# Study Shows How Secondhand Smoke Injures Babies' Lungs

August 15 2006

UC Davis researchers today described in unprecedented biochemical and anatomical detail how cigarette smoke damages the lungs of unborn and newborn children.

The findings illustrate with increased urgency the dangers that smokers' families and friends face, said UC Davis Professor Kent Pinkerton, and should give family doctors helpful new insight into the precise hidden physical changes occurring in their young patients' lungs.

"Smoke exposure causes significant damage and lasting consequences in newborns," Pinkerton said. "This research has a message for every parent: Do not smoke or breathe secondhand smoke while you are pregnant. Do not let your children breathe secondhand smoke after they are born."

Pinkerton added that the results from this study are further proof that secondhand smoke's effects on children are not minor, temporary or reversible. "This is the missed message about secondhand smoke and children," he said. "Parents need to understand that these effects will not go away. If children do not grow healthy lungs when they are supposed to, they will likely never recover. The process is not forgiving and the children are not going to be able to make up this loss later in life."

The 2006 Surgeon General's Report on secondhand smoke estimates that more than 126 million residents of the United States age 3 or older are exposed to secondhand smoke. Among children younger than 18 years of

age, an estimated 22 percent are exposed to secondhand smoke in their home; estimates range from 11.7 percent in Utah to 34.2 percent in Kentucky.

To get the word out to parents about the dangers of secondhand smoke, two states (Arkansas and Louisiana) have made it illegal to smoke in a car with young passengers. In California, a similar bill, AB 379, is currently under consideration in the state Legislature.

The new UC Davis research is reported in today's issue of the American Journal of Respiratory and Critical Care Medicine. The lead author is Cai-Yun Zhong, a former UC Davis graduate student now working at ArQule Biomedical Institute in Boston; the co-authors are Ya Mei Zhou, also a former UC Davis graduate student and now investigating breast cancer signaling pathways at Buck Research Institute in Novato, Calif.; Jesse Joad, a UC Davis pediatrician who studies children's lung development and cares for sick children in the UC Davis Health System; and Pinkerton, a UC Davis professor of pediatric medicine and director of the UC Davis Center for Health and the Environment.

The Pinkerton research group is one of the few groups in the nation capable of studying the effects of environmental contaminants on unborn and newborn animals. Their 15 years of studies on mice and rats have yielded greater understanding of how air pollution affects human lungs and health through experiments that attempt to reproduce true exposure conditions to environmental air pollutants.

The new study was done with rhesus macaque monkeys, in order to obtain the best possible understanding of what happens in people. Pregnant macaques were exposed to smoke levels equal to those that a pregnant woman would breathe if someone in her home or workplace smoked. Newborn macaques were exposed to secondhand smoke levels similar to those a human baby would breathe if it was cared for by a

moderate-to-heavy smoker.

What the researchers found is that environmental tobacco smoke wreaks havoc in babies at a critical time in the development of lungs -- when millions of tiny cells called alveoli (pronounced al-VEE-o-lye) are being formed.

Alveoli are the place where oxygen passes from the lungs into the bloodstream. Human infants are born with only about one-fifth of the 300 million alveoli they will need as adults. They construct almost all those 300 million alveoli between birth and age 8.

Pinkerton's group had previously shown that rats exposed to secondhand smoke while in the womb and after birth developed hyper-reactive, or "ticklish," airways, which typically occurs in children and adults with asthma. The airways in those rodents remained hyper-reactive even when the secondhand smoke exposure stopped. Thus, this early exposure to environmental tobacco smoke created a long-lasting and perhaps permanent asthma-like condition.

In the new study, the researchers analyzed step-by-step how the alveolar cells' inner workings reacted to cigarette smoke. They found the normal orderly process of cell housecleaning had gone haywire.

In healthy people, cells live and die on a schedule. Programmed cell death, called apoptosis (a-pop-TOE-sis), is regulated by genes that increase or decrease various chemical reactions in the cell.

But in this study, when baby monkeys were exposed to cigarette smoke before and after birth, apoptosis went awry. Critical cellular controls regulating cell death turned off. Alveolar cells died twice as fast as they should have.

"If you are killing cells at a higher rate during a critical developmental stage, when they are supposed to be proliferating in order to create new alveoli, the lungs may never be able to recover," Pinkerton said.

Source: UC Davis