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Glucose administration attenuates spatial memory deficits induced by chronic low-power-density microwave exposure

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ABSTRACT

Extensive evidence indicates that glucose administration attenuates memory deficits in rodents and humans, and cognitive impairment has been associated with reduced glucose metabolism and uptake in certain brain regions including the hippocampus. In the present study, we investigated whether glucose treatment attenuated memory deficits caused by chronic low-power-density microwave (MW) exposure, and the effect of MW exposure on hippocampal glucose uptake. We exposed Wistar rats to 2.45 GHz pulsed MW irradiation at a power density of 1 mW/cm^2 for 3 h/day, for up to 30 days. MW exposure induced spatial learning and memory impairments in rats. Hippocampal glucose uptake was also reduced by MW exposure in the absence or presence of insulin, but the levels of blood glucose and insulin were not affected. However, these spatial memory deficits were reversed by systemic glucose treatment. Our results indicate that glucose administration attenuates the spatial memory deficits induced by chronic low-power-density MW exposure, and reduced hippocampal glucose uptake may be associated with cognitive impairment caused by MW exposure.

1. Introduction

The public concern regarding the potentially hazardous effects of microwave (MW) exposure on human health has grown over the past decade with rapid application of devices and systems employing MWs, such as ovens, 3 G telecommunications and Wi-Fi systems. Based on experimental data, exposure to MW has been postulated to produce a variety of adverse effects on the central nervous system, including headache, sleep disturbance, neuronal damage, increased blood–brain barrier (BBB) permeability, disturbances in neurotransmitter release, and cognitive alterations in humans and animals [1–5]. The possibly neurobehavioral changes after chronic exposure to MW have received attention. Some studies have demonstrated that spatial learning and memory that depend on the hippocampus are impaired by chronic exposure to MW irradiation in rodents [6–9], whereas others do not, even the spatial learning and memory was improved [10–12]. These controversial conclusions warrant further studies. However, the underlying mechanisms by which this MW exposure causes cognition alteration are poorly understood.

Glucose is the major energy source utilized by the brain, and it is essential for synthesis of neurotransmitters [13] and cholesterol [14], which is the substrate of neurosteroids. Besides these basic roles, glucose has been correlated to cognitive function. Fluorodeoxyglucose positron emission tomography has demonstrated that glucose uptake and metabolism are reduced in several brain areas including the hippocampus in Alzheimer's disease (AD) [15,16]. Learning impairment in aged rats is correlated significantly with a decrease in glucose utilization in regions of the limbic system [17]. In addition, multiple memory deficits are attenuated by glucose administration. For example, glucose treatment attenuates memory deficits in aged rodents, humans and patients with AD [18–20], as well as memory deficits produced by high-fat diets and drug treatments including γ-aminobutyric acid (GABA) receptor agonist, scopolamine and atropine in rodents [21–24]. Furthermore, hippocampal glucose uptake plays a crucial role in spatial learning and memory. Several studies have reported that hippocampal glucose uptake increases after training on spatial memory tasks [25–27], which is supported by increased glucose transporter expression in the hippocampus after a learning task [28]. Inhibition of glucose uptake using the central injection of glucose transporter inhibitors leads to memory impairment in chicks [29]. Intraseptal infusion of morphine, which induces spatial memory impairment, prevents hippocampal glucose uptake [30].

A recent study has shown that the metabolic rate of glucose in the brain is significantly reduced by short-term exposure to GSM radiation [31]. Whereas, another study arrives at a converse conclusion that acute cell phone exposure is associated with increased brain glucose

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metabolism in the region closest to the antenna [32]. Some other studies have shown that MW exposure results in alterations which suggest adverse effect of MW exposure on brain glucose metabolism. For example, MW exposure reduces energy metabolism in the rat brain, indicated by a reduced concentration of ATP and creatine phosphate [33], and blood glucocorticoids, which decrease brain glucose utilization, are elevated by 2.45 GHz irradiation [6,34]. However, the effects of MW exposure on brain glucose metabolism are inconsistent at present and the effect of chronic exposure has not been reported.

In the present study, we investigated whether glucose administration attenuated spatial learning and memory impairment induced by chronic exposure to 2.45 GHz low-power-density MWs. We also explored the effects of MW exposure on hippocampal glucose uptake, which may be partially stimulated by insulin [35,36]. And the levels of blood glucose and insulin were determined after MW exposure.

2. Materials and methods

2.1. Animals

Adult male Wistar rats (3 months old) were obtained from the Experimental Animal Center of Third Military Medical University (Chongqing, China) and weighed 270–310 g. The rats were housed in standard conditions of room temperature (22 ± 2 °C) and humidity (60–65%) under an artificial 12-h light and dark cycle (lights on at 7 a.m.). The rats were allowed *ad libitum* access to rodent chow and water during the experiment. All experimental procedures were approved by the Local Animal Use Committee and were carried out in accordance with the Guidelines on the Handling and Training of Laboratory Animals of Third Military Medical University.

2.2. MW exposure

The circular waveguide exposure system was used which has been described by Chou et al. [37] and used by Li et al. [6]. The individual cylindrical waveguides were connected to a single MW source (model 81133A; Agilent, Santa Clara, CA, USA) through a power divider and were simultaneously excited. The MW signals were amplified by an amplifier (model P7000s; Yamaha, Japan). Each circular waveguide was constructed of galvanized wire screen in which a circularly polarized TE_{11}-mode field configuration was excited. A cylindrical transparent plastic chamber (length 23.6 cm, diameter 17.6 cm, floor width 14.5 cm) which had ample ventilation holes was located in the middle of the waveguide. A rat could move freely inside the chamber.

The animals were subjected to MW or sham exposure in the plastic chambers. Seven groups of rats were exposed to circularly polarized, pulsed (10 μs pulse, 800 pps) 2.45 GHz MWs at a spatially averaged power density of 1 mW/cm^2. The rats were exposed to MWs for 3 h/day for 30 days. Simultaneously, Rats of three sham groups were exposed for the same time as the MW-exposed rats in similar conditions except the waveguides were not activated. In addition, three groups of rats that were never in contact with the exposure system were used as naive control. They were housed in the same room and subjected to the same procedures as were the MW-exposed and sham-exposed groups, but not subjected to MW or sham exposure. Each group contained six rats. All exposures were executed between 8 a.m. and 11 a.m. to eliminate the possible effect of circadian variation. The whole body specific absorption rates (SARs) were measured using a microprocessor-controlled twin-cell calorimeter with fresh rat carcasses and local SARs in the brain were obtained from time-temperature profiles measured with a non-interacting thermistor probe [6,38]. The average whole body SAR was 0.2 W/kg for up to 30 days, and the average brain SAR was estimated to be 0.7 W/kg for up to 30 days according to the obtained brain SARs of rats fixed in different orientations and locations.

2.3. Glucose administration

Four MW-exposed groups were used to evaluate the effect of glucose administration on memory impairment induced by MW exposure, two groups for Morris water maze (MWM) test and two groups for the radial arm maze (RAM) test. On the day before beginning MWM and RAM test, all rats received an intraperitoneal (i.p.) injection of saline to familiarize them with the injections. In the MWM and RAM testing, one group of MW-exposed rats received an i.p. injection of glucose (100 mg/ml) at a dose of 100 mg/kg 30 min before the daily training session. Another group of MW-exposed rats received a daily i.p. injection with the same volume of saline 30 min before training session.

2.4. Behavioral assessments

MWM and RAM tasks, which were sensitive to hippocampus dysfunction, were used to evaluate the spatial learning and memory of the rats 24 h after the final MW exposure. Three exposed groups were used for MWM, other three exposed groups were used for RAM.

The MWM testing was carried out according to the procedures developed by Morris [39]. The MWM (Chengdu TME Technology, China) consisted of a circular pool (130 cm in diameter, 50 cm deep) whose interior was painted gray. A circular escape platform (10 cm in diameter, 25 cm height) was located on one quadrant center of four hypothetical quadrants at a depth of 2 cm below the water surface. The water was made opaque with milk powder and maintained at 25 ± 2 °C. Each rat was subjected to one training session daily for six consecutive days. In each session, a rat was respectively placed into the water from the wall of four starting points in a random order. Therefore, each session consisted of four training trails. The rats were given a maximum time of 60 s to find the hidden platform. If a rat failed to find the platform in 60 s, it was gently guided to the platform. After landing or being placed on the platform, rats were allowed to remain there for 30 s before the next trial. On day 7, all animals were subjected to a probe trail, in which the platform was removed and the animals were placed into water from the quadrant opposite to the previous platform location and allowed to search the platform for 60 s. The variables of latency time and time spent in target quadrant were recorded and analyzed by an image analysis system throughout the testing.

The RAM system (Chengdu TME Technology, China) consisted of eight arms (45×12 cm) that radiated horizontally from a central cylindrical platform (26 cm in diameter) with a concave food well at the end of each arm. The floor and 10-cm high borders of the maze were made of black Plexiglas. The platform and arms were covered with transparent Plexiglas. Sixteen infrared photocells of diffuse reflection (two per arm) enabled a computerized analysis system to track and record movements of the rats. Testing procedures for the RAM were based on those of Winocur and Gagnon [19]. Before testing, the rats were reduced to 85% of normal body weight through food restriction. The animals were given a preliminary training for four consecutive days and broken pieces of food were scattered throughout the apparatus. For the first 3 days, animals were placed in pairs and allowed to probe the maze for 15 min. On the fourth day, the procedure was repeated, except rats were placed individually. Between rats, the maze was cleaned with 2.5% cider vinegar [40]. Following preliminary training, all rats were subjected to one testing trail per day for 10 consecutive days. Each trail consisted of baiting all eight arms with a piece of food and individually placing a rat on the cylindrical platform. After all eight arms had been entered, or 10 min had elapsed, the rats were removed. The number of errors which were indicated as re-entered arms that had been completely visited was recorded.

2.5. Hippocampal glucose uptake assay

Another one exposed group was used. The hippocampal glucose uptake was measured according to the methods of Fernando et al.

[41] and Shibata et al. [42]. The rats were deeply anesthetized with pentobarbital and killed by decapitation 24 h after the final MW exposure. The brains were rapidly removed into ice-cold, carbogen-bubbled artificial cerebrospinal fluid (aCSF, NaCl 124 mmol, KCl 3 mmol, NaH_2PO_4 1.25 mmol, $MgCl_2$ 2 mmol, $NaHCO_3$ 26 mmol, $CaCl_2$ 2 mmol, and glucose 10 mmol). Then, the hippocampal hemispheres were rapidly dissected out. All aCSF used subsequently was bubbled with carbogen. The hippocampal slices were prepared using a vibratome (DTK-3000W; Dosaka, Kyoto, Japan) in ice-cold aCSF and the slices from each hemisphere were kept together. Thereafter, hippocampal slices were transferred to fresh aCSF and incubated at 37 °C for 1 h. After incubation, the slices from each hemisphere were placed in the aCSF containing 0.1 μCi/ml [^{3}H]2-deoxy-d-glucose (^{3}H-2DG, specific activity 8 Ci/mmol; PerkinElmer, Waltham, MA, USA) supplemented with or without 100 nmol human recombinant insulin (Sigma, St Louis, MO, USA), and were incubated at 37 °C for 45 min. The uptake of ^{3}H-2DG was terminated by rapidly washing the slices four times in ice-cold PBS. The slices were transferred to preweighed filter paper and allowed to dry. The slices were then weighed and solubilized with SOLVABLE (PerkinElmer). An aliquot (200 μl) was added to Ultra Gold scintillation fluid (PerkinElmer) and counted in a Beckmam LS6500 scintillation counter (Beckman, USA).

2.6. Assay of blood glucose and insulin

All blood samples were taken from the rats 24 h after the final MW exposure. The animals were anesthetized with pentobarbital and blood samples (3 ml) were collected by heart puncture, then the rats were decapitated and used for hippocampal glucose uptake assay. Blood samples were centrifuged at 3000×*g* for 10 min. Serum glucose concentrations were determined by the glucose oxidase method using a glucose assay kit (Sigma-Aldrich, St Louis, MO, USA) and serum insulin levels were measured by radioimmunoassay with a Rat Insulin RIA Kit (Millipore, USA).

2.7. Statistical analysis

All data are presented as mean ± SEM and all the statistical comparisons were performed by SPSS 18.0. The data of escape latency and errors were compared by a repeated-measures analysis of variance (ANOVA) with the factor treatment and the repeated measure factor training days. Other data were analyzed by one-way ANOVA or Student's t test. All post hoc tests were performed by Fisher's LSD multiple comparisons tests. A value of $P<0.05$ was considered to be statistically significant.

3. Results

3.1. Effect of chronic low-power-density MW exposure on spatial learning and memory

To investigate the effect of MW exposure on spatial learning and memory, we determined the performance of rats in MWM and RAM tasks after MW exposure for 30 days. In the MWM task, repeated-measures ANOVA revealed a significant reduction in escape latency during the six training sessions [$F(5,75)=72.089$, $P<0.01$] and a significant difference between the groups (naive, sham, MW exposure) [$F(2,15)=4.104$, $P=0.038$], but no significant difference in interaction between factors [$F(10,75)=1.369$, $P=0.211$] (Fig. 1A). Chronic MW exposure induced significant prolongation in latency time on the fourth, fifth, and sixth days ($P<0.05$ and $P<0.01$ versus sham group). The sham group did not show a significant difference in latency time throughout the training period as compared with naive group. In the probe trail, the time spent in the target quadrant of MW-exposed rats was significantly reduced compared with the sham-exposed animals ($P<0.05$), but there was no significant difference in time spent in the target quadrant between sham and naive animals (Fig. 1B). Also, in the RAM test, repeated-measures ANOVA of errors showed a significant day effect [$F(9,135)=7.279$, $P<0.01$)] and a significant treatment effect [$F(2,15)=8.391$, $P=0.004$], as shown in Fig. 1C. The errors in the MW exposure group on days 5, 7, 9 and 10 were significantly reduced compared with the sham group ($P<0.05$). Throughout the 10 training days, no significant differences in errors were observed between the sham and naive groups.

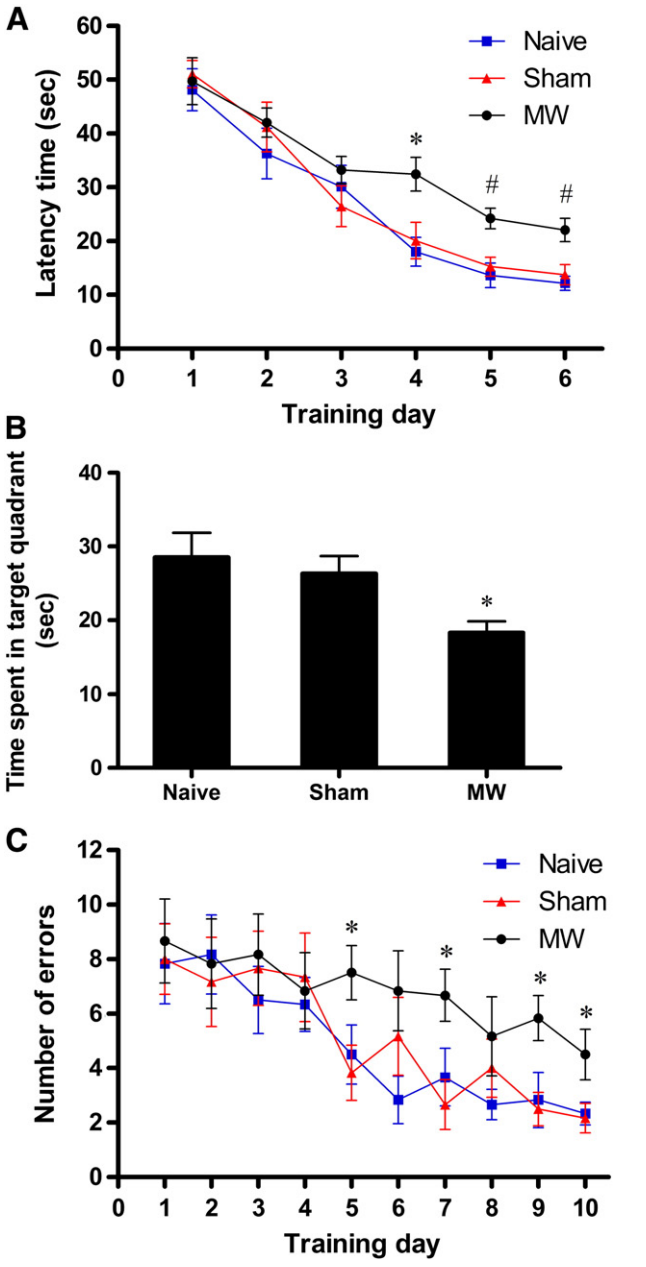

Fig. 1. Effects of MW exposure on performance in the MWM and RAM tasks. The rats exposed to MWs or sham exposed rats were subjected to the tasks at 24 h after the final exposure, and a group of naive control (never had contact with the exposure system) was used simultaneously. (A) Latency time during the six initial MWM training sessions with four trails in each session. (B) Time spent in the target quadrant in the probe trial. (C) Number of errors (re-entry into an arm already visited) in the RAM test. Data are means ± SEM, six animals per group, $^*P<0.05$, $^\#P<0.01$ versus corresponding values in sham group.

3.2. Effect of glucose administration on spatial memory deficits induced by MW exposure

To evaluate whether glucose treatment attenuated the spatial learning and memory deficits induced by MW exposure, the rats

exposed to MW were intraperitoneally injected with glucose or saline 30 min before the training sessions. In the MWM test, as shown in Fig. 2A, repeated-measures ANOVA revealed a significant reduction in escape latency during the training course [$F(5,50)=52.453$, $P<0.01$] and a significant effect of chronic MW exposure [$F(1,10)=7.265$, $P=0.022$]. The latency of the glucose-treated group on days 4–6 was significantly reduced compared with the saline-treated group ($P<0.01$). In the probe trail, the glucose-treated rats spent a longer time in the target quadrant as compared with the saline-treated rats ($P<0.05$) (Fig. 2B). In the RAM task, repeated-measures ANOVA revealed a significant decrease in errors during the training period [$F(9,90)=3.792$, $P<0.01$] and a significant effect of glucose treatment [$F(1,10)=6.048$, $P=0.034$]. Also, the errors in the glucose-treated group on days 5, 6 and 8–10 were significantly reduced compared with the saline-treated group ($P<0.05$) (Fig. 2C).

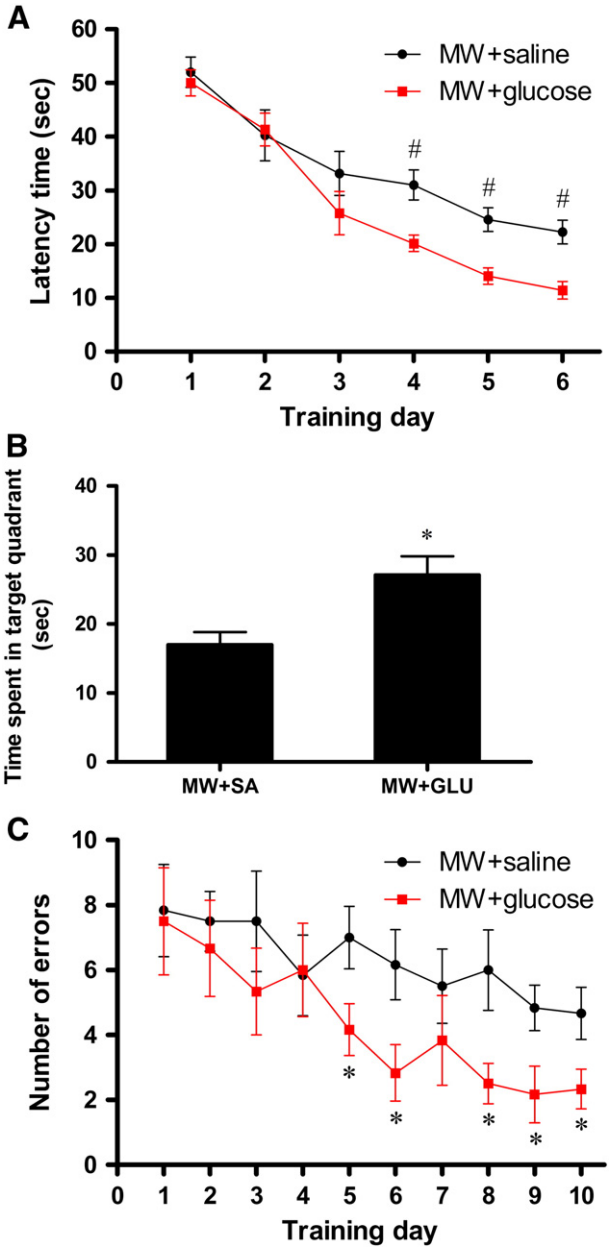

Fig. 2. Glucose administration attenuated the spatial memory impairment induced by MW exposure. The tests were performed 24 h after the last MW expose. Throughout the MWM and RAM testing, the rats were intraperitoneally injected with 100 mg/kg glucose or an equal volume of saline 30 min before daily training session. (A) Latency time during the six initial MWM training sessions with four trails in each session. (B) Time spent in the target quadrant in the probe trial. (C) Number of errors (re-entry into an arm already visited) in the RAM test. Data are means ± SEM, six animals per group, $^{*}P<0.05$, $^{\#}P<0.01$ versus corresponding values in sham group.

3.3. Effect of chronic low-power-density MW exposure on hippocampal glucose uptake

We used co-incubation of hippocampal slices and ^{3}H-2DG to investigate the effect of MW exposure on hippocampal glucose uptake. As shown in Fig. 3, one-way ANOVA showed that the glucose uptake in hippocampal slices of rats exposed to MW significantly decreased compared with the sham-exposed rats ($P<0.01$), but there was no significant difference between the sham and naive groups. Moreover, hippocampal glucose uptake was detected in case insulin was present in aCSF, basing on the reports that the brain glucose metabolism could be regulated by insulin. Also, chronic MW exposure induced a significant reduction in hippocampal glucose uptake in slices in the presence of insulin ($P<0.01$ versus sham group), whereas the difference in hippocampal glucose uptake was not significant between sham-exposed and naive rats.

3.4. Effect of chronic low-power-density MW exposure on blood glucose and insulin levels

The hippocampal availability of glucose might be influenced by changed blood glucose and insulin levels after MW exposure. Therefore, we examined the levels of blood glucose and insulin in rats. The blood glucose and insulin levels were not affected by chronic low-power-density MW exposure, as shown in Fig. 4A and B.

4. Discussion

Public concern regarding potentially deleterious effects of MW exposure on health has grown with the rapid increase in environmental MW radiation. The current study demonstrated that the hippocampus-dependent spatial learning and memory of rats was impaired by chronic exposure to 2.45 GHz low-power-density MWs. This cognitive deficit was attenuated by systemic glucose administration. A potential explanation of the cognitive deficit was given by an additional study that showed a decreased glucose uptake in hippocampal slices after chronic MW exposure. Although more explorations are needed, this result suggests that the spatial learning and memory deficits induced by chronic low-power-density MW exposure might be associated with reduced glucose uptake in the hippocampus.

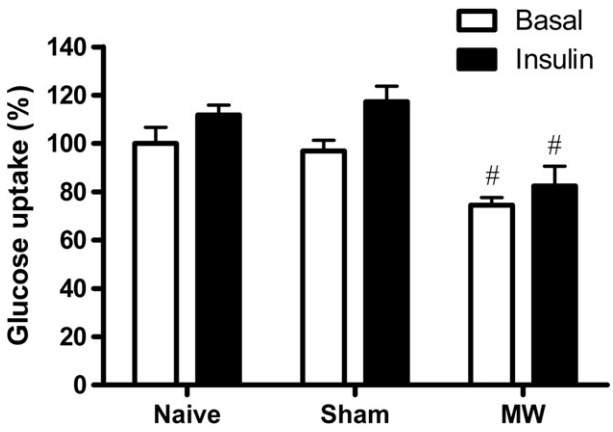

Fig. 3. Effects of MW exposure on hippocampal glucose uptake. Hippocampal slices were prepared 24 h after the final MW and sham exposure, and a naive control group was used. The slices were cultured for 45 min in aCSF containing 0.1 μCi/ml ^{3}H-2DG supplemented with or without (basal) 100 nmol insulin. Data are expressed as percentage and are means ± SEM, six animals per group, $^{\#}P<0.01$ versus corresponding values in sham group.

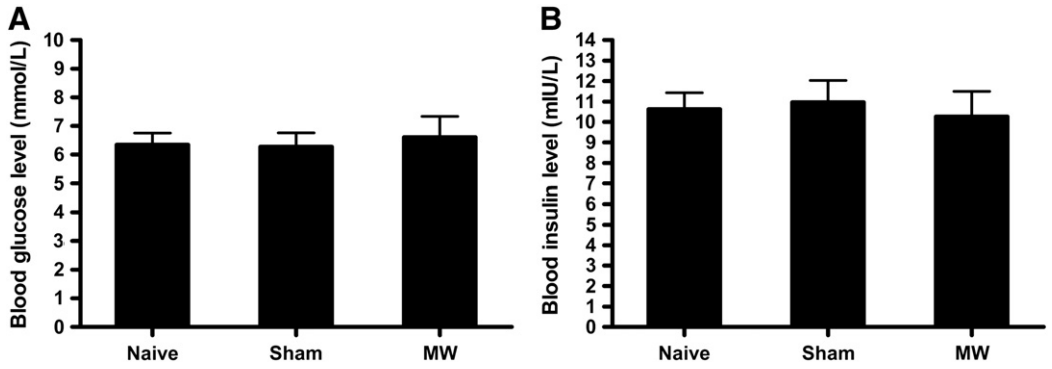

Fig. 4. Effects of MW exposure on blood glucose and insulin levels. The blood samples were collected 24 h after the final MW and sham exposure, and a naive control group was used. (A) Levels of blood glucose. (B) Levels of blood insulin. Data are expressed as means ± SEM, six animals per group.

However, the neurobehavioral effects of MW exposure are still a matter of debate. Several studies have reported that memory performance of rats is not affected by MW exposure [10,11,43]. These findings are in contrast with the present study. However, it should be noted that the MW exposures applied in these studies were either short-term or other frequencies. Several other studies have demonstrated significant effects of MW exposure on spatial memory [8,40,44]. Besides, our results are consistent with recent reports that chronic exposure to low-level MWs induces spatial memory deficit [8,45]. These conflicts might be due to diverse methods and designs such as frequency, orientation, modulation, power density and exposure duration, and even the different maze apparatus. Therefore, further research is needed to bring a definite conclusion about the behavioral effects of MW exposure.

Extensive evidence has shown that glucose administration, either systemic treatment or direct infusion into the brain, enhances memory in humans and rodents [46,47]. Most learning and memory processes facilitated by glucose administration are associated with hippocampal function, such as inhibitory avoidance conditioning [48], spontaneous alternation [49], delayed recall [50], RAM and spatial non-matching-to-sample tasks [19]. Therefore, glucose administration is postulated to reverse the hippocampus-dependent spatial learning and memory deficits caused by MW exposure. As expected, glucose treatment effectively attenuated the spatial learning and memory impairment following MW exposure in our study. Augmented synthesis and release of acetylcholine (ACh) in the hippocampus may account for glucose attenuation of cognitive deficits induced by MW exposure. Much research has revealed that increased ACh function in the hippocampus is involved in the memory-enhancing effect of glucose [51–53]. For instance, systemic, intraseptal and intrahippocampal administration of glucose augments ACh release in the hippocampus [49,54,55]. Meanwhile, increased ACh function in the brain typically facilitates learning and memory [56,57]. Furthermore, acetylcholinesterase (AChE) inhibitors mimic the reversing effects of glucose on memory deficits caused by septal GABA receptor activation [58]. Of interest, several studies have reported that low-level MW irradiation decreases cholinergic function and release of ACh in the hippocampus [59,60], and pretreatment using physostigmine, an AchE inhibitor, reverses the memory deficits produced by MW exposure [40]. The ability of ACh to cause an increase in intrasynaptosomal free calcium ion concentration was found to decrease by 60% in MW exposed rats [61]. Therefore, it is reasonable that glucose reverses MW-induced cognitive deficit by increasing intrasynaptosomal free calcium ion concentration through enhanced ACh function.

Hippocampal glucose availability is associated with spatial memory performance. Thus, we examined the hippocampal glucose uptake in slices and then confirmed that it was reduced by chronic exposure to low-level MWs. Although the incubated hippocampal slices in vitro cannot strictly represent the endogenous conditions, our result is consistent with a recent report that GSM radiation results in reduced glucose metabolism in the temporoparietal junction and anterior temporal lobe of the cerebral hemisphere ipsilateral to the exposure [31], but inconsistent with another similar research that the glucose metabolism is increased by cell phone exposure in the region closest to the antenna [32]. Although the exposure designs, such as frequency and duration, and brain regions are different in these studies including our current research, this conflict demands further studies on the effects of MW exposure on brain glucose metabolism and uptake. Also, our current results extend the findings that memory deficits are associated with reduced hippocampal glucose uptake and metabolism [15–17,62,63]. Therefore, impaired hippocampal glucose uptake might be a potential mechanism for MW-induced spatial learning and memory deficits. A decrease of extracellular glucose in the hippocampus during spatial working memory testing indicates an increase of glucose uptake and demand by hippocampal cells during memory processing [25,64]. From this point of view, glucose treatment may attenuate MW-exposure-induced cognitive deficit partially by increasing hippocampal glucose uptake. This hypothesis is supported by the fact that morphine-induced spatial memory deficit and decrease of hippocampal glucose uptake are reversed by small doses of glucose injected into medial septum [30]. This low dose of glucose administration may increase the hippocampal glucose uptake through regulating GABA and ACh neurons projecting from the medial septum to the hippocampus. A recent study reported that cognitive impairment in Alzheimer's mice was reversed by long term exposure to 918 MHz radiation [65], which may be a result of enhanced brain mitochondrial function [66]. Whereas previous studies have demonstrated that the hippocampal glucose metabolism and uptake decreased significantly in the patients with AD and glucose administration improved the cognitive functions of AD. These results indicate that reduced hippocampal glucose utilization correlates with and partially accounts for the cognitive deficits in AD. Similarly, our investigation showed that the hippocampal glucose uptake decreased parallel with spatial memory impairments after chronic MW exposure, and glucose treatment reversed these cognitive impairments. However, the present results simply manifest MW-induced impairment of hippocampal glucose uptake which may partially account for MW-induced cognitive impairment rather than a direct proof, although the connection between brain glucose metabolism and cognition has been well established.

Based on the reports that the brain glucose metabolism could be regulated by insulin [36], hippocampal glucose uptake might be influenced by altered blood insulin and glucose which are probably induced by the exposure in vivo. A recent study showed that long-term 50 Hz magnetic field (MF) exposure increased BBB permeability in a diabetic rat model, and insulin treatment decreased the effect of MF on BBB and reduced the blood glucose level [67]. So, it is possible that MW exposure affects glucose metabolism by altered blood insulin level which will result in alterations of blood glucose and BBB permeability. Thus the blood insulin levels were examined after

the MW exposure, whereas both the levels of blood glucose and insulin were not altered by MW exposure in the present study. Therefore our experiments offer no evidence that MW exposure would affect the glucose uptake and metabolism by impairing the insulin system in vivo. Also this result means that the MW exposure will not affect the hippocampal glucose availability through reduced glucose supply, and glucose attenuates the cognitive impairment by other mechanisms rather than direct supplement of glucose supply. Accordingly, some other mechanisms whereby MW exposure induces the decrease of hippocampal glucose uptake expect for further investigations, such as electromagnetic field exposure induced glucocorticoids elevation and neuronal mitochondria damage [6,68], both of which play important roles in brain glucose utilization.

In summary, the present study demonstrates that glucose administration attenuated the spatial learning and memory deficits caused by chronic low-power-density MW exposure, and indicates a potential relationship between decreased hippocampal glucose uptake and the MW-induced cognitive deficit. Although this association needs further studies to be confirmed, our results provide a promising approach for further exploration into the neurobehavioral effects of MW exposure.

Acknowledgments

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