# EFFECTS OF AFLATOXIN ON HUMAN AND ANIMAL HEALTH IN THAILAND*

# ผลของอัฟฟล่าท๊อกซินต่อสุขภาพของคนและสัตว์ในประเทศไทย

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## บทคัดย่อ

กล่าวถึงบทบาทของสารพิษอัฟฟล่าท๊อกซิน ที่เกิดจากเชื้อราที่มีต่อสุขภาพของคนและสัตว์ในบ้านเรา อัฟฟล่าท๊อกซินนับเป็นสารพิษและสารก่อมะเร็ง ที่สามารถตรวจพบได้ในวัตถุดิบที่ใช้เป็นส่วนผสมของอาหารสัตว์และในอาหารสัตว์ และเท่าที่พบและพิสูจน์อย่างเด่นชัดว่าสารดังกล่าวทำให้เกิดโรคในสัตว์นั้นมีอยู่เพียงสองครั้งเท่านั้น สำหรับคนไทยแล้วกลุ่มอาการไร้ย์ในเด็กและโรคมะเร็งของตับในผู้ใหญ่ อาจมีสาเหตุส่วนหนึ่งมาจากสารตัวนี้ได้

## Abstract

The role of aflatoxin, a secondary metabolite of certain strains of the mold *Aspergillus flavus* and *Aspergillus parasiticus* have been discussed on human and animal health in Thailand. Aflatoxin were detected in several feed ingredients and feedstuffs collected from several provinces with only two episodes that showed evidence of

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aflatoxicosis. Aflatoxin and other factors were also indicated as possible causes of Reye's syndrome and hepatocellular carcinoma in Thailand.

The term aflatoxicosis is given to the intoxication caused by the aflatoxin, a secondary metabolites of certain strains of the mold *Aspergillus flavus* Link (*Aspergere*, L. to scatter). The aflatoxins $B_1$ and $B_2$ produced blue fluorescence and $G_1$ and $G_2$ produced green fluorescence under ultraviolet light on thin layer chromatographic plates. Other aflatoxin fractions have been isolated and characterized; the major structures have been identified as difuranocoumarin derivatives

## Animal Aflatoxicosis

**A. General information.** Although the $LD_{50}$ is only an indication of the acute toxicity nevertheless its determination gives a valuable piece of comparative information (See Table 1). Different species vary in their susceptibility to acute poisoning with $LD_{50}$ values varying from 0.3–17.9 mg/kg. Mature animals tend to be less susceptible than young animals. These observations may indicate differing pathways of metabolism and clearance of the aflatoxin. In oder of decreasing susceptibility to acute aflatoxin poisoning, livestock may be classified as followed, pig > cows > sheep; poultry are classified as, duckling > turkey > chicken whereas cat > dog > monkey in pets and rabbit > guinea pig > rat > mouse > hameter in laboratory animals. Trouts are also susceptible to aflatoxin than salmon.

*Table 1.* Single dose $LD_{50}$ of $AFB_1$ in animals.

| Type of animal | Species | $LD_{50}$ (mg. $AFB_1$/kg) |
|---|---|---|
| *Livestock* | pig | 0.62 |
| | Calf | 1.6 |
| | Sheep | 2.0 |
| *Poultry* | Duckling | 0.33 |
| | Turkey | 1.36 |
| | Chicken | 6.5–16.5 |
| *Pet* | Cat | 0.55 |
| | Dog | 0.5–1.0 |
| | Monkey | 2.2 |
| *Laboratory animal* | Rabbit | 0.30 |
| | Guinea pig | 1.40 |
| | Rat, weanling male | 5.5 |
| | weanling female | 7.4 |
| | adult male | 7.2 |
| | adult female | 17.9 |
| | Mouse | 9.0 |
| | Hamster | 10.2 |
| *Fish* | Trout | 1.0 |
| | Salmon | 12.5 |

Aflatoxin liver damage is periportal in the rat, duckling and cat, midzonal in rabbit and centrolobular in the pig, quinea pig, sheep, baboon, dog, chicken and

monkey (Table 2). These findings, therefore, indicate that aflatoxin is metabolically handled differently by different species of animals. It is usual for the toxin requiring activation to result in centrolobular lesion while those detoxified directly produce periportal damage. Those toxins which result in detoxification through intermediates which are more toxic than the parent compound and its ultimate metabolites, produce midzonal necrosis.

*Table 2.* Area of aflatoxin induced liver injury in animals.

| Periportal[1] | Midzonal[3] | Centrolobular[2] |
|---|---|---|
| Rat | Rabbit | Pig, guinea pig, |
| Duckling | | dog, baboon, |
| Cat | | horse, monkey, |
| | | sheep, chicken |

[1]Not require activation

[2]Require activation

[3]Require intermediate activation

**B. Animal aflatoxicosis in Thailand.** The first knowledge of a disease in animals subsequently associate with aflatoxin has been known in Thailand since the mycotoxin research team form Massachusette Institute of Technology (MIT) Boston, headed by Dr. Shank and Dr. Wogan in co-operation with a group of Thai investigators headed by Dr. Natth Bhamarapravati from Mahidol University was established in 1967. During that time, the study was concentrated on the problem of aflatoxin in human health. The first objective of their studies was to determine the number of toxigenic fungi in food

destined for local consumption in our country. The second phase was to find out the distribution of aflatoxins as regards foods and geographic regions of Thailand. The third task was to investigate a possible correlation between aflatoxin ingestion and the incidence of liver cancer in the population.

In 1972 our own laboratory was set up in Mahidol University to study the mycotoxin problem. Most of the unit's work is the analysis of aflatoxin in foods and feeds. Pathologic examinations are also done on cases of acute and chronic toxicity of aflatoxin in experimental and domestic animals.

*Field studies.* The diagnosis of aflatoxin poisoning is far from easy in the first period of our study. We found that co-operation between practicing veterinarians, farmers and laboratory is essential because the definite diagnosis will depend on an assessment of the clinical examination, mycological identifcation and chemical analysis of feeds, pathological findings and animal toxicity tests.

Our field studies divided into two proposes. The first objective is to find out the level of aflatoxin consumed by animals in suspected cases. For this propose, 1-4 kilograms of animal feeds are always sent to our laboratory from different parts of the country. The second task of our field studies is to find out the definitely diagnosed cases of aflatoxicosis by pathologic studies when there is clinical evidence of aflatoxicosis and a high level of aflatoxin contamination in the feed was detected in our laboratory.

The results from the analysis of 22 samples of ingredients used for animal feeding of suspected cases of aflatoxicosis are shown in Table 3. Aflatoxin $B_1$ was detected in 13 samples with levels ranging from 0.8 to 6,500 $\mu$g/kg and a mean concentration of 541.7 $\mu$g/kg. Peanut meal, an ingredient used in horse feed, was reported on

cases of naturally occurring equine aflatoxicosis from Thailand, had the highest aflatoxin content.

*Table 3.* Aflatoxin content in ingredients use for animal feeding, 1975–1980.

| Materials | No. of samples | No. of samples containing aflatoxin | Conc. of aflatoxin $B_1$ ($\mu$g/kg) |
|---|---|---|---|
| Fish meal | 2 | 0 | 0 |
| Rice meal | 8 | 3 | 1, 10, 31.5 |
| Peanut meal | 2 | 2 | 6.5, 6,500 |
| Maize meal | 8 | 7 | 0.8, 10, 22.7, 30 43.7, 51.2, 325 |
| Soyabean meal | 2 | 1 | 10 |
| TOTAL | 22 | 13 | |

Thirty two feedstuffs were associated with field cases of suspected aflatoxicosis and 28 of the samples contained aflatoxin (Table 4) with levels ranging from 0.8 $\mu$g/kg (in duck feed) to 934 $\mu$g/kg (in pig feed). The feed samples were recieved from all parts of the country, i.e. Chanthaburi and Chon Buri in the eastern part, Nakohn Pathom and Pathum Thani in the central part, Udon Thani and Nakhon Ratchasima in the northeastern part and Surat Thani in the southern part. We also informed by some colleaques from the northern part especially Chiang Mai for the incidence of high contamination of aflatoxin in animal feeds during 1977 – 1978 but no incidence of aflatoxicosis was confirmed.

*Table 4.* Aflatoxin content of feed samples, 1975–1980.

| Feedstuff | No. of samples | No. of samples containing aflatoxin | Concentration of aflatoxin $B_1$ ($\mu g/kg$) |
|---|---|---|---|
| Duck pellets[1] | 8 | 7 | 0.9, 2.3, 3.7, 10, 15.6, 70, 70 |
| Duck feed[2] | 7 | 6 | 0.8, 2.5, 2.9, 3.9, 5, 16.8 |
| Layer Crumbles[3] | 1 | 1 | 10 |
| Grower Crumbles[4] | 1 | 1 | 15 |
| Pig feed[5] | 8 | 7 | 3.2, 7, 8.7, 15, 17.5, 20 8, 934 |
| Cattle feed[6] | 1 | 1 | 700 |
| Horse feed[7] | 1 | 1 | 216 |
| Dog feed[8] | 5 | 4 | 6.2, 9.9, 15, 18.7 |
| TOTAL | 32 | 28 | |

Origin : [1,2]Chanthaburi, Chon Buri, Chai Nat, Chachoengsao, Nakhon Pathom, Phetchabun, Surat Thani. [3]Udon Thani. [4]Nakhon Pathom. [5]Chon Buri, Nakhon Pathom, Pathum Thani, Phetchabun, Ratchaburi, Saraburi, Udon Thani. [6]Nakhon Pathom. [7]Phetchabun and [8]Nakhon Ratchasima.

Table 5 showed the sample of feedstuffs which we believed were associated with suspected cases of aflatoxicosis in 1975–1979. There were only two episodes that showed evidence of aflatoxicosis which have been reported elsewhere. The details are as followed:

On March 22, 1976, we received a lot of canned dog foods locally produced with several ill puppies from a military dog unit [21]. There were at least 66 puppies, aged 2-3 month old, of mixed German shepherd and Labrador-retriever breed affected and 24 puppies died within 2-3 days after onset of illness. The diet composed of meat and peanut meal. The young animals from 10 litters had clinical signs of anorexia, bloody diarrhea and jaundice without fever. Some of them had sign of ascites. Treatment with antibiotics or sulfonamide had little effect. Necropsy revealed large swollen fatty livers, anemic carcass. Some of them had nodular liver surface. Histopathologic findings revealed fatty degeneration, centrolobular necrosis and bile duct proliferation and some of them had cirrhosis. Congestion of the gastrointestinal tract were frequently seen. Dogs that showed early sign of aflatoxicosis could recover when had supportive treatment and change to the other food. By using the method of Eppley[13] the range of aflatoxin $B_1$ detected in these foods are 5.1-63.1 $\mu$g/kg and a mean concentration of 21.42 $\mu$g/kg or 35.31 $\mu$g/kg in term of total aflatoxin. We found that our findings was almost the same in animals recieving at a daily dose of 20 $\mu$g/kg body weight, 5 days per week for 10 weeks except our animals aged younger.[2]

On January 23, 1978, The second episode occurred in a horse-breeding farm.[5] The horses were gave a feed mixture composed of 70% maize and peanut meal. A number of young animals had clinical sign of illness and 12 yearling colts died. Necropsy revealed swollen fatty livers, pale and swollen kidneys, and hemorrhagic enteritis. The brain was swollen with compress sulci. Histopathologic findings consisted of various degree of fatty degeneration and centrolobular necrosis of the livers. Fatty infiltration of epithelial lining of the proximal tubules was seen as well as focal and diffuse degeneration of myocardial fibers. Microscopic examination of brain revealed cerebral edema and neuronal degeneration, mainly in the cerebral cortex.

During the episode in horse, the price of rice was high, so maize and peanut meal were substituted for rice in larger than usual proportions and caused the high concentration of aflatoxins in the mixed ration i.e. 216 μg/kg $AFB_1$ or 411 μg/kg total aflatoxins.

*Table 5.* Example of feedstuffs associated with suspected cases of aflatoxicosis, 1975-1979.

| Feedstuff | Animal with signs | Concentration of aflatoxin, μg/kg | | |
|---|---|---|---|---|
| | | $B_1$ | $B_2$ | $G_1$ |
| Layer crumbles (May, 1974) | *Laying hens :* a number of 1,562 hens showed sign of inappetite, cachexia, drop in egg production. Some had nervous sign, convulsion and death. Necropsy revealed severe hemorrhage of intestine and muscle | 10 | – | – |
| Dog food (March 22, 1976) | *Puppies (2–4 months) :* sign of anorexia, bloody diarrhea with jaundice and 24 puppies died. Necropsy revealed large swollen fatty livers | 63.1 | 2.8 | – |
| Cattle feed (October 10, 1976) | No information supplied | 700 | 210 | 17 |
| Duck feed (September 2,1977) | *Ducks :* 20 out of 65 ducks died within 8 month with primary hepatic cancer | 6.75 | 0.52 | – |
| Chick starter crumbles (Nov. 4, 1977) | *Ducks :* a 0.5% of 30,000 chicken died after ingesting feed from one company | 70 | 7.5 | – |

| | | | | |
|---|---|---|---|---|
| Duck feed (Nov. 24, 1677) | *Ducklings (10 days):* sign of distress, nervous symptoms and convulsion. Death occurred about 200 out of 5,000 ducklings with 20% morbidity. Necropsy revealed hepatomegaly, aerosacculitis and mild enteritis [2] | 70 | 3.75 | – |
| Horse feed (Jan. 23, 1978) | *Horse (7 months):* a number of animals had clinical signs of illness and 12 yearling colts died. Necropsy revealed swollen, fatty livers, pale swollen kidneys and hemorrhagic necrosis | 216 | 195 | – |
| Pig feed (Sept. 5, 1978) | *Pig (3–5 months):* a 50% of 10,000 pigs died after ingesting stored feed (more than 1 year) | 934 | – | – |
| Parboiled rice (Oct. 16, 1978) | *Ducklings (2–5 days):* signs of distress and death occurred a few days after onset of symptoms. Necropsy revealed large and yellow red liver[3]. | 10 | – | – |
| Maize meal (April 17, 1979) | *Pigs:* 30 out of 200 pigs died within 30 days after ingesting feed. Necropsy revealed anemic carcass, swollen liver with fine granulation on the surface. Swollen gall bladder wall and hemorrhagic gastroenteritis[3] | No feed samples were available at the time of episode. | | |

Recently, we found a report of experimental induced aflatoxicosis in pony from the Iowa group[11]. They reported deaths occurred at 12 and 16 days, 25 and 32 days, and 36 and 39 days after 3 groups of 2 adult male Shetland ponies were given aflatoxin daily at levels of 0.3, 0.15 and 0.075 mg/kg respectively. The histopathologic findings were similar to naturally affected horses of our report.

*Experimental studies.* The general nature of aflatoxicosis is similar in all affected species, there is considerable interspecies difference in susceptibity. Ducklings, cats, rabbits and piglets are highly susceptible, dogs and turkey poults are moderately susceptible, while chicken and sheep are least susceptible as measured by the criterion of single oral dose $LD_{50}$. Because of the quantities of toxin involved, we try to use small animals instead of large animals in our experimental studies. The experimental designs and pathological findings in one day old male Pekingese ducklings and one day old male hatch chick of super harco breed were studied in order to use for our references in the natural cases in our laboratory.

A single oral dose $LD_{50}$, 0.33 mg/kg body weight was administered to one day old duckling followed by using semisynthetic diet for 7 days. Survivors were sacrificed at the end of the experiment. Gross and microscopic findings of the liver was studied as well as their serum chemistry activities. It was found in Table 6 that mean dietary intake and body weight gain in duckling treated with $AFB_1$ were lower than the control group, while relative liver weight and serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT) and Alkaline phosphatase (ALP) activities were higher than those of control. Histopathologic findings were similar to those reported elsewhere.

*Table 6.* Summary of body and relative liver weight, dietary intake and serum chemistry in one day old duckling treated with a single oral $LD_{50}$ dose of aflatoxin $B_1$.

| $AFB_1$ dose level (mg/kg) | No. of animals | Mean diet intake (gm) | Body weight (gm) at day 0 | 2 | 4 | 7 | Relative liver weight (%) | Serum chemistry* SGOT | SGPT | ALP |
|---|---|---|---|---|---|---|---|---|---|---|
| 0 | 4 | 25.0 | 48.7 | 53.7 | 58.7 | 57.5 | 4.00 | 56 | 31 | 4.4 |
| 0.335 | 6 | 22.5 | 55.8 | 50.8 | 60.1 | 60.0 | 5.48 | 170 | 36 | 9.5 |

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One day old male chicks were also treated in similar manner as described in duckling and the result is similar to those of duckling except there were 4 out of 6 duckling died on day 1,2,3 and 5 but only 1 out of 6 chicks died on day 3 (Table 7).

*Table 7.* Summary of body and relative liver weights, dietary intake and serum chemistry in one day old chicks treated with a single oral $LD_{50}$ dose of aflatoxin $B_1$.

| $AFA_1$ dose level (mg/kg) | No. of animal | Mean diet intake (gm) | Body weight (gm) at day 0 | 2 | 4 | 7 | Relative liver weight (%) | Serum chemisiry* SGOT | SGPT | ALP |
|---|---|---|---|---|---|---|---|---|---|---|
| 0 | 4 | 43.7 | 38.7 | 45.0 | 56.0 | 57.5 | 3.65 | ND | ND | ND |
| 11.5 | 6 | 18.3 | 40.0 | 41.6 | 53.0 | 51.0 | 5.66 | 424 | 96 | 100 |

*Sigam Frunkel Units

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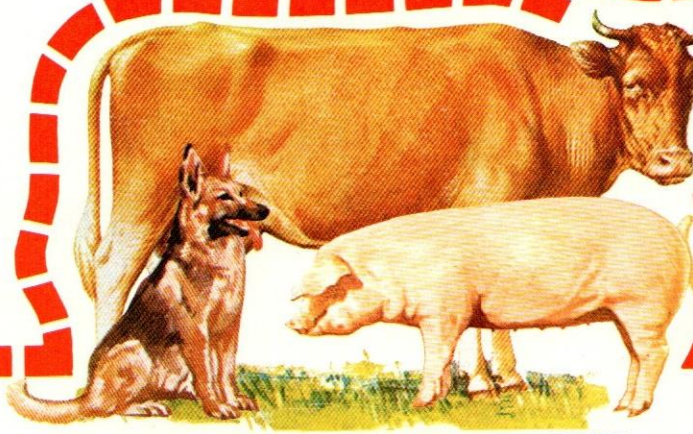

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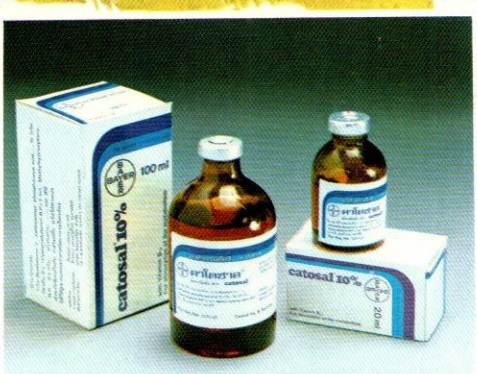


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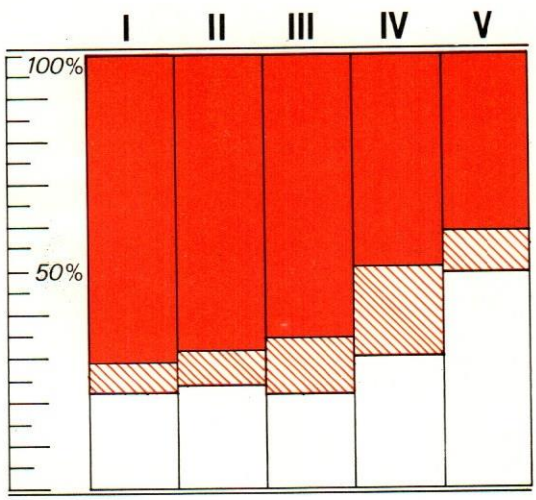


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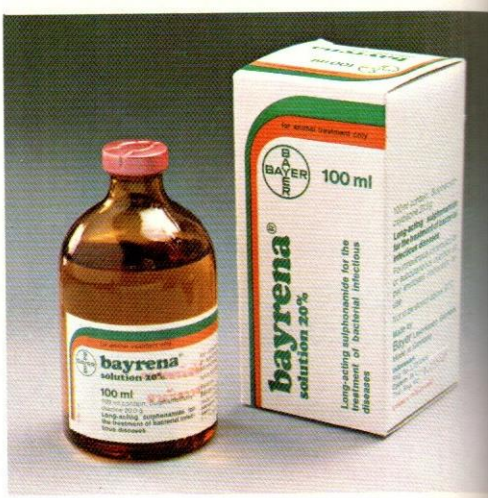


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## Human Aflatoxicosis

**A. General information** Some strain of *Aspergillus flavus* or *Aspergillus parasiticus* produced aflatoxins in our staple foods, and these can be ingested by man either directly from foods or indirectly after cousumption of products from animals fed aflatoxin contaminated feeds. Until recently, aflatoxin has been considered as a serious health hazard principally of under developing countries, while there is little health effects in developing countries because of the insistence of the consumer for clean and undamaged products. The problems of aflatoxin in human health will be discussed later on the possibility that aflatoxin may be involved in the etiology of Reye's syndrome in Thai children or human liver cancer in Thai people.

**B. Human Aflatoxicosis in Thailand.**

*Human liver cancer*. Primary liver cancer is a major problem in the developing countries, i.e. Thailand, The high incidence of the tumor in rural, tropical areas points to naturally occurring agents as being the most important. Aflatoxin $B_1$ and the hepatitis B virus are the most important of those other naturally occurring agents.[15] We still believed that mycotoxin hypothesis proposed by Oettle[16] fitted the known incidence data better than any suspect etiologic factors except for hepatitis B virus. There were several reports about aflatoxin induced primary liver cancer in animals such as trout, guppy, duck, rat, guinea pig, ferrets, mice, tree shrews and monkeys. With a total dose ranging from 99 to 840 mg, aflatoxin can induced liver cancer in monkey within 4 to 6.1 years.[1,14,22] There were 3 cases with hepatocellular carcinoma, 1 case with cholangiocarcinoma and 1 case with hemangioendotheliosarcoma found in these experimental monkeys.

Interaction between environmental contaminants and liver cancer induced by aflatoxins seems very important. In one of our experiment[7] we added dimethylnitro-

samine (DMN, a naturally occurring hepatic carcinogen) with aflatoxin $B_1$ ($AFB_1$, a very highly potent and naturally occurring hepatic carcinogen). We found that DMN enhanced the liver tumors induced by $AFB_1$. This liver tumor is the result of the combined effect of DMN and $AFB_1$. Conversely, however, when $AFB_1$ was fed simul taneously with alpha benzenehexachloride (∝-BHC, a derivative of lindane, an organochlorine insecticide), we found that the liver tumor was totally inhibited by ∝-BHC.[4, 6]

The studies of Dr. Shank in Thailand in 1967 - 1969 were the part of our country wide survey for aflatoxin and liver cancer incidence. Food was sampled from randomly chosen families in representative villages in Ratchaburi, Sing Buri and Songkhla. Significant contamination in That staple food is shown in Table 8. There are dried chilli peppers and dried fish. Estimated aflatoxin intake from food in our country and crude liver cancer rate are shown in Table 9 and these data demonstrate that an increased intake of aflatoxin is possible associated with liver cancer incidence.

*Table 8.* Aflatoxin contamination of selected Thai foods market sample, 1967-1969.[19]

| Foodstuffs | % contamination | Aflatoxin Content | |
|---|---|---|---|
| | | mean | maximum (ppb) |
| Peanuts | 49 | 1,530 | 12,156 |
| Corn | 35 | 400 | 2,730 |
| Chilli peppers | 11 | 125 | 966 |
| Millets | 11 | 67 | 248 |
| Dried fish | 5 | 166 | 772 |
| Mung beans | 5 | 16 | 112 |
| Rice | 2 | 20 | 98 |

*Table 9.* Dietary aflatoxin intake and liver cancer incidence in Thailand.[20]

| Province | Mean aflatoxin intake ($\mu$g/kg b.wt./family) | Liver cancer incidence (new cases/$10^5$/yr) |
|---|---|---|
| Songkhla | 5-8 | 2 |
| Ratchaburi | 45-77 | 6 |
| Sing Buri | 73-81 | ND |

*Reye's syndrome.* Reye's syndrome is an acute and often fatal childhood illness which is characterized by encephalopathy and fatty degeneration of the viscera the so-called EFVD. Clinically, the main features of this syndrome are vomiting, convulsions and coma. Hypoglycemia, hypoglycorrhagia and elevated serum transaminese are the most constant biochemical abnormalities. The major autopsy findings are fatty change in the liver and kidneys with cerebral edema. Patients with similar clinicopathological features are observed in Thailand as Udon encephalopathy by physicians in Udon Thani province before 1969.

The cases of Reye's syndrome reported from our country are shown in Table 10. This data indicates that the disease is prevalent throughout the entire northeastern part, lesser in the northern and central parts and rare in the southern part of Thailand.[3]

*Table 10.* Distribution of Reye's sydrome in Thailand, 1963–1976.[3]

| Region | Province | Number of cases |
|---|---|---|
| North | Chiangmai | 5 |
| South | – | – |
| Central | Bangkok Metropolis | 98 |
| Northeast | Khon Kaen | 270 |
| | Nakhon Ratchasima | 29 |
| | Udon Thani | 251 |
| | Chaiyaphum | 33 |
| | Kalasin | 6 |
| | Maha Sarakham | 33 |
| | Ubon Ratchathani | 86 |
| | Si Sa Ket | 9 |
| | Yasothon | 2 |

A comparison of Reye's syndrome in city and rural areas of Thailand is reported elsewhere[3] and the definite etiologic agents of Reye's syndrome is not known at the present time. However, no virus could be isolated from autopsy tissue obtained from 26 cases in the northeast.[17] Aflatoxin and other mycotoxins were indicated as possible causes of Reye's syndrome, e.g. aflatoxin $B_1$ was detected in one or more specimens from 22 out of 23 cases of the syndrome.[18] In addition similarities between Reye's syndrome and those changes produced by experimental aflatoxin $B_1$ intoxication in monkey have been reported.[9] It has been suggested that some cases of the syndrome reported in New Zealand,[8] Czechoslovakia,[12] and the United States[10] may also be related to aflatoxins.

*Table 11.* Liver aflatoxin in Reye's syndrome.[10]

| Country | Age | Sex | $AFB_1$ ($\mu$g/kg liver) |
|---|---|---|---|
| Thailand | 2 yr | Male | 93 |
| | 13 yr | Female | 47 |
| New Zealand | 22 mo | Male | 5–50 |
| | 8 mo | Female | 5–50 |
| Czechoslovakia | 1 yr | Female | ND |
| | 22 mo | Female | ND |
| US. | 15 yr | Female | 22.5 |

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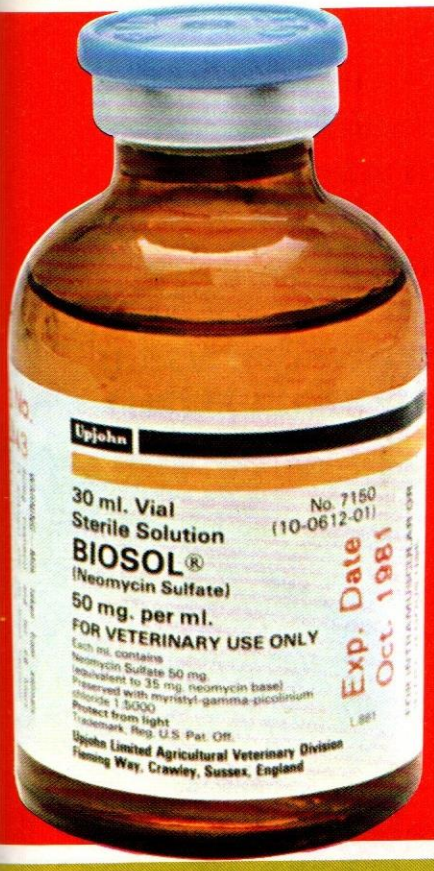


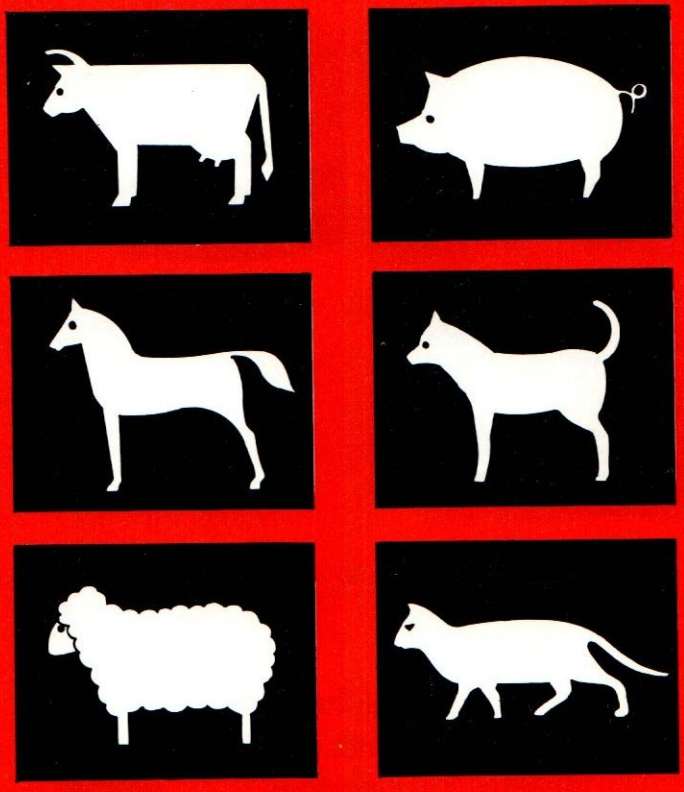

**วิธีใช้**

**ในวัว ควาย ม้า แพะ แกะ สุกร สุนัข แมว ใช้ 2-5 ม.ก.**ต่อน้ำหนัก 1 ก.ก. **หรือ 1 ซี.ซี.** ต่อน้ำหนักตัว 10-20 ก.ก.
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**ใช้ 10 ม.ก.** ต่อ น.น.ตัว 1 ก.ก.
**หรือ 1 ซี.ซี.** ต่อ น.น.ตัว 5 ก.ก.

**ใน เป็ด ไก่ ห่าน**
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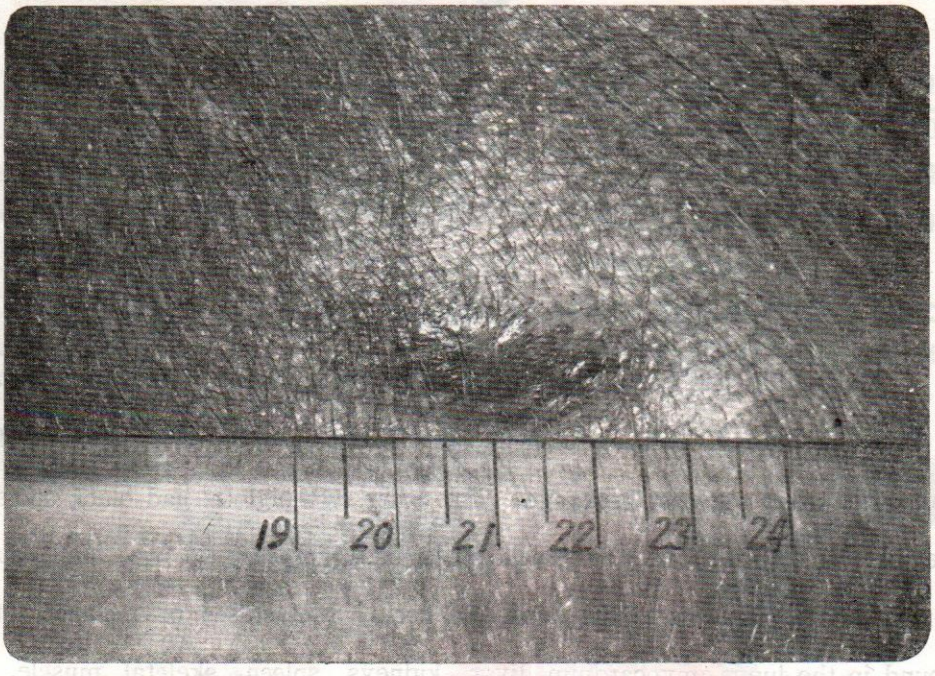
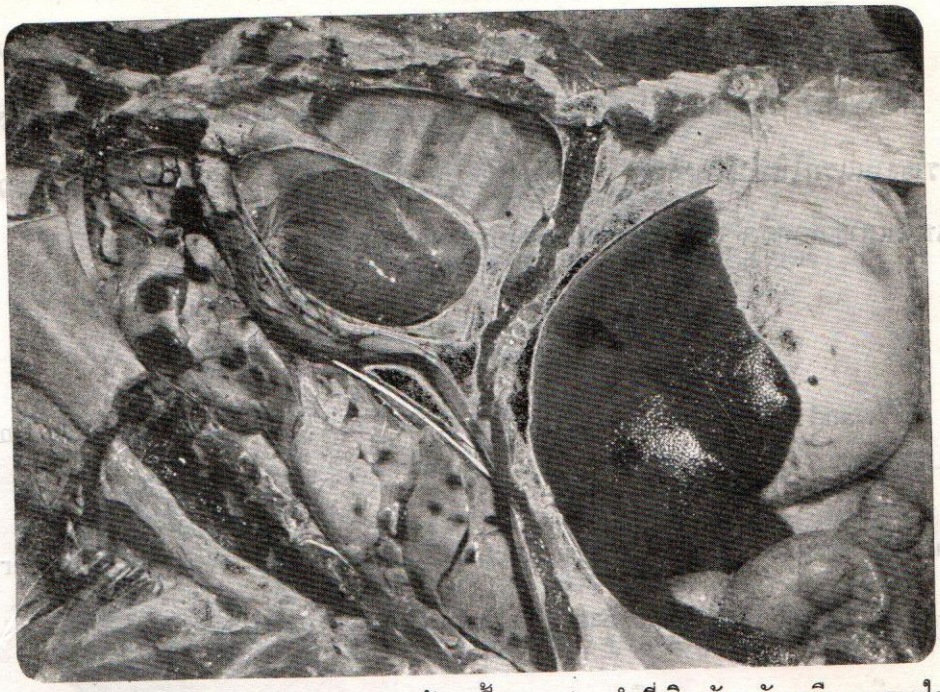
จับดูนิ่ม ขนาด 3.5 x 1.5 ซม. (รูปที่ 1) ต่อมาก้อนเนื้องอกสีดำนี้จะแตก มีน้ำสีดำไหลออกมา และเป็นแผลเรื้อรัง รักษาไม่หาย ส่วนที่เหลืออีก 2 ตัวพบเป็นจุดสีดำที่ผิวหนังบริเวณสวาบ เป็นลักษณะนูนขึ้นมาจากผิวหนังปกติ หนึ่งในจำนวนนี้ตายเมื่ออายุได้ 2 สัปดาห์ เมื่ออายุประมาณเดือนเศษจะพบว่า จุดสีดำที่ผิวหนังแผ่ขยายใหญ่ขึ้นและกระจายไปบริเวณอื่นทั่วร่างกาย เช่น ขาหน้า-หลัง บริเวณหัว ลำตัว เป็นต้น ต่อมน้ำเหลืองที่บริเวณสวาบและบริเวณคอขยายใหญ่ขึ้น ลูกสัตว์ไม่เจริญเติบโตเท่าที่ควร




**รูปที่ 1** ลักษณะก้อนเนื้องอกปานดำที่ผิวหนัง เมื่อแตกจะมีน้ำสีดำไหลออกมาและเป็นแผลเรื้อรัง รักษาไม่หาย

**การผ่าชาก**

นอกจากก้อนเนื้องอกสีดำใต้ผิวหนังซึ่งเห็นได้จากภายนอกแล้ว ยังพบจุดสีดำนี้ได้แผ่กระจายไปทั่วร่างกายเช่น กล้ามเนื้อ ต่อมน้ำเหลือง หัวใจ ปอด ตับ ผนังของกระเพาะอาหาร ม้าม ไต (รูปที่ 2) และยังพบในโพรงจมูกด้วย ต่อมน้ำเหลืองต่าง ๆ มีขนาดใหญ่ขึ้น



**รูปที่ 2** แสดงลักษณะการแพร่กระจายของก้อนเนื้องอกปานดำที่เกิดกับอวัยวะอื่น ๆ ภายในร่างกาย ในภาพจะเห็นเกิดขึ้นที่ปอด ตับและผนังของกระเพาะอาหาร

จากการศึกษาด้วยกล้องจุลทรรศน์พบว่า ก้อนเนื้องอกปานดำแผ่กระจายเข้าไปในเนื้อเยื่อต่าง ๆ เหล่านั้น และเป็นชนิดร้ายแรง

**วิจารณ์**

Gase ได้รายงานการพบเนื้องอกปานดำในลูกสุกรอายุ 5 สัปดาห์ เมื่อปี ค.ศ. 1964 (อ้างโดย Dunne and Leman, 1975) โดยพบเป็นก้อนเนื้อสีดำที่ผิวหนังบริเวณก้น ขนาดเส้นผ่าศูนย์กลาง 7-8 ซม. จากการผ่าซากพบการกระจายของเนื้องอกไปยังต่อมน้ำเหลืองทั่วไป ตับ ไต ปอด หัวใจ สมอง และกล้ามเนื้อหัวใจ นอกจากนี้ Kara and Leipold ได้รายงานไว้เมื่อปี 1981 (In : Veterinary Referenec Service, 1982) ว่า จากการผ่าซากลูกสุกรพันธุ์ดูรอคเจอร์ซี่ 3 ตัว อายุประมาณ 3 เดือน พบเนื้องอกปานดำแต่กำเนิดที่ผิวหนังบริเวณไหล่และสวาบ ทั้งยังกระจายไปต่อมน้ำเหลืองต่าง ๆ ปอด ม้าม สมอง ผนังกระเพาะอาหาร กล้ามเนื้อหัวใจ ลูกสุกรตัวหนึ่งพบมีน้ำสีดำในก้อนเนื้องอกด้วย

จากการพบเนื้องอกปานดำในลูกสุกรพันธุ์ดูรอคเจอร์ซี่แต่กำเนิดที่สถานีบำรุงพันธุ์สัตว์ตาก มีลักษณะภายนอกและจากการผ่าซากตรงกับรายงานที่มีผู้พบและรายงานไว้ก่อนแล้วข้างต้น แต่เป็นรายงานแรกในประเทศไทย สาเหตุการเป็นครั้งนี้ สันนิษฐานจากพันธุกรรมร่วมกับการผสมพันธุ์ในสายเลือดชิด ซึ่งจะได้ติดตามในครอกต่อ ๆ ไปอย่างใกล้ชิด

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