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

John B. Classen*
Classen Immunotherapies, Inc., 6517 Montrose Avenue, Baltimore, MD 21212, United States

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, 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.

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 an 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
type 1 diabetes may result from slowly progressive autoimmune immunity 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.

**References**


[14] Classen JB, Classen DC. Clustering of cases of IDDM occurring 2–4 years after vaccination is consistent with clustering after infections and progression to IDDM in autoantibody positive individuals. JPEM 2003;16:495–508.