EXHIBIT 2
DATE 2-18-05
HB 661

Testimony Montana Legislature
Second Hand Smoke

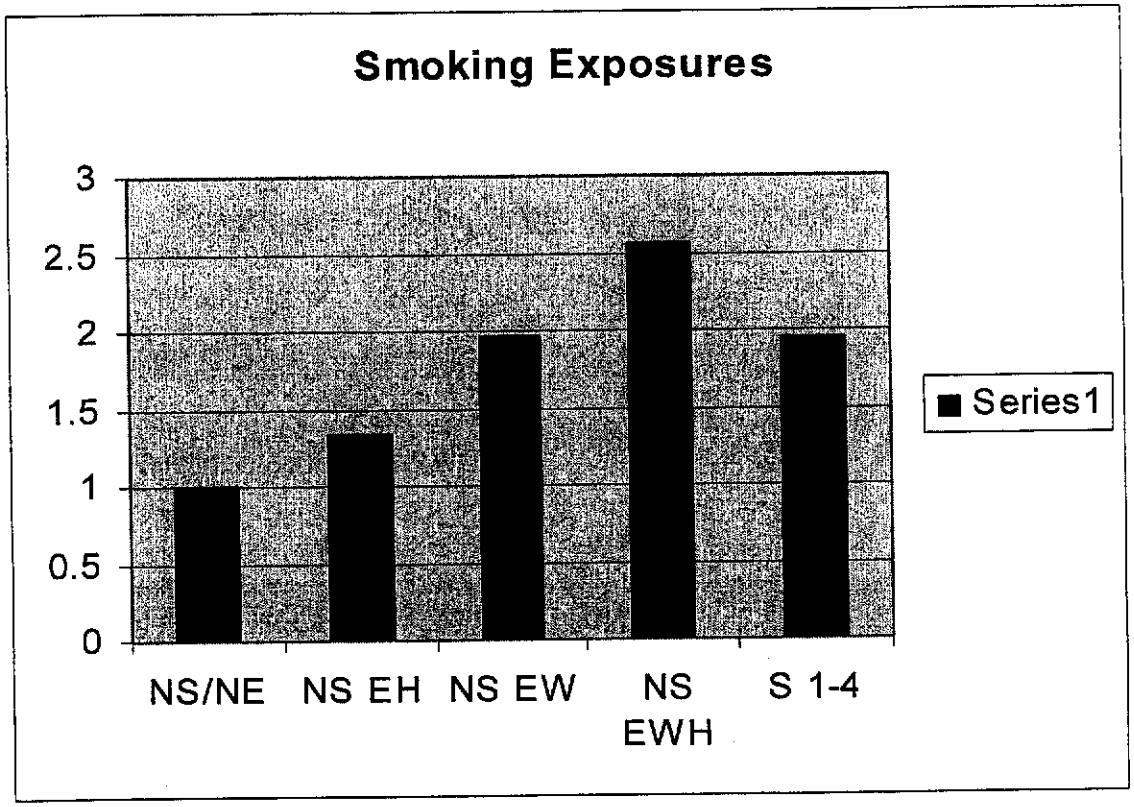
Let me thank all of you for your time today. I appreciate the opportunity to discuss this major public health issue – Second Hand Smoke. The study conducted by my partner and I and published in the British Medical Journal has generated a lot of interest and, most fun of all, a lot of controversy.

I hope, in the few minutes I have today, to show you not only why the results we found are plausible, but actually why they should have been expected. Indeed, it would have been more surprising if we had found nothing.

Starting in early 1990, many studies were done that showed individuals exposed to second hand smoke had a higher risk of heart attacks than people not exposed. This is the same type of data as the recent studies on Vioxx. People who took Vioxx had a higher risk of heart attacks than did people who did not. People exposed to second hand smoke have a higher risk of heart attacks than people not exposed. What does this mean?

Take a sample group of 1000 people and assume that none of them are exposed to second hand smoke. This group will have a certain number of heart attacks, say 100. Now take a second group of people, identical to the first except for the one issue we are measuring (either Vioxx or second hand smoke) and count the number of heart attacks in the second group. Let's say the second group has 130 heart attacks. We can now say the second group has an increased risk of 30%. We also express this as a relative risk of 1.3. There are over 50 studies in the medical literature that document an increased risk of heart attack with exposure to second hand smoke. Let's look at one. (Pitsavos C, et al Tob Control 2002 Sep; 11(3):220-5)

This study divided people into 4 groups. All groups were 100% non-smokers. The first group has no second hand smoke exposure anywhere. The second group is exposed only at home, the third only at work and the fourth at both home and work. The risk for each group is shown in the attached graph. (NS/NE = Non-smoker Not-exposed, NS EH = Non-smoker Exposed at Home, NS EW = Non-smoker exposed at work, NS EW = Non-smoke exposed at work and at home. For reference, a study of light smokers (1-4 cigarettes per day is also included.)



Note that if you exposed your spouse to your cigarette smoking, your spouse has an increased risk of heart attacks of 30%. Note also, that people exposed at work have almost twice the risk of a heart attack as people who are not exposed at work.

This shows the consistent finding from all studies that exposure to second hand smoke is associated with increased risk. Note also, that the greater the exposure, the higher the risk. This is a dose response curve. Whenever we find a "dose response curve", the probably that the factor looked at is causative is increased.

Now we can ask the rhetorical question: "If second hand smoke is the causative factor, would removing the second hand smoke reduce the risk?" The importance of our study is that it was the first time this question had been asked.

Whenever scientists find an association like this, the next question asked is "What are the mechanisms? How does cigarette smoke, whether first hand or second hand, cause heart disease?"

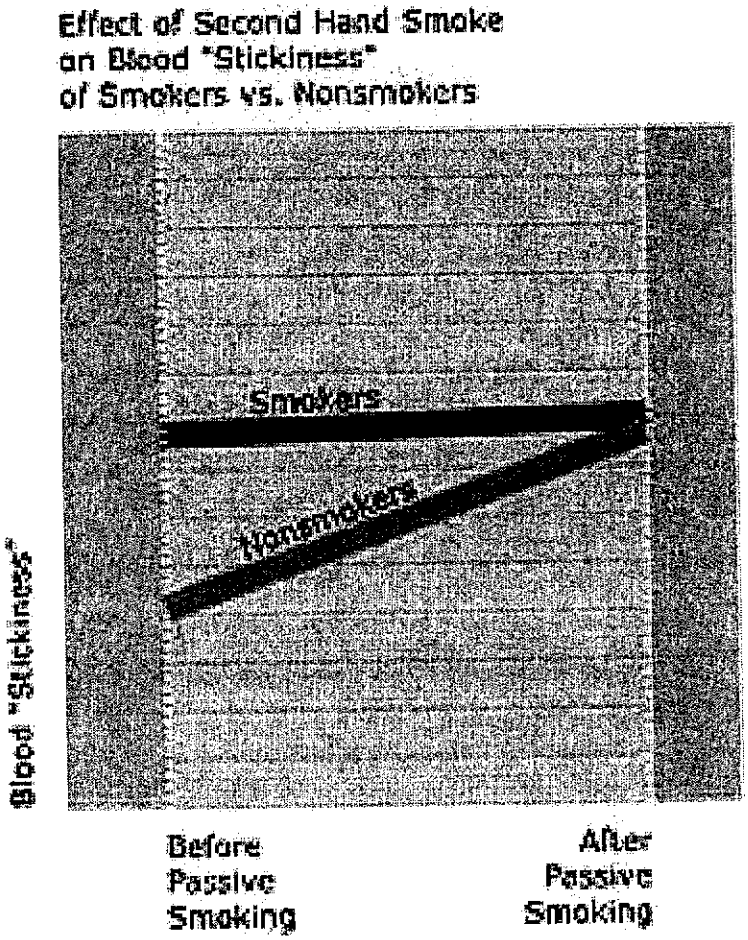
Let me give you a brief high level overview of the answer. All of the answers are laboratory proven. These experiments have actually been done on people and animals. This is not "statistics".

First, let me review the mechanisms of heart attacks. There are three different processes that can occur, either alone or together, to cause a heart attack. First, you can have

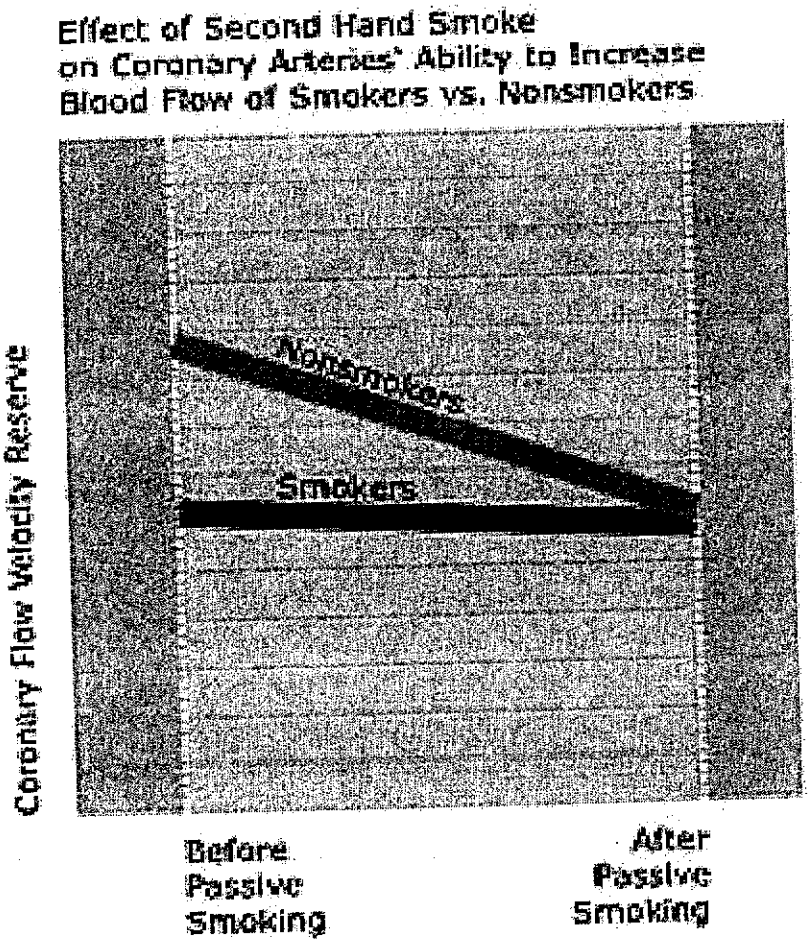
cholesterol build-up in the arteries, like scale in a pipe, until the artery is closed. You can have a clot form in the artery, just like clots in a vein in your leg, and suddenly plug up the artery like a cork in a bottle. Lastly, the artery can spasm and close so tightly as to prevent any blood flow.

Laboratory studies in animals show that rabbits exposed to second hand smoke for six months will have twice as much cholesterol build-up in their arteries as rabbits not exposed. And ultrasound studies in humans have shown that chronic exposure to second hand smoke results in thicker arterial walls. Thus second hand smoke contributes to faster cholesterol build up in our arteries.

Now about platelets: Platelets are small fragments of cells floating around in our blood stream. Their function is to trigger clotting. One mechanism of heart attacks is clot formation. That is why taking aspirin daily reduces heart attacks; it slows down the clotting mechanism, by weakening our platelets so that they do not clump together as quickly. We can measure this effect in the laboratory. Now what happens to platelets when they are exposed to second hand smoke? They become activated. That is, they are primed to clot. Smokers have platelets that are maximally activated. Like a hair trigger on a gun, they are ready to fire. When volunteers are exposed to second hand smoke, their platelets become just as activated as a smoker's platelets. The striking finding is that this takes only 20 minutes. Again, this is an experimentally confirmed finding and is illustrated in the attached graph. If you enter a smoky atmosphere, and stay for 20 minutes, your platelets are ready to clot. It is no wonder you are primed for a heart attack. This effect persists for hours after exposure.

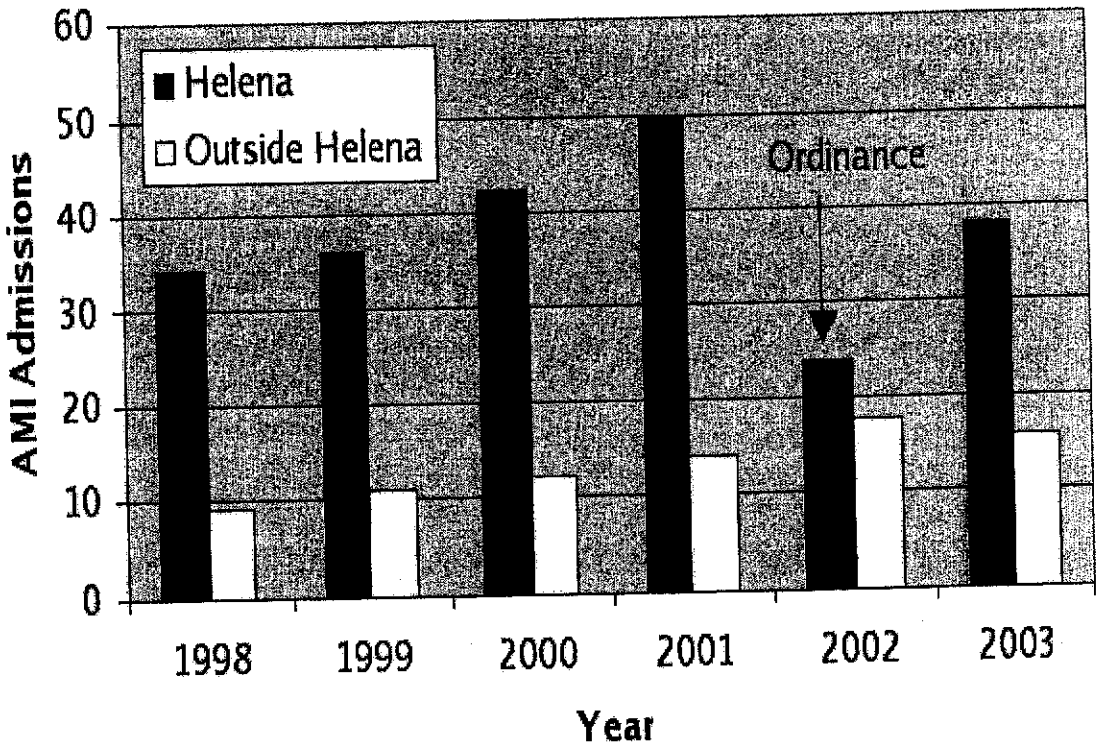


The third mechanism is arterial spasm. The walls of our arteries have muscles that allow the artery to expand to increase blood flow, or contract to shift blood flow to some other area of the body. If those muscles spasm, (much like a Charlie-horse), they can squeeze the artery so tightly that blood flow stops. In one study, they measured the blood flow through the coronary arteries of volunteers, some smokers and some non-smokers. The smokes had 30% less blood flow through their coronary arteries than non-smokers. The researchers then exposed both the smokers and non-smokers to second hand smoke. There wasn't much change in the smoker's arteries, they were already constricted. The striking change was in the non-smokers. Their arteries reduced their blood flow by 30%, to the same lower level as smokers. This took only 30 minutes exposure and persisted for hours after exposure.



What we have now is the "perfect storm", increasing cholesterol build up, activated platelets ready to clot, and narrowed coronary arteries reducing blood flow. And the last two effects occurring very quickly and lasting for hours. Think now about a meal in a smoky restaurant, you are there for a couple of hours, and then you go home and wake up 4 hours later with a heart attack. It is no wonder we never connected these events.

Now to our study: What we did was very simple. We just counted the number of heart attacks to our hospital. We are a small town, isolated and the closest general hospital to ours is 60 miles away, and the closest hospital with cardiac facilities better than ours is 90 miles away. This enabled us to capture all of the heart attacks in our community. What we found is that the number dropped, and dropped significantly, during the six months the ordinance was in effect. It rebounded after the ordinance was suspended. Like a light switch on and off. Was this possibly just chance? Yes, but when we analyzed the probability that this occurred by chance, it was less than 5 in 100 or 1 in 20. Whenever an association occurs with this low probability, it is considered statistically significant.



At the same time, we counted the number of heart attacks outside of Helena. This number did not change. So the association was both temporal (i.e. started with and stopped with the ordinance) and geographical (i.e., it occurred only in the region of our community affected by the ordinance).

Lastly, I would like to point out that the raw data and our analysis was extensively reviewed by the editorial board of the British Medical Journal, one of the most widely respected journals in the world. It was also reviewed by the CDC.

The CDC published an editorial in the same issue of the journal as our paper. They said, and we agree, it is a first study. It had small numbers. It needs to be repeated. We agree on all accounts. They also reviewed a lot of additional information and concluded that the result is biologically plausible and consistent with everything medical science knows about tobacco smoke and heart disease. This lead to the following comments:

- "Even without future studies or replications of these findings the data are sufficient to warrant caution regarding exposure to secondhand smoke. Clinicians should be aware that such exposure can pose acute risks, and all patients at increased risk of coronary heart disease or with known coronary artery disease should be advised to avoid all indoor environments that permit smoking."

- "Additionally, the families of such patients should be counseled not to smoke within the patient's home or in a vehicle with the patient."

I would like to mention that studies of lung cancer and second hand smoke show very similar information. The statistics show that lung cancer is increased in non-smokers exposed to second hand smoke. If you live with a smoker, your risk of lung cancer is increased between 25% and 30%. If you work in an office with a smoker, your risk is about doubled. If you work in a bar, your risk of lung cancer is 3 times higher than workers who are not exposed. The EPA has reported that air quality in bars is six times worse than the air in office buildings. Indeed, a rational, scientific policy would exempt office buildings and restrict smoking in bars.

Just like heart disease, we have made a lot of progress understanding how second hand smoke causes heart disease, we now understand a lot more about how second hand smoke causes lung cancer. We know now carcinogens in tobacco smoke attach to DNA molecules, how the DNA mutates (changes). Using current DNA techniques, we can show that these changes are different when the cancer is caused by cigarette smoke than cancer is caused by other factors. Soon, we will be able to trace the chemicals in cigarette smoke to specific genes and specific mutations in cancer. We will have the smoking gun, the molecular fingerprint proving second hand smoke causes cancer.

Before I finish, I would like to emphasize that a number of different diseases are related to second hand smoke. All of the following diseases have been significantly related to second hand smoke:

- **Respiratory**
  - **Acute LRI children**
  - **Asthma Induction/Exacerbation**
  - **Eye/Nasal Irritation Adults**

  - **Middle Ear infections Children**
- **Developmental Effects**
  - **Low Birth Weight Infants**
  - **SIDS**
- **Carcinogenic Effects**
  - **Nasal Sinus Cancer**
- **Developmental Effects**
  - **Spontaneous Abortion**
  - **Adverse Impact on Cognition and Behavior**
- **Carcinogenic Effects**
  - **Cervical Cancer**
- **Respiratory**
  - **Exacerbation of Cystic Fibrosis**
  - **Decrease Pulmonary Function**

Today, you have the opportunity to protect the citizens of our state from exposure to second hand smoke. You have to ask yourself, is the residual doubt in your mind about these effects worth the risk to your families, your friends, and your neighbors. If just one person dies of a heart attack that you could have prevented, is it worth the risk?

Respectfully Submitted:

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