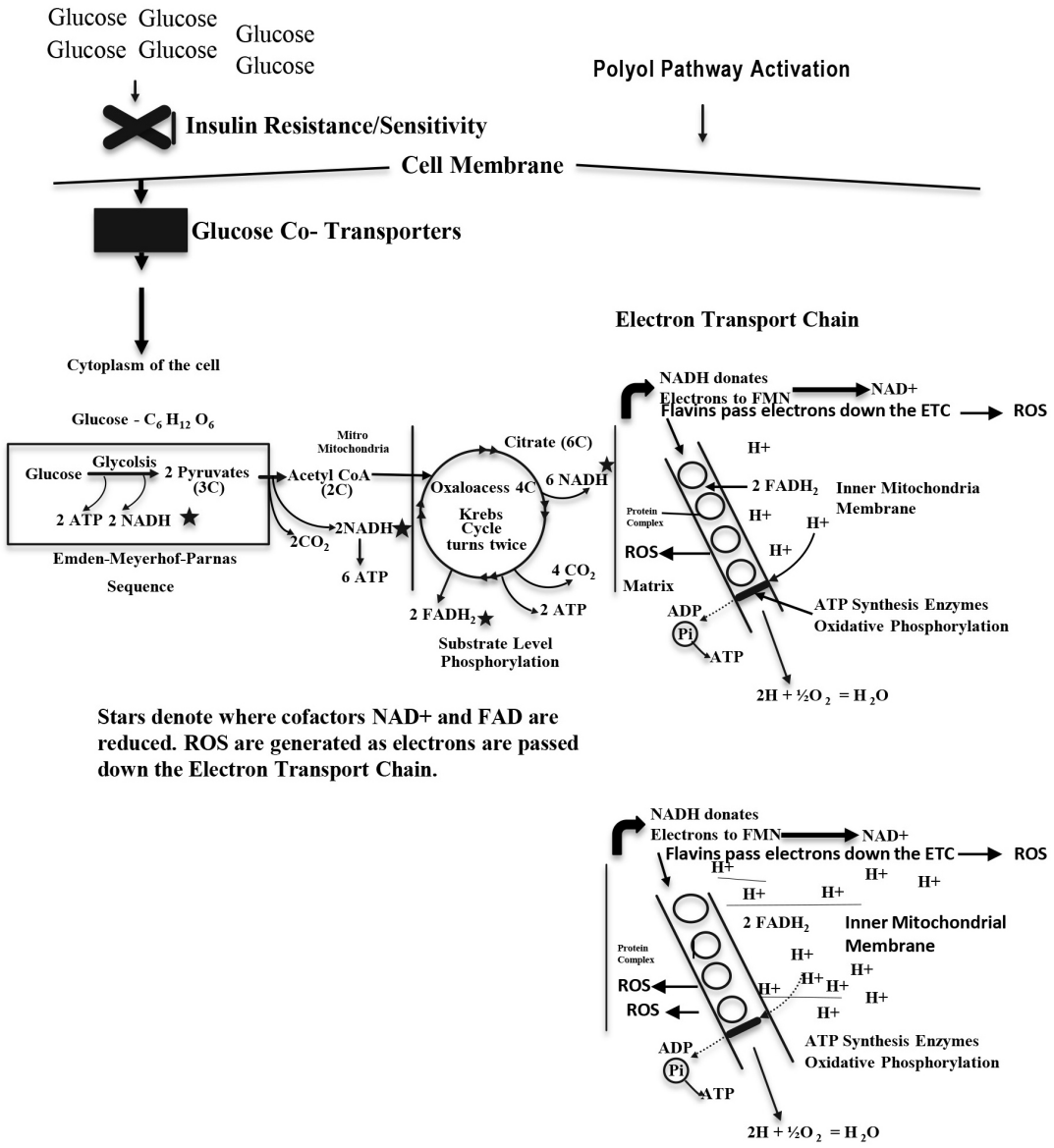


Fig. 3. Hyperglycemic, insulin-resistant/sensitive glucose metabolism. Stars (★) denote sites where cofactors oxidized nicotinamide adenine dinucleotide (NAD^+) and flavin adenine dinucleotide (FAD) are reduced. ROS are formed as electrons are passed down the electron transport chain. The lower diagram depicts what occurs under extreme hyperglycemic conditions when proton levels reach a high concentration and flow across the inner membrane without producing adenosine triphosphate (ATP).

Under the extreme hyperglycemic conditions in the study, a proton (H+) imbalance of the inner membrane changes the proton motive force (Δp) and allows protons to move across the membrane rather than through the channel protein to drive adenosine triphosphate (ATP) synthase. This results in loss of ATP yield, in effect, a physiological uncoupling of phosphorylation from oxidation (Fig. 3). Considering the extreme hyperglycemic condition observed in high dose streptozocin (T1D) skin and the extreme weight loss in these treated animals, the complete suppression of carcinogenesis may be related to limitation of metabolic fuel (ATP) to support normal cell growth and development and the cancer phenotype.

Regarding tumor suppression, our attention has been alerted to the Two-sample Mendelian study of Type 1 diabetes and the risk for 22 site-specific cancers [25]. The risks for some major cancer sites increased, some were not causally associated, whereas the risk for nonmelanoma skin cancer decreased. The authors concluded that the underlying mechanisms are multifaceted, involving inflammation, immune dysregulation, altered genetic foci, and metabolic disturbances, e.g., hyperglycemia and insulin related pathways. It must be recognized that humans suffering from Type 1 diabetes will be undergoing various therapies that will affect the response. For example, it has been reported that patients receiving Metformin have a decreased risk for

developing NMSC. The Mendelian study involved only European populations. Conversely, an Asian study of diabetic patients reported an increased risk for skin cancer [26]. Clearly, individual patients must be followed carefully as the clinical response will depend upon variables of ethnicity and therapy. Patients should be closely monitored for dermatological changes that require early treatment.

Limitations and Strengths of the Current Study

A limitation of the study is, as the title indicates, prospective and is based upon indirect evidence of a metabolic dysfunction of mitochondrial ATP formation that results in the metabolic equivalent of caloric restriction that suppresses skin cancer in Type 1 diabetic skin. The failure to measure food intake in the original study leaves the possibility that caloric restriction could have resulted from toxicity of Streptozocin or a self- imposed caloric restriction of the animals. However, the strength of the current study is that it is based on quantitative measurement of metabolic parameters (severe hyperglycemia, redox imbalance, and severe body weight loss) that logically indicate that the observed body weight loss is a result of ROS-induced proton motive force that diverts protons from normal channel proteins to drive ATP synthase and ATP yields become limited and cannot support normal cell growth, development, and the cancer phenotype. Supporting evidence could be obtained by measurement of Phosphorylation/Oxidation ratios (P/O ratios) from mitochondria of T1D epidermis at the designated time intervals and compared to controls.

4. Conclusions

Eliminating the Fructose-6-P shunt and the PKC signal transduction pathway involvement for T1D in the current study leaves the second pathway by which glucose is metabolized and produces ROS, i.e., normal glycolysis. As noted, under euglycemic conditions upwards of 10% of the oxygen consumed in oxidation of foodstuffs may be lost as ROS as the electrons are transferred from the reduced cofactors NADH and reduced flavin adenine dinucleotide ($FADH_2$). Under the extreme hyperglycemic conditions in the study, a proton (H^+) imbalance of the inner membrane changes the proton motive force (Δp) of the membrane and allows protons to move across the membrane rather than through the channel protein to drive ATP synthase. This results in loss of ATP yield, in effect, a physiological uncoupling. These data demonstrate a dramatic elevation of hyperglycemia; a small, but statistically insignificant, increase in OS in non-living stratum corneum, indicating that ROS levels were from endogenous origin; and a drastic loss of body weight that provides insight into the mechanism by which experimentally induced T1D suppresses UVR-induced skin cancer.

Abbreviations

CR, caloric restriction; OS, oxidative stress; ROS, reactive oxygen species; T1D, Type 1 diabetes; T2D, Type 2 Diabetes; DM, Diabetes Mellitus; UVR, ultraviolet light radiation; MED, Minimal Erythemal Dose; NMSC, non-melanoma skin cancer.

Availability of Data and Materials

All data reported in this paper will also be shared by the lead contact upon request.

Author Contributions

HSB designed the research study and performed the quantifications. MCR performed the original research from which the current study is based and provided the original data for quantitation. HSB and MCR contributed to editorial changes in the manuscript. HSB and MCR read and approved the final manuscript. HSB and MCR have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

Not applicable. This study was a secondary analysis of previously published animal data. No new animal experiments were performed. The published study has obtained approval from the relevant ethics committee.

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Conflicts of Interest

The authors declare no conflicts of interest.

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