

## The Influence Of Aflatoxins On Prevalence Of Neonatal Jaundice In Qalyobia Governate, Egypt

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### ABSTRACT

**Background:** Aflatoxins(AF) are commonly environmental toxicant in developing tropical countries. **Aim of the work:** This study set out to investigate the prevalence of aflatoxins and their metabolites in sera of jaundiced neonates and their mother's sera and breast milk using Thin Layer Chromatography (TLC). **Subjects and methods:** Samples were obtained from 200 jaundiced neonates and their mothers, and 60 non jaundiced controls and their mothers admitted to Benha university hospital, located in Qalyobia governate, Egypt. **Results:** AF levels in cases were highly significant ( $P<0.001$ ) comparing with controls, the highest percentage of aflatoxin type in the studied groups was for M1, then M2 then B1 respectively but no correlation between severity of jaundice and aflatoxins levels. There was significant negative correlation ( $P<0.001$ ) in birth weight with aflatoxin level in jaundiced neonates. The rates of detection of aflatoxins in neonates were: significant higher ( $P<0.001$ ) in breast feeding than bottle feeding neonates, significant higher ( $P<0.001$ ) in hot wet than dry cold months and significant higher ( $P<0.05$ ) in samples from those living in rural than urban areas.

**Conclusion:** aflatoxins are risk factor for neonatal jaundice and low birth weight in hot wet months in those living in rural areas.

**Keywords:** aflatoxins, neonatal jaundice, Qalyobia

### INTRODUCTION

Aflatoxins are secondary metabolites produced by some species of filamentous fungi called *aspergilli*. The major *aspergillus* species of fungi that can produce aflatoxins are *Aspergillus flavus* and *Aspergillus parasitus*, both of which are found worldwide. These molds are ubiquitous in nature and accordingly contaminate a wide variety of foods and feeds and grow on a variety of substrates, thereby producing aflatoxins. The most biologically potent toxin of this group is aflatoxin B1 (AFB1) (1).

Aflatoxins are detected as natural contaminants of food and other agricultural commodities. Aflatoxins may enter food either by direct contamination as a result of molds growth on the food or by indirect contamination through the use of contaminated ingredients in the processed food or by using animal product as milk and milk products that contain aflatoxin residues caused by giving moldy meal to animals (2).

Corn and peanut represent the major potentially contaminated commodities especially in tropical climatic conditions which favor fungal

growth and legation of secondary metabolites. Aflatoxins occurred in noteworthy amounts in market place samples of edible nuts and grains as well as their derived products and figs (3).

Although, aflatoxins (AF) were known for more than 30 years, there is still considerable controversy about their human effect. Food-borne exposure to aflatoxins B1 (AFB1) has implicated in association with acute hepatic injury in humans as well as carcinogenesis (4).

During the last decade, a lot of epidemiological and laboratory investigations have shown a closed association between dietary exposure of pregnant women to aflatoxins and increased incidence of neonatal jaundice (5).

Aflatoxins play a role in induction of neonatal physiological jaundice in some tropical countries where the level of AF is high *Wild and Turner* (6). *Bakry et al.* (7) who detected six types of aflatoxins (AFB<sub>1</sub>, AFB<sub>1</sub>+AFG<sub>1</sub>, AFM<sub>1</sub>, AFM<sub>2</sub>, AFG<sub>1</sub> and AFG<sub>2</sub>) in 22 out of 200 breast milk samples (11%) collected from Qalyobia governate, Egypt. Moreover, the same authors

examined forty samples of children's blood (1-15 years old), where all contained AFB1.

The progressive increase in number of neonatal physiological jaundice in Egypt has attracted the attention to study the effect of some factors including food habits that could be implicated in the pathogenesis of such condition.

This study was designed to investigate the prevalence of aflatoxins in the blood of neonates with physiological jaundice and their mothers in order to determine the possible relationship between perinatal aflatoxins exposure and unexplained neonatal jaundice and low birth weight.

## MATERIAL AND METHODS

### Subjects

This study was conducted on 260 neonates and their mothers. All neonates were selected from outpatient clinics in Benha university hospital, pediatric department, neonatology unit located in Qalyobia governate, Egypt. The subjects were divided into two groups.

**Group I:** This group included 200 full term neonates with physiological jaundice. This group contained 134 males and 66 females aged from 3-5 days, and their mothers.

**Group II (control group):** This group included 60 healthy neonates with normal bilirubin level. It contained 30 males and 30 females aged from 3-5 days, and their mothers.

Ethical approval was obtained from the parents of the neonates who underwent this study. Informed consent was also taken from all mothers taking part in this study.

In this study all cases and controls were subjected to:

I) Questionnaire to collect relevant data regarding time of neonatal birth at 4 months of 2006 (cold dry months i.e. Jan. and Feb. and hot wet months i.e. Aug. and Sept.), their residence and neonatal diet habit whether breast feeding or bottle feeding. Also questionnaires were asked to their mothers about medical history (to exclude infectious diseases, chronic disease and drugs intake during pregnancy) and Full dietetic

history were taken from all mothers especially aflatoxins contaminated food (i.e. cereals foods, milk, milk products, etc).

II) Careful physical examination: gestational age assessment by Ballard's score, birth weight and local organ examination to exclude any other causes of pathological jaundice.

III) Laboratory investigation: blood samples were obtained from neonates and their mothers. Breast milk samples were obtained from each study case and control. These samples were used for evaluation of aflatoxin and serum bilirubin levels.

### Exclusion criteria

- \* Mother with any acute or chronic illness.
- \* Neonate with pathological jaundice.
- \* Neonate with any congenital anomalies.
- \* Preterm neonate.

## METHODS

**A-Serum bilirubin :** 2-3 ml of neonatal venous blood samples under sterile aseptic condition were withdrawn and left to clot for 2 hours at room temperature then centrifuged for 10 minutes at 5000 rpm, serum separated and kept at -20 C till determination (8).

### B-Aflatoxins determination

- (1) Aflatoxin standards: aflatoxins B1, B2, B1, G1 and G2 together with their metabolites M1, M2, M2a, Q and Ro were obtained from Sigma Chemical Company (Sigma Aldrich Corporation), P.O. Box 14508, St. Louis, Missouri, 63178, USA.
- (2) C18 sep. Pak.: These packs were obtained from Associates Inc. Milford, MA, 01757, USA.
- (3) Confirmatory tests of major aflatoxins and their metabolites in the blood and milk samples of all cases and controls were carried out by TLC *Peterson and Ciegler (9)*.
- (4) Quantitative determination of aflatoxins: positive samples and standard spots were measured with fluorometer then the amount of each aflatoxin present in blood and milk samples was calculated (10).

### Statistical method (11)

The methods used for statistical analysis of the collected data were the following:

- 1- t-test: for comparison between the means of two means  $\pm$  standard deviations. P values less than 0.05 considered significant.
- 2- One way ANOVA test (F test) for comparison between more than two means  $\pm$  standard deviations.

## RESULTS

Two hundred and sixty neonates and their mothers were selected, 200 of them served as cases and 60 as controls, to determine the aflatoxin levels in neonatal and maternal blood and breast milk.

There was a high significant level ( $P < 0.001$ ) of aflatoxin in blood of neonates with neonatal jaundice and their maternal blood and breast milk compared with controls [Table 1 & Fig.(1)]. In jaundiced neonates (cases), aflatoxin  $M_1$  was the highest percent (81.5%),  $M_2$  was less common (68.9%) while the least incidence was

aflatoxin B1 (67.5 %). But in controls,  $AFM_2$  was the highest percent (31.1%) followed by  $AFB_1$  (30%) then  $AFM_1$  (18.5%) [ Table 2]. There was no correlation between levels of neonatal blood aflatoxins and billirubin ( $P > 0.05$ ) [Table 3].

There was a highly significant ( $P < 0.001$ ) reduction in birth weight of jaundiced neonates in comparison with controls [Table 4 & Fig.(2)]. Also, there was negative significant correlation ( $P < 0.001$ ) between aflatoxin levels and birth weight [Table 5 & Fig. (2)].

There was a highly significant correlation ( $P < 0.001$ ) between aflatoxin levels and breast feeding neonates in comparison with bottle feeding neonates [Table 6 & Fig.(3)]. Aflatoxins level was highly significant ( $P < 0.001$ ) in hot humid months (Aug. and Sept.) when compared with cold dry months (Jan. and Feb.) [Table 7 & Fig.(3)]. Additionally, there was a significant correlation between aflatoxin levels and residence from rural areas than those from urban areas [Table 8 & Fig. (3)].

**Table 1. Comparison of aflatoxins level in neonatal blood of jaundiced neonates, (N=200) and their maternal blood and breast milk compared with controls (N=60).**

Aflatoxin	X $\pm$ SD	t	P
1-Neonatal blood cases control	2.83 $\pm$ 1.38 0.16 $\pm$ 0.02	19.47	<0.001
2-Maternal blood cases control	2.8 $\pm$ 1.38 0.16 $\pm$ 0.02	19.7	<0.001
3-Breast milk cases control	2.55 $\pm$ 1.32 0.12 $\pm$ 0.02	18.4	<0.001

**Table 2. The percentage of different types of aflatoxin detected in studied groups (St. Gp.).**

AF	Cases		Controls		Total		t	P
	No	%	No	%	No	%		
M1	132	81.5	30	18.5	162	100	7.3	<0.001
M2	40	68.9	18	31.1	58	100	2.2	<0.05
B1	28	57.0	12	30	40	100	1.95	<0.05
Total	200	76.9	60	23.1	260	200		

**Table 3. The correlation between aflatoxin and serum billirubin levels in jaundiced neonates.**

Bl.AF \ S. Bil.	$\bar{X} \pm SD$	t	P
M1 (n=132)	8.01 $\pm$ 2.82	t1=0.058	>0.05
M2 (n=40)	8.39 $\pm$ 2.48	t2=0.15	>0.05
B1 (n=28)	8.14 $\pm$ 2.91	t3=0.26	>0.05

One way ANOVA F=.014 t1= M1 Vs M2 t2= M1 Vs B1 t3= M2 Vs B1

**Table 4. Comparison between reduction of birth weight among cases and control .**

St.Gp. \ B.W.	$\bar{X} \pm SD$	t	P
Cases (n=200)	2542.6 $\pm$ 567.1	8.01	<0.001
Control (n=60)	3120 $\pm$ 244.1		

**Table 5. The correlation between aflatoxin levels and birth weight in jaundiced neonates.**

B.W \ Neonatal AF	$\bar{X} \pm SD$	t	P
LBW(n=132)	3.32 $\pm$ 0.69	9.85	<0.001
Normal Wt. (n=68)	1.3 $\pm$ 1.14		

LBW : Low birth weight

**Table 6. The correlation between aflatoxins level and types of neonatal feeding habit (FH) in jaundiced neonates.**

FH \ N. AF	$\bar{X} \pm SD$	t	P
Breast feeding (n=118)	3.7 $\pm$ 0.62	10.3	<0.001
Bottle feeding (n=82)	1.59 $\pm$ 1.21		

**Table 7. The correlation between aflatoxins level and seasonal variation (SV). in jaundiced neonates.**

Season	$\bar{X} \pm SD$	t	P
Hot wet months (Aug. & Sep.) (n=100)	3 $\pm$ 1.02	7.22	<0.001
Cold dry months (Jan. & Feb.) (n=100)	1.12 $\pm$ 1.14		

**Table 8. The correlation between aflatoxins level and residence in jaundiced neonates.**

Residence \ N. AF	$\bar{X} \pm SD$	t	P
Rural (n=150)	8.60 $\pm$ 2.5	2.31	<0.05
Urban (n=50)	7.50 $\pm$ 1.9		

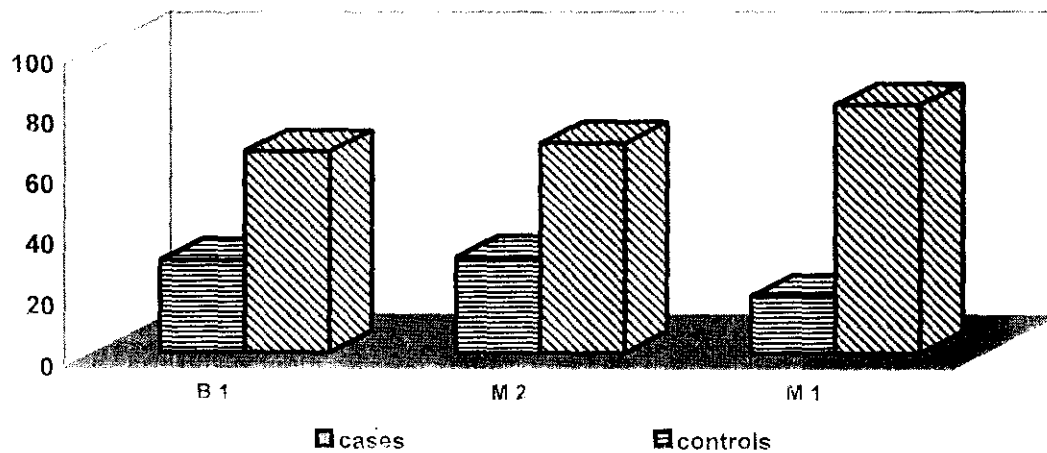


Fig.1 Percentage of different types of aflatoxins among cases and controls

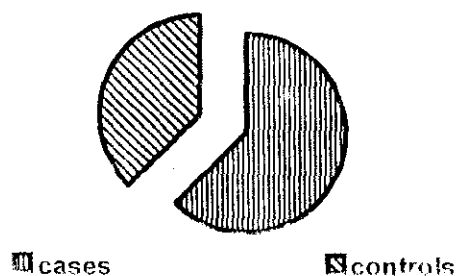


Fig.2. Reduction in body weight among jaundiced neonates in cases and controls

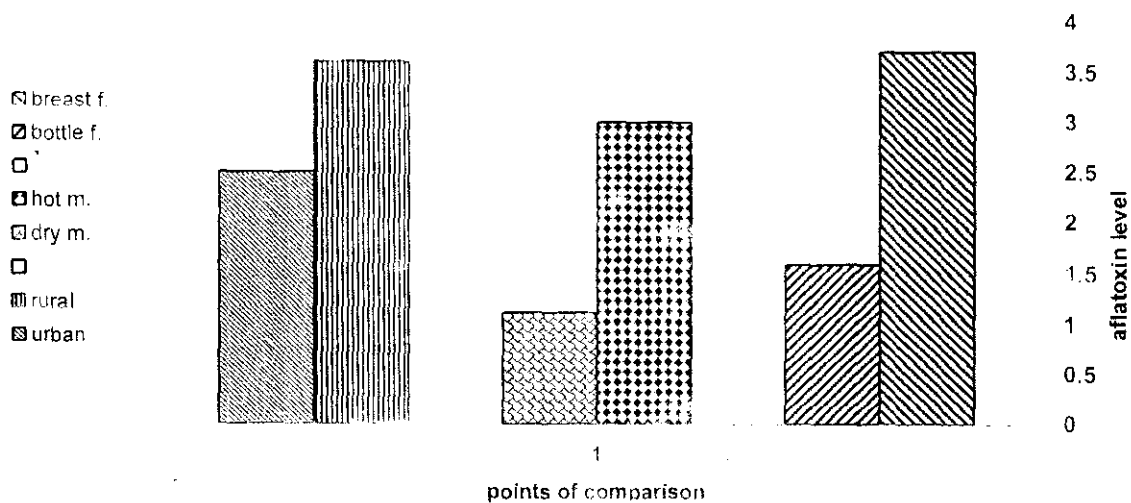


Fig 3. Correlation of aflatoxins level and type of feeding, date of birth and residence in jaundiced neonates.

## DISCUSSION

Human exposure to aflatoxins may begin prenatal, during breast feeding and continues in adult life. It's postulated that aflatoxins play a role in kwashiorkor, increase neonatal susceptibility to infection and malignancy (12,13).

This study was set out to investigate the prevalence of aflatoxins in the sera of babies with neonatal jaundice and their mothers in order to determine whether they contribute to the occurrence of unexplained neonatal jaundice in Qualyobia governate, Egypt. In the present study Aflatoxins were significantly detected in neonatal blood, maternal blood and breast milk in studied cases compared with controls. There was no correlation of aflatoxins concentration in neonatal blood or maternal blood and severity of jaundice. These results coincide with previous report *Ahmed et al. (14)* which reported that aflatoxins were detected in blood over 50% of neonates with jaundice of unknown aetiology and there was no correlation between level of bilirubin and aflatoxins level.

In Sierra Leone it has been found that 58% of cord samples from pregnant women contained aflatoxins. In Ibadan, aflatoxins were detected in the blood of 27.4% of jaundiced infants. In Kenya, 53% of women seen at hospital were found to be positive for aflatoxins at anytime during pregnancy and 37% of cord blood contained aflatoxins. In Thailand, 48% of cord blood samples were found to contain aflatoxins *Jonsyn et al. (15)*.

In contradiction to results of the present study *Abdulrazzaq and his associates (12)* who failed to demonstrate any effect of aflatoxins on neonatal jaundice. They reported that there was no association between aflatoxin (M1) concentration in maternal blood or cord blood and jaundice or infection.

In a trial to explain the possible mechanism of aflatoxin in induction of neonatal jaundice it has been reported that the liver is the principal target organ for toxicity *AAPSH (16)*. The liver is the predominant site

of metabolic transformation of AFB1. Cytochrome P-450 and 1A2 catalyze the epoxidation of AFB1 to a highly reactive intermediate AFB1 exo-8, 9- epoxide (17). This intermediate binds with high affinity to guanine bases in DNA to form guanyl -N7 adducts (18). The formation of DNA adducts explain the toxic dynamic effects of AFB1. Acute cytotoxic effects of high-level AFB1 exposure may be mediated by binding to functional cellular protein. Several investigators (4,19) reported that in neonates hepatic excretory capacity is low because of low concentration of the binding protein ligand in hepatocytes and because of low activity of glucoronyl transferase.

In the present study, there was a significant reduction in birth weight of neonatal jaundice compared with controls. Also, a highly significant negative correlation ( $P < 0.001$ ) was found between level of aflatoxins and birth weight especially in breast feeding neonates born in hot wet months. These results are in accordance with several reports (20,21) which indicated that aflatoxins exposure in tropical areas occur in 30% of pregnancies resulting sometimes in high levels of aflatoxins in cord blood, leading in turn to jaundice and possibly low birth weight and perinatal death. The biochemical, immunological and metabolic derangments caused by aflatoxin in the fetus could lead to low birthweight and intrauterine growth retardation (22,23). On the other hand *Maxwell et al., (24)* who failed to demonstrate any effect of aflatoxins on birth weight.

The concentration and frequency of plasma aflatoxins present in pregnant women delivering in hot wet months (August and September) was significantly higher than those delivering in dry months (January and February). The pattern of seasonal variation for detection of aflatoxin probably reflecting the ingestion of contaminated grains stored in summer when humidity and temperature are very high which favor fungal growth. These finding is in agreement with the result of studies carried out recently (12, 25) and explained the effects of seasonal variation, and

the frequency of aflatoxins detected by the high contamination of foods during periods of high humidity and temperature but *Allen et al. (26)* were disagreeing with this result.

From our results we noticed that the higher percentage and concentrations of aflatoxins were present in rural areas (inhabited by farmers) than in urban areas in examined samples. This may be due to the improper storage of cereals which leads to growth of the fungus and the formation of the toxin. The bad behavior of farmers in using this contaminated cereals in their food were noticed in most of positives cases and recorded in history of our cases during questionnaire.

It has been suggested that the possibility of removing the potential carcinogen aflatoxin from environment is certainly difficult but the progressive urbanization is occurring in developing countries may afford an opportunity of determining whether the liver cancer is decreasing with urbanization and the changes in food habits and source of dietary staples (27). Very little variation between the rural and urban populations sampled with slight higher level in rural areas was also cited (28).

**In conclusion:** This study demonstrates that the presences of serum aflatoxins are risk factor for neonatal jaundice occurred in the wet hot months. There was also a strong negative correlation between aflatoxins and birthweight but there was no correlation between aflatoxin concentration and severity of hyperbillirubinemia.

*Further studies are in need to determine:* {1} the extent of pre and postnatal exposure to aflatoxins and their effects on fetal and neonatal health, {2} long term exposure to aflatoxins in childhood is necessary especially in areas where protein-energy malnutrition is endemic, {3} the modifying effect of dietary intake of lipotropes, proteins or vitamins on aflatoxins, {4} identification of biological markers of exposure and toxicosis as the presence of aflatoxins in human blood and tissues and {5} the methods and mechanisms responsible for

modulation and protection of neonates from aflatoxins exposure.

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## الملخص العربي

### تأثير الإفلاتوكسينات على تواجد الصفراء في الأطفال حديثي الولادة في محافظة القليوبية

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تعتبر الإفلاتوكسينات من السموم البيئية في الأقطار النامية في المنطقة الإستوائية وتم تصميم هذه الدراسة لفحص مدى تواجد الإفلاتوكسينات ومشتقاتها في سيرم دم الأطفال حديثي الولادة المصابين بالصفراء وكذلك سيرم أمهاتهم ولبن الثدي بواسطة الاختبار الكروماتوجرافى ذو الطبقة الرقيقة (TLC).

وتم تجميع العينات من ٢٠٠ من الأطفال حديثي الولادة المصابين بالصفراء وأمهاتهم و ٦٠ من الأطفال الغير مصابين بالصفراء وأمهاتهم كضوابط للبحث من المترددين على المستشفى الجامعى بينها بمحافظة القليوبية . وقد وجد أن مستوى الإفلاتوكسين في الأطفال المصابين بالصفراء أعلى معنوياً ( $P<0.001$ ) بالمقارنة بالأطفال غير المصابين . وقد وجد أن أعلى نسبة من أنواع الإفلاتوكسين هي أفلاتوكسين م١ ب٢ م١ ولكن لا توجد علاقة بين حدة الإصابة ومستوى الإفلاتوكسين. كما استنتجت علاقة سلبية معنوية ( $P<0.001$ ) بين الوزن عند الولادة زمستوى الإفلاتوكسين في الأطفال المصابة بالصفراء. كذلك وجد أن الإفلاتوكسين في الأطفال حديثي الولادة أعلى معنوياً ( $P<0.001$ ) في التغذية على لبن الثدي من التغذية الصناعية وكذلك أعلى معنوياً ( $P<0.001$ ) في العينات المؤخدة من الافراد المقيمين في الريف عنها في الحضر.

ونستخلص من هذه الدراسة أنه يمكن استبيان أن الإفلاتوكسين عامل مسبب لإصابة الاطفال حديثي الولادة بالصفراء ونقص الوزن عند الولادة في الأشهر الحارة الرطبة عنها في الأشهر الباردة الجافة.