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Public Health Open Access
ISSN: 2578-5001

Low-Density Polyethylene Microplastics in the Blood Seems not Induce Alzheimer's Disease in Wistar Rat

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Editorial

Volume 7 Issue 2

Received Date: September 18, 2023

Published Date: September 28, 2023

DOI: 10.23880/phoa-16000250

Keywords: Health Problems; Microplastic; Food

Editorial

Microplastic particles <5 mm in the blood pose health problems to humans. One of the entry points for low density polyethylene microplastics in the human blood is through consumption of contaminated food. These low density polyethylene microplastic particles are found in table salt, canned sardines, beer, sea fish, honey, sugar, tea bags, minerals and drinking water. These findings estimated between 37 to as high as billion microplastic particles from those various food products. The number of low density polyethylene microplastic particles that contaminate food and beverages will continue to increase along with the increase in plastic debris in the environment.

Low density polyethylene microplastics in the digestive tract will be phagocytosed into the blood and the lymph circulation. These particles will try to be destroyed by cellular defense mechanisms through both oxygen-dependent and oxygen-independent mechanisms. Furthermore, low density polyethylene microplastics in the blood will circulate to all parts of the body. These particles have been reported in the liver, lungs, heart, muscles, kidneys and brain. Some microplastic particles will be excreted with the feces and urine.

Low density polyethylene microplastics in the body will trigger oxidative stress due to high free radicals. A decrease in antioxidant enzymes is seen in studies using low density polyethylene microplastics as an exposure material. Free radicals induced by low density polyethylene microplastics come from three components, namely plastic monomers, endogenous additives included during the production process (such as phthalate, bisphenol A, nonylphenol, polybrominated diphenyl ethers) and environmental pollutants absorbed while in nature (such as polychlorinated biphenyls; polycyclic aromatic hydrocarbons; 1,1,1-trichloro-2,2-bis (p-chlorophenyl) ethane, 1,1dichloro-2,2 bis (chlorophenyl) ethylene, and heavy metals). These various toxic compounds also enter the body along with the entry of the microplastics.

Disruption of the cell barrier due to reactive oxygen species (ROS) will initiate cell damage and death. Damage due to ROS also occurs in brain neurons. If neuronal damage occurs continuously, it causes the remaining cells to be unable to maintain the normal function of the brain organs. This condition is known as the pathophysiology of Alzheimer's Disease. Cognitive function impairment occurs due to the number of damaged brain neurons. Low density polyethylene microplastics have been known to trigger neuronal damage, but whether these pollutants cause Alzheimer's disease remains to be investigated. In theory, the toxicity of low density polyethylene microplastics requires the accumulation of a certain dose and time to cause an effect other than the influence of particle size.

Amyloid beta 1-42 (Aβ1-42) is used as a marker to assess cognitive abilities in Alzheimer's disease. Aβ1-42 protein is derived from the fragmentation of amyloid precursor protein (APP). APP plays a role in the growth, survival, and repair of damaged brain neurons. Increased accumulation of Aβ1-42 protein in blood serum indicates that there has been damage to neurons in the brain. Furthermore, this protein will undergo fibrillation to become amyloid plaque in brain neurons. This causes a decrease in the accumulation of Aβ1-42 in the blood serum. The large number of amyloid plaques or the low accumulation of Aβ1-42 in the blood serum indicates that there has been a decrease in cognitive abilities. This condition indicates Alzheimer's disease has occurred.

High levels of ROS in the body will increase the amount of amyloid plaque deposits in the brain which significantly reduces cognitive abilities due to the death of brain neurons. Amyloid plaques will bind to the ends of dendrites so that impulses cannot be received by dendrites, synapses no longer function for signal transmission. In addition, the accumulation of Aβ1-42 in blood serum also reduces synapse plasticity. These various conditions initiate the death of brain neurons.

Our study was a purely experimental study with post-test group design by using 42 Wistar rats which were divided equally into six groups. The control group was no exposed to low density polyethylene microplastics, study group 1 (X1) was given 0.0375 mg/days, 2 (X2) was given 0.075 mg/day, 3 (X3) was given 0.15 mg/day, 4 (X4) was given 0.3 mg/day, and 5 (X5) was given 0.6 mg/day. low density polyethylene microplastics dry powder <20 μm in size mixed with 2 cc of distilled water and given through a probe for 90 days. The result showed that low density polyethylene microplastic particles have been found in the blood of Wistar rats, which were in higher number along with the group exposed to high doses of low density polyethylene microplastics. There was an increase in Aβ1-42 levels in blood serum in the study group exposed to small doses, whereas decreased in the study group exposed to larger doses. Statistical analysis between two variables using by Spearmen's rho test showed p value >0.05. It is concluded that there is no evidence that microplastic particles in the blood lead to increase Aβ1-42 levels in blood serum to assess cognitive abilities in Alzheimer's disease. Then low density polyethylene microplastic particles in the blood do not induce Alzheimer's disease in Wistar rats.