
Iodide Protects Against Reperfusion Injury



Researchers at Fred Hutchinson Cancer Research Center (Seattle, WA, USA) have found that iodide, a safe and common chemical, can reduce reperfusion injury by up to 75 percent in mice and may hold potential for humans. Reperfusion refers to the restoration of blood flow to the heart muscle that has had its blood supply cut off, following a heart attack.

Reperfusion injury is a life-threatening flood of inflammation and cellular destruction that has baffled scientists for 40 years. Now, the potentially groundbreaking study by Fred Hutch researchers suggests that the worst effects of reperfusion injury may be prevented with a safe, simple solution: a dose of iodide, a chemical form of the element iodine that is added to ordinary table salt.

"If this turns out to be positive, it could transform medicine and the leading cause of death in the Western world," according to Dr. Mark B. Roth (pictured), the Fred Hutch cell biologist whose lab came up with the new technique. The study has been published in the journal *PLOS ONE*.

The extent of harm from reperfusion injury in humans is difficult to estimate, said Dr. Graham Nichol, a professor of medicine at the University of Washington. However, in animal models, it may contribute to up to a third of the size of the infarct, or heart damage.

In the Fred Hutch study, mice with induced heart attacks were given IV infusions of sodium iodide five minutes before reperfusion, reducing myocardial infarction (MI) damage by as much as 75 percent. Also, when mice were given sodium iodide in their drinking water for two days before the procedure, they showed similar significant protection against the damage.

"A 75 percent reduction in infarct size would be significant and important if it translates into humans," Dr. Nichol said.

The new technique is still a long way from being used in the 720,000 Americans who suffer heart attacks yearly, or in helping the 600,000 in the U.S. who die of heart disease – the top cause of death – each year. However, if the protective effect of the iodide infusions and oral iodide shown in mice is borne out in other animal models – and, eventually, in people – it could eventually change how physicians treat heart attacks and perform cardiac bypass procedures. "If this works in other animals as effectively as it does in our own hands, I would guarantee you that it would be a big deal," Dr. Roth pointed out.

No one in Dr. Roth's team could say how soon the iodide could – or would – proceed to trials in humans. The researchers cautioned that it is still too early for people with heart problems to start ingesting iodide before surgery.

Dr. Roth said the Seattle company he started, Faraday Pharmaceuticals, is moving the work forward. With further research, the promise of dramatically reducing perfusion injury in people could become a reality, the doctor added.

Dr. Roth's findings have caught the interest of other experts. "It's truly incredible, in my opinion," said Dr. Rakesh Kukreja, a member of the operations committee of CAESAR, the National Institutes of Health's Cardioprotection Consortium.

The study by Dr. Roth and his colleagues, Dr. Akiko Iwata and Dr. Mike Morrison, provided a thoroughly researched and documented potential solution to the longstanding paradox of reperfusion injury, said Dr. Kukreja, who is also director of the molecular cardiology programme at Virginia Commonwealth University.

"Even today, after 40 years of research and spending billions of dollars of NIH money, there's no accepted method of reducing infarct size," he said, referring to the area of damage caused by a heart attack. "These mouse studies are very, very encouraging."

In addition to confirming the effect of iodide on reperfusion in other experiments, scientists must nail down exactly how the chemical element works. In the paper, the authors suggested that iodide might impact thyroid hormone function: "... The benefit of iodide may be conferred by depressing thyroid hormone production and as a result decreasing cardiac metabolism that is linked to reperfusion injury."

The iodide did not have the same protective effect in mice with depleted thyroid function, Dr. Roth said, adding that is only a theory and must be proven in future studies.

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Image Credit: Paul C. Miller

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