EFFECT OF ALDICARB PESTICIDE ON SOME ELECTRICAL PROPERTIES OF RAT TISSUES AND MUSCLE

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Abstract. The present work focused on investigating the effect of the different doses of Aldicarb on total body, extracellular and intracellular water content and dielectric properties of brain, heart, liver, kidney and leg muscles in Swiss-Albino rats. Control group was fed by standard diet and treated group was divided into two subgroups: Subgroup (2a) orally administered a single dose of aldicarb (0.5 mg/kg bw) 24 hour prior to measurements and Subgroup (2b) orally administered a single lethal dose 50 of aldicarb (lethal dose LD_{50} = 0.93 mg/kg bw). Percentage of total body water to body weight, percentage of extracellular water and intracellular water to total body water were calculated. Determination of aldicarb concentrations in the blood was measured. Permittivity and conductivity of control group and experimental groups at multiple frequencies were measured. Results of this study revealed a significant increase in total body water percentage from 58.68 to 64.10 of group (2a) while a non-significant increase in total body water from 58.68% to 60.38% of group (2b) compared to Control group was obtained; meanwhile, a significant increase in extracellular water percentage from 31.62% to 34.41% of group (2a), but a significant decrease from 31.62% to 28.37%: of group (2b) compared to control group was noticed. A significant decrease in intracellular water percentage from 68.38 to 65.59 of group (2a), but a significant increase from 68.38% to 71.63% of group (2b) compared to control group was also noticed. Concentrations of aldicarb in blood of rats (2a) were of 0.1388 ± 0.0168 mg/L, while blood of rats (2b) presented 5.48 ± 0.322 mg/L. The dielectric properties showed different behavior in permittivity and conductivity of internal organs such as the brain, heart, liver, kidney and leg muscles.

Key words: aldicarb, extracellular water, intracellular water, total body water, permittivity, conductivity, brain, heart, liver, kidney, muscles.

INTRODUCTION

Human health is in danger due to possible food and groundwater contamination. Modern societies cannot exist without either synthetic or naturally

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occurring chemicals. Agricultural soils during the course of cultivation and planting need some chemicals, as for example pesticides, to control certain insects, mites, and nematodes. Without these chemicals, a significant percentage of the crops will be lost. Although the benefits regarding the protection of food sources against chemical contamination are obvious, the use of pesticides, unfortunately, results in poisonings in many animals and humans every year [27].

Environmental monitoring of pesticides has started attracting increasing attention due to the increasing use of pesticides throughout the world. Among these pesticides aldicarb gained special importance due to its high toxicity. Aldicarb is a carbamate ester that is moderately persistent, water soluble and mobile in soil. In surface water, its half-life is 5 to 10 days, but in underground water the half-life can be of months. The general population is exposed to aldicarb and its toxic metabolites mainly through food and contaminated water. The ingestion of contaminated food may lead to poisoning incidents from aldicarb and its toxic metabolites (oral LD_{50} is 0.93 mg/kg bw in rats). It is initially metabolized to sulphoxide and sulphone metabolites, which then undergo further metabolism [2, 24].

Poisoning by acute high-level exposure to certain pesticides has well-known neurotoxic effects, but whether chronic exposure to moderate levels of pesticides is also neurotoxic is more controversial. Most studies of moderate pesticide exposure have found increased prevalence of neurologic symptoms and changes in neurobehavioral performance, reflecting cognitive and psychomotor dysfunction [14]. Carbamate inhibits brain and plasma cholinesterase in birds and rabbits [11, 25]. Carbamates share organo-phosphates in having single pharmacological properties chiefly manifested by the inhibition of cholinesterase (AChE) which plays a decisive part in the transmission of nerve impulses and stimulate the parasympathetic nervous system [15].

During the past two decades several suggestions have been published on how to perform risk assessment of pesticides. The clinical signs are an important criterion in the diagnosis of suspected poisoning of carbamate [9]. Body cell mass (BCM) is an important indicator of nutrition status; however, its measurement in the clinic has been limited. BCM can be estimated by the measurement of intracellular water (ICW). The assessment of extracellular water (ECW) is also important because many clinical populations undergo alterations in fluid distribution, particularly individuals exposed to a number of chemicals *via* food and environment [7].

In diseased states where body water is affected, the compartments that have changed can give clues to the nature of the problem. Ideally, the evaluation of the toxicological properties of aldicarb requires detailed information on the composition of the body water content. Total body water (TBW) estimations have

been used to estimate body composition, particularly extracellular and intracellular water, to aid in nutritional interventions and to monitor toxic status. Considering several diseases are associated with altered fluid balance in the body (including nutritional status, kidney failure, cardiovascular function and neurobehavioral performance) quick and accurate quantification of changes in cellular water status may be an invaluable clinical and diagnostic tool. For this reason, bioimpedance spectroscopy (BIS) has become an attractive technique for studying cellular water due to its noninvasive nature, simplicity of use, relatively low cost and allowing repeated measurements. BIS can provide estimates of total body water (TBW), intracellular water (ICW) and extracellular water (ECW) [5, 21, 26].

The objective of the current experiments is to determine if there were significant fluctuations in body water and dielectric properties of rat tissues and muscle after aldicarb consumption.

MATERIALS AND METHODS

ALDICARB

Aldicarb was purchased from Sigma-Aldrich, Germany. Aldicarb is an oximecarbamate insecticide. It has a molecular weight of 190.3; the chemical name is 2-metyl-2-(methyltio(propionaldehydo o-(methylcarbamoil) oxyme. Aldicarb is obtained as powder (store at room temperature) and dissolved in distilled water for use.

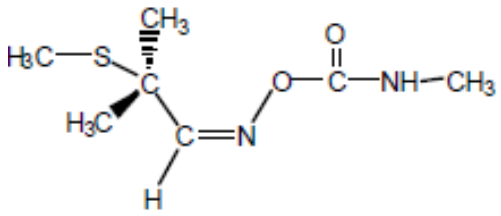

Fig. 1. The structural formula of aldicarb.

EXPERIMENTAL ANIMALS

The study protocol was approved by the Committee on Scientific Activities of Medical Research Institute, where this study was performed. A total of 30 male albino Wistar rats, 8 weeks old and weighing 79 ± 5 g were housed in standard cages at room temperature on a 12 h light and 12 h dark cycle.

Experimental groups

Two main experimental groups were used in this study:

1. Control group (10 rats) was fed by standard diet.

2. Experimental group (20 rats). This group was divided into two subgroups:

– Subgroup (2a), of 10 rats, was fed with standard diet and a single dose of aldicarb (0.5 mg/kg bw) was orally administered by gastric tube 24 hours prior the measurements.

– Subgroup (2b), of 10 rats, was fed with standard diet and a lethal dose 50 of aldicarb (LD_{50} = 0.93 mg/kg bw) was orally administered and the rats left (nearly about half an hour) until death. Rats were subjected to measurements then sacrificed.

Studying the effect of aldicarb on total body water, extracellular and intracellular water, aldicarb concentration in the blood and dielectric properties of rat tissues and muscle were carried out.

DETERMINATION OF BODY WATER

Rats were anesthetized with ether. Hair was removed from the dorsal surface of the head and body for electrode placement. The rats were placed in prone recumbency on a nonconductive surface to eliminate interference of electrical induction. Body orientation was standardized according to Hall *et al*. [10]. On the midline, source electrode 1 was placed at the anterior edge of the orbit, Source electrode 2 was placed 4 cm from the base of the tail, detector electrode 1 was placed at the anterior opening of pinna and detector electrode 2 was placed at mid-pelvis of the rat. The weight of the rats was recorded to the nearest 0.1 g. Body length was measured from the narium to the pelvic-caudal junction. LCR meter (Model ISO-9001, GOOD WILL instrument Co, LTD Hsin, TIEN CITY, Taiwan) was used to measure the electrical resistance and capacitance of rats using a tetrapolar electrode at low frequency of 1 KHz and at high frequency of 1 MHz.

Total body water weight (W_{TBW}) can be estimated from the empirical formula of Hall *et al*. [10]

$$W_{TBW} = 15.47 + \frac{97.44\,L^2}{WBR}, \tag{1}$$

where: L is body length (cm) and WBR is whole body resistance (ohm) from single-frequency bioimpedance measurements.

At low frequency (1 KHz) the whole body resistance is represented by only the extracellular fluid (R_e) [12].

$$R_e = WBR. \tag{2}$$

At high frequency (1 MHz) the whole body resistance becomes the parallel between resistances of the intra and the extracellular fluid, ($R_i \| R_e$). The resistance of ICW can be calculated from the electrical circuit model of a parallel combination of a resistor and capacitor [12]

$$\frac{\left(R_e + R_i\right)}{R_i} = \frac{R_e}{WBR}. \tag{3}$$

The ratio of *ICW* to *ECW* volume $\frac{V_{ICW}}{V_{ECW}}$ (mL/mL) can be estimated by solving the equation (4), which was derived by De Lorenzo *et al*. [5].

$$\left[1 + \frac{V_{ICW}}{V_{ECW}}\right]^{\frac{5}{2}} = \left[\frac{R_e + R_i}{R_i}\right]\left[1 + k_\rho \frac{V_{ICW}}{V_{ECW}}\right] \tag{4}$$

where: R_e = resistance of extracellular water measured at low frequency, R_i = resistance of intracellular water measured at high frequency, and $k_\rho = \frac{\rho_{ICW}}{\rho_{ECW}}$ is the ratio of resistivity of *ICW* to *ECW*.

Using the V_{ICW}/V_{ECW} ratio, equation (4) can be rewritten simpler as:

$$\left[1 + Q\right]^{\frac{5}{2}} = \left[\frac{R_e + R_i}{R_i}\right]\left[1 + k_\rho Q\right] \tag{5}$$

where $Q = \frac{V_{ICW}}{V_{ECW}}$.

Equation (5) can be solved through an iterative procedure for the value of Q without information on body geometry. The mean value of k_ρ was set at 3.60 based on human data (3.82 for men and 3.40 for women) [5], because the corresponding data for rats were unavailable.

De Lorenzo *et al*. [5] scaled ρ_{ICW}, ρ_{ECW} and k_ρ based on the dilution study in humans. Hence, k_ρ was scaled to the mole amount of water rather than the physical volume of fluid. Therefore, *ICW* and *ECW* weights were calculated from equations (6) and (7) as follows:

$$W_{ICW} = \left[\frac{Q}{1+Q}\right] W_{TBW} \tag{6}$$

$$W_{ECW} = \left[\frac{1}{1+Q}\right] W_{TBW} \tag{7}$$

where: W_{ICW} is *ICW* weight (g), and W_{ECW} is *ECW* weight (g).

Using the values of total body water weight W_{TBW} (g), extracellular water weight W_{ECW} (g) and intracellular water weight W_{ICW} (g), we can calculate percentage of total body water, percentage of extracellular water of total body water and percentage of intracellular water of total body water, respectively, as follows:

$$TBW\% = \frac{W_{TBW}}{W} \times 100 \tag{8}$$

$$ICW\% = \frac{W_{ICW}}{W_{TBW}} \times 100 \tag{9}$$

$$ECW\% = \frac{W_{ECW}}{W_{TBW}} \times 100 \tag{10}$$

DETERMINATION OF ALDICARB CONCENTRATION IN THE BLOOD

Stock solution of aldicarb at concentrations of 1 mg/mL was prepared in acetonitrile and stored at 4 °C. Different concentrations ranging from 0.1 to 10 μg/mL were prepared from stock solution, and then acidified to pH 4.0 with concentrated HCL. Control and calibration samples were prepared by spiking aldicarb-free blood samples with standard solutions.

High performance liquid chromatography (HPLC) system with a Model HP1100 pump from Agilent, USA, was used for the determination of aldicarb concentration in blood according to Covaci *et al.* [4]. Blood samples were harvested during sacrificing from hearts of control and experimental groups. Whole blood were extracted three times with dichloromethane and filtered on anhydrous sodium sulphate. Supernatants were evaporated to dryness under a slow stream of nitrogen, at 40 °C. The dried extracts were reconstituted with 50 μL water acidified with HCl (pH= 3) and an aliquot (20 μL) was injected into the HPLC system. Samples were analysed in the following instrumental conditions: RP-C18 column (250×4.6 mm i.d., 5 μm); acetonitrile: water (35:65, v/v), isocratic, as mobile

phase; flow rate 1.5 mL/min; ultraviolet detection at 210 nm; retention time of 7.2 min. The limit of detection obtained was of 0.05 μg/L.

DIELECTRIC MEASUREMENTS OF RAT TISSUES

To demonstrate the effect of aldicarb on the dielectric properties of the brain, liver, heart, kidney and leg muscle tissues, permittivity and conductivity of control group and aldicarb-treated groups at multiple frequencies were measured. The change in conductivity at low frequencies can be explained by a change in the conductivity of the extracellular fluid, whereas change at high frequencies can be explained by a change in the conductivity of the extracellular and intracellular fluid.

Rats from the group (2a) were dissected 24 hours after administration of a single dose of aldicarb, while for the group (2b), rats were dissected immediately after death (nearly half an hour). Samples of brain, heart, kidney, liver and leg muscle were removed and stored at –4 °C up to 24 hours. At the measurements time, samples were left to reach the room temperature. Slices of about 0.2 cm thick were carefully cut using special jig to ensure uniform known thickness and parallel cut surfaces. In order to perform measurements on samples using LCR Bridge, an electric cell was designed for this purpose (Fig. 2). This cell consists of two parallel silver electrodes impeded on a plexiglass. The samples were connected to the LCR meter by means of two circular silver electrodes with a radius of 0.5 cm each and the distance between the two electrodes was of 0.2 cm. Ag-Ag electrodes were coated with silver chloride, which provides a good contact transfer with minimum polarization.

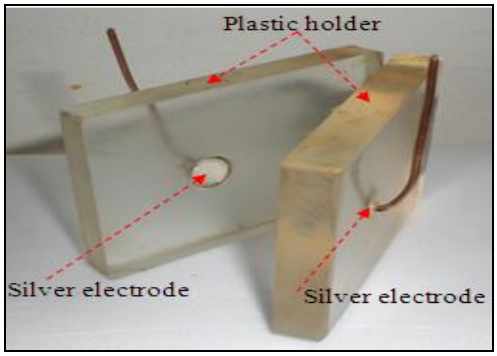

Fig. 2. The constructed dielectric cell.

Capacitance (*C*) and resistance (*R*) were recorded over a frequency range of 50 Hz up to 1 MHz. By using capacitance and resistance, the relative permittivity ε', the electrical conductivity σ (Siemens/m), the imaginary part of complex

permittivity ε" and the imaginary part of complex conductivity σ", were calculated using equations (12) and (13).

$$C = \frac{\varepsilon' \varepsilon_0 A}{d} \tag{11}$$

$$G = \frac{\sigma A}{d} \tag{12}$$

where C (expressed in farad) and G (Siemens) are the capacitance and conductance of the capacitor between the two measuring electrodes, A (m²) is the surface area of the electrodes, d (m) the separation between the two electrodes, ε' – the relative permittivity (Farad/meter), ε_0 the permittivity of vacuum (8.85×10^{-12} F/m), and σ – the electrical conductivity (Siemens/m).

The imaginary part of complex permittivity ε" and conductivity σ" were calculated according to equations (14) and (15).

$$\varepsilon'' = \frac{\sigma' - \sigma_L}{2\pi f \varepsilon_0} \tag{13}$$

$$\sigma'' = 2\pi f \varepsilon_0 \left(\varepsilon' - \varepsilon_h\right) \tag{14}$$

where σ_L is low frequency limiting conductivity taken at 50 Hz, and ε_h is the high frequency limiting permittivity taken at 1 MHz. Also total tissue fluid is calculated from Cole-Cole [12] diagram as follows:

$$\frac{W_{ECW}}{W_{TBW}}\% = \frac{R_{1M}}{R_{50k}} \times 100 \tag{15}$$

where R_{1M} is resistance at high frequency and R_{50} at low frequency, respectively.

STATISTICAL ANALYSIS

Data were presented and analyzed using SPSS software package, version 16.0. The data were presented as mean ± standard error (M ± SE). Statistical significance ($p < 0.05$) for each variable was estimated by a one-way analysis of variance (ANOVA). Comparisons of body water contents (*TBW, ECW* and *ICW*) for control and administered aldicarb groups were performed by paired *t*-test.

RESULTS

Immediate behavioral changes were observed after injection of aldicarb. The animals showed signs of toxicity, including tremors.

BODY WATER DISTRIBUTION

The total body water, intracellular water and extracellular water results obtained from bioimpedance spectroscopy data are systematically presented in Tables 1 and 2.

Table 1

The percentage of total body water to body weight for the groups (*TBW*%)

	Body length (cm)	Body weight (g)	Control	Subgroup (2a)	Subgroup (2b)
Min	14.4	73.5	54.29	57.63	55.75
Max	15.6	84	63.15	77.34	63.91
Mean	15	78.2	58.50	64.10	60.38
SD	0.45	4.38	3.84	6.71	3.07

Table 2

The percentage of intracellular water (*ICW*%) and extracellular water (*ECW*%) to total body water for the groups

	Control			Subgroup (2a)			Subgroup (2b)		
	ICW%	*ECW*%	*TBW*%	*ICW*%	*ECW*%	*TBW*%	*ICW*%	*ECW*%	*TBW*%
Min	66.56	29.85	54.29	63.77	33.00	57.63	71.01	27.70	55.75
Max	71.51	33.44	63.15	67.11	36.23	77.34	72.30	28.99	63.91
Mean	68.38	31.62	58.50	65.59	34.41	64.10	71.63	28.37	60.38
SD	1.73	1.73	3.84	1.08	1.08	6.71	0.38	0.38	3.07

CONCENTRATION OF ALDICARB IN THE BLOOD

Table 3 represents the mean values of aldicarb concentration in the blood of rats for 0.5 mg aldicarb/kg and lethal dose of aldicarb treated group.

Table 3

Aldicarb concentration (mg/L) in the blood of rats treated with with different doses of aldicarb

	Subgroup (2a)	Subgroup (2b)
Min	0.115	4.9
Max	0.165	5.9
Mean	0.1388	5.48
SD	0.0168	0.322

DIELECTRIC MEASUREMENTS

Complex permittivity and conductivity diagrams

Figures (3a,b) illustrate the diagrams of complex permittivity and conductivity of the brain, heart, kidney, liver and muscle of rat tissues for the groups. As seen in these diagrams, the degree of depressed center and the maximum values of the real and imaginary permittivity and conductivity depend on the dose of aldicarb used. The complex permittivity diagram follows the Cole-Cole semi-circle pattern. The maximum permittivity peaks of the brain, heart and kidney corresponding to group (2a) were more decreased than in the control group, while the maximum permittivity peak corresponding to group (2b) than those of control group. Permittivity of the liver and muscle samples decreased in both experimental groups compared to control group.

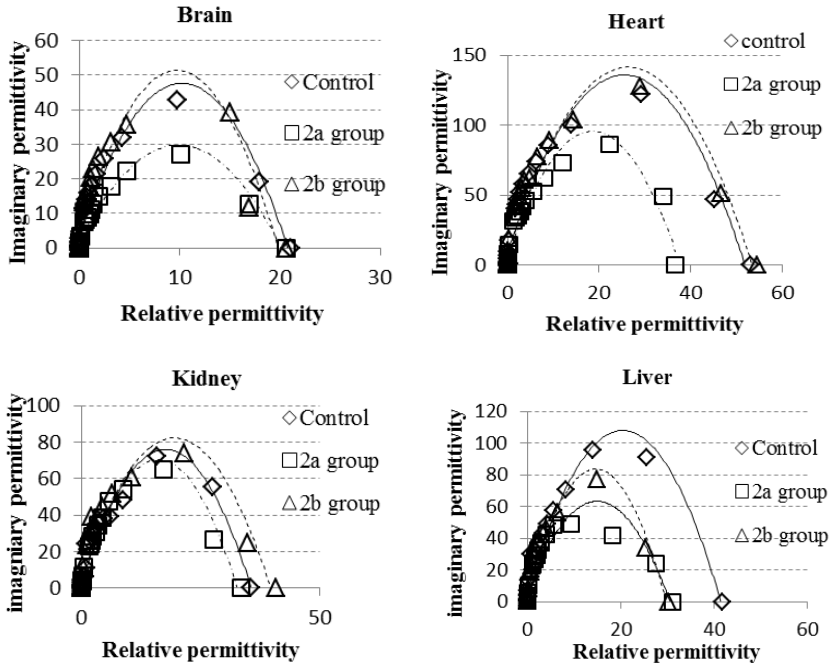

Fig. 3a

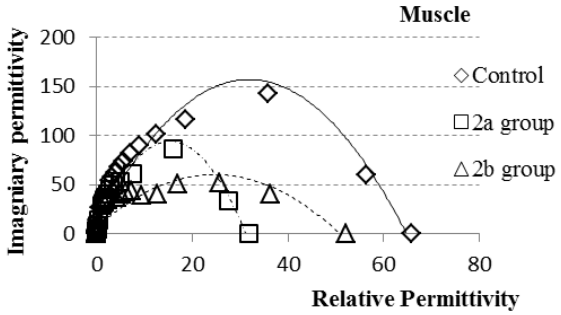

Fig. 3a (continued). Inter-relation between relative and imaginary permittivity of different tissues for different groups.

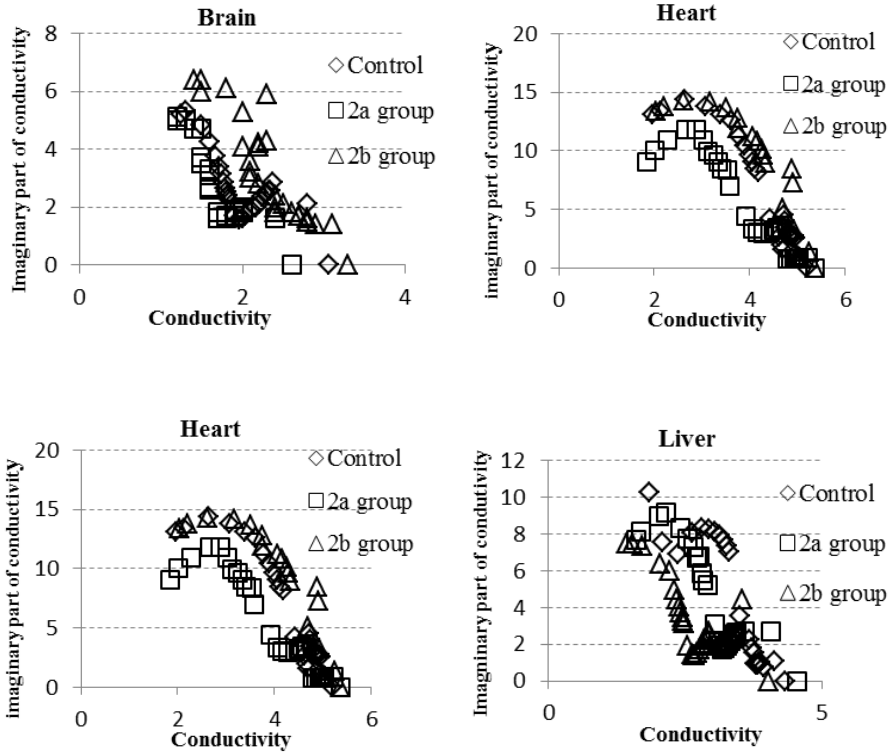

Fig. 3b

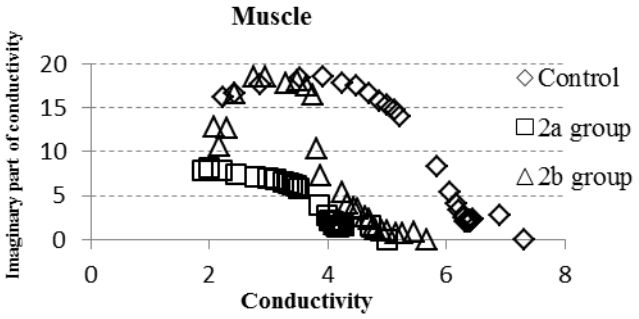

Fig. 3b (continued). Inter-relation between conductivity and imaginary conductivity of different tissues for different groups.

The values of the total body water volumes obtained after BIS are shortly presented in Table 4.

Table 4

Percentage volumes of total body water (*TBW*%) for each tissue and group

Tissue / Group	Brain	Heart	Kidney	Liver	Muscle
Control	64.3153	84.19015	66.32964	85.93598	83.10016
(2a)	66.4802	75.94667	67.27582	68.30081	79.41636
(2b)	70.1255	91.74726	68.26713	65.33563	74.89721

DISCUSSION

Aldicarb sulfoxide and aldicarb sulfone induce high acute toxicity by the oral route [3]. In the present study behavioral changes of rats were observed within half an hour after administration of a lethal dose of aldicarb and significant effects were still observed at 24 hours after a 0.5 single dose. This is in agreement with previous studies, in which the maximum inhibition of acetylcholinesterase activity was observed for maternal and fetus blood [3].

The primary mechanism of aldicarb toxicity is acetylcholinesterase inhibition. Carbamate insecticides are known to directly affect the enzyme acetylcholinesterase (AChE), which is associated with the outer surface of membranes [20]. Signs and symptoms of AChE inhibition and subsequent accumulation of ACh in nervous tissue and effector organs mimic the muscarinic, nicotinic, and CNS actions of ACh and may be categorized as follows:

1. Muscarinic Signs. The stimulation of muscarinic receptors (which are found primarily in the smooth muscle, the heart, and exocrine glands) results in the following symptoms: A tightness in the chest and wheezing due to bronchoconstriction, increased bronchial secretions, salivation, lacrimation, and sweating; increased gastrointestinal tone, with consequent development of nausea, vomiting, abdominal cramps, diarrhea, and involuntary defecation; frequent contraction of smooth muscle of the bladder, resulting in involuntary urination; bradycardia that can progress to heart block and constriction of the pupils.

2. Nicotinic Signs. The accumulation of ACh at the endings of motor nerves to skeletal muscle and autonomic ganglia results in the following symptoms:
Muscular effects, including easy fatigability and mild weakness, followed by involuntary twitching and cramps. Weakness affects the muscles involved in respiration and contributes to dyspnea, hypoxemia, and cyanosis. Nicotinic actions at autonomic ganglia may, in severe intoxication, mask some of the muscarinic effects. Thus, tachycardia caused by stimulation of sympathetic ganglia may override the usual bradycardia due to muscarinic action on the heart [8]. Also, according to Verster' study [28] elevation of blood pressure and hyperglycemia reflect nicotinic action at sympathetic ganglia.

BODY WATER DISTRIBUTION

Assessment of body fluid distribution would be useful to evaluate nutritional status, renal and cardiovascular function [13]. The results obtained for the body water contents of normal rats *TBW* percentage was 58.5%, *ICW* percentage was 68.38% and *ECW* percentage was 31.62% according to Tables 1&2 which are in agreement with results reported by DePalma *et al*. [6] and Marieb *et al*. [16], in which total body water percentage was 60% of body weight, about 2/3 of *TBW* was intracellular water and 1/3 of *TBW* was extracellular water.

There are three types of extracellular fluid: tissue fluid, blood plasma and transcellular fluid. Transcellular fluid includes cerebrospinal fluid, aqueous humor of the eye, secretion of the digestive tract and associated organs (saliva, bile pancreatic juice), renal tubular fluid and bladder urine, synovial fluid and sweat [22]. A significant increase in total body water from 58.5% to 64.10% and extracellular water percentage from 31.62% to 34.41% of rats given a single dose of 0.5 mg aldicarb/kg bw compared to control group, at $p < 0.05$, may be due to increased bronchial secretions, pulmonary edema and salivation, as reported by Dan Becker [1].

A non-significant increase in total body water from 58.5% to 60.38% of rats given lethal dose 0.93 mg aldicarb/kg bw, significant increase in intracellular water from 68.38% to 71.63% and significant decrease in extracellular water from 31.62% to 28.37%, at $p < 0.05$ compared to control group was also observed. The significant increase in intracellular water and significant decrease in extracellular

water may be due to activation of the acetylcholine esterase, resulting in an influx of sodium ions, which, in turn, causes the depolarization of muscle cell and subsequent opening of voltage-gated sodium channels. This ion influx then travels down the cell membranes via T-tubules and, via calcium channel complexes, leads to the release of calcium from the sarcoplasmic reticulum. When levels of calcium inside the muscle cell are high enough, muscle will contract. This could be due to changes of sodium ions which causes flow of water inside the cell [23].

DIELECTRIC PROPERTIES OF TISSUE

There is a definite paucity of information of aldicarb effect on dielectric properties from direct experimental testing. The passive electrical properties, *e.g*., conductivity and permittivity of biological material have their origin on the biochemical and structural composition of biological tissue [19]. Electrical bioimpedance measurements have been used for many years to study the electrical properties of biological tissue and to measure physiological events, being applied in several clinical areas, including body composition. Any changes in tissue physiology should produce changes in the tissue electrical properties .The electrical properties of any material, including biological tissue, can be broadly separated into two categories: conducting and insulating. In a conductor, the electric charges move freely in response to the application of an electric field, whereas in an insulator (dielectric), the charges are fixed and not free to move. In practice, most materials, including biological tissue, actually display some characteristics of both insulators and conductors because they contain dipoles as well as charges that can move, but in a restricted manner [18, 19].

To demonstrate the effect of aldicarb on dielectric properties of the brain, liver, heart, kidney and leg muscle tissues, permittivity and conductivity of control group and aldicarb treated groups were measured at multiple frequencies. The changes in conductivity at low frequencies can be explained by a change in the conductivity of the extracellular fluid, whereas changes at high frequencies can be explained by a change in the conductivity of the extracellular and intracellular fluid.

Complex diagram of relative – imaginary permittivity and conductivity-imaginary conductivity (Fig. 3a,b) exhibited lower values for the brain and heart tissue of rats orally administered 0.5 mg aldicarb /kg bw compared to control group. This may be due to muscarinic effect, which causes bradycardia and hypotension, which means lower fluid in the brain and heart. While in the kidney, it may be due to weakness and frequent urinary bladder contraction, which causes involuntary urination that means loss of water. In contrast, in rats orally administered with 0.93 mg aldicarb / kg bw, higher values of permittivity and

conductivity for the brain and heart tissue than in control group were observed. This may be due to nicotinic effect, which causes tachycardia and hypertension which means higher fluid to heart and brain. The results of the present study are in agreement with the study of Ragoucy *et al*. [20] which reported that Aldicarb can cause hyperactivity at nicotinic sites, especially at the skeletal muscle junctions, causing muscle fasciculations, weakness and paralysis that may lead to significant morbidity and mortality. Hyper-stimulation at the nicotinic receptors of the autonomic ganglia may cause tachycardia, mydriasis and hypertension, instead of bradycardia and hypotension that are seen when muscarinic stimulation at these sites predominates.

Complex diagrams of relative – imaginary permittivity and conductivity-imaginary conductivity (Figures 3a and 3b) exhibited lower values for liver and muscle tissue of rats orally administered 0.5 and 0.93 mg aldicarb /kg bw compared to control group. This may be due to the consumption of water in the hydrolysis step of aldicarb metabolism in the liver. This is due to the formation of aldicarb oxime, where its formation rate is larger than the rate of aldicarb nitrile produced in the dehydration step that produces water. As a result, a shortage of liver water was induced, which increases in the lethal dose group. In muscle, the transmission of electrical impulses between nerves and at myoneural junctions generally occurs through the release of chemical transmitters which bind with specific receptors on the postsynaptic terminal or motor end plate, respectively. As the chemical transmitter, acetylcholine in certain nerve synapses and at neuromuscular junctions binds to the receptor sites; an esterase (AChE) rapidly hydrolyzes the ACh into acetyl and choline fractions so that the stimulated nerves or muscles are not continually excited. Essentially, aldicarb and other cholinesterase inhibitors in some way prevent the breakdown of ACh and the subsequent return to a more normal or resting state for the nerve and/or muscle cells [22].

CONCLUSION

The present study proved that oral administration of pesticide aldicarb to rats induced a significant increase in total body water and extracellular water and a significant decrease in intracellular water of the group orally administered with 0.5 mg/kg bw. A significant decrease in extracellular water and a significant increase in intracellular water of the group orally administered with LD_{50} also appeared. Increasing real conductivity and relative permittivity of the brain, heart and kidney tissues in the group administered with DL_{50}, while decreasing in the group injected with 0.5 mg/kg bw were noted. The decrease in renal tissue conductivity and relative permittivity of liver and leg muscles in the group orally injected with lethal dose and injected with 0.5 mg/kg bw was noted. Pathological effects of aldicarb

depend largely on the dose of aldicarb administered, so measuring the body water content and dielectric properties of some internal organs can be used as a monitor of severity of aldicarb toxicity. Considerable evidence supports the view that the toxicological responses of laboratory animals to aldicarb and its metabolites are similar or identical to those of humans.

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