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The alteration in the neuromasts of the system of the lateral line of a freshwater fish "*Gambusia affinis*" by various xenobiotics

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Abstract

This work aims to alter the system of the lateral line by different treatment with the ototoxic antibiotic (gentamicin), the heavy metal which is cadmium and the pesticide Methyl parathion. The description of the cells of the lateral line system in a fish exposed or not exposed to different xenobiotics. A topographical and anatomical study of mechanoreceptors in the lateral line of the head of a teleostéen *Gambusia affinis* was conducted before and after exposure of the latter to specific doses of Gentamicin. Photonics microscopy shows that exposure to a daily mechanoreceptor dose of 80 mg of Gentamicin for 15 days engenders a separation of the cilia from the apex of the cell. However, the various component organ areas seem to keep structural integrity. However, the observation of the ultra-structure of ciliated sensory cell specimens treated with Gentamicin shows an alteration of the cell illustrated by the loss of the cilia of the apex and those synaptic structures of the basal cytoplasm. However, afferent and efferent endings are maintained. The increase in the dose of Gentamicin causes the acceleration of the degenerative process of the ciliated cell, but stopping the treatment brings about the unleashing of a regenerative process, which reveals the reversibility of the effect of the antibiotic on the ciliated sensory cell. Mechanoreceptor's exposure to methyl parathion and cadmium in high doses 1 mg/l and 5 µg/l for a time interval of 7 days does not cause any change in the apical part of the cell. However, different areas making up the body seem to have retained their structural integrity.

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Introduction

Environmental factors, whether natural, biotic or abiotic, or anthropogenic can strongly influence sensory perceptions and individual adaptive responses of animals in their respective environments. The behaviour of an animal is closely related to the nature of its sensory equipment, which is the lateral line system. The fish has a very developed sensory equipment which is the lateral line used for the identification and the location of obstacles (Campenhausen Von *et al.*, 1981; Blaxter and Batty, 1985; Bleckmann, 1993).It equally seems involved in the detection of moving objects as potential predators or preys, through the vortex generated by movement (Bleckmann,1993; Abboud and Coombs, 2000; Kanter and Coombs, 2003).

Its importance in nutrition is also reinforced by the marked preference for mobile teleostean, live prey (Pohlmann *et al.*, 2004). The side lines replace the vision when the latter is deficient, both in the absence of proper eye development due to the absence of sufficient brightness (Bensouilah and Denizot, 1991; Schemmel,1967). The lateral system is directly exposed to the environment permanently; this position begs the question about the extent to which the change, natural or anthropogenic nature of the environment may taint its operation and thus the survival of the fish in its natural environment. The aims of this study is the examination of the alteration of the lateral line system through the use of various xenobiotics (Gentamicin, Cadmium and Methyl parathion), and the description of the cells of the system of lateral line in a fish *Gambusia affinis (*Baird and Girard, 1853) untreated and treated based on the figure obtained by using a transmission electronic microscope.

Gentamicin is an aminoglycocide antibiotic that inactivates the lateral line system (Song *et al.*, 1995; Coombs *et al.*, 2001). Ototoxic antibiotics have the property of moving the calcium ions from their binding site; they block the cationic channels located at the apex of the stereo cilia of sensory ciliated cells in the lateral system of fish neuromasts (Kroese *et al.*, 1989; Forge and Schacht, 2000) and also act on synaptic transmission which takes place on the part basolateral the same cell (Forge and Schacht, 2000). The presence of metal cations (cadmium, lead, mercury, nickel, cobalt.) in the milieu, which compete with calcium cations at the channels transductions located on the apical portion of each hair cell stereo cilia (Sand, 1975; Janssen, 2000), would likely engender alterations of sensory hair cells that impact fish behaviour.

Methyl parathion is an organophosphate pesticide, which exerts its neurotoxicity through an action mechanism linked to the phosphorylation of the enzyme acetyl cholinesterase (Verhaar *et al.*, 1992; Milson, 1998). This causes the inhibition of acetyl cholinesterase (AChE) and a skin of the neurotransmitter acetylcholine in the synapses of the central and peripheral nervous system resulting in overstimulation of the cholinergic muscarinic and nicotinic synapses.

The aim of this work is to alter the system of the lateral line by an aminoglycoside "Gentamicin", a heavy metal "Cadmium" and organophosphorus pesticide "Methyl parathion"

Material and methods

Treatments to different xenobiotics

Treatment of fish to the antibiotic "aminoglycocide" Gentamicin

Fish lots, the species Gambusia affinis, of equal size are each placed in aquarium containing a liter of water aerated with the aid of a pump. In this study, the treatment of fish to gentamicin was performed according to two protocols:

The first treatment consisted of exposing a group of fish (n=10) to a daily dose of Gentamicin sulfate 80 mg we dilute in water with a capacity of one litre container (equivalent to a concentration of 0.2 %); after 24 hours of action of aminoglycoside on the fish, the water is renewed and the fish receives a new dose of gentamicin; the operation is repeated as many times as required by the protocol; therefore, the fish

used in this experiment are continuously exposed to a dose of 80 mg of gentamicin over a period of 25 days. After stopping treatment, the fish is sacrificed for the observation of the impact of gentamicin on the ciliated sensory cell mechanoreceptor.

The second treatment was to be diluted in water where the fish two 80mg ampoules gentamicin sulfate, which is equivalent to 160 mg Gentamicin sulfate, corresponding to a concentration of 0.4%.

The first aquarium of fish are sacrificed after exposure times ranging from 3h, 6h, 9h, 12h, 15h, 18h and 21h (time t = 0 is the moment where begins aminoglycoside dilution). The second groups of the fish exposed to a dose of 160mg/l for 24 hours only. The fish are kept alive for 45 days.

Treatment of methyl parathion

The steam pressure of parathion is 5X10 $^{-3}$ Pa at 20°C and its solubility in water is 24mg/l at 25°C (Worthing, 1983). In the exposure conditions used in this study (pH = 8.86, T ° = 25°C) the lethal dose (100% mortality) is 2 mg /l, and the sublethal dose is from 0,25mg/l, 0,5mg/l and 1mg/l. The fish are divided into four experimental groups, by having 60 specimens per aquarium.

One of those groups serves as a testimony and thus will not receive insecticide treatment. After 48 hours acclimatization, the water in each aquarium is renewed; the final concentrations of methyl parathion around 0,25mg/l, 0,5 mg/l and 1 mg/l. The exposure times are 7days, 15 days, 21days and 30 days.

The fish treatment of cadmium

The sub lethal cadmium doses are based on acute toxicity data reported in the literature (0,5 mg/l to500mg/l). Under our experimental conditions (pH = 8.86; T° = 25 ° C), the lethal dose (100% mortality) is of 10 g / l and sub lethal are 0,1 µg/l, 1 µg/l and 5 µg/l. The fish are divided into four experimental groups at 60 individuals per aquarium; one of them was a control. After 48 hours of acclimatization of fish, half the water each aquarium is renewed and cadmium is introduced at levels to obtain final concentrations of approximately 0,1µg/l, 1 µg/l and 5 µg/l. The time of exposure to this heavy metal is 7 days, 15days, 21 days and 30 days.

Preparation of the ultra-structure of mechanoreceptors

The epidermis fragments for anatomical study of cutaneous sensory organs were fixed by immersion in a 2% glutaraldehyde solution in 0.05M phosphate buffer overnight at 4 °C, then post fixed by immersion in a 2% osmic acid solution in the same buffer for one hour at 4 ° C. After post-fixing, the parts were washed in the same phosphate buffer.

The pieces are dehydrated by passing through increasing degrees of alcohol baths (70, 80, 90, 100 °) and immersed in 3 epoxy -1-2 propane bathroom to 10 minutes each and then left overnight in a mixture of equal parts of Araldite epoxy propane 1-2. The inclusion of parts in Araldite was achieved after treatment for one day in Araldite epoxy, supplemented with 1-2 propane (1ml Araldite epoxy + 1 drop 1-2 propane). The specimens were included in the freshly prepared and pure Araldite and degassed under vacuum and put to polymerize in an oven at 60°C for 24 to 48 hours.

The epidermis debited to ultra-microtome semi thin sections of 1 to 2 microns thick glued on histological slides and thin sections (70 to 80 nm), collected on copper grids, were stained for 1 to 2 minutes at 60 ° C (by placing the slides on a hot plate), by toluidine blue dissolved in sodium tetraborate at 1% in water (Trump and *al.*, 1961). Sections were contrasted 30 minutes by uranyl acetate, and after washing, lead citrate according to the method of Reynolds (1963). The observation was conducted through the use of a transmission electron microscope Philips CM10 (neurophysiology laboratory, CNRS, Gif sur Yvette, France).

Results

Morpho anatomy of the organ

Observation by photonic microscopy of serial sections of epidermis Gambusia affinis treated with a daily dose of 80 mg of Gentamicin over a period of fifteen

days shown at the neuromaste uncoupling of the cilia of the apex of the sensory cell, eyelashes detach from the hair cell and end up in the cup that covers the organ. However, the various components the organ areas seem to maintain their structural integrity (Fig. 1E).

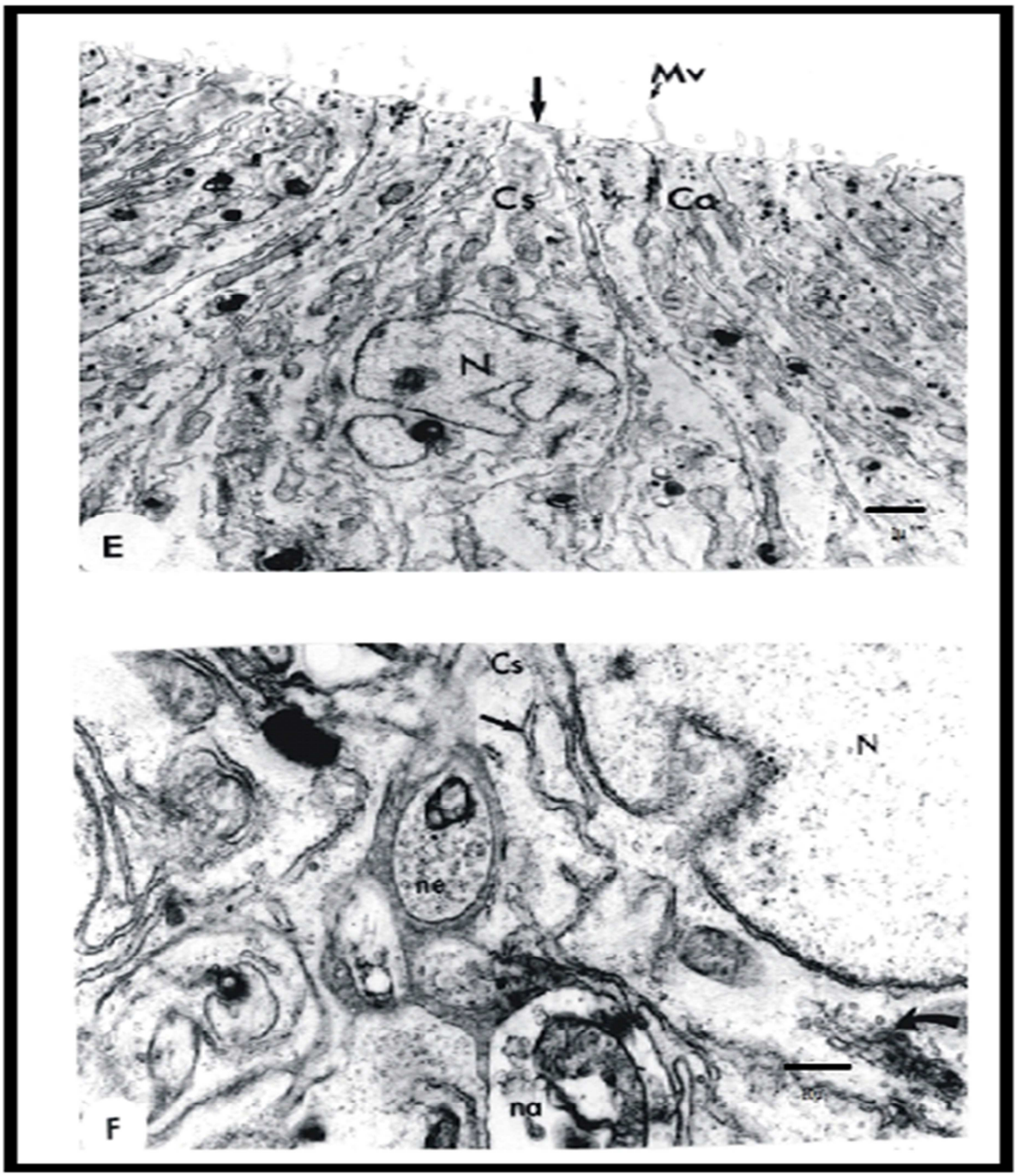

Fig. 1. Cups of fine mechanoreceptor lateral line *Gambusia affinis* from treated with gentamicin sulfate (80 mg daily dose of/l for 15 days). Staining with uranyl acetate and lead citrate. E: Overview of the member E-View; Note the disappearance of cilia from the apex (arrow) of the sensory cell (Cs), however the microvilli (mv) remain on the apex accessory cells (Ca); N: nucleus (X6600). F: Detail basal cytoplasm of sensory cell (Cs) and its innervations: note the presence of the afferent nerve ending (na) but the absence in the cytoplasm of sensory synaptic ribbons and synaptic vesicles persist only the endoplasmic reticulum (arrow) and the Golgi apparatus (curved arrow), N: nucleus (X 33000).

Ultra structure of the organ

After daily exposure to 80mg/l Gentamicin: Exposure of mechanoreceptor at a daily dose of 80 mg/l Gentamicin for a 15 days period causes the disappearance of cilia from the apex of sensory cell ciliated. We note, however, that the microvilli of the apex of the support cell is not affected (Fig. 1E).

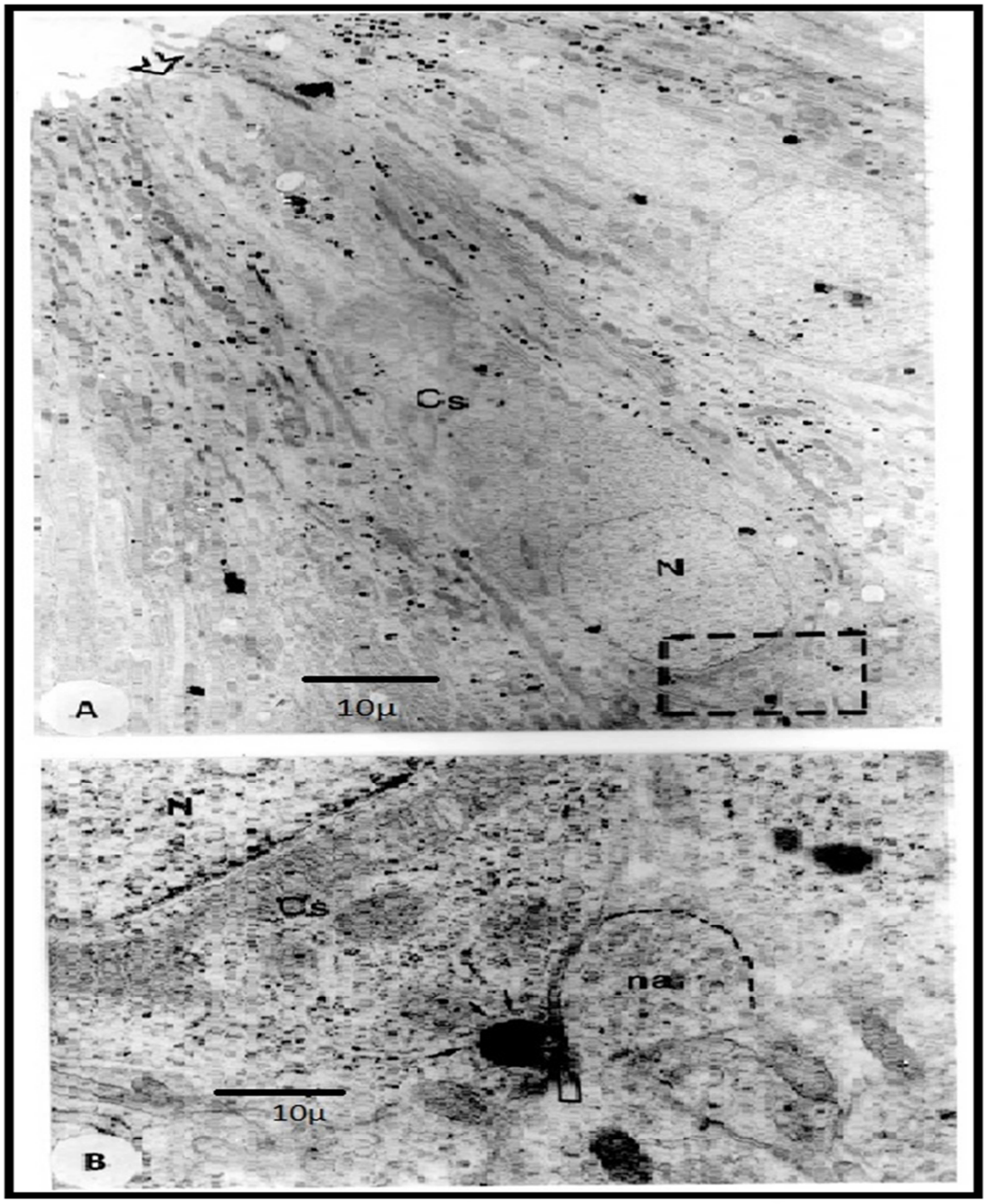

Fig. 2. Coups of fine neuromaste *Gambusia affinis* 9 hours after treatment with Gentamicin (at a rate of 160 mg/l) (staining with uranyl acetate and lead citrate). A: Entire body View: Note the separation of the cilia of the apex (empty arrow) sensory cell (Cs); N: nucleus (X7400); B: Inset of Figure A showing the presence in the cytosol of a basal synaptic ribbon (curved arrow) surrounded by synaptic vesicles (arrows) in the vicinity of the afferent nerve ending (na). (Cs): sensory cell; N: nucleus (X31000).

The detailed description of basal cytoplasm of sensory cell shows the absence saccule in the cytosol of the latter, although the efferent nerve ending is present; otherwise, the synaptic ribbon and synaptic vesicles surrounding disappeared from the cytoplasm of single cell sensory or the Golgi apparatus and endoplasmic reticulum persists (Fig.1F). Mitochondria and vesicles are fewer in the cytoplasm of the sensory treated cell compared to untreated cells.

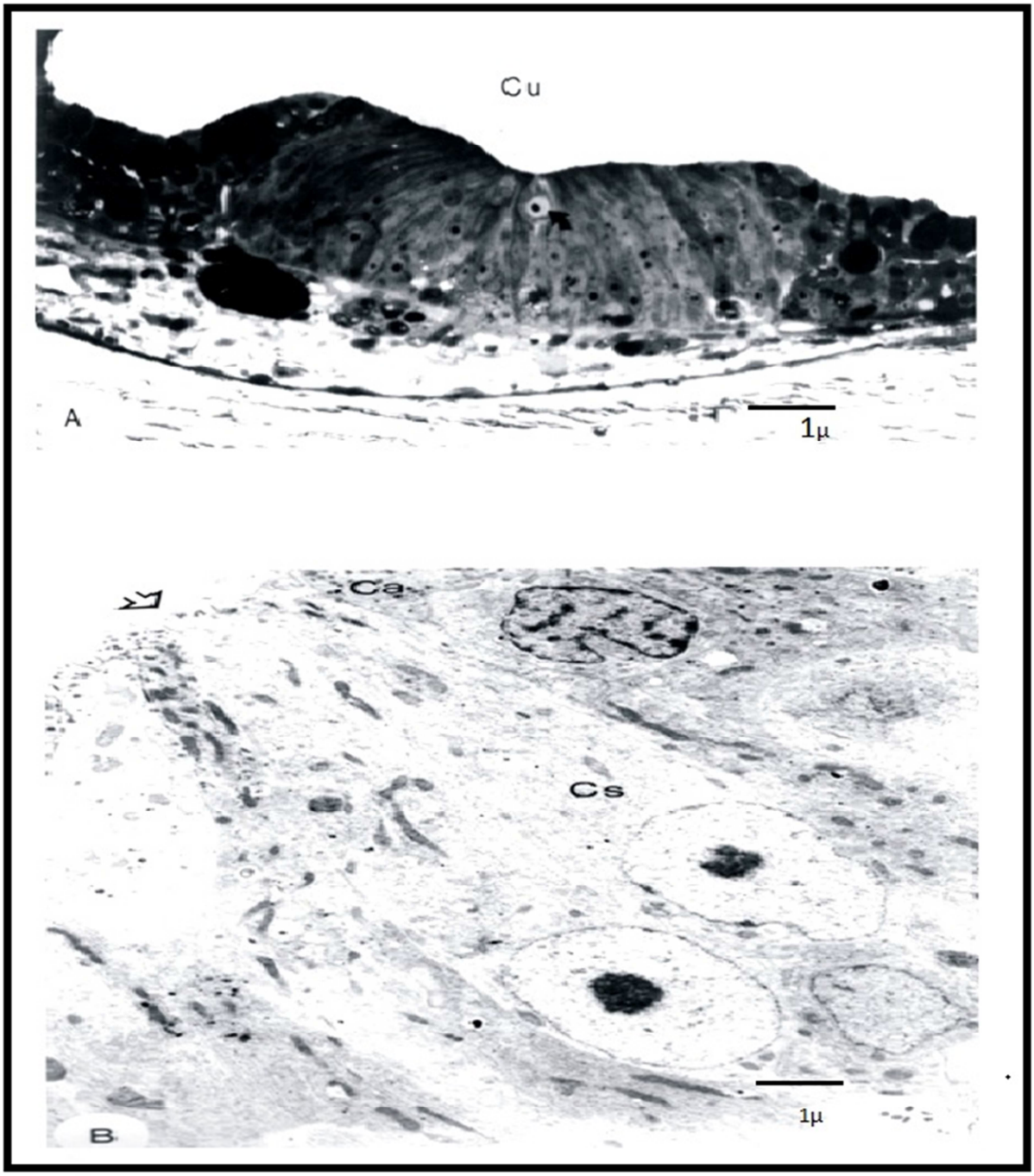

Fig. 3. *Gambusia affinis* Mechanoreceptor of 15 days following discontinuation of treatment with gentamicin. A: Cup semi showing thin in the central area of the body the presence of a sensory cell (Cs) in the regeneration phase (curved arrow). Note, in the clear presence of a nucleus nucleolus (osmiophilic task). (Cu: Cupule) (Toluidine blue staining) (X 2600); B: Fine cuts of the epithelium showing sensory cells (Cs) in regeneration phase, illustrated by the presence of the osmiophilic task in the kernel (marking the nucleolus and the reappearance of the cilia on the apex (empty arrow). (Ca: accessory cell) (Staining with uranyl acetate and lead citrate) (X5400).

The exposure of fish to a single dose of 160 mg/l Gentamicin: The exposure of fish to a single dose of 160 mg/l Gentamicin causes acceleration in the mechanoreceptor degenerative process; loss of cilia was observed after 9 hours of exposure to the antibiotic; however, synaptic structures are maintained, (Fig.2A and B).

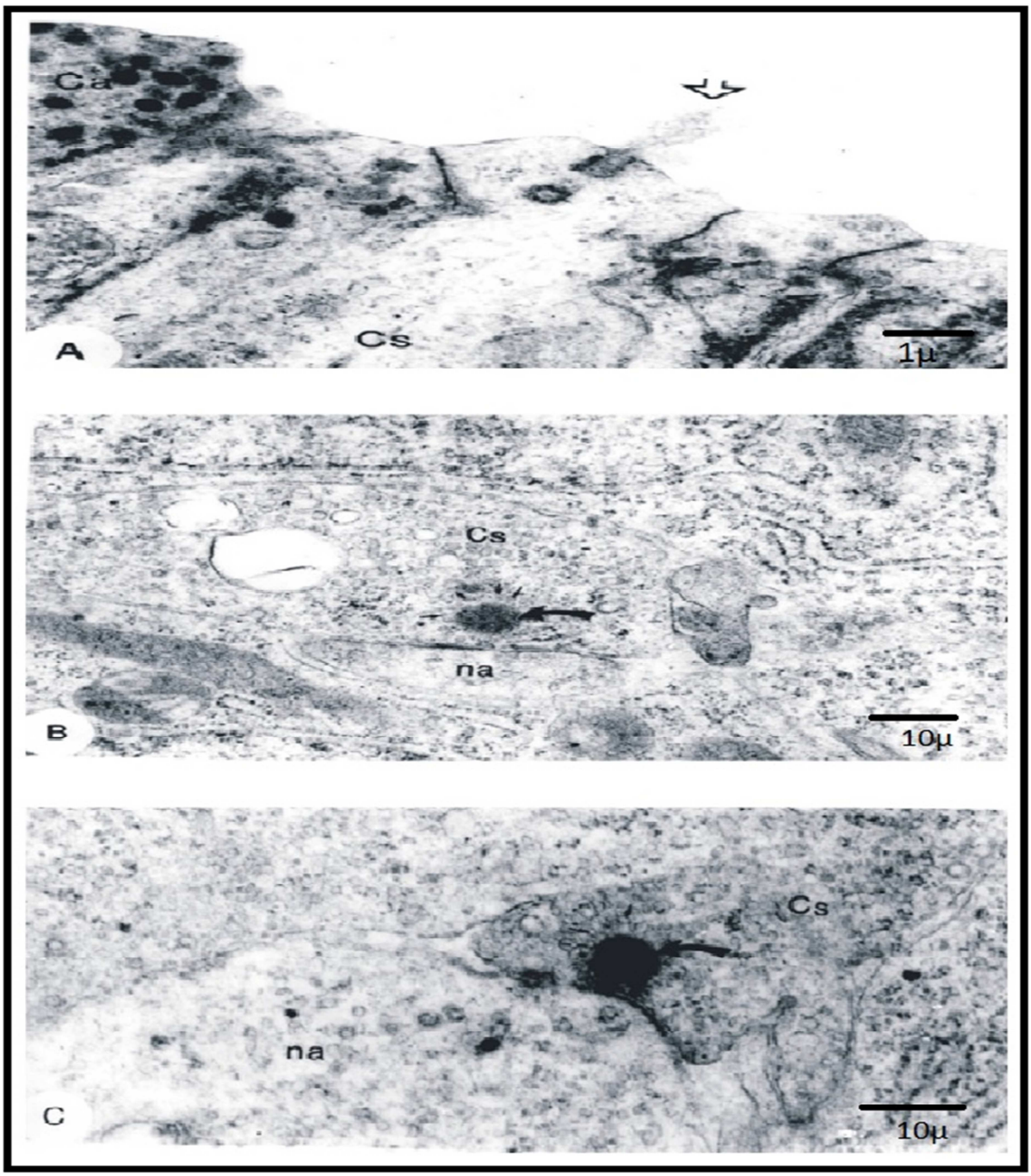

Fig. 4. The ultra structure of *Gambusia affinis* mechanoreceptor of 15 days following discontinuation of treatment with Gentamicin (Staining with uranyl acetate and lead citrate). A: Detail of the apex of the sensory cell (Cs) showing the presence of an eye (open arrow) training. (Ca): accessory cell (X20000); B: Details of basal cytosol of sensory cell (Cs) showing the establishment (low contrast) of synaptic spherule (curved arrow) and synaptic vesicles (arrows) adjacent to the afferent nerve fibre (na: afferent nerve) (X 56000); C: Basal cytoplasm of sensory cell (Cs) showing the presence of tape (curved arrow) and synaptic vesicles (arrows) affixed to nerve afferent fibre (na) (X 46000).

The observation of specimens treated with a 160 mg/l dose of Gentamicin for 15 hours shows, in addition to the disappearance of cilia, the absence of synaptic structures; we note, however, the presence of nerve endings close to the cell (Fig. 3A and B).

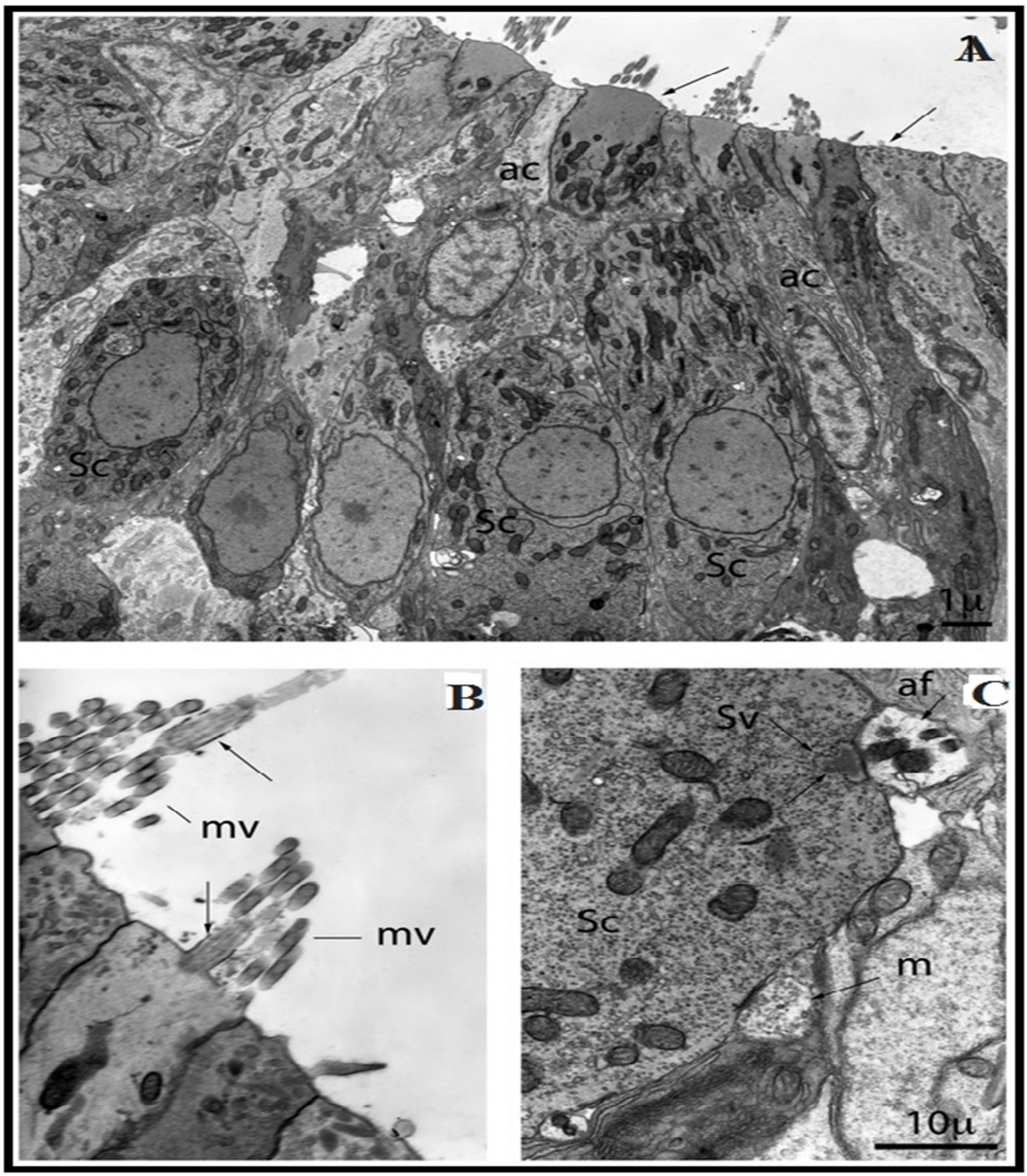

Fig. 5. Cups thin epithelium mechanoreceptors lateral line from head *Gambusia affinis* of Cadmium treated at 5 µg/l. (Staining with uranyl acetate and lead citrate). A: General view showing the distribution of sensory cells (Sc) and accessory cells (ac) constituted the epithelium of mechanoreceptors. The apical part of the cells (arrows) is in contact with the aquatic medium (X 2750); B: Enlargement of the apical part of the sensory cells showing the presence of a cohort of microvilli (mv) and kinocilium (arrows) (X13400). C: Enlarging the area of innervations of the sensory cells. The base of the sensory cell (SC) remains in contact with nerve endings of sensory fibres (af afferent fibre) and motor (m). In the cytoplasm of sensory cell, noted before the sensory nervous button, the presence of a synaptic tape (arrow) surrounded by synaptic vesicles (sv) (X 13400).

After stopping exposure to Gentamicin: The observation of thin cuts of fish skin taken 15 days after discontinuation of treatment with a single dose of Gentamicin sulphate 160 mg reveals the unleash of a regenerative process (Fig. 4); the latter is illustrated by the appearance of lashes in the apex of the cell (Fig. 4A) and vesicles and synaptic spherules in the basal cytoplasm (Fig. 4B and C).

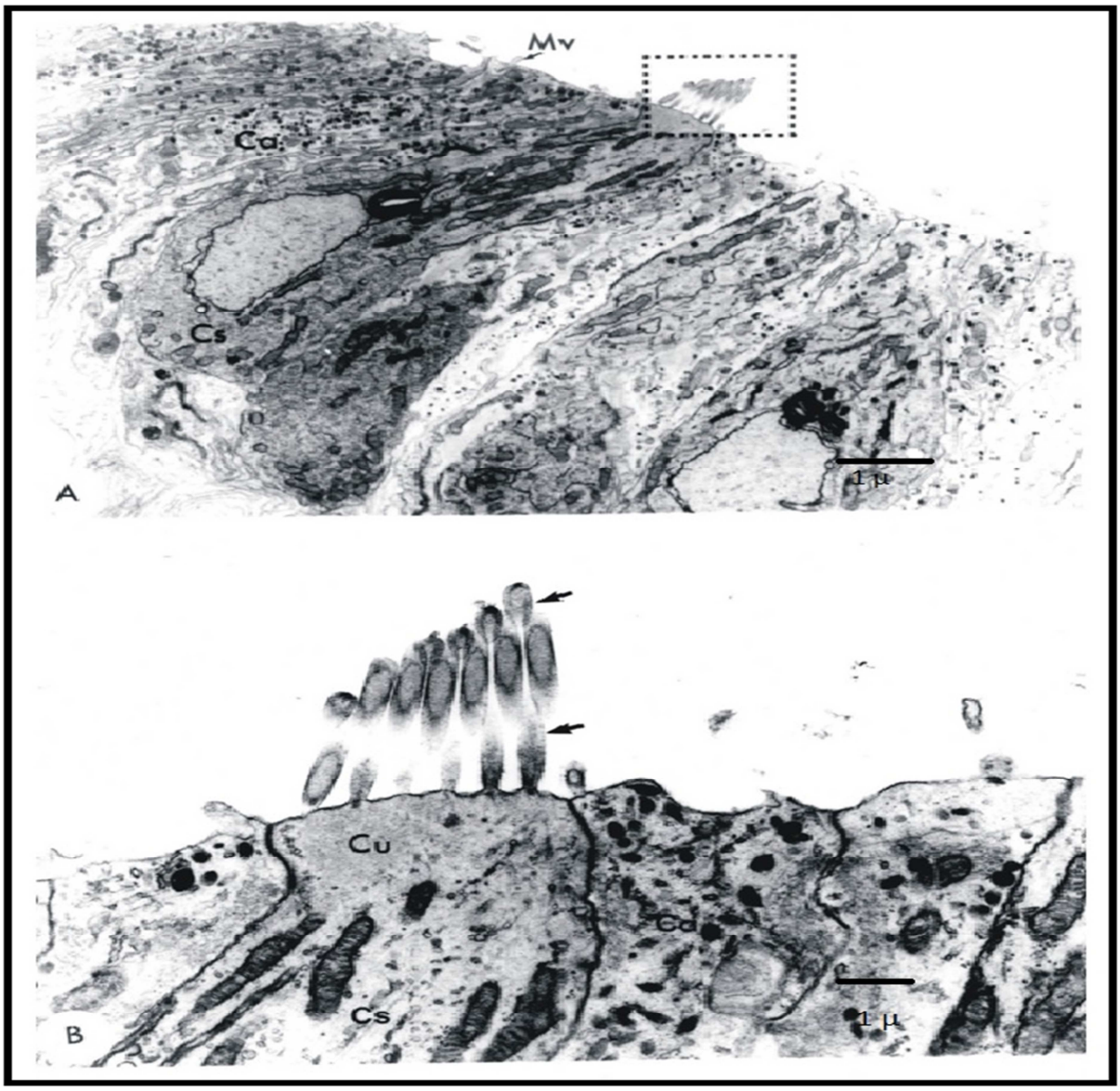

Fig. 6. Cups of fine mechanoreceptor lateral line *Gambusia affinis* of Methyl parathion treated dose (1mg/l) (Stained with uranyl acetate of lead). A: Overview mechanoreceptor; Note the presence of cilia (rectangle) to the apex of the sensory cell (Cs) and microvilli (arrow) at the apex accessory cells (Ca) .the core support needs the lower third of the sensory cell (X5200); B: Inset of Figure A: cilia (arrow) of the sensory cell is rooted in the cuticle (Cu) present on the apical part of the sensory cell (Cs) (X24500).

Heavy metal treatment "cadmium"

Morpho-anatomy of the organ: Observation, by photonic microscopy, of serial sections of fish exposed fish to cadmium at 0,5 and 5g/l during a time interval of 7 days shows no change of the part apical cell; moreover, we notice that the different zones that make up the body seem to have maintained their structural integrity.

Ultra structure of the body exposed to cadmium: The analysis, transmission electron microscope, of the epithelium of the mechanoreceptors of the side line of the head *Gambusia affinis* exposed to different doses

of cadmium shows the absence of damage to the components that body structures. In fact, the anatomical organization of the epithelium is normal even in the high dose of 5µg/l; The apical part of the sensory cell, despite the direct contact with the heavy metals tested, still has a cohort of microvilli and kinocilium; It is further noted that the treatment has no effect on the innervations system located at the base of these mechanoreceptor to sensory function; nerve endings of sensory and motor fibers maintain their contact with the base of the cell (Fig. 5 A, B and C).

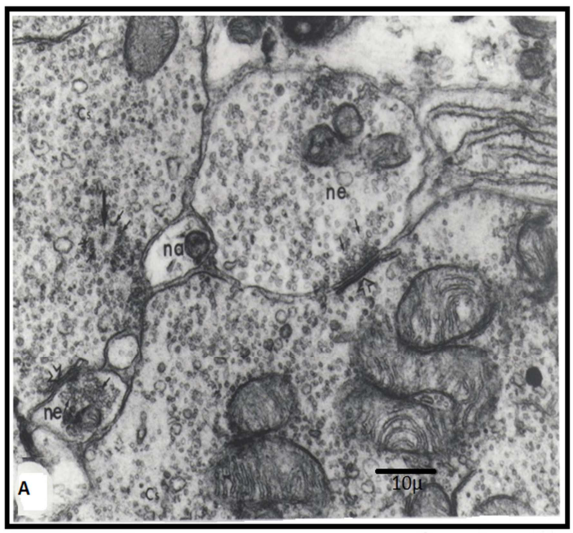

Fig. 7. Mechanoreceptor innervations of the lateral line *Gambusia affinis* of Methyl parathion treated dose (1mg/l) (Staining with uranyl acetate lead) B: Innervations of the basal portion of the cell showing the sensory afferent terminal (na) or the synaptic ribbon are observed (arrow) and vesicles (small arrows); efferent ending (do) rich in vesicles (small arrow), synaptic ribbon (open arrow).

Methyl parathion treatment

Morpho-anatomy of the organ: The observation, of serial sections of fish exposed to methyl parathion at doses of 0,25mg/l 0,5mg/l and 1mg/l for a time interval of 7days shows no change of the apical portion of the cell with retention of structural integrity of the body component different areas.

Ultra structure of the organ: The side line of the head of *Gambusia affinis* exposed to different doses of methyl parathion shows the absence of alteration of components structures this body. In fact, the anatomical organization of the epithelium is normal even in the high dose of 1 mg/l; the apical part of the sensory cell, despite the direct contact with the pesticide, always present a cohort of microvillosities and kinocilium. In the basal part of the sensory cell to mechanoreceptors function (Fig. 6 (A.B)), the nerve endings of sensory and motor fibers maintain their contact with the cell, the processing is therefore no effect on the system of innervations (Fig 7.B).

Discussion

The observation, the transmission electron microscopy, and the ultrastructure of sensory ciliated cell, after exposure to gentamicin, reveals impaired sensory cell ciliated illustrated by the disappearance of cilia of the apex and the absence of cytoplasmic components of the base components synaptic structures such as synaptic ribbon, synaptic vesicles and the head respectively saccule transmission of afferent and efferent neural information.

The results of our research are original because they provide an unprecedented and fairly detailed description of the structure of the ciliated sensory cells intact and treated with aminoglycoside.

Until today, authors have especially based their efforts on scanning electron microscopy observations to describe fish neuromasts damaged by exposure to aminoglycocide they relate disorganization eyelash apex macula and bare lashes (Song *et al.*, 1995; Montgomery *et al.*, 1997; Coombs et *al.*, 2001; Faucher *et al.*, 2006).

We used Gentamicin and streptomycin to inactivate the whole lateral system in *H. bleheri.* Our SEM data are indicative of successful inactivation, as 97% of the superficial neuromasts appeared damaged. Although we observed only the superficial neuromasts by SEM, exactly the Saami treatment has-been shown to damage Both the superficial and the channel neuromasts in various fish species, inactivating the whole lateral system (Song *et al.*, 1995; Coombs *et al.*, 2001).

Our observations also reveal that the speed of alteration of the sensory ciliated cell is a function of the aminoglycoside dose. Indeed, the application of a

single dose of 160mg/l gentamicin sulfate for 24 hours generates the same cellular changes than those obtained after 15 days of treatment with a daily dose of 80 mg/l gentamicin sulfate. The application of a strong and unique dose of gentamicin (160 mg/l) revealed a certain period elapses in the process of alteration of the sensory cell ciliated illustrated by the separation of the cilia apical after 9 hours exposure followed by alteration of synaptic structures after 15 hours of treatment at the same dose. This chronology in the action of the aminoglycoside is supported by the data Aran *et al*., (1995) who observe the effects of the antibiotic on the 2nd day of treatment in a region near the cuticular plate of hair cell cilia inner ear of mammals.

According to some authors, sensory hair cells of fish neuromaste would present a relatively higher sensitivity than that observed in other vertebrates. The ciliated sensory cells of the fish neuromaste die after about two hours of exposure to a concentration of 10µM neomycin then it takes 1mM to kill a ciliated cell of vestibular organ crow (Richardson and Russel, 1991; Li *et al*., 1995).

Furthermore, Kaus, (1987), and William Holder (2000) reported that administering a dose of 10 µM inhibited neomycin function ciliated sensory cell and the 300 µM kills the fish. These observations bolster the idea of selective toxicity of aminoglycosides overlooked the ciliated sensory cells and show that ciliated sensory cells of mechanoreceptor in *Gambusia affinis* are the type 1 because the latter have a pattern of destruction and regeneration similar to what is described in Type 1 ciliated cells of the utricle striolaire region and lagena's Oscar after treatment with gentamicin (Song *et al*., 1995; Faucher, 2004).

Our data show that the effect of gentamicin on ciliated sensory cells in "*Gambusia affinis*" is reversible, resulting in the establishment of a regenerative process 15 days after discontinuation of treatment. This process is illustrated by the appearance of the cilia at the apex and synaptic structures in the basal cytosol.

Similar observations are reported by William and Holder, (2000), in the zebrafish embryo, 15 days after exposure to a dose of 10 µM of Neomycin. This reversible loss of sensitivity of the lateral line is also described in Aplocheilus lineatus after cessation of exposure to aminoglycosides (Kaus, 1987).

At the Oscar fish *Astronotus ocellatus*, Song *et al*.(1995) reported regeneration neuromast channels 8-12 days after discontinuation of treatment with gentamicin; Coombs *et al*. (2001), noted that process, in mottled sculpin (*Cottus bairdi*), 20 days after the end of treatment.

This reversibility of the sensitivity would be correlated with the regenerative process of ciliated sensory cells that we see in *Gambusia affinis* few days after stopping treatment with aminoglycoside. According Tanyeri *et al*. (1995), the spatio-temporal gradient of cell proliferation following the process of degeneration of ciliated cells suggests that some aspects of the degeneration of hair cells give signals triggering proliferation. Furthermore, Kaus (1987) explains this reversibility of the sensitivity by maintaining the afferent and efferent nerve endings in the vicinity of the sensory cells whose synaptic cytoplasmic structures are altered by the antibiotic. This data confirms the results of our observations by transmission electron microscopy revealed that the retention of afferent and efferent nerve fibers near the sensory cells treated with gentamicin despite the absence of structures that make up the synapses in both afferent that efferent the cytoplasm of the latter.
The side line of the head *Gambusia affinis* treated with cadmium at a dose of 5 µg/l, shows a normal anatomical organization of sensory cells and accessories thereof. We note, in fact, that after treatment with cadmium, the apical part of the sensory cells still present his cohort microvilli and kinocilium despite their direct contact with the tested metal. Moreover, the treatment appeared to have no effect on the system of innervation of these cells to mechano receptor function that the base of the sensory cell maintains its contact with the nerve endings of afferent and efferent fibres.

The different doses of cadmium used in this study does not lead to alterations in the structure and ultrastructure of mechanoreceptors, our results diverge from the findings in the work of Atchison *et al.* (1987), The alteration of fish mechanosensory abilities by metal ions has been reviewed. Nevertheless, the impact of a cadmium exposure is fish behaviour had been shown mainly on freshwater fish.

Many behavioural consequences have been described: swimming alterations (Yorulmazlar and Gül, 2003), intraspecific interactions (Sloman *et al.*, 2003 a, b; Tilton *et al.*, 2003). Predator/prey interactions (Sullivan *et al.*, 1978; Scherer *et al.*, 1997; Scott *et al.*, 2003) and avoidance responses (Mc Nicol *et al.*, 1996, 1999). One study pointed out that it could induce cadmium in freshwater fish sensory deficiencies in both olfaction and in the lateral line system (Baker and Montgomery, 2001). However, apart from this, very few studies have demonstrated the impact of cadmium is seawater fish behaviour. For example, in the catfish *Ictalurus nebulosus* (Lesueur, j1819), 40µg/l cadmium electro-deteriorated performance orientation by blocking calcium channels in the basal membrane of electroreceptors (Neuman *et al.*, 1991). In addition, the white sea bass *Lates calcarifer* (Bloch,1790) and the flounder *Pleuronectes flesus* (Linnaeus, 1758) presented erratic swimming in response to an acute cadmium exposure at high concentration (10 mg/l) (Larsson *et al.*, 1976; Tophon *et al.*, 2003). Associated with this abnormal behaviour, the white sea bass excessive mucus producing exhibited year on the opercular area, hyperventilation and a lower feeding rate (Tophon *et al.*, 2003). Many studies concerning fish contamination by heavy metals, especially cadmium, have focused on the bioaccumulation of this metal in the organs of the fish (Tophon *et al.*, 2003). It appears that in fish, cadmium accumulates mainly in gill, liver, kidney and muscle. Cadmium in fish generally induces profound disruption of major bodily functions such as reproduction (Waldichuk, 1979), embryonic development and embryo post (Voyer *et al.*, 1979).

In the liver, this metal also induces the production of metallothionein and is responsible for an activity of the detoxifying enzym EROD (Lemaire-Gony *et al.*, 1995; Cattani *et al.*, 1996).

The different doses of methyl parathion used in this work do not lead to alterations in the structure and ultrastructure of mechanoreceptors. According to Zinkl *et al.* (1991); Saglio *et al.* (1996) the inhibition of AChE-induced carbamates leads to accumulation of acetylcholine in cholinergic synapses phenomenon can cause behavioural disturbances affecting more particularly locomotion and balance.

According to the works of Bretaud *et al.*, (2001), the swimming activity was studied in juvenile *Carassius auratus* exposed to carbofuran, a carbamate insecticide. Observations were made after short exposures (2, 4, 6 and 8) at concentrations of 25, 50 and 100 mg/l; exposure to carbofuran has changed significantly the swimming activity of the fish.

An increase in locomotors' activity was observed after 4 hours of exposure to a concentration of 25µg/l. Conversely, a decrease in locomotor activity is observed in response to the higher concentrations (after 6h and 8h to 50µg/l and after 2, 4 and 6 h at 100 µg/l). Rosic *et al.*, (1974) were able to show that a subcutaneous injection of an organophosphorus (the armine) in a Serranidae (*Serranus scriba*) causes behavioural disturbances for cholinesterase activity decreased by only 20%. They also noted that this disturbance persisted even after the AChE activity has returned to a normal state. Dutta *et al.*, (1992), report that the swimming activity is more affected than the acetyl cholinesterase activity in perch exposed to diazinon.

Ortiz and Lutz (1995) showed that there was a relationship between these neurotransmitters and swimming activity in a Sciaenidae (*Sciaenops ocellatus*) (Linnaeus, 1766).

Conclusion

The exposure of neuromast to a cytotoxic antibiotic such as gentamicin causes alteration of ciliated sensory cells is illustrated in this alteration transmission electron microscope by the destruction

of the cilia of the apex of the ciliated sensory cell synaptic structures and present in basal cytosol. Depending on the antibiotic dose, the more the dose, the more the degenerative process is fast; however, it is reversible after the discontinuation of treatment stopped. By neuromast against exposure to a heavy metal such as cadmium and pesticides such as Methyl parathion does not affect the epithelium of sensory cells and accessories.

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