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Sensory Disturbance by Six Insecticides in the Range of μg/L in *Caenorhabditis elegans*

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Using *Caenorhabditis elegans* as an animal model, the possible toxic effects of six insecticides (dinotefuran, thiamethoxam, thiacloprid, nitenpyram, acetamiprid, and sulfoxaflor) commonly used in agriculture on sensory perception were examined. The sensory behaviors of thermotaxis, avoidance of copper ion, chemotaxis to NaCl, and chemotaxis to diacetyl were measured to investigate the damage on sensory perceptions in nematodes exposed to the examined insecticides in the range of micrograms per liter (μg/L). Exposure to dinotefuran, thiamethoxam, thiacloprid, nitenpyram, acetamiprid, or sulfoxaflor at concentrations of 10–100 μg/L resulted in severe deficits in sensory perceptions to temperature, copper ion, NaCl, and diacetyl. The relative neurotoxicity of the six insecticides examined to *C. elegans* were shown as dinotefuran > thiamethoxam > thiacloprid > nitenpyram > acetamiprid > sulfoxaflor. Moreover, post-treatment with the antioxidant ascorbate effectively suppressed the production of reactive oxygen species and damages of sensory perceptions induced by the six insecticides, indicating that the activation of oxidative stress can act as an important cellular contributor to the observed damage of the examined insecticides in affecting sensory perceptions. Our data highlighted the potential toxicity of the six insecticides at low concentrations in inducing sensory disturbance to environmental organisms.

Keywords: neurotoxicity, sensory perception, insecticides, *Caenorhabditis elegans*, oxidative stress

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INTRODUCTION

The application of insecticides has made great contributions to the development of agriculture around the world. Among insecticides, most of the neonicotinoid insecticides are considered as classic neurotoxins since they can irreversibly target to nicotinic acetylcholine receptors and cause paralysis and eventual death in most arthropods rather than in vertebrates (Matsuda et al., 2005; Bradford et al., 2020). Moreover, diamide insecticides could damage muscle contraction *via* activating ryanodine receptors and releasing stored calcium from the sarcoendoplasmic reticulum, which is an important mechanism to control lepidopteran pests (Cordova et al., 2006). Sulfoximine insecticides (such as sulfoxaflor) could kill insects by damaging Ca^{2+} homeostasis in muscle cells (Guo et al., 2019).

After release into the environment, insecticides potentially cause toxicity to environmental organisms. As reported in previous studies, organochlorines, organophosphates, carbamates,

pyrethroids, and neonicotinoid insecticides could cause damage on reproduction, feeding, and avoidance of predation in poultry populations (Walker, 2003). In addition, exposure to insecticides also poses a great threat to some soil organisms—for example, cycloxaprid, a novel neonicotinoid insecticide, could induce neurotoxicity in earthworms, such as neurological dysfunctions, stress responses, and damage on calcium binding (Qi et al., 2018).

Due to the properties of short lifespan, small size, short life cycle, and high sensitivity to pollutants (Leung et al., 2008; Haegerbaeumer et al., 2018; Queirós et al., 2019; Wang, 2020; Zhang et al., 2022), *Caenorhabditis elegans* has been used as an ideal model for examining the response to environmental toxicants or stresses (Wang et al., 2021a; Liu et al., 2021c; Sun et al., 2021). Recently, it was reported that some neonicotinoid insecticides could cause a dysfunction in the development, reproduction, and locomotion behaviors of *C. elegans* (Bradford et al., 2020). Moreover, *C. elegans* is an important animal model for the developmental study of nervous systems because of its simple nervous system structure composed of 302 neurons in adult hermaphrodite (Bargmann, 1998; Corsi et al., 2015). *C. elegans* is helpful for investigating different functions of the nervous system, such as learning and memory (Ardiel and Rankin, 2010; Bargmann et al., 1993). In addition, *C. elegans* is a powerful animal model for assessing neurotoxicity induced by different toxicants (Wang, 2019; Liu et al., 2021a)—for example, in *C. elegans*, the development of dopaminergic nervous systems and their functions (such as sensory perception behaviors) were found to be affected by exposure to nanoparticles (Qu and Wang, 2020). Considering the fact that ~80% of *C. elegans* proteome has human homologous genes, specifically neurodevelopmental genes (Riddle et al., 1997), *C. elegans* is an important animal model for both biomedical and environmental toxicology. Due to the availability of some disease models (Moy et al., 2009; Griffin et al., 2017), *C. elegans* is also useful for pharmaceutical screening.

Sensory perception is an important function for organisms to sense and detect the existence and alteration of environmental stimuli. Nevertheless, the possible toxic mode of action on sensory perception behaviors induced by exposure to insecticides remains largely unclear in organisms. Thus, the aim of this study is to compare the possible damage of six insecticides (acetamiprid, nitenpyram, thiacloprid, thiamethoxam, dinotefuran, and sulfoxaflor) on the sensory perception behaviors of *C. elegans* as representative for other nematodes. Among them, acetamiprid, nitenpyram, thiacloprid, thiamethoxam, and dinotefuran are neonicotinoid insecticides. Sulfoxaflor belongs to sulfoximine insecticides. *C. elegans* has been widely used for the assessment of pesticide toxicity (Gomez-Eyles et al., 2009; Roh and Choi, 2011; Ruan et al., 2012; Du et al., 2015; Yu et al., 2020). The well-described backgrounds of development and functions of the nervous system of *C. elegans* provide a strong support for this research. The sensory perception behaviors of thermotaxis, avoidance to copper ion, chemotaxis to NaCl, and chemotaxis to diacetyl were examined in this study. Thermotaxis reflects a sensory perception to physical stimuli. The chemotaxis to NaCl and avoidance to copper reflect a gustatory perception to soluble chemicals. The chemotaxis to diacetyl indicates an olfactory perception to volatile chemicals. Our results demonstrated the potential of exposure to the examined insecticides in the range of micrograms per liter (μg/L) in causing damage on sensory perceptions to different degrees in nematodes. The obtained data highlights the toxicity of long-term exposure to the examined insecticides at low concentrations in inducing damage on sensory perceptions in environmental organisms.

MATERIALS AND METHODS

Reagents

The standards for six insecticides (**Figure 1**) were obtained from Shanghai Pesticide Research Institute Co. (Shanghai, China). Acetamiprid (purity, 98.0%), nitenpyram (purity, 99.1%), thiacloprid (purity, 99.4%), thiamethoxam (purity, 98.0%), dinotefuran (purity, 99.0%), and sulfoxaflor (purity, 99.7%) were dissolved in distilled water to obtain stock solutions (1 g/L). The working concentrations of the examined insecticides are shown in **Supplementary Table S1**.

Strains and Maintenance

The wild-type (N2) nematodes were obtained from *Caenorhabditis* Genetics Center. Nematode growth medium (NGM) plates seeded with *Escherichia coli* OP50 were used to maintain the examined worms (Brenner, 1974). To obtain age-synchronous L1-larvae nematodes, the gravid worms were lysed using a bleaching mixture solution containing 2% HOCl and 0.45 M NaOH to release enough eggs from the body (Yang et al., 2021b). After that, the eggs were allowed to develop into L1-larvae on new NGM plates.

Exposure

Nematodes were exposed to six insecticides with the addition of OP50 (4×10^6 colony-forming units, CFUs) from L1-larvae to adult stage (adult day-1, approximately 4.5 days). The age-synchronous L1-larvae were used for the exposure. Insecticides were diluted into the examined concentrations using K-medium to determine their effects on different sensory perception behaviors.

Thermotaxis Assay

Radial temperature gradient was employed to perform the thermotaxis assay as described by Ye *et al.* (2008). A steeper temperature gradient in the range from 17°C (at the center) to 25°C (at the periphery) was prepared. To generate this radial temperature gradient, a vial containing frozen acetic acid was put on the bottom of 9-cm culture plates, and the culture plates were incubated at 26°C (the preferred temperature for nematodes) for 90 min. After exposure to

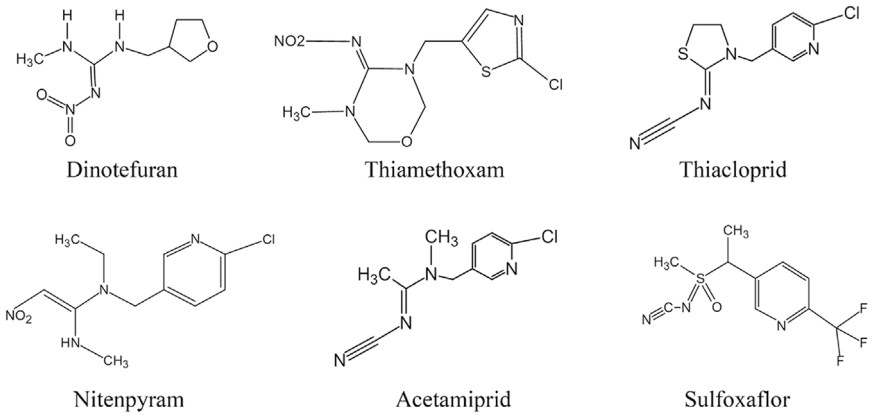

FIGURE 1 | Structures of the six insecticides examined.

insecticides and the following washing with M9 buffer, the individual worms were transferred on the agar surface of the prepared 9-cm culture plates having a thermal gradient. On the agar surface, the worms were allowed to move for 2 h. After removing the worms, the traces of movement that the examined worms left on the agar surface were captured by camera. The percentages of worms performing isothermal tracking (IT) were counted. Approximately 30 nematodes were examined for each exposure. Three replicates were performed.

Avoidance of Copper

Under normal conditions, the nematodes will avoid the copper on the NGM assay plate (Sambongi et al., 2000). An assay of avoidance of Cu^{2+} was performed as described previously (Sambongi et al., 2000). A 9-cm culture plate was divided into four equal parts. Among these four equal parts, normal NGM medium was added into two opposite sides. Meanwhile, NGM medium containing 100 μM Cu^{2+} was added into the other two opposite sides. After exposure to the insecticides and the following washing with M9 buffer, the worms were transferred onto the surface of Cu^{2+}-free parts for 1 h. The avoidance index was calculated as the value of the number of worms on NGM containing Cu^{2+}/total number of worms. Approximately 100 nematodes were examined for each exposure. Three replicates were performed.

Chemotaxis to NaCl

Chemotaxis to NaCl (a water-soluble chemoattractant) was performed as described previously (Saeki et al., 2001). An agar plug containing NaCl (100 mM) was placed on the off-center surface of an agar plate prepared with agar (20 g/L), potassium phosphate (5 mM, pH 6.0), $CaCl_2$ (1 mM), and $MgSO_4$ (1 mM). After overnight treatment, the NaCl plug was removed. In order to anesthetize the nematodes, 1 μl sodium azide (0.5 M) was added on position 4 cm away from NaCl plug position (control) and NaCl plug position. After 1 h, the chemotaxis index (CI) was calculated as the value of (number of worms within 1.5 cm of center of NaCl spot — number of worms within 1.5 cm of the control spot) / (total number of worms). Approximately 100 nematodes were examined for each exposure. Three replicates were performed.

Chemotaxis to Diacetyl

Chemotaxis to diacetyl was performed as described previously (Li et al., 2011). One microliter of diacetyl (10^{-2}) was added on the surface of the assay plates. Meanwhile, 1 μl sodium azide (0.5 M) was added on diacetyl position and the position 4 cm away from the diacetyl position (control). After 1 h, the CI was calculated as the value of (number of worms within 1.5 cm of the center of the diacetyl spot—number of worms within 1.5 cm of the control spot) / (total number of worms). Approximately 100 nematodes were examined for each exposure. Three replicates were performed.

Reactive Oxygen Species Production

Reactive oxygen species (ROS) production was used to reflect the activation of oxidative stress (Liu et al., 2021b). To detect the production of ROS, the control and exposed nematodes were labeled with CM-H_2DCFDA (1 μM) in the dark for 3 h (Yang et al., 2021a). After that, the nematodes were washed with M9 buffer three times. *C. elegans* was then mounted on a 2% agar pad and analyzed for their fluorescence signals at 510 nm (emission filter)/488 nm (excitation wavelength) using laser scanning confocal microscopy. The intestinal fluorescent intensities were determined by normalization against the autofluorescence. For each treatment, 50 nematodes were used. Three replicates were performed.

Pharmacological Assay

The nematodes were first exposed to six insecticides (100 μg/L) with the addition of OP50 (4×10^6 CFUs) from L1-larvae to adult day-1. After that, the nematodes were treated with 10 mM ascorbate (an antioxidant) for 24 h. Three replicates were performed.

Lethality Assay

For the lethality assay, 100 worms were added into each well in a 24-well plate with 250 μl insecticide at concentrations of 0.1, 1, 10,

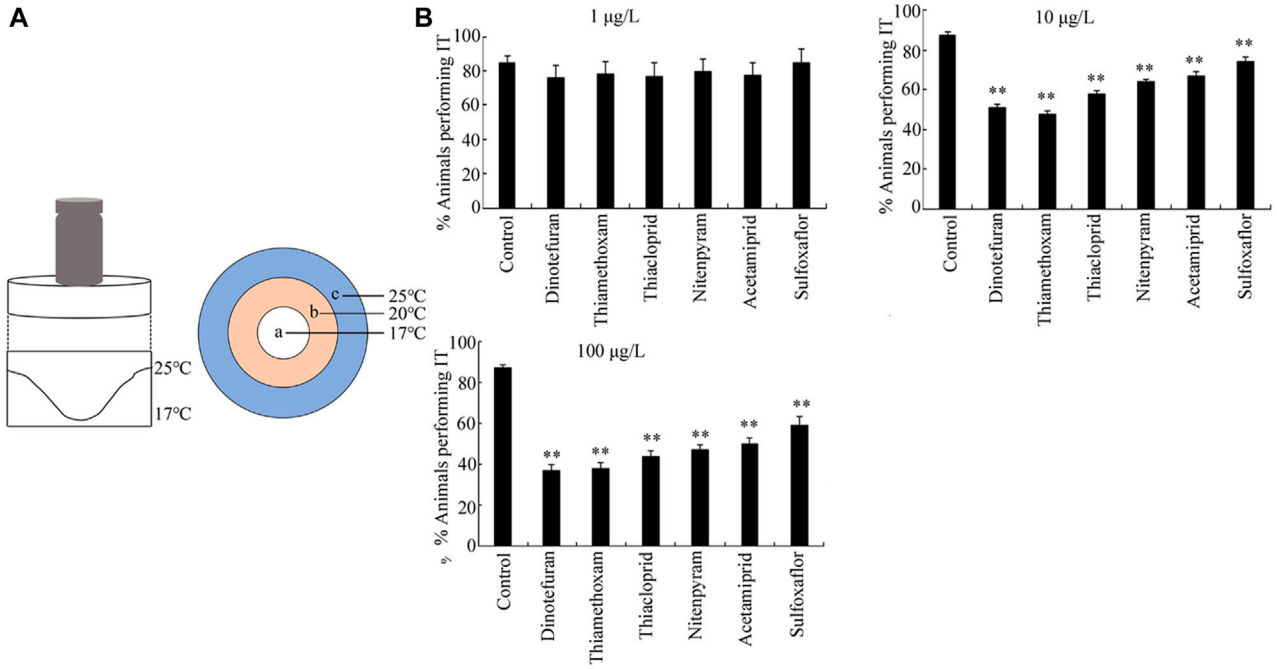

FIGURE 2 | Toxicity of insecticides in affecting thermotaxis. **(A)** Assay model for thermotaxis. **(B)** Toxicity of the six insecticides examined in affecting thermotaxis ability. IT, isothermal tracking. Nematodes were exposed to insecticide with the addition of OP50 from L1-larvae to adult day-1. Bars represent means ± SD. $^{**}p < 0.01$ *vs*. control.

or 100 mg/L. After exposure, the nematodes were transferred to a fresh NGM plate. After gently touching with a needle, the nematodes showing immobilization without recovery were considered dead. Three replicates were performed in each exposure.

The LC_{50} and 95% confidence interval of the examined insecticides were analyzed using SPSS 13.0 software. The LC_{50} and 95% confidence interval of the examined insecticides are shown in **Supplementary Table S2**.

Body Bend Assay

Body bend refers to the alteration in bending direction at the mid-body as described by Wang *et al.* (2021b). Forty nematodes were analyzed per treatment. Three replicates were performed.

Statistical Analysis

Statistical analysis was performed using SPSS 12.0 software. The probability level of 0.01 (**) was considered to be statistically significant. The one-way analysis of variance, followed by *post-hoc* Bonferroni test, was performed for group comparisons.

RESULTS

Toxicity Comparison of Six Insecticides in Affecting Thermotaxis

The first sensory perception response to the exposure of six insecticides examined was thermotaxis (**Figure 2A**). At the concentration of 1 μg/L, none of the six insecticides induced significant changes in thermotaxis in nematodes. However, after exposure to all the examined insecticides at concentrations of 10–100 μg/L, the thermotaxis ability of nematodes decreased significantly compared to the control ($p < 0.01$) (**Figure 2B**), and the order for the toxicity of the six insecticides in reducing thermotaxis was dinotefuran > thiamethoxam > thiacloprid > nitenpyram > acetamiprid > sulfoxaflor (**Figure 2B**).

Toxicity Comparison of Six Insecticides in Affecting Avoidance of Copper Ion

The second sensory perception response to the exposure of six insecticides examined was avoidance of copper ion (**Figure 3A**). Exposure to the six insecticides examined at the concentration of 1 μg/L did not significantly affect the avoidance index (**Figure 3B**). However, after exposure at concentrations of 10–100 μg/L, all the six insecticides examined could cause a significant increase in the avoidance index compared to the control ($p < 0.01$) (**Figure 3B**). At concentrations of 10–100 μg/L, the order for the toxicity of the six insecticides in reducing ability to avoid copper ion was dinotefuran > thiamethoxam > thiacloprid > nitenpyram > acetamiprid > sulfoxaflor (**Figure 3B**).

Toxicity Comparison of Six Insecticides in Affecting Chemotaxis to NaCl

The third sensory perception response to the exposure of six insecticides examined was chemotaxis to NaCl (**Figure 4A**). After

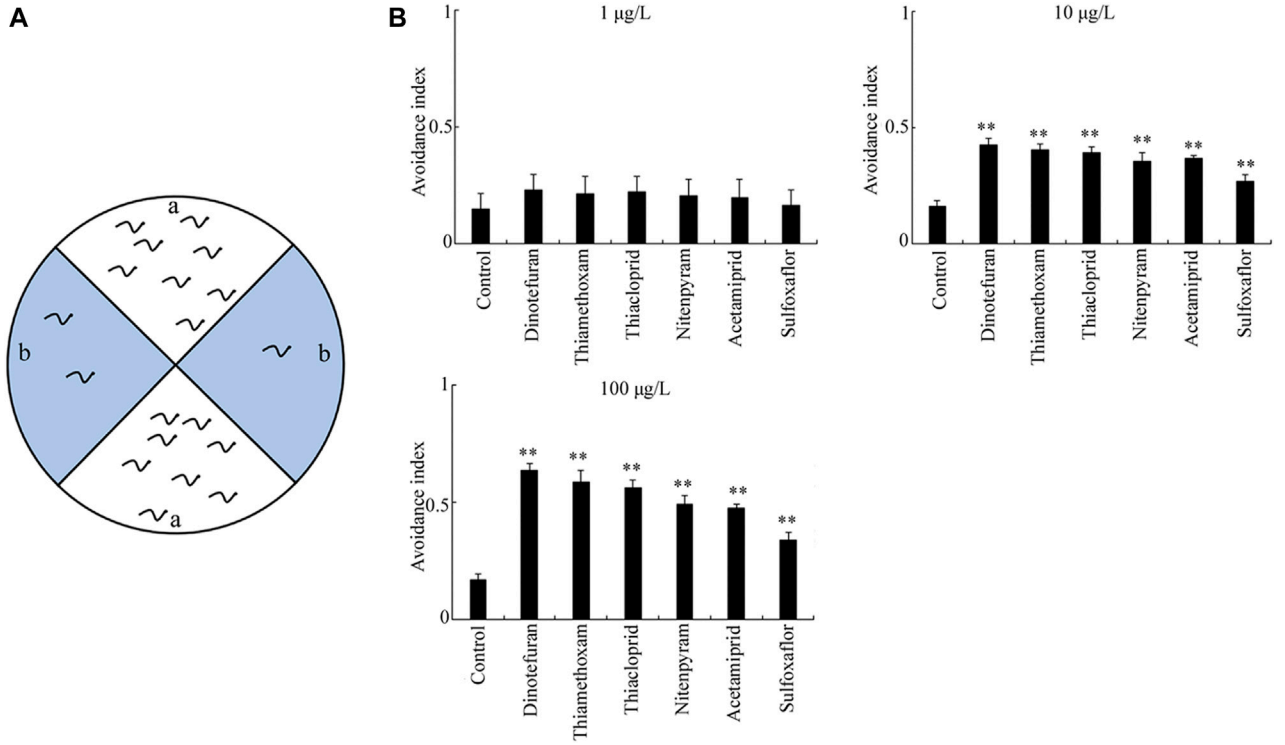

FIGURE 3 | Toxicity of insecticides in affecting avoidance of copper ion. **(A)** Assay model for avoidance of copper ion. **(B)** Toxicity of the six insecticides examined in affecting the avoidance of copper ion. Nematodes were exposed to insecticide with the addition of OP50 from L1-larvae to adult day-1. Bars represent means ± SD. $^{**}p < 0.01$ *vs.* control.

exposure at the concentration of 1 μg/L, dinotefuran, thiamethoxam, thiacloprid, nitenpyram, acetamiprid, and sulfoxaflor did not alter chemotaxis to NaCl obviously (**Figure 4B**). However, after exposure at concentrations of 10–100 μg/L, all the examined insecticides significantly decreased the chemotaxis to NaCl compared to the control ($p < 0.01$) (**Figure 4B**). Additionally, after exposure at concentrations of 10–100 μg/L, the order for the toxicity of the six insecticides in inhibiting chemotaxis to NaCl was dinotefuran > thiamethoxam > thiacloprid > nitenpyram > acetamiprid > sulfoxaflor (**Figure 4B**).

Toxicity Comparison of Six Insecticides in Affecting Chemotaxis to Diacetyl

The fourth sensory perception response to the exposure of six insecticides determined was chemotaxis to diacetyl (**Figure 5A**). After exposure at the concentration of 1 μg/L, dinotefuran, thiamethoxam, thiacloprid, nitenpyram, acetamiprid, and sulfoxaflor did not remarkably influence chemotaxis to diacetyl (**Figure 5B**). Differently from this, the exposure to all the examined insecticides at concentrations of 10–100 μg/L significantly inhibited chemotaxis to diacetyl compared to the control ($p < 0.01$) (**Figure 5B**). Moreover, in the concentration range of 10–100 μg/L, the order for the toxicity of the six insecticides in suppressing chemotaxis to diacetyl was dinotefuran > thiamethoxam > thiacloprid > nitenpyram > acetamiprid > sulfoxaflor (**Figure 5B**).

Treatment With Antioxidant Suppressed the Damage of Insecticides on Sensory Behaviors

Exposure to the six insecticides (100 μg/L) examined from L1-larvae to adult day-1 caused significant ROS production ($p < 0.01$) (**Figure 6A**). In contrast, treatment with 10 mM ascorbate from adult day-1 for 24 h could not induce obvious ROS production. Moreover, after exposure to insecticides, post-treatment with 10 mM ascorbate could obviously suppress ROS production in nematodes exposed to the six insecticides examined ($p < 0.01$) (**Figure 6A**).

In nematodes, treatment with 10 mM ascorbate alone could not alter the sensory perception of thermotaxis, avoidance of copper ion, chemotaxis to NaCl, and chemotaxis to diacetyl (**Figures 6B–E**). Moreover, after exposure to insecticides, post-treatment with 10 mM ascorbate could further significantly inhibit the damage induced by exposure to the six insecticides examined in reducing thermotaxis, suppressing avoidance of copper ion, decreasing chemotaxis to NaCl, and reducing chemotaxis to diacetyl (**Figures 6B–E**).

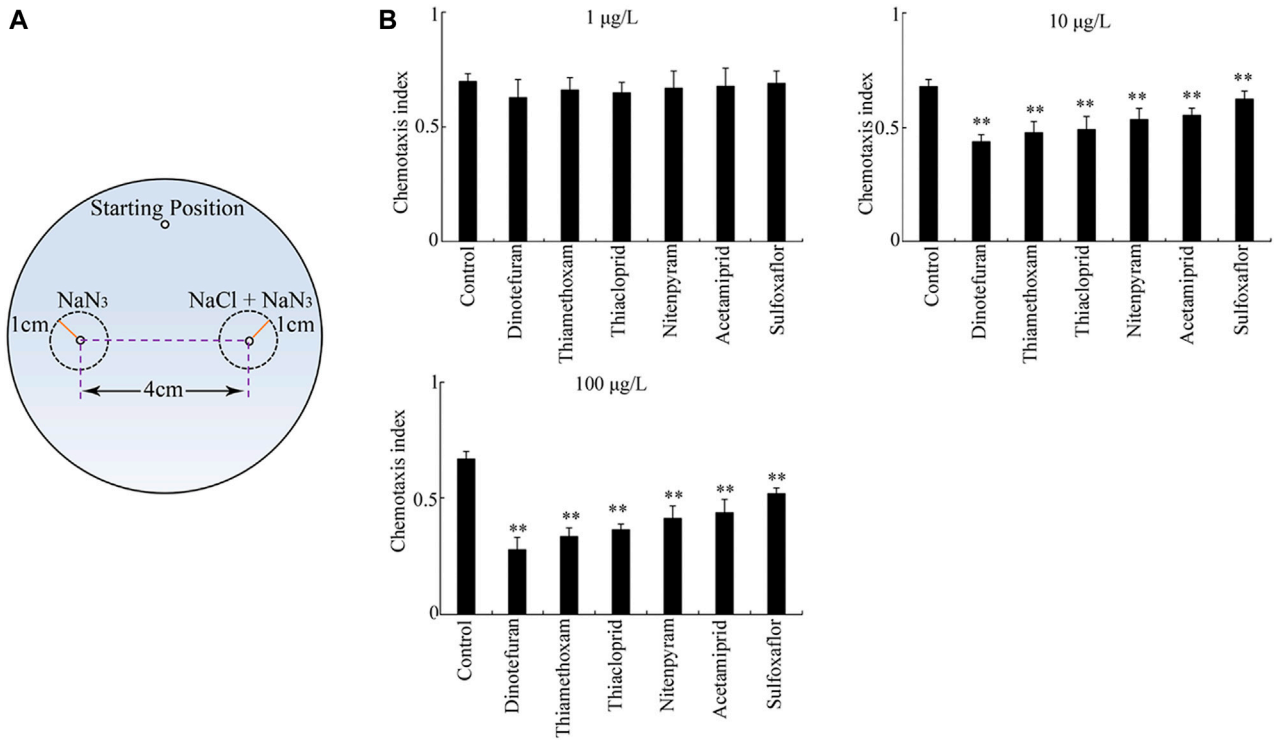

FIGURE 4 | Toxicity of insecticides in affecting chemotaxis to NaCl. **(A)** Assay model for chemotaxis to NaCl. **(B)** Toxicity of the six insecticides examined in affecting chemotaxis to NaCl. Nematodes were exposed to insecticide with the addition of OP50 from L1-larvae to adult day-1. Bars represent means ± SD. $^{**}p < 0.01$ *vs*. control.

DISCUSSION

Normally, the environmental concentrations of insecticides in surface water were in the range of microgram or nanogram per liter (μg/L or ng/L) (Faria et al., 2020). This is one of the important reasons for our focus on insecticide concentrations in the range of μg/L—to determine the effect of insecticide exposure on sensory perception in this study. Besides this, according to our study, exposure to the six insecticides examined at a concentration of 1–100 mg/L caused significant lethality, but exposure to the six insecticides (0.1 mg/L) examined did not induce significant lethality (**Supplementary Figure S1**). Thus, the observed damage on sensory perceptions in nematodes exposed to the six insecticides examined at a concentration of ≤100 μg/L might be largely not due to the lethal effect. This is another reason for our focus on insecticide concentrations in the range of μg/L—to determine the effect of insecticide exposure on sensory perception in nematodes.

Using *C. elegans* as an animal model, it has been shown that exposure to certain insecticides (such as neonicotinoid insecticides) could result in oxidative stress and damage on reproduction, locomotion behaviors, and growth (Rajini et al., 2008; Han et al., 2017; Kudelska et al., 2017; Zeng et al., 2017; Bradford et al., 2020). In this study, we found that prolonged exposure to insecticides in the range of μg/L potentially caused damage on multiple aspects of sensory perception in nematodes.

Temperature is an important environmental factor for the survival of organisms. Meanwhile, temperature is also an important environmental stimulus for environmental animals to sense. *C. elegans* has highly sensitive and sophisticated thermosensory mechanisms to detect environmental temperatures (Takeishi et al., 2020). As shown in **Figure 2**, exposure to neonicotinoid insecticides potentially noticeably affected thermotaxis behavior in nematodes. Sulfoxaflor had the weakest toxic effect on thermotaxis behavior among the six insecticides examined.

Avoidance of copper ion reflects a form of gustatory perception of worms, and this form of gustatory perception is controlled by ASH sensory neurons (Wang, 2019). All the six insecticides examined (10–100 μg/L) suppressed the avoidance of copper and showed the following toxicity order: dinotefuran > thiamethoxam > thiacloprid > nitenpyram > acetamiprid > sulfoxaflor (**Figure 3**). The heavy metal Cu is neurotoxic for worms (Wang and Xing, 2009; Zhang et al., 2010). These observations suggested that exposure to the examined insecticides in the range of μg/L may cause the difficulty for organisms to avoid harmful compounds or noxious stimuli in the environment. It was also reported that exposure to some insecticides might adversely influence the avoidance of predation in the poultry populations (Walker, 2003).

Chemotaxis towards NaCl is another form of gustatory perception of worms, and ASE sensory neurons controlled this form of gustatory perception (Bargmann and Horvitz, 1991).

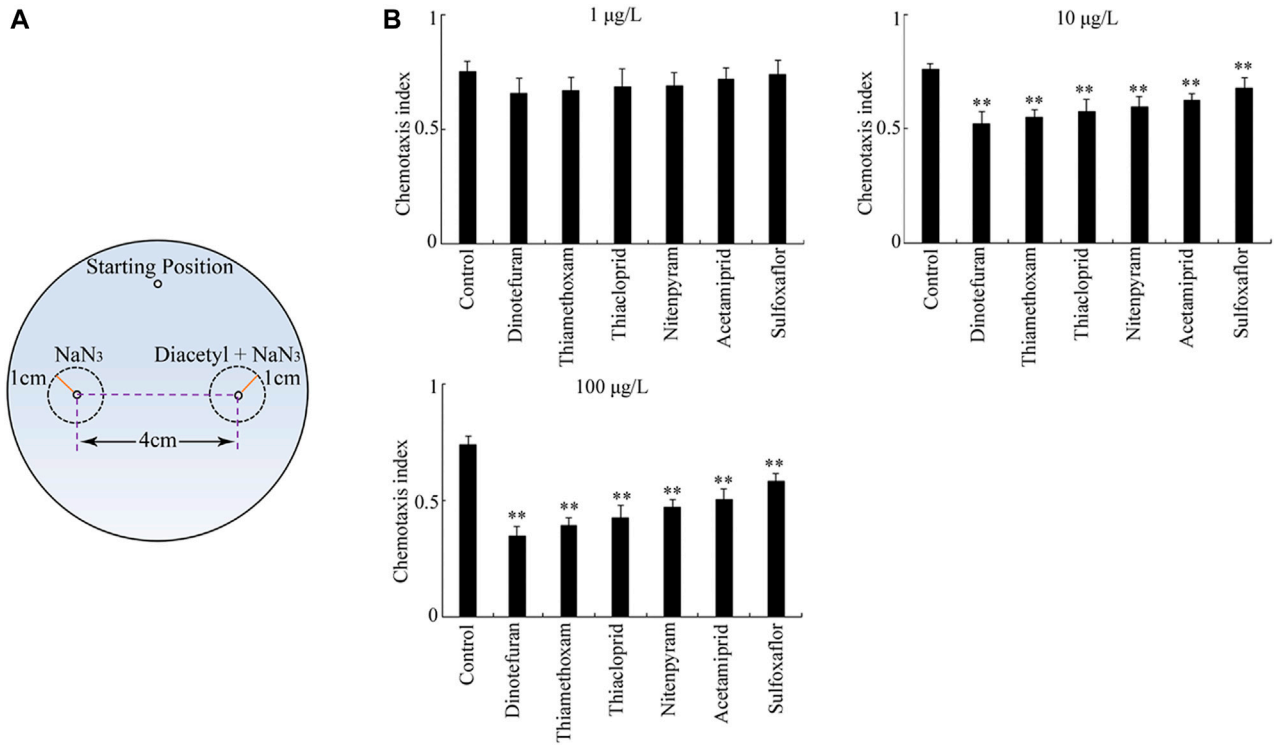

FIGURE 5 | Toxicity of insecticides in affecting chemotaxis to diacetyl. **(A)** Assay model for chemotaxis to diacetyl. **(B)** Toxicity of the six insecticides examined in affecting chemotaxis to diacetyl. Nematodes were exposed to insecticide with the addition of OP50 from L1-larvae to adult day-1. Bars represent means ± SD. $^{**}p < 0.01$ *vs.* control.

Similar to the observations on thermotaxis behavior and avoidance of copper ion, these six insecticides at concentrations of 10–100 μg/L caused a remarkable deficit in chemotaxis to NaCl and exhibited the following toxicity order: dinotefuran > thiamethoxam > thiacloprid > nitenpyram > acetamiprid > sulfoxaflor (**Figure 4**). In honeybees, exposure to thiamethoxam also caused a deficit in sensory perception of sugar (Aliouane et al., 2009).

Chemotaxis to diacetyl reflects the ability of olfactory perception in worm, and this sensory perception is mainly controlled by AWA sensory neurons (Metaxakis et al., 2018). After exposure at concentrations of 10–100 μg/L, the toxicity order for the six insecticides examined in affecting chemotaxis to diacetyl was dinotefuran > thiamethoxam > thiacloprid > nitenpyram > acetamiprid > sulfoxaflor (**Figure 5**). These observations demonstrated that, in worms exposed to the examined insecticides in the range of μg/L, both gustatory perception and olfactory perception would be noticeably damaged. Besides the four forms of sensory perceptions examined, there are also some other forms of sensory perceptions in *C. elegans* (Metaxakis et al., 2018). The possible effects of exposure to the examined insecticides on other forms of sensory perceptions still remain unclear in nematodes.

After exposure, 100 μg/L of the six insecticides examined obviously decreased the locomotion behavior as reflected by the endpoint of body bend (**Supplementary Figure S2**). In contrast, dinotefuran, thiamethoxam, thiacloprid, nitenpyram, acetamiprid, and sulfoxaflor at concentrations of ≤100 μg/L did not affect the locomotion behavior (**Supplementary Figure S2**). Therefore, the observed damage of the six insecticides examined at concentrations ≤100 μg/L on sensory perceptions was not due to the deficit in locomotion behavior.

In nematodes, exposure to insecticides (such as lindane) could induce oxidative stress (Yu et al., 2021). Ascorbate is a normally used antioxidant against oxidative stress in nematodes (Li et al., 2012). We further found that post-treatment with the antioxidant ascorbate could effectively suppress damage by the six insecticides examined in reducing themotaxis, in suppressing avoidance of copper ion, in decreasing chemotaxis toward NaCl, and in reducing chemotaxis toward diacetyl (**Figures 6B–E**). Meanwhile, post-treatment with the antioxidant ascorbate could also obviously suppress the ROS production induced by exposure to the six insecticides examined (**Figure 6A**). Therefore, the activation of oxidative stress can act as an important cellular contributor to the observed damage of the examined insecticides in affecting sensory perceptions. In nematodes, the ROS signals are mainly detected in the pharynx and the intestinal cells (Yang et al., 2021a). This implied that the examined insecticides might potentially bind to certain group(s) of biomolecules so as to induce oxidative damage in primary biological barriers, such as pharynx barrier, in nematodes. After that, neurotoxicity on sensory perceptions

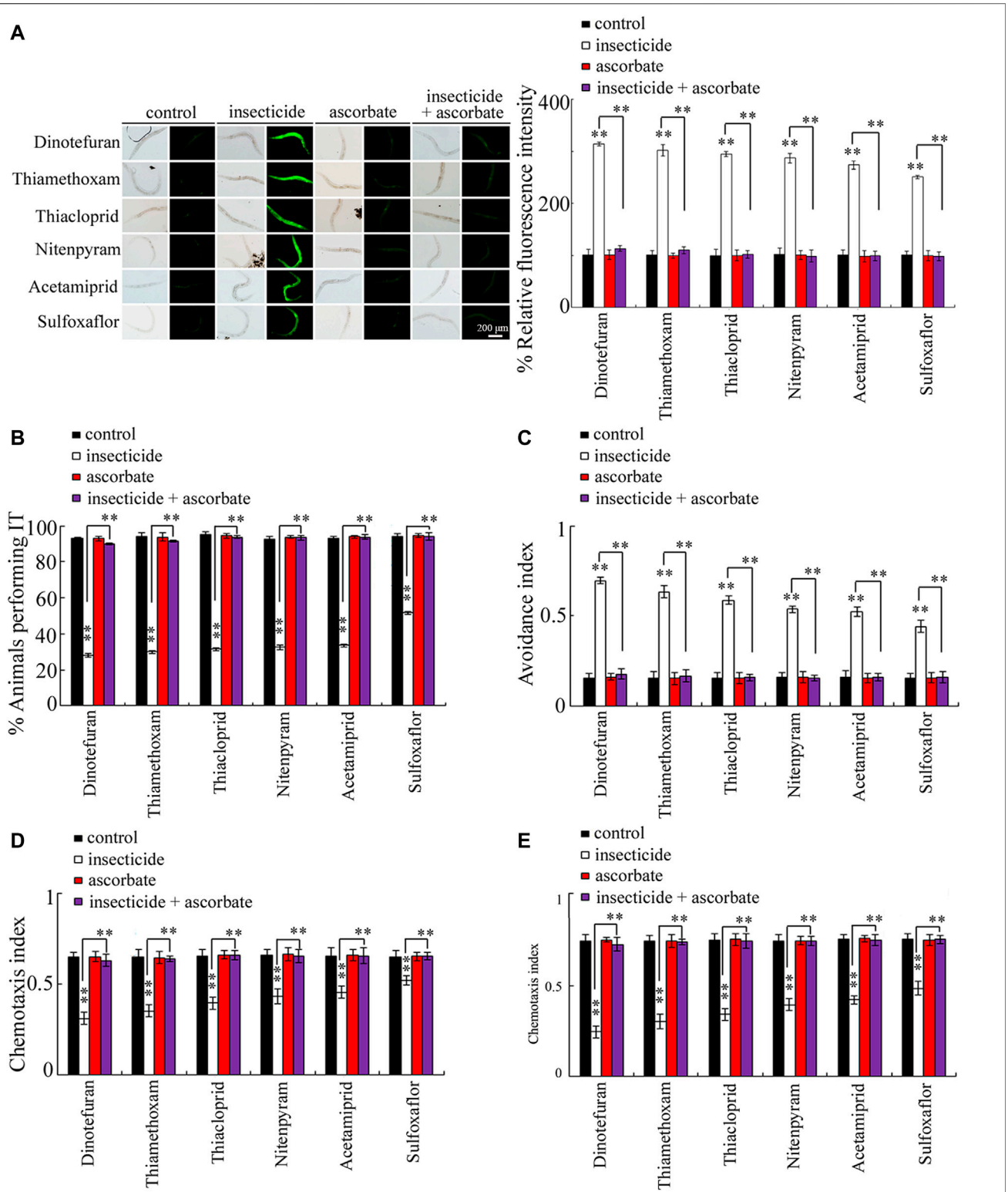

FIGURE 6 | Effect of treatment with ascorbate on the toxicity of the examined insecticides in affecting sensory behaviors. **(A)** Effect of treatment with ascorbate on the toxicity of the examined insecticides in inducing reactive oxygen species production. **(B)** Effect of treatment with ascorbate on the toxicity of the examined insecticides in affecting thermotaxis ability. **(C)** Effect of treatment with ascorbate on the toxicity of the examined insecticides in affecting the avoidance of copper ion. **(D)** Effect of treatment with ascorbate on the toxicity of the examined insecticides in affecting chemotaxis to NaCl. **(E)** Effect of treatment with ascorbate on the toxicity of the examined insecticides in affecting chemotaxis to diacetyl. Nematodes were first exposed to 100 μg/L insecticides with the addition of OP50 from L1-larvae to adult day-1. After that, the nematodes were treated with 10 mM ascorbate (an antioxidant) for 24 h. Bars represent means ± SD. $^{**}p < 0.01$ *vs.* control (if not specifically indicated).

will be further induced after insecticide exposure—that is, the damage on the examined four forms of sensory perceptions might be largely due to the fact that both sulfoxaflor and neonicotinoid insecticides could cause the activation of oxidative stress. This is also helpful to explain the fact that the sensory perceptions were affected in the same way by the examined insecticides.

The possible adverse effects of insecticides at various aspects have also been determined with other organisms, such as *Daphnia magna* and *Ceriodaphnia dubia* (Sheets et al., 2016; Raby et al., 2018). Moreover, it was observed that exposure to neonicotinoid insecticides could result in developmental neurotoxicity (Sheets et al., 2016)—that is, at least at high concentrations, the observed damage on sensory perceptions to temperature, copper ion, NaCl, and diacetyl might also be possibly due to the developmental deficits in related neurons and/or neuronal circuit(s) governing these sensory perceptions in nematodes.

CONCLUSION

The potential damage on sensory perceptions by exposure to six insecticides (dinotefuran, thiamethoxam, thiacloprid, nitenpyram, acetamiprid, and sulfoxaflor) was compared in *C. elegans*. Exposure to these insecticides at concentrations of 10–100 μg/L caused severe deficits in sensory perceptions to temperature, copper ion, NaCl, and diacetyl. Activation of oxidative stress acted as an important cellular mechanism for the observed damage of the six insecticides examined in affecting sensory perceptions. Therefore, our results suggested the potential of long-term exposure to the examined insecticides in the range of μg/L in inducing damage on sensory perceptions in organisms.

DATA AVAILABILITY STATEMENT

The original contributions presented in the study are included in the article/**Supplementary Material**, further inquiries can be directed to the corresponding author.

AUTHOR CONTRIBUTIONS

RZ and YY contributed to study design, experimentation, and initial draft writing. WZ and DW contributed to experimentation. YB and YW contributed to data analysis and result visualization. YB took charge of funding acquisition. All authors reviewed the final version of the manuscript and approve it for publication.

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SUPPLEMENTARY MATERIAL

The Supplementary Material for this article can be found online at: https://www.frontiersin.org/articles/10.3389/fenvs.2022.859356/full#supplementary-material

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