



Contents lists available at ScienceDirect

# Diabetes & Metabolic Syndrome: Clinical Research & Reviews

journal homepage: [www.elsevier.com/locate/dsx](http://www.elsevier.com/locate/dsx)



## Brief communication

# Evidence childhood epidemics of type 1 and type 2 diabetes are opposite extremes of an immune spectrum disorder induced by immune stimulants. Role of race and associated cortisol activity as a major determining factor of the type of diabetes

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## ARTICLE INFO

Article history:  
Available online xxx

Keywords:  
Type 2 diabetes  
Type 1 diabetes  
Vaccine  
Cortisol  
Immunization

## ABSTRACT

**Design:** Prior publications have provided evidence that type 1 and type 2 diabetes are opposite extremes of an immune spectrum disorder.

**Method:** The risk of type 1 diabetes was correlated with the risk of type 2 diabetes in different races.

**Results:** Races with high risk of developing type 2 diabetes have an decreased risk of developing type 1 diabetes (correlation coefficients  $-0.5$  or  $-0.85$  depending on age,  $p < 0.05$ ). Maori, American Indians, Asians, Australian Aboriginals and US Black children have a higher risk of developing type 2 diabetes but a low risk of type 1 diabetes compared to White children.

**Conclusion:** Population data is consistent with type 1 and type 2 diabetes being opposite ends of an immune spectrum disorder.

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## 1. Research design and method

There is an global epidemic in children of both type 1 diabetes, an autoimmune disease, and type 2 diabetes/metabolic syndrome, disorders which have been previously suggested to be opposite ends of an immune spectrum disorder [1,2]. Epidemiology data was sought comparing the incidence of both type 1 and type 2 diabetes in different races of children to determine if there was a consistent negative correlation between the risk of type 1 and type 2 diabetes. Medline was searched to find papers on the incidence of both type 1 and type 2 diabetes in children. Key search words included type 1 diabetes, type 2 diabetes, incidence, children, adolescents. Abstracts and papers were read to find papers that contained incidence of both type 1 and type 2 diabetes. Incidence data was matched by age to allow more accurate comparisons. Statistics were performed using software Statistica, Stat Soft, Tulsa, OK. Pearson Product-Moment Correlation was used to look for a possible correlation between the risk of type 1 diabetes and a risk of type 2 diabetes.

## 2. Results

Three papers were found containing studies measuring the incidence of both type 1 and type 2 diabetes in children where the

results were recorded by race. The studies included subjects from New Zealand [3] where the incidence was compared between Whites and Maoris, Australia [4] where the incidence was compared between Whites and Aboriginals, and the US [5] where the incidence was compared between Whites, African Americans, Asians, Hispanics, and American Indians.

Table 1 compares the incidence of type 2 diabetes and type 1 diabetes. The incidence of type 2 diabetes showed a negative correlation with the incidence of type 1 diabetes. The correlation coefficient was  $-0.85$  ( $p < 0.05$ ) in children aged 10–19 and  $-0.5$  in children age 0–14 ( $p < 0.05$ ).

## 3. Conclusions

There are simultaneous epidemics of type 1 and type 2 diabetes/metabolic syndrome in children and a single cause is likely. Evidence has been presented that that type 1 diabetes and type 2 diabetes/metabolic syndrome are opposite extremes of an immune mediated disorder induced by a rise in iatrogenic immune stimulation [1,2]. The current data further supports previously published evidence that type 1 and type 2 diabetes are opposite ends of an immune spectrum disorder.

The current data demonstrated a negative correlation between risk of type 1 diabetes and type 2 diabetes. An increased risk of type 2 diabetes is associated with a reduced risk of developing type 1 diabetes. The protective effect of type 2 diabetes is not explained by traditional theories of diabetes causation. First

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**Table 1**  
Incidence of type 1 diabetes versus type 2 diabetes by race.

Age 10–19			Age 0–14		
Race	Yearly incidence/100,000		Race	Yearly incidence/100,000	
	Type 1	Type 2		Type 1	Type 2
Indians	5.94	36.96	Indians	6.1	9.39
Asians	7.44	17.1	Asians	7.37	4.8
Hispanics	14.98	12.79	Hispanics	14.11	3.39
Blacks	15.34	20.98	Blacks	15.18	8.61
US Whites	24.1	4.25	Whites	26.79	1.16
Aboriginals	15.1	12.7	Maori	5.6	1.78
Australian Whites	21.4	2.1	Non-Maori	21.7	0.39
Correlation coefficient –0.85			Correlation coefficient –0.5		
$p < 0.05$			$p < 0.05$		

The incidence of type 1 and type 2 diabetes in different races based on age.

type 1 diabetes may result from slowly progressive autoimmunity and may initially present as type 2 diabetes. In these cases there is a positive correlation between the risk of type 1 and type 2 diabetes. Second, many type 2 diabetics have insulin resistance. A pre-type 1 diabetic with declining insulin secretion should reach a level of insufficient insulin secretion sooner if the person is insulin resistant. Again there should be a positive correlation between the risk of type 1 and type 2 diabetes.

It has previously been proposed that the epidemics of type 1 diabetes and type 2 diabetes/metabolic syndrome are caused by an increase in iatrogenic immune stimulation, the increase in vaccines [1,2]. The mechanism by which general immune stimulation can lead to specific autoimmune diseases such as type 1 diabetes has been presented [6]. Certain individuals, such as Japanese children, secrete high levels of cortisol following immune stimulation which protects them from developing autoimmune diseases but increases their risk of type 2 diabetes/metabolic syndrome. Previous publication have reviewed the evidence linking type 2 diabetes/metabolic syndrome with cortisol release [1,2]. Small increases in cortisol activity are associated with metabolic disturbances including increased glucose levels, insulin resistance, increased blood pressure, obesity and hyperlipidemia. Metabolic syndrome and related type 2 diabetes resemble mild Cushingoid syndrome [7] and several have suggested that metabolic syndrome is caused by increased cortisol activity [8]. Decreases in cortisol production caused by adrenalectomy leads to increased rates of type 1 diabetes in mice [9]. Cortisol activity is also consistent with the phenotypes of diabetes. Type 2 diabetics tend to be obese, consistent with excessive cortisol activity, while type 1 diabetics tend to be slender, consistent with low cortisol activity.

The racial differences in the incidences of type 2 and type 1 diabetes in this paper can be explained by cortisol activity. Asians [10] and Australian Aboriginals [11] have been reported to have increased cortisol production compared to Whites while American Indians may have increased sensitivity to cortisol [12]. Variation in genes coding for MHC have been used to explain the racial differences in type 1 diabetes but they do not explain the racial differences in type 2 diabetes. Nor does the variation in MHC explain the differences in BMI (body mass index) between type 1 and type 2 diabetics. However, genes affecting cortisol activity have been linked to the risk of type 2 diabetes [13] and could alter the risk of type 1 diabetes as well [9].

The most productive way of stopping the simultaneous epidemics of type 1 diabetes and type 2 diabetes/metabolic syndrome is to prevent exposure to unnecessary immunological challenges. Previous papers have provided evidence that a single agent, BCG immunization, was associated with an increased risk of type 1 diabetes (an autoimmune disease) or type 2 diabetes

depending on race [1]. BCG vaccination of school age Europeans was associated with an increased risk of developing type 1 diabetes while immunization of school age Japanese children was associated with an increased risk of type 2 diabetes. The discontinuation of BCG immunization in Japan was followed by a rapid decrease in type 2 diabetes. Similar declines in the incidence of type 1 diabetes in Denmark occurred following the discontinuation of the BCG vaccine [14]. Such steps will likely be fruitful in pets as well. Recently an epidemic of obesity has been recorded in primarily grass fed horses [15], creatures that are heavily immunized and where the etiology of the epidemic cannot be confused by the presence of fast food, sodas, television and video games.

## Disclosure

The author is an employee and shareholder of Classen Immunotherapies, Inc. which holds patent applications and patents regarding testing vaccines for their ability to cause type 1 and type 2 diabetes.

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