Can residential pesticides be one of the risk factors for developing diabetes in infants?

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Abstract: Exposure to pesticides may result in abnormal glucose metabolism, increasing risk of diabetes. The study was to investigate the relationship between maternal and infants residential pesticides exposure and the onset of diabetes in these infants. Both the diabetic infants and the control groups were recruited from the Outpatient Clinic for Diabetic children and Well Baby Clinic, Assiut University Children Hospital. The study included 72 infants divided into 2 groups study group (40 diabetic infants) and the control group included (32 infants). A structured questionnaire sheet was designed to collect data about mothers and their infants. The study showed that there was statistically significant difference considering the items of no consanguinity among parents of infants in the study group compared to those in the control group (52.5 % Vs. 40.6%, respectively P ≤ 0.02.).The percentage frequency of maternal exposure to residential pesticides was significantly higher for the patients group in comparison to the control group. (65% Vs. 31.2%, respectively, P- value 0.0001).Similarly, the percentage frequency of the diabetic infants exposed to residential pesticides was significantly higher than that for the control group. (47% Vs. 28.1%, respectively, P – ≤ 0.01). Moreover, the percentage frequency of exposure of infants to residential pesticides per week was significantly higher among the study group in comparison to their partners in the control group. P- ≤ 0.0001. In conclusion: Exposure of infants to residential pesticides both during pregnancy and after birth may be considered as a risk factor for developing diabetes mellitus in these infants. The study recommended that, further studies are needed to study the relationship of exposure to residential pesticides for developing diabetes by using the biochemical markers to detect the level of metabolites of these pesticides in these populations.
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1. Introduction

The World Health Organization (WHO) estimated that the incidence of diabetes under age of 20 years of age 215,000, and about 1 in every 400 children and adolescents has type 1 diabetes. In 2007, diabetes was listed as the underlying cause on 71,382 death certificates and was listed as a contributing factor on an additional 160,022 death certificates. This means that diabetes contributed to a total of 231,404 deaths (American Diabetes Asssociation, 2011).

There is a growing evidence of the adverse impact of exposures to ambient and indoor air pollutants on fetal growth and early childhood neurodevelopment (Perera *et al*., 1999). Human and experimental studies showed that the fetus and infant are more sensitive than adults to many environmental toxicants, including environmental tobacco smoke (ETS), polycyclic aromatic hydrocarbons (PAHs), and residential pesticides such as diazinon and chlorpyrifos (Mott *et al*., 1994 and Whyatt and Perera, 1995).

Organophosphorus (OP) compounds are cholinesterase-inhibiting chemicals used as pesticides. Exposures to OPs cause a significant number of poisonings and deaths each year. Exposure to pesticides may result in abnormal glucose metabolism, increasing the risk of diabetes. One of the reported adverse effects in human exposure to OPs is hyperglycemia (Abdollahi, *et al*, 1995; Abdollahi, *et al*, 1995; Abdollahi, *et al*, 1996 and Shadnia, 2005).

In a cohort study of Australian outdoor workers, mortality from diabetes was elevated among those with high pesticide exposures compared with the general population (Beard *et al*., 2003). Studies of dioxin exposure (a contaminant of pesticides) are also suggestive of increased risk for type 2 diabetes, hyperglycemia, and hyperinsulinemia (Henriksen *et al*., 1997 & Longnecker *et al*., 2000 & Cranme *et al*., 2000). While many studies have examined the relationship between pesticides and diabetes (.Abdollahi *et al*., 2004 & Pournourmohammadi *et al*., 2005 & Abu-Basha EA *et al*., 1999), none have focused on type 1 diabetes. Most researches focused on agricultural or food pesticides and limited number focus on residential pesticides, hence the need for this study.

Aim of the study:

The aim of this study is to investigate the relationship between maternal and infants residential pesticides exposure and the onset of diabetes in these infants.

2. Subjects and Methods

Research Design:

Exploratory research design was used for this study.

Subjects:

Setting:

This study was conducted in Outpatient Clinic for Diabetic children and Well Baby Clinic, Assiut University Children Hospital for the study and control groups respectively.

Sample:

The study included 72 infants divided into 2 groups study and control groups; all infants with diabetes who attended the outpatient clinic within a year (the study group 40 infants). The control group (32 infants) included infant from the Well Baby Clinic, Assiut University Children Hospital.

Tools: A structured questionnaire sheet was designed by the researchers to collect data about mothers and their infants. It included the following data:

1. Biosocial data of infants as age, sex and birth order.
2. Biosocial data of mothers as name, age, educational level, occupation and residence.
3. Date of onset of disease, exposure to residential pesticides, duration of exposure, and types of pesticides used.
4. Pregnancy history regarding the exposure to residential pesticides during pregnancy (duration and types) and history of gestational diabetes.
5. Family history of diabetes, other siblings with diabetes, degree of consanguinity, family history of hyperlipidemia or hypertension and young age stroke

Methods:

1. A pilot study was carried out on 10 infants to test the clarity and applicability of the sheet and reliability of the Arabic version. The tool was relevant and applicable but some words have been modified. The reliability of the Arabic version was tested using Cronbach alpha test. Cronbach alpha coefficient of internal consistency was reported to be .0.85

2. Data collection:

An official permission was obtained from the Manager of Assiut University Children Hospital and written consent was obtained from mothers of the studied sample. All mothers were reassured that information obtained will be confidential and used only for the purpose of the study. Data was collected during the period from January 2010 to December 2010. Each mother was interviewed individually using the questionnaire sheet. The time taken for fulfilling the sheet ranged from 30 to 45 minutes depending on the response of the participants.

Data was gathered regarding mother's age, education, residence and working condition. then, they were asked about infant's age, sex and birth order. History of diabetes among infants' mothers was taken including: date of diagnosis, family history and degree of consanguinity. After that history of gestational diabetes and pregnancy semester of its diagnosis was taken. Data about exposure of the mothers of infants to residential pesticides during pregnancy as well as exposure of their infants to residential pesticides, frequency of its using and its types was recorded.

Analysis of Data

Personal computer (Pc) was used to store and analyze data and, to produce graphic presentation for some important results. Comparison of percentages: qualitative variables are expressed as percentages and compared among two groups using the chi square test. The same test is used for comparison of 2 or more groups. The Fischer's exact formula is used with small frequencies to avoid false significance (Munro, 1997). Comparison of means: the t-test is used for comparison of means. The output of t-value (probability of error) is obtained from the t-test. Correlation coefficient (r) was used to study the correlation between two quantitative variables (Munro, 1997).

-For each test level of significant (P) was considered as follows:

P> 0.05 → Insignificant
P< 0.05 → Significant
P< 0.01 → Highly significant
P< 0.001 → Very highly significant

Limitations of the study:

We used trade names of pesticides because the types of pesticides were not labeled on their package and no available information obtained.

3. Results:

This study included 72 infants (40 in the study group and 32 in the control group). The mean age of mothers' infant in study group was 34.02 ± 0.85

years while, the mean age of mothers' infant of the control group was 30.97 ± 5.14. No statistically significant differences were found as regards family history of premature stroke, hypertension and hyperlipidemia for both study and control groups.

Table 1 shows distribution of mothers' personal data for both study and control groups. No statistically significant differences were found regarding all items of mother's personal data (mother's age, education, residence and working condition) between both study and control groups.

Table 2 shows frequency distribution of infants' personal data in both study and control groups. As regards infant's age; the majority of infants in the study and control groups were belonging to the age group of 6 – 12 months, 90 % and 87.5%, respectively. Approximately half of infants in both study and control groups were female. About one-third of infants in the study and control groups were first birth order (32.5 % and 28.1%, respectively) with no statistically significant differences between the two studied groups regarding all previous items.

Table 3 shows frequency distribution of history of diabetes and gestational diabetes among infant's mothers in both study and control groups. No statistically significant difference was found between study and control groups as regards diabetic status of the mothers included in the study. Approximately one – quarter of infants' mothers in study group were diabetic compared to about one – fifth of infants' mothers in the control group (27.5% Vs. 21.9%, respectively).

Regarding duration of diagnosis of diabetes per year, it was found that more than half of infants' mothers' were recently diagnosed (less than 3 years ago) compared to 28.6 % in the control group. While, more than half of infants mothers' in the control group diagnosed from 3 to less than 10 years ago (57.1 % compared to 18.1 % in the study group). Moreover, no statistically significant differences were found between infants' mothers of study and control groups regarding having gestational diabetes and time of its diagnosis. The highest frequency of diagnosis of gestational diabetes was detected in the third trimester of pregnancy for both study as well as control groups (66.7% Vs. 75%, respectively).

Table 4 shows Comparison between the study and control groups related to frequency and degree of consanguinity. It was found that statistically significant difference was detected considering the items of no consanguinity among parents of infants in the study group than those in the control group (52.5 % Vs. 40.6%, respectively P ≤ 0.02.) Similarly, close consanguinity was found to be significantly lower among parents of infants in the study group compared to the control group (22.5% Vs. 43.5%, respectively).

Table 5 shows Frequency distribution of frequency of exposure to residential pesticides and its types among infants in the study and control groups. A statistically significant difference was found between the study and control groups with higher frequency of using residential pesticides during pregnancy in the study group (65% Vs. 31.2%, respectively P- ≤ 0.01).

As regards frequency of using residential pesticides, it was found that a statistically significant difference between the study and control groups was detected in using residential pesticides from 1- 2 times / day. While, no single mother in the control group used residential pesticides from 1- 2 times / day. On the other hand only 3.8% of the study group use residential pesticides 4 times per year compared to 60% in the control group. This difference proved to be statistically significant (P- ≤ 0.0001).

Regarding type of residential pesticides, the more frequently used residential pesticides was ants pesticides in the study group compared to 10% in the control group. While, the least frequent type of residential pesticides used by the mothers of the study group were liquid pesticides, Raid and Flit compared to 10 % for the control group, respectively. (P- ≤ 0.0001).

Table 6 shows comparison between the study and control groups as regards direct exposure of infants to residential pesticides, frequency of its exposure and its types. It was noticed that about half of infants exposed to residential pesticides in the study group compared to about one- quarter in the control group with statistically significant differences between the two groups (47% Vs. 28.1%, respectively, P – ≤ 0.01).

The percentage frequency of exposure of infants to residential pesticides per week was significantly higher among the study group in comparison to their partners in the control group. (P- ≤ 0.0001). According to types of residential pesticides used, it was found that percentage frequency of using Pyrosol was significantly higher than other types both for the control and patients groups (47.4% Vs. 55.6%, respectively).

Figure 1 shows Frequency of exposure of infants in the study group to residential pesticides. The more the frequency of exposure of infants to residential pesticides, the higher the number of infants developing diabetes.

Figure 2 shows Distribution of types of residential pesticides to which infants in the study group were exposed. The most frequent type of residential pesticides to which diabetic infants was exposed was Pyrosol followed by Raid and Pyrosol followed by Raid.

Table (1): Distribution of mothers' personal data in both study and control groups

Items	Study group N = 40		Control group N = 32		X^2	p- value
	No	%	No	%		
*Mother's age:						
20- > 30	9	22.5%	11	34.4%		
20- >40	24	60%	16	50%	1.26	0.533
40 and more	7	17.5%	5	15.6%		
Mean ± SD	34.02+0.85		30.97 ± 5.14			
*Mother's education:						
Illiterate	14	35%	10	31.2%		
Read and write	2	5%	0	0.0%		
Primary education	0	0.0%	0	0.0%	4.64	0.327
Preparatory education	0	0.0%	1	3.1%		
Secondary education	18	45%	12	37.5%		
Higher education	6	15%	9	28.1%		
* Residence						
Urban	24	60%	20	62.5%	0.455	0.500
Rural	16	40%	12	37.5%		
*Mother's working condition						
House wife	33	82.5%	28	87.5 %		
Working mother	7	17.5%	4	12.5%	0.066	0.80

Table (2): Frequency distribution of infants' personal data in both study and control groups

Items	Study group N = 40		Control group N = 32		X^2	p- value
	No	%	No	%		
Infant's age:						
Less than 6 months	4	10%	4	12.5%		
6- 12 months	36	90%	28	87.5%	0.002	0.97
Infants gender						
Male	18	45%	17	53.1%	0.201	0.654
Female	22	55%	15	46.9%		
-Birth order						
First	13	32.5%	9	28.1%		
Second	11	27.5%	6	18.8%		
Third	7	17.5%	7	21.9%	4.18	0.382
Fourth	2	5%	6	18.7%		
Mor e than fourth	7	17.5	4	12.5%		

Table (3): Frequency distribution of history of diabetes and gestational diabetes among infants' mothers in both study and control group

Items	Study group N = 40		Control group N = 32		X^2	p- value
	No	%	No	%		
*Mothers having diabetes						
Diabetic	11	27.5%	7	21.9%	0.68	0.40
Not	29	72.5%	25	78.1%		
*Duration of diagnosis of diabetes/year						
More than 3 years	6	54.5%	2	28.6%	32.65	0.0001***
3-10 years	2	18.1%	4	57.1%		
10 years and more	3	27.3%	1	14.3%		
-Gestational diabetes						
Yes	6	15%	4	12.5%	0.149	0.70
No	34	85%	28	87.5%		
- Diagnoses of gestational diabetes						
First trimester	0	0.0%	0	0.0%		
Second trimester	2	33.3%	1	25%	1.294	0.255
Third trimester	4	66.7%	3	75%		

Table (4): Comparison between the study and control groups related to frequency and degree of consanguinity

Items	Distribution percent				X^2	p- value
	Study group		Control group			
	No	%	No	%		
No consanguinity	21	52.5%	13	40.6%	8.32	0.02*
Close consanguinity	9	22.5	14	43.5%		
Remote consanguinity	4	10	5	15.9%		

Table (5): Maternal exposure to residential pesticides in both study and control groups.

Items	Study group N = 40		Control group N = 32		X^2	p- value
	No	%	No	%		
-Use of residential pesticides during pregnancy :-						
Yes	26	65%	10	31.2%	21.28	0.0001***
No	14	35%	22	68.8%		
-Frequency of using residential pesticides :-						
1-2 times / day	8	30.8%	0	0.0%		
Twice a week	4	15.4%	0	0.0%		
Once a week	2	7.7%	0	0.0%	115.6	0.0001***
Twice a year	7	26.9%	1	10%		
3 times per year	4	15.4%	3	30%		
4 times per year	1	3.8%	6	60%		
Type of residential pesticides						
Raid	3	11.5%	1	10%		
Pyrosol	7	26.9%	3	30%		
Powder for ants and cockroach	6	33%	1	10%	59.9	0.0001***
Raid and Pyrosol	2	7.7%	0	0.0%		
Kito	3	11.5%	0	0.0%		
Liquid pesticide	1	3.8%	1	10%		
Raid and Flit	1	3.8%	0	0.0%		
Flit and Pyrosol	3	11.5%	2	20%		
Ragon	0	0.0%	2	20%		

Table (6): Frequency distribution of frequency of exposure to residential pesticides and its types among infants in the study and control groups

Items	**Study group N = 40**		**Control group N = 32**		**X^2**	**p- value**
	No	**%**	**No**	**%**		
-Direct exposure of infants to residential pesticides						
• **Yes**	**19**	**47%**	**9**	**28.1%**	**6.95**	**0.01***
• **No**	**21**	**52.5%**	**23**	**71.9%**		
Frequency of exposure of infants to residential pesticides/week						
• **Once**	**1**	**5.3%**	**6**	**66.7%**		
• **Twice**	**1**	**5.3%**	**2**	**22.2%**		
• **3 times**	**3**	**15.8%**	**1**	**11.1%**	**64.71**	**0.0001*** **
• **4 times**	**3**	**15.8%**	**0**	**0.0%**		
• **5 times**	**5**	**26.3%**	**0**	**0.0t%**		
• **6 times**	**6**	**31.6%**	**0**	**0.0 %**		
Mean+ SD	**0.0625±0.3535**					
Types of residential pesticides						
• **1- Raid**	**3**	**15.8%**	**4**	**44.4%**		
• **2- Pyrosol**	**9**	**47.4%**	**5**	**55.6%**		
• **3-Raid and Pyrosol**	**4**	**21.1%**	**0**	**0.0%**	**50.88**	**0.0001*** **
• **4- Kito**	**1**	**5.3%**	**0**	**0.0%**		
• **5- Raid and Flit**	**1**	**5.3%**	**0**	**0.0%**		
• **6-Flit and Pyrosols**	**1**	**5.3%**	**0**	**0.0%**		

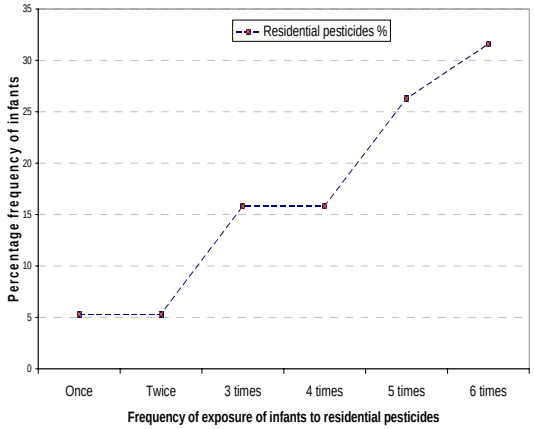

Figure (1): Frequency of exposure of infants in the study group to residential pesticides

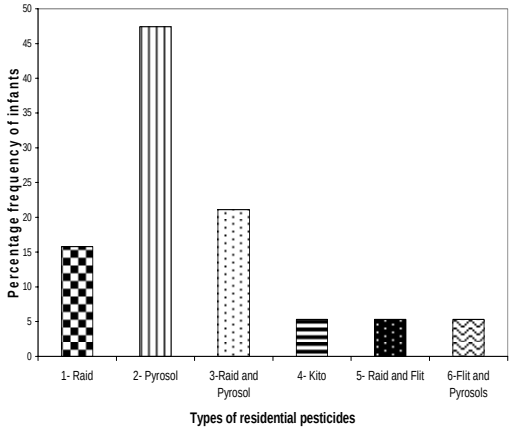

Figure (2): Distribution of types of residential pesticides to which infants in the study group were exposed**.**

4. Discussion

Type 1 diabetes incidence is now rising even in countries with historically low incidence, suggesting a catch-up phenomenon. (Gale 2002b). Levels of most persistent organic pollutants have declined recently in developed countries. In developing and some former Soviet countries, however, some persistent organic pollutants (like DDT) are still in use, and contamination due to open dumping is also a concern. Levels of some organochlorine pesticides (such as DDT) are now higher in people living in developing countries than in developed nations (Tanabe and Kunisue 2007). Perhaps contamination resulting from industrialization contributed to the rising incidence in many now-developed, high-incidence countries, and other countries, where contamination began later, are now "catching up."

Children with a parent or sibling who has type 1 diabetes are at increased risk for developing the condition. Children with two parents that have type 1 diabetes are at slightly higher risk. However, family history is not the primary factor in developing type 1 diabetes. Studies have shown that despite having identical genes, one identical twin may develop type 1 diabetes while the other does not. This indicates that there is an environmental factor that can increase the risk of having the condition, and not only genes. (National Diabetes Information Clearinghouse: National Diabetes Statistics 2011)

It was observed that more than half of infants in both study and control groups were living in urban areas (60% and 62.5%, respectively) as shown in table 1.This is in agreement with (Haynes *et al*. 2006) who found that type 1 diabetes incidence tends to be higher in more urban areas as compared to more rural or remote areas and also Arab, 1992 stated that there is distinct geographical difference in the prevalence of diabetes mellitus: 5.7% in urban areas, 4.1% in rural agricultural areas

Worldwide, the incidence of diabetes tends generally not to be differed between genders, this fact is cleared in our study which revealed that gender had no effect among the diabetic patients group as shown in table 2. This is in agreement with results of Mariatte *et al*., 2006 who demonstrated that the incidence of type 1 diabetes is rising in children of both genders.

Although it is well known that there is a relationship between the occurrence of type 1 diabetes and family history of diabetes, the present study noticed no statistically significant difference between the two groups with about one-fourth of mothers of infants in the study group were diabetic compared to only 21.9% in the control group. as shown in table 3. This is in contrast with Arab, (1992) who found that a high risk of Diabetes Mellitus (DM) is associated with family history of the disease, obesity, premature atherosclerosis and hypertension. This can be explained by the fact that there are two types of type 1 diabetes Type 1A, where antibodies attack and destroy the islet cells, which produce insulin in a child's pancreas. It is unclear why the immune system attacks the pancreas, but it is believed that the child must have the genetic predisposition (or ability) to develop Type 1, and it emerges following an environmental trigger like a virus. (Williams, 2011)

It is also explained by the results obtained from table 4 which showed that there was no consanguinity among fifty percents of infants in the study group.

In the present study, there is no statistically significant difference between the study and control groups regarding presence of gestational diabetes in infants mothers. While, approximately two- thirds of mothers in the study group used residential pesticides during pregnancy compared to about one –thirds of mothers of infants in the control group with highly statistically significant differences (P≤ 0.0001) as shown in table 5. This is in contrast to results reported by Saldana *et al*., 2007 who examined the association between pesticide use during pregnancy and gestational diabetes mellitus (GDM) among wives of licensed pesticide applicators and showed no association between residential pesticide exposure (applying pesticides in the home and garden during pregnancy) and Gestational Diabetes Mellitus (GDM).

Pesticides may also cause harm. Some can damage the environment and accumulate in ecosystems, depending on dose; some pesticides can cause a range of adverse effects on human health, including cancer, acute and chronic injury to the nervous system, lung damage, reproductive dysfunction, and possibly dysfunction of the endocrine and immune systems. (Saldana *et al*., 2007)

Our study found a statistically significant difference between the study and the control groups as regards infants' direct exposure to residential pesticides ($p \leq 0.01$) as shown in table 6. This could be explained by the fact that there is an environmental factor that can increase the risk of having diabetes, and not only genes. (National Diabetes Information Clearinghouse: National Diabetes Statistics 2011). This study also showed that there is a significant positive correlation between frequency of infants' exposure to residential pesticides and number of diabetic infants. (P- Value 0.0001). The more frequent the infants' exposure to residential pesticides, the more the susceptibility of infants to develop type 1 diabetes. This is in agreement with Daily, 2008 who reported that use of any of the pesticides studied for more than 100 days in a lifetime increased diabetes risk 17%. This study, also revealed that the most common type of residential pesticides used was Pyrosol followed by Raid and Pyrosol followed by Raid

Conclusion:

This study concluded that:

- Exposure of infants to residential pesticides both during pregnancy and after birth may be considered as a risk factor for developing diabetes mellitus in these infants.
- The most common type of residential pesticides to which these infants were exposed was Pyrosol followed by Raid and Pyrosol followed by Raid.

Recommendations:

- Educational program for pregnant women to avoid exposure to residential pesticides during all semesters of pregnancy.
- Educational program for mothers to avoid exposure of their infants to residential pesticides to their infants.
- Further studies are needed to study the relationship of exposure of residential pesticides to developing diabetes using the biochemical marker to detect the level of metabolites of these pesticides in the populations.

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References

Abdollahi M, Donyavi M, Pournourmohammadi S, Saadat M: Hyperglycemia associated with increased hepatic glycogen phosphorylase and phosphoenolpyruvate carboxykinase in rats following subch ronicexposure to malathion. Comp Biochem Physiol C Toxicol Pharmacol 137: 343–347, 2004

Abdollahi, A. Jafari and N. Jalali, Chronic toxicity in organophosphate exposed workers, M. J. I. R. I. 9 (1995), pp. 221–225.

Abdollahi, A. Jafari, N. Jalali, B. Balali-Mood, A. Kebriaeezadeh and S. Nikfar, A new approach to the efficacy of oximes in the management of acute organophosphate poisoning, Irn. J. Med. Sci. 20 (1995), pp. 105–109. View Record in Scopus | Cited By in Scopus (25)

Abdollahi, M. Balali-Mood, M. Akhgari, B. Jannat, A. Kebriaeezadeh and S. Nikfar, A survey of cholinesterase activity in healthy and organophosphate-exposed populations, Irn. J. Med. Sci. 21 (1996), pp. 63–66.

Abu-Basha EA, Yibchok-Anun S, Hopper DL, Hsu WH: Effects of the pesticide amitraz and its metabolite BTS 27271 on insulin and glucagon secretion from the perfused rat pancreas: involvement of alpha2D-adrenergic receptors. Metabolism 48:1461–1469, 1999

American Diabetes Association (2011): National Diabetes Fact Sheet (released Jan. 26, 2011)

Arab M: Diabetes mellitus in Egypt. World Health Stat Q. 1992;45(4):334-7, university of Alexandria, Egypt.

Beard J, Sladden T, Morgan G, Berry G, Brooks L, McMichael A: Health impacts of pesticide exposure in a cohort of outdoor workers.EnvironHealthPerspect111:724– 730, 2003

Cranmer M, Louie S, Kennedy RH, Kern PA, onseca VA: Exposure to 2, 3,7,8-tetrachlorodibenzo- p-dioxin (TCDD) is associated with hyperinsulinemia and insulin resistance. Toxicol Sci 56:431–436, 2000

Daily G (2008): http://www.precaution.org/lib/08/prn_tributyltin_and_diabetes.081203.h m

Gale EA. 2002b. The rise of childhood type 1 diabetes in the 20th century. Diabetes 51(12):3353-3361. PM:12453886.

Henriksen GL, Ketchum NS, Michalek JE, Swaby JA: Serum dioxin and diabetes mellitus in veterans of Operation Ranch Hand. Epidemiology 8:252–258, 1997

Longnecker MP, Michalek JE: Serum dioxin level in relation to diabetes mellitus among Air Force veterans with background levels of exposure. Epidemiology 11:44–48, 2000

Marjatta Karvonen, PHD, Maarit Viik-Kajander, MSC, Elena Moltchanova, MSC, Ingrid Libman, MD, PHD, Ronald LaPorte, PHD, Jaakko Tuomilehto, MD, PHD for the Diabetes Mondiale (DiaMond) Project Group: Incidence of Childhood Type 1 Diabetes Worldwide; Diabetes Care. 2006;23(10)

Mott *et al*., 1994 L. Mott, F. Vance and J. Curtis, Handle with care: children and environmental carcinogens, Natural Resources Defense Council, New York, NY (1994).

Munro BH (1997): Statistical methods: For health care research. 3rd ed., Philadelphia, J.B Lippincott Company, PP. 100,: 254-88.

National Diabetes Information Clearinghouse: National Diabetes Statistics (2011): Health Information Services. National Diabetes Information Clearinghouse

Perera *et al*., 1999 F.P. Perera, W. Jedrychowski, V. Rauh and R.M. Whyatt, Molecular epidemiologic research on the effects of environmental pollutants on the fetus, Environ. Health Perspect. 107 (1999), pp. 451–460. View Record in Scopus | Cited By in Scopus (111)

Pournourmohammadi S, Farzami B, Ostad SN, Azizi E, Abdollahi M: Effects of malathion subchronic exposure on rat skeletal muscle glucose metabolism. Environl Toxicol and Pharmacol 19:191–196, 2005

Saldana I, Knott C, Basso O, Blair A *et al*.: Pesticides exposure and self reported gestational diabetes mellitus in the agricultural health study. Diabetes Care: 2007; 30 (3).

Shadnia, E. Azizi, R. Hosseini, S. Khoei, S. Fouladdel, A. Pajoumand, N. Jalali and M. Abdollahi, Evaluation of oxidative stress and genotoxicity in organophosphorus insecticide formulators, Hum. Exp. Toxicol. 9 (2005), pp. 439–445. Full Text via CrossRef | View Record in Scopus | Cited By in Scopus (30)

Tanabe S, Kunisue T. 2007. Persistent organic pollutants in human breast milk from Asian countries. Environ.Pollut. 146(2):400-413. PM:16949712.

Whyatt and Perera, 1995 R.M. Whyatt and F.P. Perera, Application of biologic markers to studies of environmental risks in children and the developing fetus, Environ. Health Perspect. 103 (1995), pp. 105–110. View Record in Scopus | Cited By in Scopus (44).

Williams A: prevalence of type I diabetes in children with diabetes: 2011; 17(30).

8/20/2011