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Table (5)- AFB1 Positivity and Percentage of contamination below and above age of 9 years

Age group		AFB1		Total
		-ve	+ve	
<9 years	count	19	8	27
	% within age group	70.4%	29.6%	100.0%
>9 years	count	45	28	73
	%within age group	61.6%	38.4%	100.0%
Total	count	64	36	100
	%within age group	64.0%	36.0%	100.0%

P=0.42

There is no statistically significant difference between 2 groups less than 9 years and more than 9 year as regard the distribution of + ve AFB1

Table (6)- Mean level of AFB1 in urine of positive subjects with consideration of sex

Sex		Number	Mean level of AFB1	Std. Deviation
AFB1	M	19	0.3384	0.4160
	F	17	0.2147	0.2078

There is no statistically significant difference between level of AFB1 in male and female t=1.10 P=0.28

Table (7)- Mean level of AFB1 in urine of positive subjects with consideration of socio economic standard level

Standard Level	Number	Mean level of AFB1	Std. Deviation
H	12	0.2857	0.3778
L	24	0.2700	0.3182

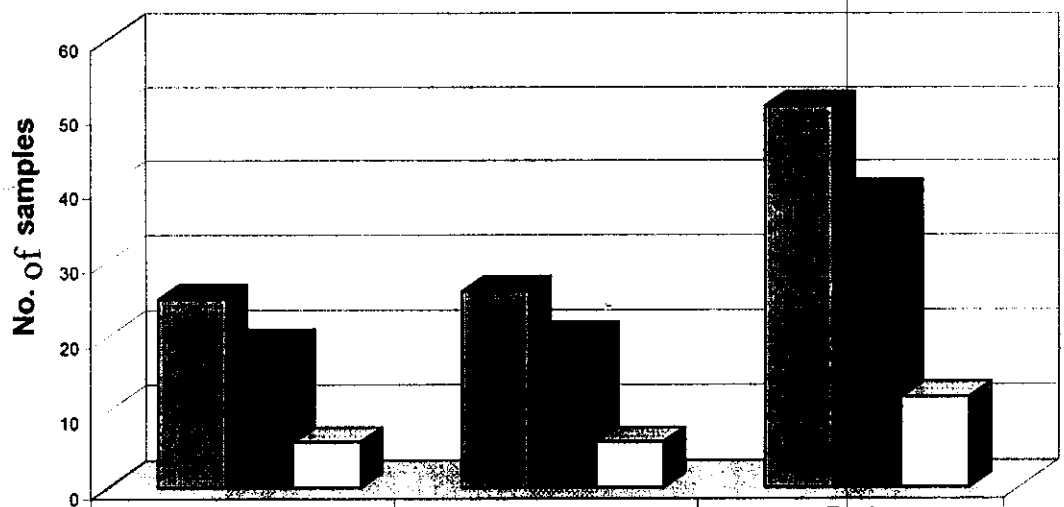
There is no statistically significant difference between mean level of AFB1in high and low standard level t =0.13 P=0.90

Table (8)- Mean age consideration and its relation to AFB1 positivity

		N	Mean age	Std. Deviation
AFB1	-ve	64	10.23	2.92
AFB1	+ve	36	10.42	2.41

There is no statistically significant difference between mean age in presence or absence of AFB1 t=0.31 P=0.75

Number of free and contaminated samples with aflatoxin B 1 in children urine samples in high social economical standard group



	Mal	Female	Total
Total no of samples	25	26	51
Not contaminated samples	19	20	39
Contaminated samples	6	6	12

Figure (3)

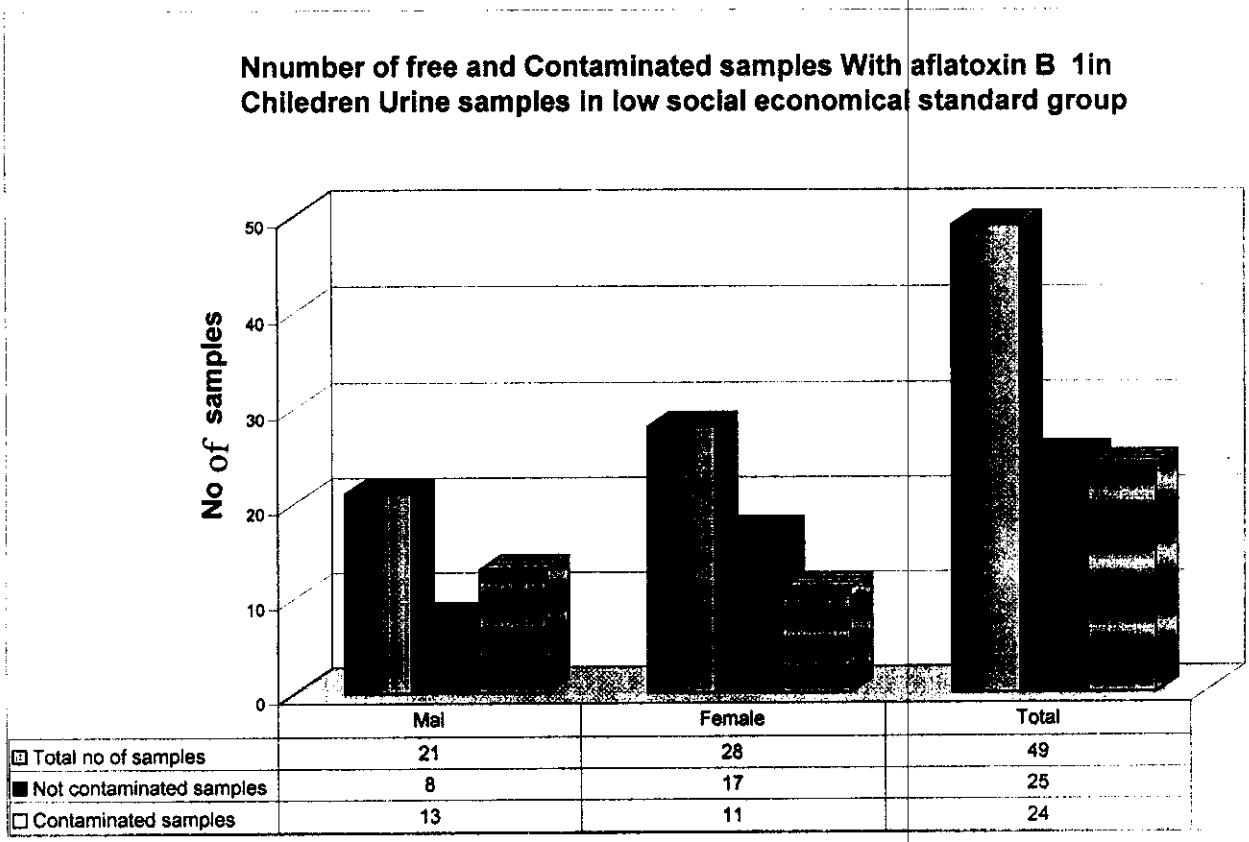
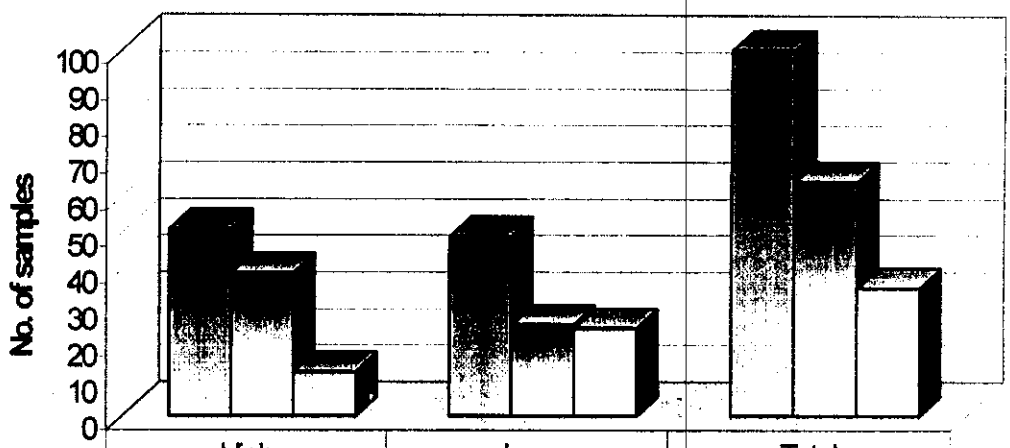

Nnumber of free and Contaminated samples With aflatoxin B 1in Chiledren Urine samples in low social economical standard group
No of samples
50
40
30
20
10
0
Mal
Female
Total
Total no of samples
Not contaminated samples
Contaminated samples
21
28
49
8
17
25
13
11
24

Figure (4)

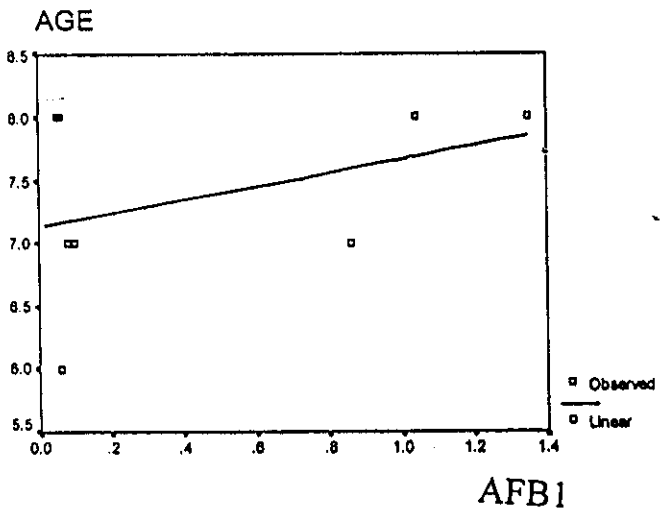
Comparison between AFB1 postivity in high and low socio economic standard group



	High	Low	Total
Total no of samples	51	49	100
Not contaminated samples	39	25	64
Contaminated samples	12	24	35

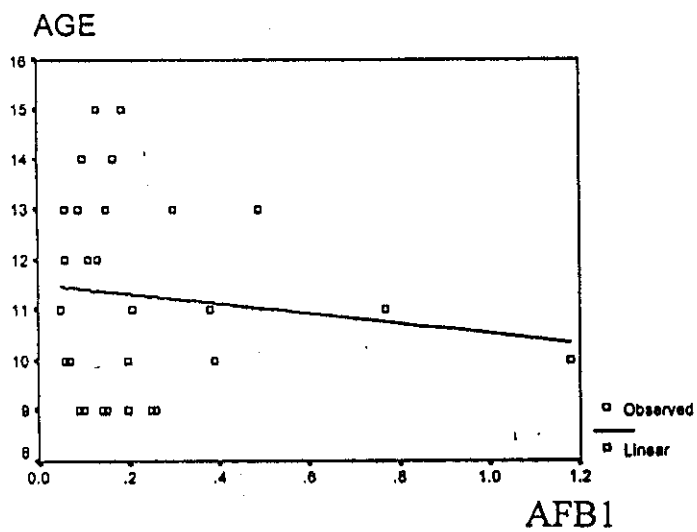
Figure (5)

Figure (6)



(Group of Age less than 9 years) there is a positive linear relation between age and afb1 (i.e. increase in one variable is associated with increase of the other one). But it did not reach statistical significance P>0.05

Figure (7)



(Group of Age more than 9 years) there is a negative linear relation between age and afb1, (i.e. increase in one variable is associated with decrease of the other one). But it did not reach statistical significance P>0.05 (no statistical correlation)

DISCUSSION

In Egypt, liver diseases in children are a growing health problem. The most common causes are attributed to viral diseases, parasitic infestations and metabolic disorders. Hepatitis B and C are considered the major hepatic problems. Both infections can lead to an acute or silent course of liver disease, progressing from liver impairment to cirrhosis and decompensate liver failure or hepatocellular carcinoma (HCC) in a 20-30 year period. In addition, hepatitis B and C infection rates differ in different settings, and prognosis may be worse in conjunction with Schistosomiasis in Egypt. ***(Attia, 1998).***

The peculiarity about these conditions is the early deterioration of these children liver status leading to de-compensation. The synergism with other hepatic insult can be incorporated in such early deterioration. A variety of insults can account to such synergism. Toxic metabolites from moulds and others should be considered.

Mycotoxicosis are diseases caused by mycotoxins, i.e.. Secondary metabolites of moulds. Although they occur more frequently in areas with hot and humid climate, favorable for growth of moulds, they can also be found in temperate zones. Mycotoxicosis often remains unrecognized by medical professionals, except when large numbers of people are involved ***(Peraica et al., 1999***). Aflatoxins are a group of highly toxic fungal secondary metabolites that occur in aspergillus species and may contaminate foods ***(Daily,et al.,2001).*** They are a potent liver carcinogens in experimental animals and frequently contaminate human diets ***(Wogan, 2000***). It is one of the most potent rodent and human liver carcinogens ***(Steller,et al., 2001).***

Apart from hepatocellular carcinoma and acute hepatic encephalopathy caused by aflatoxins, other liver diseases may occur such as hepatitis leading to hepatic fibrosis, affecting age ranges from 4 to 6 years, and juvenile cirrhosis affecting age Group around 5 years ***(Amla et al., 1971).***

The role of aflatoxin in producing cirrhosis in humans remains inconclusive although probable in some settings ***(Zimmerman, 1999).***

Mowat, (1994)c reported that there is a marked geographical variation in age at infection with HBV, and the higher the adult carrier rate, the higher the rate of acquisition in childhood. In much of Asia and Africa where adult carrier rates are greater than 10 % and, to a lesser extent, in Mediterranean countries and South America where 2- 7 % of adults are carriers, HBV is predominantly a disease acquired in early infancy and childhood. Fetal exposure could contribute to the high prevalence and early onset of primary hepatic carcinoma and infectious diseases, including hepatitis B and malaria in West Africa ***(Wild et al., 1991).*** So AFB1 may carry certain predisposition to HBV, and may be to other hepatitis

Alvarez et al., (1995). Mentioned that aflatoxins are a well known carcinogenic, mutagenic and teratogenic substances, likewise its association with hepatitis B virus leading to the high incidence of hepatocellular carcinoma in such viral infection.

In 1967, there was an outbreak of apparent poisoning of 26 persons in two Taiwan rural villages ***(Ling et al., 1967).*** The victims had consumed moldy rice for up to 3weeks; they developed edema of the legs and feet, abdominal pain, vomiting, and palpable livers, but no fever. The

three fatal cases were children between 4 and 8 years. Autopsies were not done, and the cause of death could not be established. In a retrospective analysis of the outbreak, a few rice samples from affected households were assayed for aflatoxins. Two of the samples contained up to 200ppb aflatoxin B1.

Sirag et al., (1981) reported that consumption of levels of AFB1 of up to 0.1to 0.6 mg/kg body weight for several days is capable of causing an acute toxic response often culminating to death. ***Ryan et al., 1979,*** stated that AFB1 in high levels in the blood was present in 40% of the patients with Rye's syndrome. Aflatoxin B1 was present in 6 livers of seven autopsies from proven cases of Rye's syndrome.

Abd El- Hamid, (1990) examined several kinds of Egyptian foodstuffs namely maize, rice cake, rice germ cake, rice bran. Wheat bran, cottonseed, cotton seed cake, ground nuts and mixed feed for the aflatoxins residues. He found that aflatoxin level in the positive examined food samples ranged from less than 100ppb. Up to 400ppb. The AFB1 was the common aflatoxin detected. The recommended levels of AFB1are 0ppb in food ***(Grybauskas, 2002).***

Accordingly, it is highly important to investigate the current situation of the exposure of Egyptian children to AFB1 in particular, as it is the most traumatizing mycotoxin to the liver.

The importance of the study of such exposure may be exaggerated with the introduction of new foods that become available to children, and promoted with the audiovisual media, to the extent that may change or at least modulate their eating habits.

For this task the present work included one hundred children of different socioeconomic standard levels in the age group from 6 to 15 years. All the studied subjects were apparently healthy with an inclusion criterion, that they were all using corn feeds like Haridy, Bozo...Etc., from time to other. The studied subjects were classified according to the socioeconomic standard level into two groups, high socioeconomic standard class children (49) (collected from those attending private schools) and low socioeconomic standard class children (51) (collected from those attending governorate schools). These two groups were further classified according to sex into males (46) and females (54), and based on age into below 9 years (27) and above 9 years (73).

Considering the socioeconomic standard in our classification is to monitor the eating habits, which is assumed to differ according to the child socioeconomic standard and the availability of the financial resources.

The second point of interest is classification according to sex, this classification is based on the idea of different social habits of males and females, even in young ages. Boys spend more time in playing outdoors than girls.

Age, is the last classification parameter included in this work. It is based on the change in eating habits with age, as what is calling the interest of a boy of 7 years may not call the same interest of a boy of 9 years old.

Many studies measured AFB1in serum samples, the use of urine samples may be recommended in children, as it is more convenient and easy to be collected. The blood samples for children may not be the best for assessment of AFB1 as much volume of blood that is required by the

chromatographic method (100-ml) may be a leading barrier.

The gained data of the present study showed that in low socioeconomic standard group,(21 males and 28 females) that were included in the study. In males group, 13 urine samples were contaminated with a mean value of 0.29ng/ml and 61.9% as a percent of contamination. In females, 11urine samples were contaminated with a mean value of 0.25ng/ml and 39.3% as a percentage of contamination(figure 4).

In high socioeconomic standard group,(25 males and 26 females) that were included. In males, 6 urine samples were contaminated with a mean value of 0.2ng/ml and 24% as a percentage of contamination. In females, 6 urine samples were contaminated and 23%as a percentage of contamination (figure 3).

Similar work to that of the present study was done by ***Autrup et al., 1983.*** They examined the urine samples which were collected in Murang a district in Kenya, in the age range of 5 to 15 years, AFB1guanine was analyzed by HPLC,6 of 81 samples had a detectable level of a compound whose fluorescence spectrum was identical to chemically synthesized AFB1-guanine as confirmed by photon counting fluorescence spectrophotometer. Although they used different technique yet they describe an incidence of random contamination about 7.4% which is lower than that described in the present study as our total percent of contamination was 36% (table 3, 4 and 5). The interesting point in the work of ***Autrup et al., 1983*** is that they can correlate the positivity of 5 samples from 6 positive samples are from the same district which have heavy rain fall and their stable diet is maize and beans, that could easily

be contaminated with AFB1 while the others were from a fairly dry district but rather under developed compared with the other locations. ***Autrrup et al., 1983,*** could also postulate that the processing of food may carry no risk of contamination and could the storage be the mean cause of contamination if not proper.

In an other study by ***Autrup et al., 1987***, rate of exposure to AFB1in various parts of Kenya was done, different age groups were involved, of all tested individuals 12.6% were positive for AFB1 exposure. Males have significantly higher rate of exposure to AFB1 than females, the male female ratio ranged from 0.9-4.5 which agree with our results. This is in accordance with the present study as the presence of contamination in males was 41.3% while in females it was 31.5% (Table 3).

Alvarez et al., 1991, examined urine samples of children suffering from chronic liver disease for AFB1. They describe incidence of 35% contamination with AFB1 in children with chronic active hepatitis. Their described incidence of contamination with AFB1 was different for those children suffering from chronic metabolic liver disease which was 25%. Although ***Alvarez et al., 1991,*** examine the urine of diseased children yet the incidence is so close to that demonstrated in the present study (36%), which carry a lot of questions about the real health status of included children in the present work. This comparison is annoying as our apparently healthy children may be in a greet risk.

The present study is also in accordance with that study by ***Makaranada et al., 1998,*** whom studied AFB1 exposure in Thailand population, urinary AFB1/creatinine ratio was measured, in different population (neonate, vegetarian and non vegetarian groups), 30% of the samples from vegetarian and 26% from non vegetarian were found to be positive.

The present study also demonstrate that low socioeconomic standard group is at higher risk of contamination than high group, this may be due to certain factors in the food or due to low price of available corn food in the market. The close findings between the study of ***Makaranada et al., 1998,*** and that described in the present work in low socio economic standard group may carry certain back ground of the food types of vegetarian included in the work of ***Makaranada et al., 1998*** as food stuffs may be the same. But the environmental condition may carry more risk of food contamination during storage (heavy rains).

From previous studies, we found that contamination in our study in low socioeconomic standard level children (49%) is greater than all previous studies in other population but in high socioeconomic children (23.5%), contamination is lower than Kenya population.

In the present study, it was found that the percentage of contamination with AFB1 was (38.4%) in children equal to or more than 9 years, with no positive correlation to age (Table 5 & Figure 7) but in children less than 9 years was (29.6%) with positive correlation between level of AFB1 and age (Table 5 & Figure 6). So it could be demonstrated that percentage of contamination increases with increasing age. The limited number of included children may be leading factor to fail to prove

a positive correlation between age and Aflatoxin level over the age of 9 years. This may be due to increase consumption of contaminated food with Aflatoxins or may be due to cumulative effect of toxins. Also there was increased in percentage of contamination in males than females being (41.3%) and (31.4%) respectively (Table 3). The social habits can give male child more time to play out door and can cause more chance to consume fast foods that may carry an explanation.