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Neurophysiological and behavioral effects of mycotoxin deoxynivalenol and fumonisin

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Two experiments were conducted to compare the effects of feeding blends of wheat grains naturally contaminated with deoxynivalenol (DON) and maize products contaminated with Fusarium (FUM) mycotoxins on brain regional concentration of brain dopamine (DA), norepinephrine (NE) and serotonin (5-HT) in hippocampus, mid brain, cortex, striatum, pons and medulla and cerebellum of male albino mice. Daily feeding of wheat or maize grains contaminated with deoxynivalenol in a dose level (803 µg/kg) or fumonisin in a dose level (1330 ppb) for six weeks caused highly significant increase in dopamine (DA), norepinephrine (NE) and serotonin (5-HT) contents, in most of the studied mice brain areas. When all the studied brain areas were compared, it can be concluded that hypothalamus-dopamine concentration was more sensitive towards the studied toxicants. On the other hand, except for norepinephrine in pons and medulla oblongata, there was a significant increase in epinephrine and serotonin levels at all the studied brain areas. Maximal concentration, however, was attained in the cortex for both neurotransmitters. Additionally, rearing behavior was found to increase following feed intake of the test feed and deoxynivalenol was found to modulate more behavioral disturbances as compared with fumonisin. The data recorded also showed a highly significant increase in the aggressive and locomotor behavior of the intoxicated albino mice.

Key words: Deoxynivalenol, fumonisin, brain areas, monoamines.

INTRODUCTION

Fusarium mycotoxins are the largest group of mycotoxins, which includes more than 200 known metabolites of fungi (Doll et al., 2008; Peracia et al., 2010). They are synthesized by many species of fungi, mainly by Fusarium (F. graminearum and F. culmorum) (Singh et al., 2008) and have been detected and abundant in buckwheat, popcorn, sorghum, triticale, and other food products including flour, bread, breakfast cereals, noodles, infant foods, pancakes, malt and beer (Sobrova et al., 2010). According to the first author, Fusarium mycotoxins are abundant in cereals and their products and have been a persistent problem to farmers and the animal husbandry industry in Eastern Europe and developing countries. Recently, however, research carried out implicated many toxin-producing fungi, such as Stachybotrys (Li and Yang, 2005), Penicillium (Ustianowski et al., 2008), Aspergillus (Wobeser, 1997) and Fusarium (Nelson et al., 1993; Kim et al., 2012) species to indoor air quality problems and building related illnesses and many are harmful to humans and animals when inhaled, ingested or even brought into contact with human skin (Hendry and Cole, 1993). Mycotoxins can cause a variety of short term as well as long-term health effects, ranging from immediate toxic response to potential long-term carcinogenic and teratogenic effects (Baines et al., 2011; Prouillac et al., 2012).

Deoxynivalenol known colloquially as "vomitoxin" is a mycotoxin that commonly contaminates cereal-based foods worldwide; it is one of several mycotoxins produced by Fusarium species that frequently infect corn, wheat, oats, barley, rice and other grains in the field or during storage (Castillo et al., 2008; Schmidt et al., 2011). The exposure risk to human is directly through foods including flour, bread, breakfast cereals (Krieger et al., 2010) or indirectly through foods of animal origin (Smith et al., 1995; Berry, 1998). Deoxynivalenol affects animal
and human health causing acute temporary nausea, vomiting, diarrhea, abdominal pain, headache, dizziness, and fever (Sobrova et al., 2010). At the molecular level, deoxynivalenol disrupts normal cell function by inhibiting protein synthesis via binding to the ribosome and by activating critical cellular kinases involved in signal transduction related to proliferation, differentiation and apoptosis (Wollenhaupt et al., 2007; Abdel-Twab et al., 2009). Relative to toxicity, there are marked species differences, with the pig being most sensitive to deoxynivalenol, followed by rodent > dog > cat > poultry > ruminants (Rottet et al., 1996). With respect to chronic effects, deoxynivalenol decreased nutritional efficiency (Girardet et al., 2011), immune function (Garcia et al., 2009) and reproduction (Gowrinathan et al., 2011). Critical areas for future deoxynivalenol research include molecular mechanisms underlying neuronal toxicity, sensitivity of neuronal cells/tissues relative to other species, emetic effects in primates, epidemiological association with gastroenteritis and chronic disease in humans, and surveillance in cereal crops worldwide. On the other hand, fumonisins are a recently discovered class of mycotoxin and have been attracting the attention of scientists and farmers where they have been shown to cause a variety of toxic effects in both experimental animals and livestock and are associated with damage to brain and pulmonary functions (Voss et al., 2011). In addition, it was observed that, the ELEM syndrome observed in equids has already been described in the subacute with fumonisin toxicity section. Till now, the effect of fumonisin on humans has not been fully established. Salas et al. (1999) described a patient who after being exposed to Aspergillus, Penicillium and Rhizopus developed fatigue, headache, slowness of thinking and severe tremors. In addition, as described by Kilburn (2003) in the mold-exposed individuals neurobehavioral manifestations are described as abnormal decrease in steady balance, reaction time, blink-reflex latency, color discrimination and visual fields. To the authors’ knowledge, Fumonisins and DON mycotoxins have been observed to occur in a different potential feed grain source and the behavioral changes of their daily exposure on the basis of neuronal monoamines concentration in different brain areas have not been previously reported. Therefore, the purpose of this research was to throw light on Fumonisins and deoxynivalenol concentrations on different collected food samples and to investigate a comparative study on the effect of their daily administration on the behavioral activity and to create exploration on the basis brain areas epinephrine (E), norepinephrine (NE) and serotonin (T3-H) changes.

MATERIALS AND METHODS

Experimental animals

The animals used in the present experiment were mice of strain MFI, of about 25-30 g BW. They were obtained from the animal house at the King Fahd Research at King Abdulaziz University. Laboratory animal welfare including environment, housing and management were carried out with recommendations for the proper care and use of laboratory animals of American Association for Accreditation of Laboratory Animal Care (AAALAC) (1996) and Al-Hazmi (2001). The animals were kept under good ventilation with 12 h light and dark cycle and fed on free drinking of water contaminated with appropriate concentration of either Fumonisin or Deoxynivalenol.

Mycotoxin samples

For deoxynivalenol analysis, 12 stored samples of wheat grains were collected from local province of El-Qassim and Najran in 2010 and for Fumonisin analysis, 45 samples were collected from Jeddah market in 2009. Immediately, the collected samples were stored at low temperature (4°C) in dark place till extraction and determination of mycotoxins concentration.

Toxin extraction and calculation of concentration

The Isolation of deoxynivalenol (Sydenham et al., 1991) and Fusarium (AOAC 2007) in the collected food samples were carried out according to standard methods. The extracted toxin concentrations were determined by HPLC analysis using fluorescence detection as described by Sydenham et al. (1991) (Figure 1). All samples were measured against reference standard of known prepared solutions of three levels of Fumonisins and Deoxynivalenol solutions. The standard samples were obtained from Sigma, St. Louis, USA.

Treatments

After the determination of the appropriate concentration of NOD and Fumonisins in the collected food samples, the animals were divided into the following groups: Group I (normal control): consisted of 12 normal mice which served as normal control and were allowed to drink normal water; Group II (Mycotoxin treated): the animals of this group (24 mice) were subdivided into two subgroups: the animals of subgroup a (12 mice) were allowed to drink water contaminated with deoxynivalenol (803 mg/kg) and the animals of Subgroup B were allowed to drink water contaminated with fumonisins (1850 µg/kg) throughout the experimental period (six weeks). At the end of the experimental periods, each was divided into two equal half, one was decapitated for catecholamine determination and the other was used for the behavioral experiment.

Catecholamines determination

At the end to the tested period (6th week), six animals of both control and intoxicated animal groups were rapidly scarified; the brains were rapidly dissected, carefully removed and quickly separated into cerebellum, brain stem, striatum, cerebral cortex, hypothalamus and hippocampus. The separated brain regions were wiped dry, weighed and wrapped into plastic films and quickly frozen in dry ice pending analysis. The catecholamines, epinephrine, norepinephrine and dopamine, were extracted and estimated according to the method of Chang (1964) and modified by Ciarlone (1978).

Behavioral tests

After six weeks on their typical water which contained the...
Table 1. Deoxynivalenol level (µg/kg) and Fumonisin (ppb) concentration detected in wheat samples and maize food products obtained from different sources in Saudi Arabia.

<table>
<thead>
<tr>
<th>Mycotoxin</th>
<th>Situation</th>
<th>Number of collected samples</th>
<th>Number of negative results</th>
<th>Number of positive results</th>
<th>Range</th>
<th>Mean value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deoxynivalenol</td>
<td>Najran</td>
<td>6</td>
<td>-</td>
<td>6</td>
<td>70.71-506.23</td>
<td>219.02±89.1</td>
</tr>
<tr>
<td></td>
<td>Qassim</td>
<td>6</td>
<td>-</td>
<td>6</td>
<td>38.88-617.39</td>
<td>237.9±119.8</td>
</tr>
<tr>
<td>Fumonis (PPb)</td>
<td>Jeddah</td>
<td>45</td>
<td>30</td>
<td>15</td>
<td>1330-1850</td>
<td>1590±260</td>
</tr>
</tbody>
</table>

mycotoxin, deoxynivalenol or Fumonis, three measures of behavior test were investigated for each intoxicated animal. The locomotory and aggressive behavioral tests were determined by 1- Cannula brake test and 2- Tube Restraint Test.

Statistical analyses

Data were analyzed as Mean±SE . Statements of statistical significance were based on P ≤ 0.05. Differences between means were considered statistically significant at p<0.05 variance (Woolson, 1987). All statistical analysis were computed by SPSS version 10.

RESULTS

Mycotoxins concentration in the collected samples

In the present study, the natural occurrence of deoxynivalenol; the most important group of fusarial mycotoxins in wheat grains harvested in 2010 from the cultivating location of Najran and El-Qassim was conducted. In addition, fumonisin mycotoxin of maize products from supermarkets in Jeddah was recorded. The number of analyzed samples for deoxynivalenol were 12 samples and for fumonisin were 45. Mycotoxin contents were analyzed using an efficient high performance liquid chromatography (HPLC) with UV visible detector set at 220 nm. Our investigation showed deoxynivalenol contamination with amounts varying from 70.71 to 506.23 and 38.88 to 617.39 µg/g in Najran and El- Qassim, respectively (Table 1). Also our results investigated the presence of 6 samples from 45 maize products (11.11%) were contaminated with Fumonis in concentration of 1330 ppb. At All the tested positive wheat samples and maize products the detected values exceeded the maximum permitted limit (1.25 µg/g for deoxynivalenol and 100 ppb for fumonisn) set by the European Commission for unprocessed cereals and maize products.

Effect on catecholamines

Tables 2 to 4 illustrate the epinephrine (E), norepinephrine (NE) and serotonin (T3-H) concentrations on brain regions (hippocampus, midbrain, cortex, striatum, pons and cerebellum) post dietary supplementation with deoxynivalenol and Fumonisins for 6th weeks. In the studied brain areas, dopamine concentration attained significant increase and hypothalamus area revealed the highest drastic effect (32.68%). On the other hand, as a result of fumonisins intoxication, all the studied brain areas exhibited elevated dopamine concentration. The highest recorded value, however, was attained in hypothalamus (35.66%) followed by striatum (33.82%) and pons (22.92%) in a decreasing order.

Results in Table 3 reveal the significant increase in brain areas epinephrine concentration as a result of daily intake of the studied mycotoxins. The potent effect was attained in the brain cortex, and the values detected increased by 50.53 and 50.03% post intoxication with deoxynivalenol and Fumonisins, respectively. On the other hand, the least affected area for epinephrine change was attained in pons and medulla brain area, at which the values detected was insignificantly (P>0.05) affected.

As regard to the effect of the studied mycotoxins on serotonin level in the studied brain areas (Table 5), data recorded, showed more or less the same pattern of increased disruption. The maximal transient increases however, was attained in the cortex post treatments with both toxins, and the values detected were significantly increased by 66.16 and 45.96% post treatments with deoxynivalenol and fuminisin, respectively. On the other hand, the least potent effect was recorded in hippocampus.

Locomotory and aggressive behavior

Other impacts related with the daily management of deoxynivalenol and fuminis are behavioral toxicity related with the nervous system which can often be recognized as animals show a disrupted abnormal behavior. As shown in the recorded
Table 2. Effect of chronic oral administration of deoxynivalenol (803 mg/kg) and fumonisin (1850 µg/kg) on dopamine (DA) content in the different brain areas of albino mice.

<table>
<thead>
<tr>
<th>Group</th>
<th>Hippocampus</th>
<th>Midbrain</th>
<th>Hypothalamus</th>
<th>Cortex</th>
<th>Striatum</th>
<th>Pons and Medulla</th>
<th>Cerebellum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>894 ± 4.7</td>
<td>489 ± 2.2</td>
<td>618 ± 3.4</td>
<td>194 ± 1.0</td>
<td>809 ± 3.3</td>
<td>515 ± 2.6</td>
<td>399 ± 1.1</td>
</tr>
<tr>
<td>DON</td>
<td>1086 ± 3.5**</td>
<td>597 ± 2.5**</td>
<td>918 ± 3.9***</td>
<td>279 ± 1.2***</td>
<td>1034 ± 5.5***</td>
<td>676 ± 3.2***</td>
<td>465 ± 2.2*</td>
</tr>
<tr>
<td>Control</td>
<td>937 ± 4.5</td>
<td>522 ± 2.7</td>
<td>645 ± 2.5</td>
<td>220 ± 1.0</td>
<td>816 ± 3.9</td>
<td>528 ± 3.0</td>
<td>414 ± 22</td>
</tr>
<tr>
<td>FUM</td>
<td>1013 ± 3.2ns</td>
<td>534 ± 2.1ns</td>
<td>875 ± 4.3***</td>
<td>266 ± 1.7**</td>
<td>1092 ± 4.3***</td>
<td>649 ± 2.7***</td>
<td>481 ± 2.9**</td>
</tr>
</tbody>
</table>

Data are represented as mean± SE (N=6). *Significant P<0.05 as compared with the control group; **significant at P<0.01 as compared with control group; ***significant P<0.001 as compared with control group.

Table 3. Effect of chronic oral administration of deoxynivalenol (803 mg/kg) and fumonisin (1850 µg/kg) on norepinephrine (NE) content in the different brain areas of albino mice.

<table>
<thead>
<tr>
<th>Group</th>
<th>Hippocampus</th>
<th>Midbrain</th>
<th>Hypothalamus</th>
<th>Cortex</th>
<th>Striatum</th>
<th>Pons and medulla</th>
<th>Cerebellum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>809 ± 3.0</td>
<td>444 ± 2.2</td>
<td>674 ± 3.3</td>
<td>198 ± 1.0</td>
<td>716 ± 3.2</td>
<td>599 ± 2.9</td>
<td>419 ± 1.6</td>
</tr>
<tr>
<td>DON</td>
<td>965 ± 4.0**</td>
<td>599 ± 3.6***</td>
<td>899 ± 3.1***</td>
<td>329 ± 1.7**</td>
<td>897 ± 3.3***</td>
<td>792 ± 3.8***</td>
<td>563 ± 2.9***</td>
</tr>
<tr>
<td>Control</td>
<td>798 ± 3.7</td>
<td>432 ± 2.4</td>
<td>659 ± 2.1</td>
<td>198 ± 1.7</td>
<td>686 ± 3.5</td>
<td>582 ± 4.3</td>
<td>449 ± 2.9</td>
</tr>
<tr>
<td>FUM</td>
<td>989 ± 4.5***</td>
<td>599 ± 3.4**</td>
<td>856 ± 4.4***</td>
<td>289 ± 1.9*</td>
<td>907 ± 4.3***</td>
<td>792 ± 3.5***</td>
<td>563 ± 2.7***</td>
</tr>
</tbody>
</table>

Data are represented as mean± SE (N=6). *Significant P<0.05 as compared with the control group; **significant at P<0.01 as compared with control group; ***significant P<0.001 as compared with control group.

Table 4. Effect of chronic oral administration of deoxynivalenol (803 mg/kg) and fumonisin (1850 µg/kg) on serotonin (5-HT) content in the different brain areas of albino mice.

<table>
<thead>
<tr>
<th>Group</th>
<th>Hippocampus</th>
<th>Midbrain</th>
<th>Hypothalamus</th>
<th>Cortex</th>
<th>Striatum</th>
<th>Pons and medulla</th>
<th>Cerebellum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>865 ± 3.4</td>
<td>529 ± 2.8</td>
<td>789 ± 3.7</td>
<td>188 ± 0.7</td>
<td>809 ± 4.4</td>
<td>555 ± 3.5</td>
<td>457 ± 2.5</td>
</tr>
<tr>
<td>DON</td>
<td>1045 ± 3.9**</td>
<td>738 ± 2.8***</td>
<td>913 ± 3.2**</td>
<td>283 ± 1.0***</td>
<td>1093 ± 3.9**</td>
<td>516 ± 2.7ns</td>
<td>554 ± 2.3**</td>
</tr>
<tr>
<td>Control</td>
<td>849 ± 3.1</td>
<td>528 ± 2.2</td>
<td>680 ± 2.7</td>
<td>189 ± 1.2</td>
<td>758 ± 2.9</td>
<td>533 ± 3.0</td>
<td>457 ± 2.3</td>
</tr>
<tr>
<td>FUM</td>
<td>964 ± 3.8*</td>
<td>641 ± 2.8**</td>
<td>854 ± 3.3***</td>
<td>293 ± 1.4***</td>
<td>893 ± 3.2**</td>
<td>509 ± 2.6ns</td>
<td>554 ± 2.9**</td>
</tr>
</tbody>
</table>

On the other hand, the aggressive behavioral test showed increase in the number of upside rears.

Data, the signs of deoxynivalenol and fumonisin neurotoxicity include increased alterations in the locomotor activity, wall rears and number of crossed squares and a decrease in immobility and
**Table 5.** Effect of deoxynivalenol (803 µg/kg) and fumonisin (1850 ppb) on the locomotor behavior of albino mice.

<table>
<thead>
<tr>
<th>Group</th>
<th>Latency to explore first hole</th>
<th>Locomotion (+++</th>
<th>Immobility (+++</th>
<th>Upside Rears (+++</th>
<th>Wall rears (+++</th>
<th>Squares crossed (+++</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 (Control)</td>
<td>2 (2-3)</td>
<td>491.5 (470-512)</td>
<td>72.5 (50-91)</td>
<td>28.5 (13-64)</td>
<td>97 (61-117)</td>
<td>659 (634-714)</td>
</tr>
<tr>
<td>Deoxynivalenol intoxicated group (803 µg/kg)</td>
<td>3 (3-4)*</td>
<td>570 (518-595)**</td>
<td>48.5 (32-79)**</td>
<td>18 (6-32)**</td>
<td>130 (87-169)**</td>
<td>806 (770-850)**</td>
</tr>
<tr>
<td>Fumonisin intoxicated group (1850 µg/kg)</td>
<td>2 (2-3)</td>
<td>549.5 (522-571)*</td>
<td>53 (32-69)**</td>
<td>20.5 (5-41)**</td>
<td>56.5 (49-112)**</td>
<td>719 (665-790)**</td>
</tr>
</tbody>
</table>

+ significant (p< 0.05) according to Kruskall Wallis test; +++ highly significant (p< 0.001) according to Kruskall Wallis test.

**Table 6.** Effect of deoxynivalenol (803 µg/kg) and Fumonisin (1850 ppb) on the aggressive behavior of albino mice.

<table>
<thead>
<tr>
<th>Dose (1850 µg/kg)</th>
<th>Latency (second) to First bite (Median with range, +++</th>
<th>Number of bites (Median with range, +++</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 (Control)</td>
<td>15 (10-25)</td>
<td>192.5 (177-233)</td>
</tr>
<tr>
<td>Deoxynivalenol</td>
<td>25 (15-40)**</td>
<td>107 (55-198)***</td>
</tr>
<tr>
<td>Fumonisin</td>
<td>25 (15-40)**</td>
<td>107 (55-198)***</td>
</tr>
</tbody>
</table>

+++ more highly significant (p< 0.001) according to Kruskall Wallis test; ** highly significant change (p< 0.01) according to Mann-Whitney U test; *** more highly significant change (p<0.001) according to Mann-Whitney U test.

bites and aggressive behavior (Table 6). Along with this, the intoxicated animals showed similar responses as increased latency to first bite and decreased latency in number of bites.

**DISCUSSION**

Mycotoxin represents one of the negative factors influencing health of animals. Recently, intense attention has been paid to deoxynivalenol and Fumonisin mycotoxins which occurs predominantly in grains such as wheat, barley, oats, rye, and maize (Domijan et al., 2005; Garcia et al., 2009; Krnjaja et al., 2012). In the present study, higher levels of both toxins had been found in the studied wheat grains and maize products. The recorded mean values were about 803 µg/kg for deoxynivalenol and 1880 ppb for fumonisin. As reported with several investigators, the incidence of both are strongly associated with species of fungi, mainly called *Fusarium* (*F. graminearum* and *F. culmorum*) (Barros et al., 2009) which grows optimally at a temperature of 21 to 25°C and at a water activity above 0.87. According to Leistner and Rödel (1976), Nelson et al. (1993) and Domijan et al. (2005), the main factors that influence growth and production of Aspergillus toxins include environmental, chemical and biological factors. Under some circumstances, these effects are additive and under others, the implication is synergistic interactions which lead to a combined effect of greater production. In Saudi kingdom, the contamination of cereal grains with mycotoxins is very common. Such a situation is chiefly favored by the red sea climate of the country, which plays a critical role in the development of toxigenic fungi and consequently, the accumulation of toxic metabolites. The great difference between the type and concentration of the studied mycotoxins, however, are mainly attributed to the climatic differences.

The current data showed that in all the studied brain regions and as regard to the non-toxicated groups, norepinephrine (NE), dopamine (D) and
serotonin (5-HT) concentrations were increased at most the studied brain regions. The present data support the work of larger alterations in neurotransmitter and metabolite concentrations, which were seen in rat brain regions in response to feeding blends grains contaminated with Fusarium mycotoxins on brain regional of different animals (Swamy et al., 2004; Girish et al., 2007; Kunio and Koji, 2011). According to Eisenhofer et al. (2004), dopamine and norepinephrine levels are significantly elevated in the caput and corpus regions as a result of mycotoxin (T-2 toxin). Also, as reported by Swamy et al. (2004), linearly increased cortex 5-hydroxytryptamine (5HT, serotonin) concentrations were increased in the brain of pigs and broiler chickens as a result of feeding on grains contaminated with Fusarium mycotoxins. Furthermore, according to Pastorová and Várady (1996), a significant increase in dopamine, norepinephrine and epinephrine levels was observed at 24 and 72 h after the mycotoxin administration. Our finding on monoamines change confirms the previously mentioned hypothesis and supports the suggestion of brain monoamines synthesis and/or turnover as a result of mycotoxins intoxication. According to Edelstein and Breakfield (1986) and Youdim et al. (1989), the stimulatory effect of monoamines synthesis is mainly associated to MAO activity of the adrenal medulla and the stimulatory effect of glucocorticoids. Otherwise, the recorded elevation in levels of the studied monoamines, particularly of norepinephrine and dopamine during mycotoxins intoxication are mainly associated with increases in catecholamine synthesizing enzymes in the adrenal medulla as well as in the peripheral sympathetic ganglia. Various acute stressful stimuli have been shown to alter catecholamine dynamics. As reported by several investigators, mycotoxins caused inflammation, contact erosion, irritation and oxidative stress (Kunio and Koji, 2011) which can be considered as the chemical stress. In the present study, the allowance for contaminated water with either deoxynivalenol or Fumonisins had a more pronounced effect on neuronal dopamine which showed a significant increase (P<0.001) in all the studied brain areas, the maximal increase was attained in the hypothalamus followed by striatum and pons and medulla in a decreasing order. On the other hand, the maximal effects for both epinephrine and serotonin were attained in the cortex. These findings, however, suggest brain areas variability in response to the studied mycotoxins. From our results and from the literature data, we can conclude that, the short term oral administration of deoxynivalenol and fumonisins causes damage to peripheral adrenergic system which is involved in the regulation of some endocrine processes. These changes enhanced behavioural responses. Behavioral analyses of intoxicated mice have generated conflicting results. We therefore analyzed the relationship between behavioral changes and increase of monoamine levels, in groups of mice intoxicated with DON or Fumonisins. The most surprising result was that the mice intoxicated with the tested doses which were naturally occurring in our food were more active to induce behavioral change, the hyperactivity observed in our mice could be due to the increase in dopamine, epinephrine and norepinephrine levels.

REFERENCES


