Journal of Mediterranean Ecology **vol. 12, 2013: 5-11**

Evaluation of the Potential Genotoxic and Mutagenic Effects of Fipronil in Rats

Shenouda M. Girgis[1,*] and Venees F. Yassa[2]

[1,*]*Cell Biology Department, National Research Center, 12622 - Dokki, Giza, Egypt.*
[2] *Animal Health Research Institute, Dokki, Giza, Egypt.*

** Corresponding author: Dr. S.M. Girgis, E-mail: shenoudagirgis10@yahoo.com*

Key word: Genotoxic, mutagenic, micronucleus, fipronil, rats.

Abstract

Fipronil is a broad use insecticide that belongs to the phenylpyrazole chemical group and is used to control a variety of insects. The present study was aimed to evaluate the potential genotoxic and mutagenic effects of this compound in Wistar albino rats exposed to two different doses of fipronil using chromosomal aberrations analysis and micronucleus assay. Rats were divided into 3 groups. The first group treated with 1/4 LD_{50} (25 mg/kg. b.wt). The second group treated with 1/2 LD_{50} (50 mg/kg. b.wt) for 24, 48 and 96 hr, besides the control group. The results of the present study revealed that structural chromosomal aberrations in somatic cells specially centromeric attenuations, fragments, breaks and deletions as well as total structural aberrations and micronuclei increased significantly in rats treated either with 1/4 or1/2 LD_{50} of fipronil for 24, 48 and 96 hours compared to the control group. However, the frequency of numerical variation (peridiploidy) in the fipronil treated groups was not significant compared to the control. The peak of chromosomal aberrations and micronuclei frequency induced by fipronil occurred at 48 hr after treatment. In conclusion, fipronil was found to be genotoxic and mutagenic to rats and induced high rate of chromosomal aberrations and micronuclei. This encourages us to seek for alternative products of natural origin that are efficient in controlling pesticides and neither toxic to living organisms in general nor to the environment.

Introduction

Fipronil is a broad use insecticide that belongs to the phenylpyrazole chemical group and is used to control a variety of insects including ants, beatles, cockroaches, flees, ticks, termites, mole crickets, thrips, rootworms, weevils and other insects (NPIC, 2009). In insects, fipronil disrupts the normal function of the central nervous system by blocking GABA (γ-aminobutyric acid) gated chloride channels. This prevents the uptake of chloride ions, causing over stimulating neurons, paralysis and death of the insects (Gant et al., 1998).

Fipronil was registered for use in the United States in May 1996. It was classified as a possible human carcinogen based on increase in thyroid follicular cells tumors in both sexes of the rats (USEPA, 2000 and Leghait et al., 2009). Fipronil is highly efficient in the control of pests, including those resistant to pyrethroid, organophosphate and carbamate insecticides (Kidd and James, 1991). In natural conditions and in mammals it can degrade and form toxic metabolites,

which are characterized by high toxicity in contrast to other insecticides or fipronil itself (Hainzl and Casida, 1996; Hainzl et al., 1998 and Parsons, 2009).

Although the chromosomal aberration assay in Chinese hamster lung cells was positive (Wright, 1995) due to increases in chromatid breaks and chromatid exchanges, the mouse micronucleus test indicated that fipronil was not clastogenic in vivo (UKDEFRA, 2004).

Ghisi et al. (2011) studied the genotoxic effect of fipronil (of 0.05, 0.10 and 0.23 g/L) in the fish Rhamdia quelen using micronucleus. They found that the smallest concentration of fipronil (0.05 g/L) was similar to the control group, while concentrations of 0.10 and 0.23 g/L caused DNA damage. These results suggested that only the high tested concentrations of fipronil cause damage in fish erythrocytes.

The micronucleus assay has been used to assess the toxicity of fipronil. The presence of micronuclei is frequently used to quantify the exposure to chemical or physical agents (Trucker and Preston, 1996 and Krishna and Hayashi, 2000).

Overall, data on mutagenic effects of fipronil on mammals are few and a little is known about its genotoxicity. Therefore, this study aimed to evaluate the potential genotoxic and mutagenic effects of fipronil on albino rats using chromosomal aberrations analysis and micronucleus assay.

Materials and Methods

1-Chemicals:

Fipronil, insecticide, was purchased from N.V.C. Agrovetzaschita, S.P. Company, Moscow, Russia.

2-Experimental animals:

A total of 105 rats used in this study were obtained from the animal house of the National Research Center, Dokki, Giza, Egypt.

3-Experimental design:

Thirty five rats were used for determination of the acute oral LD_{50} of fipronil. Fifteen rats of them were used in the preliminary pilot experiment to determine the range in which the LD_{50} of the insecticide presents and the remaining twenty rats were used for the actual estimation of the $LD_{50.}$ The other seventy rats were used for determination of the genotoxic effect of fipronil and divided as follow: Ten rats were served as control (0 hr.) and sixty rats were divided into two equal groups: Group (1) treated with 1/4 LD_{50} of fipronil and Group (2) treated with 1/2 LD_{50} of fipronil. Ten rats of each group were sacrificed after 24 hr, 48 hr and 96 hr of fipronil exposure (5 rats for chromosomal analysis and 5 for micronucleus assay).

4-Methods:

Determination of acute oral LD_{50} of fipronil was performed mathematically according to the method described by Finney (1964).

Cytogenetic analysis and micronucleus assay of bone marrow cells were performed at the end of each time (24, 48 and 96 hr) using standard protocols. The slides were stained with Giemsa stain and analyzed for structural and numerical chromosomal aberrations (50 metaphases/rat) as described by Agarwal et al. (1994) and for micronucleus assay (two thousands cells/rat, two slides per animal of each treatment) as indicated by Hayashi et al. (1990).

5-Statistical analysis:

Results were expressed as means ± standard errors. The obtained data were analyzed using analysis of variance (ANOVA) according to method of Miller (1997).

Results

In the current work, the acute oral LD_{50} of Fipronil was calculated as 100.35 mg/kg bw in albino rats (Table 1) according to Finney (1964).

Table (1): Determination of acute oral LD_{50} of fipronil in albino rats.

Group	Dose (mg/kg)	Number of rats in each group	Number of dead animals	Mortality %
1	25	5	-	-
2	50	5	1	20
3	100	5	3	60
4	200	5	5	100

Calculation of LD_{50} was as follows:

M = x1+1/2d-dr1/N

Log LD_{50} =Log 200+1/2 Log 2-9/20

= 2.30103+0.150515-0.45

= 2.001545

Therefore, the LD_{50} = 100.35638 mg fipronil /kg. b.wt.

Structural and numerical chromosomal aberrations in somatic cells were listed in Table (2) and Fig. (1). The present results show that structural chromosomal aberrations in somatic cells specially centromeric at-

Table (2): Mean frequency of chromosomal aberrations and micronuclei in bone marrow cells of albino rats treated with fipronil.

Treated group		Structural aberrations					Total structural aberrations	Numerical (peridiploidy)	Micronuclei
		C. A	Frag.	Gap	Break	Del.			
control		**0.40**^{c} ±0.24	0.6^{b} ±0.24	**0.40**^{b} **±0.24**	**0.4**^{b} **±0.24**	**0.6**^{c} ±0.24	**2.6**^{c} ±0.24	**1.4**^{a} ±0.68	**15.4**^{c} ±0.51
24 hr	G1	**1.8**^{b} ±0.20	1.8^{a} ±0.37	**1.00**^{b} ±0.32	**2.6**^{a} ±0.2	**2.4**^{b} **±0.24**	**9.6**^{b} ±0.24	**2.6**^{a} ±0.75	**130.4**^{b} ±3.11
	G2	**2.8**^{a} ±0.3	2.0^{a} ±0.45	**1.4**^{b} ±0.6	**3.2**^{a} ±0.37	**4.4**^{a} ±0.24	**14.2**^{a} ±0.49	**2.4**^{a} ±0.4	**137.2**^{a} ±1.39
48 hr	G1	**2.8**^{b} ±0.2	1.8^{a} ±0.37	**1.6**^{a} ±0.24	**3.2**^{a} ±0.37	**3.00**^{a} ±0.45	**12.4**^{b} ±0.24	**2.4**^{a} ±0.24	**138.2**^{b} ±0.86
	G2	**5.8**^{a} ±0.73	**1.8**^{a} ±0.2	**1.6**^{a} ±0.40	**3.4**^{a} ±0.51	**3.4**^{a} ±0.60	**16.0**^{a} ±0.55	**3.2**^{a} ±0.49	**141.2**^{a} ±1.16
96 hr	**G1**	**2.6**^{b} ±0.4	**1.8**^{a} ±0.37	**0.6**^{b} ±0.24	**4.4**^{a} ±0.4	**3.2**^{a} ±0.58	**12.8**^{b} ±0.58	**2.2**^{a} ±0.2	**141.2**^{b} ±1.17
	G2	**4.8**^{a} ±0.58	**2.0**^{a} ±0.45	**0.6**^{b} ±0.24	**4.4**^{a} ±0.24	**4.4**^{a} ±0.24	**16.2**^{a} ±0.58	**3.2**^{a} ±0.58	**153**^{a} ±2.3

Values with different small superscript letters are significantly different.
Values are expressed as means + S. E.
G1 =1/4 LD50 of fipronil; G2 = 1/2 LD50 of fipronil.
C. A = Centromeric attenuation.
Frag. = Fragment.
Del. = Deletion.

tenuation, fragments, breaks and deletions as well as total structural aberrations increased significantly in rats treated either with 1/4 or 1/2 LD_{50} of fipronil for 24, 48, and 96 hr compared to the control group.

However, there was a slight increase in the frequency of numerical variation (peridiploidy) in the fipronil treated groups compared to the control group, but this difference was not significant.

Concerning the effect of time on chromosomal aberrations induced in fipronil exposed rats (Table 3), the results revealed that there was a significant increase in the frequency of chromosomal aberrations in the low dose fipronil treated groups (24, 48 and 96 hr treated groups) compared to the control group. Approximately, the same effect (values) was obtained in the 48 and 96 hours treated groups within the low dose where, there was no significant difference in the frequency of chromosomal aberrations between the two groups (12.4a $\pm$ 0.24 and 12.8a $\pm$ 0.58). The same

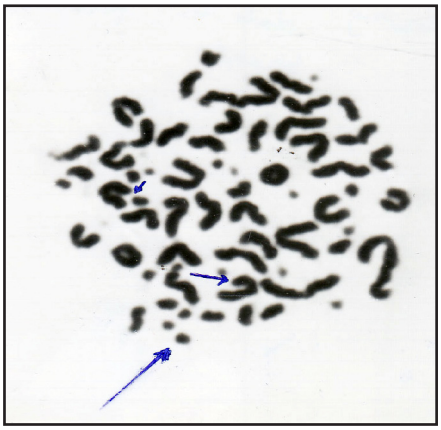

Fig. (1): Metaphase spread showing fragment (large arrow), deletion (medium arrow) and break (small arrow).

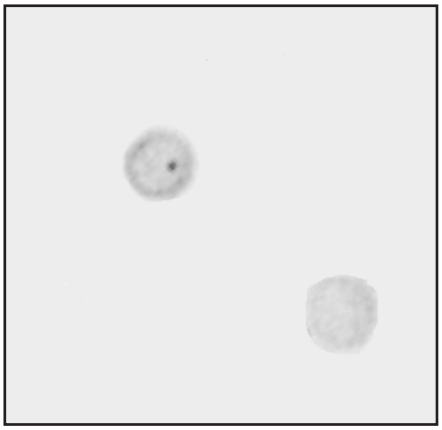

Fig. (2): A micrograph showing micronucleated (left) and normal (right) rat bone marrow cells.

Table (3): Effect of time on the frequency of chromosomal aberrations and micronuclei induced in fipronil exposed rats.

Time	Chromosomal aberrations frequency		Micronuclei frequency	
	G1	G2	G1	G2
Control	2.6c ±0.24	2.6c ±0.24	15.4c ±0.51	15.4d ±0.51
24 hr	9.6b ±0.24	14.2b ±0.49	130.4b ±3.11	137.2c ±1.39
48 hr	12.4a ±0.24	16.0a ±0.55	138.2a ±0.86	141.2b ±1.16
96 hr	12.8a ±0.58	16.2a ±0.58	141.4a ±1.17	153.0a ±2.35

- Values with different superscript letters are significantly different.
- Values are expressed as means ± S. E.
- G1 = 1/4 LD_{50} of fipronil; G2 = 1/2 LD_{50} of fipronil

effect was achieved with the highest dose of fipronil treated groups (24, 48 and 96 hr treated groups) when compared to the control one.

In regard to the micronucleus assay in bone marrow cells of the rats (Table 2 and Fig. 2), the number of micronuclei at 24, 48 and 96 hr increased significantly in all groups treated with either 1/4 or 1/2 LD_{50} of fipronil compared to the control one.

Table (3) confirmed the effect of time on micronuclei induced in fipronil exposed rats in a time dependent manner, where there is a significant increase in micronuclei frequency up to 48 hr exposure for 1/4 LD_{50} and up to 96 hr for 1/2 LD_{50}. Therefore, the maximal response was observed after 96 hr of fipronil administration. The difference between the 48 and 96 hr treated groups within the low dose was not significant, but significant within the high dose.

Discussion

The available data indicate that millions of liters of pesticides are applied every year worldwide (USEPA, 2001), and approximately 3 million cases of intoxication by pesticides occur annually resulting in 220 thousand deaths around the world (WHO, 1992). Many of these pesticides are mutagenic (Galloway et al; 1987), and have been associated with the development of cancers (Leiss and Savitz, 1995) and developmental problems (Arbuckel and Server, 1998).

In the current work, the acute oral LD_{50} of fipronil was calculated as 100.35 mg/kg. b.wt in rats. Similar results were recorded by USEPA (1996) who found that the LD_{50} of fipronil in rats was > 97 mg/kg b.wt and Tomlin (2006) who estimated it as 97mg/kg. b.wt.

Several studies have demonstrated that pesticides can have genotoxic effects in both directly and indirectly exposed non-target organisms including humans (Nehez et al., 1988). The genotoxic effects of fipronil were assessed using chromosomal aberrations analysis and micronucleus assay in bone marrow cells of rats exposed to two doses (1/4 and 1/2 LD_{50}) of this insecticide for 24, 48 and 96 hr.

A low frequency of chromosomal aberrations and micronucleated cells was observed in the control group, while at 24 hr treatment with 25 or 50 mg/kg. b.wt of fipronil, genotoxic and mutagenic effects were observed and characterized by the presence of high frequency of structural chromosomal aberrations and micronuclei. This suggests the adverse action of fipronil on DNA, inducing the formation of micronuclei during the first cell cycle after treatment as well as a genetic damage.

Forty-eight hours after treatment showed a high frequency of micronucleated cells indicating that the DNA damage was much intense than that of the control group. The peak of chromosomal aberrations and micronuclei frequency induced by fipronil occurred 48 hr after treatment. Fipronil does not act directly on DNA, as it needs to be metabolized in order to induce DNA damage (Rogers, 1994). This may explain the higher frequency of chromosomal aberrations and micronucleated cells found 48 hr after treatment rather than 24 hr.

Ninety-six hours after the treatment, fipronil still induced damage to exposed animals as indicated by the high frequency of chromosomal damage and micronuclei formation compared to the control. However, approximately no difference was found compared to the 48 hr treated group especially in the frequency of chromosomal aberrations. This may refer to that the micronucleus assay is more comparable than chromosomal aberration analysis in detection of genotoxicity

(Kliesch et al., 1981). As well, the frequency of chromosomal gaps was declined at 96 hr after treatment, this refer to that most organisms possess some capacity to repair their DNA and DNA is the only macromolecule which is repaired by cells through 3 different mechanisms: damage reversal, damage removal and damage tolerance as reported by Bohr et al. (1993).

Limited work was done cocerning the genotoxic effect of fipronil in rats. However, Kevekordes et al. (1996) reported that the treatment of mice with different doses of 1,3-dichloropropene pesticide induced the formation of micronucleated cells. The same findings were achieved in rodent bone marrow (Agarwal et al., 1994; Celik et al., 2003), human peripheral lymphocyte cultures (Surralles et al., 1990) and in aquatic organisms (Campana et al., 1999 and Ghisi et al., 2011).

Al-Sabti and Metcalfe (1995) demonstrated that the maximum induction of micronuclei normally occurs one to five days after exposure to fipronil pesticide. These differences seem to be associated with the kinetics of cell removal (Cavas and Ergene-Gozukara, 2003) or with the development of adaptive mechanisms of tolerance to stress caused by toxic chemicals that cause an increase in the replacement rate of dead or damaged cells to maintain normal physiological conditions (Mersch et al, 1996). In addition, the elevation in genetic damage and micronuclei formation may be mediated by free radical generation as reported by Vijayalaxmi et al. (1999).

These results indicate that such insecticide may have mutagenic effect and causing chromosomal aberrations which agree with Wright (1995) in Chinese hamster, leading to tumor formation in rat exposed to fipronil and suggesting a possible human carcinogen (Lyons, 2000).

There was no evidence of aneugenic (numerical changes) effect of fipronil in mice at any dose or at any harvest time as reported by (USEPA, 2003), which coincide with our findings concerning the numerical changes induced in rats exposed to fipronil as well.

It could be concluded that although fipronil is an important insecticide, which is a part of the composition of last generation synthetic insecticide/ acaricides, it is genotoxic to non-target organism and also induces high rate of chromosomal aberrations and micronuclei. Moreover, it is present in concentrations extremely higher than those needed to eliminate the ectoparasite, a fact that causes concern and encourages us to seek for alternative products of natural origin that are efficient in controlling pesticides and at the same time not being as toxic to organisms in general or to the environment.

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تقييم التأثير السمي الوراثي والطفري لمبيد الفيبرونيل في الفئران

د. شنودة مارون جرجس، د فينيس فاروق يسى *

المركز القومي للبحوث * معهد بحوث صحة الحيوان

يعتبر الفيبرونيل من المبيدات الحشرية التابعة لمجموعة الفينيل بيرازول و التي تستخدم في مقاومة العديد من الحشرات لذلك يهدف هذا البحث إلى تقدير السمية الوراثية لمبيد الفيبرونيل في الفئران و ذلك عن طريق دراسة الإختلالات الكروموسومية و الأنوية الصغيرة الناتجة عن المعاملة بهذا المبيد.

تم تقسيم الفئران تحت الدراسة إلى مجموعتين, عوملت إحداهما بربع الجرعة النصف المميتة من الفيبرونيل و الأخرى بنصف الجرعة النصف المميتة للمبيد و ذلك لمدة 24 48 96 ساعة بالأضافة إلى المجموعة الضابطة.

و قد وأضحت النتائج حدوث إختلالات كروموسومية في خلايا النخاع العظمي للفئران فى كلا المجموعتين المعاملتين بالمبيد, و منها الإنفصال السنترومتري و الشظايا و الكسور و النقص الكروماتيدي بالإضافة إلى زيادة معدل حدوث النويات الصغيرة وقد كان معدل حدوث هذه الطفرات يزداد معنويا مقارنة بالمجموعة الضابطة أما بالنسبة للتغيرات الكروموسومية العددية فقد كان التأثير غير معنويا في الفئران المعرضة للفيبرونيل مقارنة بالمجموعة الضابطة وقد لوحظ زيادة في معدل حدوث الأختلالات الكروموسومية و النويات الصغيرة حتى وصل أقصاه بعد 48 ساعة من التعرض للمبيد.

و قد خلصت هذه الدراسة إلى أن المعاملة بالمبيد الحشري الفيبرونيل أدت إلى حدوث سمية وراثية للفئران تمثلت في زيادة إحداث الأختلالات الكروموسومية و النويات الصغيرة في الفئران المعرضة للمبيد و لذلك توصى هذه الدراسة بالبحث عن بدائل طبيعية لمقاومة الحشرات بدلا من المبيدات ذات الأصل الكيميائي لما لها من الأثر الضارعلى الانسان و الكائنات الحية المختلفة و البيئة المحيطة.