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Session 102D
The Impact of Second-Hand Smoke

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Summary: A smoking spouse and a smoky house. Smoky bars and smoky cars. What does this environment do to your health?

Mr. Timothy F. Harris: This is a session on second-hand smoke or secondary smoke, side-stream smoke or environmental tobacco smoke, whatever you want to call it, which has been a topic of interest for quite a while now. The topic seems to be heating up. There are some current issues that we'll address during our presentations, and we've put together a well-rounded panel to address the topics. We have an M.D. with experience in the area, we have an underwriter, and we have an actuary, of course, talking about the numbers side of things. We're going to lead off with the doctor to address the medical aspects. Dr. Charlotte Lee has an M.D. from Indiana University and performed her internship and residency in Phoenix. She also has a clinical cardiology fellowship from the University of Minnesota. Dr. Lee has spent several years as medical director of a few insurance companies in the Minnesota area, and in the recent past was medical director at Lincoln National Reinsurance. She is presently medical director for Osborn Laboratories.

Dr. Charlotte A. Lee: The issue of passive smoking is steadily coming into the realm of insurance medicine and in the whole insurance industry because of people

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who are found to be smokers by laboratory results but are claiming that they are not smokers. There are also people who are claiming that they are passive smokers or they live with a smoker or they work in a smoky environment. They use many excuses to hide the fact that they use tobacco products. We are going to give you some insight into this whole issue. I think I have the easy job of addressing the clinical findings. What are the doctors seeing? What are the nurses seeing? What are the people seeing about themselves and how passive smoking affects their health?

Is discussion about this subject just a lot of hot air? Is this all a smoke screen? Are we just trying to cover up the fact that we don't know what we're doing when we're trying to underwrite these people? Or is there, indeed, a heated debate? This session is not a debate. For those of you who thought there would be some fiery word-slinging, we really didn't find a lot to debate on this issue. I'm going to present the pure medical aspect of this and then the other two speakers will address it from an underwriting point of view and an actuarial point of view.

Passive smoking has some other terms that we use now and then to describe it. One term is second-hand smoke; it is also called environmental tobacco smoke. This refers to the smoke that's present in the workplace and in the home and in social situations where we are. Involuntary smoking means that we don't mean to smoke, but we're still inhaling smoke fumes. Exhaled smoke is smoke from the smoker himself or herself. There's another term that is similar to the first term and that is side-stream smoke. Side-stream smoke is the smoke that's actually coming off the end of a burning pipe or cigarette. If a cigarette is sitting in an ashtray on a table and nobody is smoking it, it's still emitting smoke and that's called side-stream smoke. That smoke contains any product in the tobacco. It should be differentiated from what we traditionally call second-hand smoke, which is the smoke that a smoker blows in your face.

Now, there are certain groups of people who society (not only the medical society, but society as a whole) commonly agrees are at risk for second-hand smoke or passive smoking. Number one would be children with or without respiratory problems. We consider children to be a captive audience. Some children aren't really aware that smoking is going on around them in many instances, although in today's schools there is such a campaign against smoking that kids are more aware of smoking. Regardless of what their level of awareness is, they're considered a captive audience to smoking. Some people have thought that smoking around children or smoking in a household with children is one of the most severe forms of child abuse, next to physical abuse.

It's also generally accepted that adults with respiratory problems are certainly at risk by being in a smoking environment. It's interesting to note that a number of adults with respiratory problems have them because of smoking. When a person smokes to the point of having emphysema or chronic obstructive pulmonary disease, he or she is still at risk for further impairment by being in a smoking environment. So even though these people have done this to themselves, they still have put themselves at higher risk for passive smoking.

Less commonly accepted to be at risk are two other categories. The first is nonhispanic blacks. These people have a much higher concentration of what we call melanocyte. Melanocytes are a pigment-producing cell in the skin that gives us our skin color. For those of you who have been lying out at the pool, the melanocytes in your skin have been activated by the sun and have been made to produce more melanin and that causes a suntan. These melanocytes are now producing more melanin pigment. People of African origin have the highest density of melanocytes of any portion of the population.

What has been found to be interesting is that melanocytes tend to cling to nicotine and nicotine components, and as we'll see later, the main component or the main metabolite is called cotinine. Melanocytes have been found to have a very high concentration of cotinine. They don't rid themselves of the cotinine nearly as readily as some other cells in the body. Therefore, it's thought that if one were to examine, for example, an African person and a Caucasian person, with the same body type, same body weight, with the same exposure to smoke, a much higher density of cotinine would be found in the black person than in the Caucasian because of the black person's higher concentration of melanocytes.

It also has been known, too, that blacks with lung cancer tend to have a more aggressive disease than the disease in the population at large. One reason might be that once they get the cotinine in the cells they cannot rid themselves of it readily, so they have a total higher body concentration of cotinine.

Males are more at risk for adverse effects of passive smoke. The Environmental Protection Agency did a study on passive smoking. This was a very extensive study on the effects of second-hand smoke. Jess is going to discuss this study, so I won't go into the details. I'll only summarize it. These are the main risks that this study has shown to prevail in persons who have been exposed to second-hand smoke. They include, as we expect, lung cancer and upper- and lower-respiratory infections. Upper-respiratory infections include the sniffles, stuffy nose, plugged ears or sore throat. Lower-respiratory infections include bronchitis, pneumonia and something called bronchiectasis, which is a dilatation of the bronchial tree. The lower respiratory infections tend to be a bit more severe when a person has them.

They certainly tend to be perpetuated by second-hand smoke, according to the EPA study.

Also included is middle-ear disease. This includes adults and children. The disease doesn't necessarily have to be an infection. The smoking environment isn't an infectious environment. These people tend to have more middle-ear fluid and more of what we feel on an airplane at higher altitudes. It feels as though the ears are plugged. This is essentially the same type of thing that people who are exposed to high degrees of second-hand smoke are found to experience.

There is also an irritative process; there is irritation of the eyes, nose and throat, or as we call it, the mucous membranes—the lining of the mouth, the lining of the nose. People get an irritated throat just from being in a smoky environment. The irritative phenomenon are more of a nuisance as opposed to something that is really a detriment to health.

Last, heart disease has been implicated, but not adequately studied. Since I have been here at Palm Desert, one of the *USA Today* issues came out with an article stating that there is now an even greater implication of heart disease resulting from second-hand smoke. The author didn't give any of the details of the study itself or all of the numbers, but the statement was made that more and more seen that women who are exposed to a smoking environment have a higher risk of developing heart disease from second-hand smoke.

I'm going to say a few words about nicotine testing. When the laboratory does what we usually conceive of as being nicotine testing, it's really not testing for nicotine. Nicotine is the component of tobacco regardless of the form, but what we actually measure in the laboratory is the chief metabolite of the nicotine, which is called cotinine. The reason we are able to measure cotinine is that, being a metabolite, it tends to hang around the body longer—long enough to be measured. It has also been found to be excreted in the urine. We tend to use the urine tests for cotinine much more than any other test even though cotinine is present in other body fluids. It can be found in saliva and it can be found in blood, but it's just easier to test in urine. Most insurance companies, when they get laboratory testing done, get a urine sample to test for other things. When I say nicotine testing what we really test for is cotinine.

The following quote is from the *Journal of the American Medical Association.* "The mean concentration of cotinine in the urine of cigarette smokers and nonsmokers living with smokers and nonsmokers living with nonsmokers is the ratio of 200 to 3 to 1." These ratios are similar to the ratios of the excess risk of lung cancer. And

this is just to drive home this point: active smokers certainly have a higher urinary cotinine level than nonsmokers or passive smokers.

Here's another quote from *The Journal of Preventive Medicine*. "Heavy passive smoking may cause levels two to three times that in nonsmokers, with infrequent, active smokers or regular active smoking of low nicotine cigarettes causing only slightly higher values." This was from a study that showed that risks from passive smoking itself, in a very smoky environment, can cause cotinine levels that equal or even exceed those of a person who is an active smoker but smokes infrequently.

By the way, when we are talking about studies, please keep in mind that all studies don't show the same results. People may have studied the same phenomenon and came out with entirely different results. Some of the results of the studies that I show may be different from some of the results that Jess or Tim have come upon.

What causes us to have false positive values? False positive means that someone who does not smoke has been shown by laboratory testing to have a high enough level to be considered a smoker, depending on the lab cut-off values. So why do we get either false positive or false negative values? False positive means that someone is called positive who really isn't a smoker. And false negative means that they are called negative and they really are smokers. One of the things that causes false negatives is smoking low nicotine cigarettes. Sometimes there is a low enough nicotine count that, depending on how long before the test was done the person smoked, the test may not pick up that level of nicotine, actually cotinine.

One interesting thing is that it has been thought that people who smoke low-nicotine cigarettes actually may be at a higher risk for developing cancer and any other negative effects associated with smoking. If they smoke the very low-nicotine cigarettes, chances are they may be smoking three or four times as many of those cigarettes in order to get the high or nicotine rush from smoking. These cigarettes contain not only nicotine but they also contain the tars and all the other carcinogens or “poisons,” as we call them. Even though they are smoking low-nicotine cigarettes, they are probably smoking more of the them and, therefore, getting twice or three times as much of the other impurities in cigarettes. When the nicotine content goes down in a cigarette, the other components stay about the same. Therefore, if people think they are safer by smoking the low-nicotine cigarettes, this isn’t necessarily so.

State of hydration affects the result. State of hydration means how much body water a person has on board. And it's well known that many people who know they are going to the laboratory or to the paramed facility to be tested drink literally gallons of water to try to dilute the urine. And, sure enough, this does work. If they can

actually flood themselves enough by the time they give the urine sample, such that it's very diluted urine, sometimes this can alter the laboratory results.

Poor chain of custody speaks for itself. Are we really testing for the person whose name is on the lab slip? There are surrogate testers that are going in—even with someone else's drivers license saying, "This is my driver's license." And the paramedic says the picture kind of looks like the person and lets them be tested.

Are there interfering substances? This is one thing that's been very well studied, and it's found that nothing interferes with cotinine. The only thing that tests positive for cotinine is cotinine. We don't have to worry about what other things can interfere. For example, there is something called nicotinic acid or niacinamide, which is something that people who have had taxable income adjustment or strokes are given. It was thought at one time that there was cross-reaction between these and nicotine, but study after study has shown that it does not. So, there is no cross-reaction when it comes to nicotine testing.

There are other sources of nicotine besides tobacco. It's well known that people who chew Nicorette Gum and Nicoderm Patches are trying to stop smoking. The nicotine is absorbed through the saliva into the bloodstream in the case of Nicorette Gum. With the patch, the nicotine is absorbed through the skin. So anybody who wears a patch or who has recently chewed Nicorette Gum will test positive for cotinine.

The results of a study conducted by Dr. Nancy Haley, Medical Director of MetLab shows the difference in the cotinine content of urine in people who had home exposure versus work exposure. This came from a 1988 study, at about the time smoking in the work place was becoming unpopular. Companies were beginning to have smoke-free environments. What this shows is that—and as I say, this is one study of many—it's really not the workplace smoke content that increased the cotinine levels. Rather, the home exposure is really what created the increase in cotinine levels.

One study was done with what we call a smoking machine. This machine puffs on four cigarettes simultaneously in a room that is about eight feet by eight feet. People in the room had a total exposure time of 80 minutes, including the time for the machine to smoke the cigarettes. A measure of the urine cotinine content following this exposure still showed the values to be less than the minimum value that's reported by laboratories to be consistent with an active smoker. This proves that people who test positive on insurance lab testing and say, "I got it from inhaled smoke. I don't smoke; I've just been in a smoky room," are incorrect. They are hard pressed to prove that because it takes much more than being confined in this

room with these cigarettes being smoked to even register on the scale to be considered a smoker by laboratory measures.

Another study by Dr. Haley confirms what I said before. The higher values for the urinary cotinine content are the ones in which there is also a smoking environment at home in addition to the workplace. The ones that say they weren't exposed at home but at work only don't have nearly as high a values as the ones that were exposed at home. Again, this is an isolated study, though.

Now I'll say a few words about a substance called thiocyanate. One test that can be done to determine whether a person is actually smoking tobacco versus having it in his mouth (chewing tobacco, snuff) is called a thiocyanate test. Thiocyanate is a metabolite of hydrogen cyanide, and hydrogen cyanide is something that is produced from smoked tobacco. It's in exhaled smoke as opposed to tobacco that's in the mouth. So a person who chews tobacco will have a negative thiocyanate level, whereas the person who smokes tobacco will have a positive one. That's one way of differentiating between a person who chews tobacco and a person who smokes tobacco. The sensitivity and specificity of the thiocyanate test is only in the 80s, and we at the laboratories like the sensitivity and specificity be in the high 90s before we consider it to be a very, very accurate test. However, in the cases where there's really a question as to whether a person does or does not smoke versus whether he does or does not chew, then the thiocyanate level can help show which type of person this is.

Another thing that is a little controversial about the thiocyanate is that some studies show that the half life is about two weeks. Others show that the half life is two days, and that the thiocyanate level can go down before the corresponding cotinine level would have gone down. In this instance, you're going to miss some of the people who smoke because the thiocyanate level might have gone down before their cotinine level went down. So you're not going to pick up all the smokers by getting the thiocyanate level.

Another study that was done in 1989 was published in the *American Journal of Epidemiology.* Boys, aged 12–14 years old, were tested because we know that is a prime age for use of chewing tobacco. These are kids who know it's not good to smoke. They can't get cigarettes readily, but somehow they can get their hands on chewing tobacco. In this study, salivary thiocyanate levels were taken and it was found that at least 40% used only smokeless tobacco as opposed to smoking cigarettes. So even in this young age group, they're getting the nicotine high by using smokeless tobacco.

Jess is now going to discuss some of the underwriting aspects of what we do at the insurance companies when we get the laboratory value showing that a person is a smoker.

Mr. Harris: Jess Mast is a second vice president and director of risk management research for Lincoln National Reinsurance Companies. He directs research activities for underwriting, product development, and pricing. He has been with Lincoln for more than 30 years and is a contributing editor of *On The Risk* and co-editor of the Society of Actuaries (SOA) Life Insurance Specialty Guide on Underwriting Individual Products.

Mr. Jess L. Mast: I'm going to share with you an underwriter's perspective on trying to recognize passive smoking or second-hand smoke in the risk classification process. As Charlotte was saying earlier, there are an awful lot of studies out there and the information is conflicting at times; so it's very difficult to reach conclusions that you feel very comfortable in making based on that data. So I'm proceeding through this for those of you who want to seriously consider trying to utilize information about second-hand-smoke exposure in your risk classification or risk-selection process.

One of the terms that we see when we look at studies is the phrase the "weight of evidence." The weight of evidence is given as an indicator of credibility. For example, in the most recent update to the nurses' health study, which was published in *The Journal of Circulation* just a few days ago, one of the conclusions was the consistent findings, with the existing totality of evidence, that increases the belief that the observed association of regular exposure to passive smoking at home or at work increases the risk of coronary heart disease among non-smoking women, and that association represents a cause and effect relationship. That just gives you a feel for the kind of hedge you have to make in being able to make a positive statement about the influence of passive smoking on health risk of disease.

The Environmental Protection Agency (EPA) and other studies show marginal impact on mortality. Even though the impact may be marginal, it's still appreciable and we can't necessarily ignore it. The sheer volume of data involved in these studies is very impressive. It cannot be ignored, as far as I'm concerned. How much exposure to passive smoking really affects mortality significantly? Does the effect depend on how often such exposure has occurred and how often it will continue to occur? Those are things that we can't necessarily measure or evaluate as part of the risk classification process because that information may be difficult to come by or difficult to get an honest response to. What parameters are realistic to use in underwriting? The parameters that we use in underwriting should be

practical, objective, reproducible, and acknowledged to be relevant for underwriting and pricing levels for both the medical and insurance communities.

How predictive are results from a single cotinine test? That test reflects only the consumption of, and/or exposure to, tobacco products during the past 24–48 hours, perhaps a little longer. It's a relatively brief window of time. So, it's hard to extrapolate from that and say that the exposure that we measure through cotinine testing is, indeed, an ongoing exposure at the level we're able to test, on a one-time-test basis.

Should insurers try to recognize other forms of pollution? I definitely think of passive smoking as a form of pollution, especially to the environment. To the extent that it turns out to be reasonable and justifiable to recognize passive smoking in underwriting, maybe we should be recognizing other forms of pollution as well.

Here are some challenges to think about along the path of trying to recognize passive smoking in the risk classification and underwriting process. What kind of questions should and can we be asking on the application form to recognize exposure to environmental tobacco smoke? The first question is, does your spouse smoke tobacco? Do you live with your spouse? However, if you don't live with your spouse, it doesn't make much difference. Do you live with anyone else who smokes tobacco? Is smoking allowed in your workplace?

What questions can we ask on the application forms? Maybe we should ask something along the lines of the number of hours that someone is exposed on a daily basis in an environment of tobacco smoke, and the number of years that a person has been exposed to smoking either at home or at work. You can ask about whether someone frequents bars, taverns, or bowling alleys, for that matter. I spent a lot of years bowling and I know how bad that is. Do you live above a bar or a tavern? Not many people live above a bowling alley. It would be too noisy.

Anyway, there are challenges with testing. Are the labs geared, for example, to detect cotinine levels that represent exposure to passive smoking? Some may and others may not. You have to ask the lab where you do business about that particular capability. The cost of such testing may or may not increase total underwriting expenses. That's something I wasn't able to get a handle on in talking to three or four different labs. Most of them hadn't yet even been asked the question whether they are even capable of recognizing cotinine levels at the passive smoking or environmental tobacco smoke level.

If you do recognize passive smoking in your premium rates, then it might result in increased policy-not-taken rates. If you do recognize passive smoking in risk

appraisal, then many of the applicants really won't know whether they're going to test positive for exposure to passive smoke. As a consequence, those who test positive may decide not to purchase insurance if the price of the coverage is greater than it would have been if the company had not been trying to recognize passive smoke exposure.

You might consider doing some cost benefit analysis to set testing limits. These tests will, indeed, be kind of challenging to evaluate from a cost benefit standpoint. Grant Hemphill is an actuary with Indianapolis Life and he mentioned a scenario for evaluating the costs and benefits of doing different tests involving game theory, which I thought was very interesting. He had just seen an article on the Internet and was trying to evaluate whether there may be some opportunity along those lines to evaluate the interactions between agents and underwriters, agents and applicants, agents and other companies and so on, in a way that you might be able to optimize an expectation that really fits the way in which you are interfacing with your market and your agents. Such analysis will recognize those interactions and from that one can estimate the value of such testing.

Another problem with trying to recognize passive smoking is that exposure to it may be unavoidable. It makes it nearly impossible, as I mentioned earlier, for the applicant to be able to predict whether the cotinine test at the time of the application is necessarily going to be negative or positive. Perhaps 50% or so of those who take the test, who call themselves nonsmokers, will test positive for environmental tobacco smoke exposure.

Let's look at some of the basic tenets of underwriting. I'll call them the fundamental challenges of underwriting. The most important one is the job of minimizing the risk of antiselection. That's probably one of the main responsibilities of the home-office underwriter. Another one is to preserve equity in the risk classification process.

Another challenge that's becoming more and more interesting or challenging is the ability to justify to regulators the need for underwriting differentials when looking at different impairments and medical histories. To justify differentials to regulators, as well as to ourselves, we need to distinguish among risks in such a way that we can reasonably anticipate that the differences we're trying to recognize do produce differences in mortality outcome. Another challenge to underwriting is to manage expenses and the use of underwriting tools in prudent ways.

It's becoming more important and challenging to explain underwriting actions to the agents and applicants.

The agent may claim that the night before the test the applicant attended a bingo party but never usually does that sort of thing. So is it really fair to penalize this person for a one-time exposure to environmental tobacco smoke? In attempting to recognize passive smoking, it will also be challenging to manage expenses and productivity in the face of increases in requests for retesting. A lot of these people are going to request to be retested and many of them will turn out to test negative for cotinine or tobacco exposure the second time around. There may be requests for rating reconsideration, too.

You can expect to see increases in policy not-taken rates for issued policies and increased cost of good business lost to other companies. There could be increases in the proportion of applicants who are truly smoke-free. When you have a risk classification that appeals to a certain segment of the population, such as those who are able to insulate themselves, you might say, from exposure to tobacco smoke, these people are going to seek out companies that offer a smoke-free policy or premium rate. Both the underwriter and actuary together must evaluate the trade-offs of increased expense of doing business versus making further refinements in the risk classification process to recognize passive smoking exposure.

Attempts to recognize passive smoking and risk classification may be tempered by a recent government survey that found that 88% of nontobacco users, which were included in the third, national health and nutrition exam survey, had detectable levels of cotinine in their serum. That's a pretty high proportion of the population that may not really qualify for smoke-free rates. And that survey included almost 17,000 individuals in a representative national sample of nontobacco users. So, even if 25–50% of the applicants can call themselves nontobacco users, maybe 22–45% of those people will still test positive for environmental tobacco smoke exposure. This sounds awfully high to me, but I think that the efforts of the EPA and nonsmoking advocates are probably going to eat away at the risk of facing environmental tobacco smoke, either in the workplace or in public places. So the risk of exposure to environmental tobacco smoke should be diminishing over the next few years.

Let's consider the practicality of underwriting for passive smoking as we would any other risk factor introduced in the risk selection and classification processes. Is such underwriting manageable? It probably is, but it will be a challenge. The risks and rewards for pursuing it must be evaluated by each company. Will it be cost effective? I think it certainly can be. That's a question each company has to evaluate in deciding whether to pursue such a risk-classification approach. Each company must evaluate the fairness of adopting the use of passive smoking as an underwriting criterion based on the company's philosophy of doing business and their financial objectives.

Mr. Harris: How many people here are active smokers right now? Can I see a show of hands? Anybody here that admits to smoking? Nobody here smokes. How many people here live with a smoker? It looks like about five or so. How many people here work in an environment where there is smoke? One. That's quite low. How many people here are exposed to smoke socially? Bars, bowling alleys, things of that nature? That's probably the largest group.

There are a couple of main points in my presentation. One of which is that cotinine is not the real indicator of the risk involved here. Cotinine is a marker. Cotinine means that there has been some exposure to secondary smoke and I can argue with the doctor about this later, but that's kind of the interpretation that I've come up with. The real negative health impacts are coming from some of the other chemicals in tobacco and in side-stream smoke.

There's actually quite a bit of data available on second-hand smoke. Much of it is available on the Internet for those of you who want to look for it. One quote on second-hand smoke says, "This toll makes passive smoking the third leading preventable cause of death in the U.S. behind active smoking and alcohol." So this is a fairly major underwriting issue. Underwriters and labs have focused on the levels of cotinine and said that they're so low for this group that we really can't underwrite it, and maybe the risk is not that high. Well, I suspect that's not the case. I think it can be underwritten, and I believe the risk is higher than what people have assumed in the past.

The testing for cotinine right now, as Dr. Lee indicated, is a test in terms of nanograms per milliliter. And the insurance lab reports right now seem to cut off at about 300 nanograms per milliliter. You're picking up smokers, but you're not picking up the people exposed to secondary smoke. However, it's my understanding that there's no reason that level of sensitivity cannot be dropped. Osborn Lab is able to do it, but you may want to check with your lab and see if they can do it as well.

Let's go over the recent chronology of events here. Probably the most useful study, from an actuarial standpoint, is a 1986 study that was done by the *National Academy of Sciences.* This is a fairly lengthy study that can be found on the Internet. It gets into quite a bit of detail. It gets into an area that I call the "force of mortality." There are a few terms here that need to be defined. Dr. Lee has already defined ETS. Another one is "relative risk," which is the ratio of the risk that people are exposed to in comparison to a standard or a base group. In this case, we're looking at a base group of the people that are totally smoke free. It is not the people that say that they're smoke free, but the people that are smoke free. There's a difference there and we'll talk about that later. The groups that we're comparing to

that smoke-free group will be the people exposed to smoke in the home, the workplace, and in social settings or a combination of those areas. We'll also look at some of the data on active smokers as well.

The 1993 EPA study has been referred to a few times. This is the one that the tobacco industry does not like. There is litigation from the tobacco industry on this topic. Actually I looked at the news wires and it looks like the tobacco industry is close to settling on the liability issue for active smokers. I don't know how many of you are aware of this, but they're talking about something in the neighborhood of $300 billion over 25 years that's going to be paid out to active smokers. They're also going to put limits on the ability of the secondary-smoke population to sue. This is something that's being negotiated in smoke-filled rooms, I suspect, in D.C.

In 1993, I wrote a smoke-free term insurance article for the *Product Development News* of the SOA, which I'll talk about if I have time. In 1996, some of us were interviewed by a reporter from *Money* magazine on this topic and the article ended up not being published at that time. It addressed the risks and the underwriting of secondary smoke.

The National Academy of Science Report in 1986 report, indicated that environmental tobacco smoke can cause lung cancer in healthy adult nonsmokers. This study was focused more on the cancer aspects of secondary-smoke exposure. It looked at smokers in different environments; the workplace, the home and the social environment. The 1996 EPA report indicated that environmental tobacco smoke is a human lung carcinogen, and it was classified at that time as a Group A Carcinogen. They also looked at some of the health problems of children who are exposed to secondary smoke. The health problems of children is one of the areas that ends up examined in a number of these studies. Other studies are focused more on the cost aspects, whether it's health care cost or the mortality cost aspects of the exposure to secondary smoke.

A study that Milliman & Robertson did recently looked at the additional health care costs of smokers. It didn't consider secondary smoke. This study estimated there is about a 31% increase in health claim costs for smokers. When you look at some of the data that I'll present later on the relative risk, you should be able to pick up some of the relative-risk adjustments and apply those to the loadings for smokers and come up with the intermediate cost of health care for people exposed to secondary smoke. It's somewhere in between the amount for the smoke-free group and the smokers, possibly in the same ratio as what we're going to see for mortality.

Following some of this data, especially the EPA report, there was what I call a war of words. The Congressional Research Service issued a report (which I've read and

didn't hold a lot of confidence in), which questioned the EPA's claims. This almost seemed to be politically motivated. Maybe someone has some comments on that. The individuals who authored the report didn't have any credentials listed and they just went through and kind of picked apart different aspects of the EPA's report, which, as I indicated, is being litigated by the tobacco industry. Then there's a Phillip Morris document, which I was unable to find. It seems to have been pulled from some of the sources that also attacked the EPA report. I think as they're getting close to settling on the tobacco litigation they may have pulled back their horns a bit on some of these issues.

Acceptance! Tobacco companies are talking settlement on the active smoking issue. The implications for those exposed to environmental tobacco smoke are not good. One of the restrictions is that if you live with a smoker, you cannot sue. And if you were a previous smoker, even though you may have some ailments that are due to secondary smoke, you're going to have difficulty suing. There may be a ten-year limit; however, after ten years of not smoking, you may have the right to sue.

The 1986 report looked at, as I said, relative exposure by location, and at the carcinogens in environmental tobacco smoke. Just as many of the other reports looked at relative risk rate by location. The thing I really liked was that they came up with a formula for the force of mortality due to secondary smoke. This is something that, as an actuary, you relate to. Some of the carcinogens in environmental tobacco smoke appear here as multiples of mainstream smoke. This is what's coming off the hot end of the cigarette as compared to what's going into the lungs of the person that's smoking. I'd like to focus on one that I can't pronounce. The ratio of this carcinogen in side-stream smoke compared to mainstream smoke ranges from 20 to 100 times. You could have 100 times this carcinogen being thrown out into the air compared to what's being sucked in by the person who is smoking. This finding, especially in relationship to cancer, is where some of the higher risk is coming from. The ratio of nicotine here is three to one. So you have got a three-to-one ratio of nicotine in secondary smoke as compared to the mainstream smoke, and you have possibly 100 times the carcinogen nitrosimine.

This is where you can get some of the increases that we'll see later in the relative risk compared to cotinine levels. This gets back to my earlier statement; you can't look at cotinine as an indicator of the level of risk. It's just a marker that someone was exposed to secondary smoke.

Here are some of the numbers that I got from another study. Some of these may differ a bit from what Dr. Lee was quoting. Nicotine exposures are measured in

grams per cubic meter. Here it's indicating that the office environment can actually be slightly higher than residential. That theme carries through some of the research I've looked at, which differs from some of the Met research. Social environment, as it shows here, can be the highest, and as we'll see in some of the relative risk rates, also can be the highest there.

Let's discuss relative risk rates for cancer that come from the 1986 *National Academy of Science Study*. The base group, again, would be the people who are totally smoke free. The household risk or residential risk is 1.3, which translates to a 30% elevation in the risk of cancer. In the workplace it is 40%, and social exposure creates an elevation of 50%. Combined is 80% (someone with two or more exposures). This person is at the same level, according to this study, of an active smoker. The relative cancer risk for an active smoker is an 80% increase. On the average, people exposed to secondary smoke were exposed to half the additional cancer risk of active smokers.

Let's look at some cotinine levels here to see how these compare. This is comparable to data that we've already seen. You can see how low some of the cotinine levels go. In the nonsmoker, we'll look at the urine because this is normally where this is tested. We'll see that for people who claim to be nonsmokers, we have 1.6 mg for the totally smoke-free group. For the environmental tobacco smoke group, we have 7.7 mg. For the smoking group we show 1,000 mg. So if the insurance companies are cutting off their testing at 300 nanograms per milliliter, you're not going to find the people who are exposed to secondary smoke, because they are well below that level.

The part I like about the 1986 report is the force of mortality. There was some question about whether or not it was a linear force of mortality or a quadratic force of mortality. The report seemed to indicate that for lower levels of exposure, it's a linear force of mortality. This is something that, as actuaries, we should be able to relate to. The way I've expressed it is a little different from the way it showed up in the report, but I've said that the mortality rate is equivalent to the relative risk rate times the mortality from cancer plus the remaining mortality; this would be mortality from other causes. This formula considers only the cancer risk, and then also you need to throw into this the heart disease risk, which I'll get to in a bit. We then have additional risk from cancer and then the additional risk from heart disease, both of which are increased, according to these studies, by secondary smoke.

One of the adjustments that does need to be made, and this is going to be an issue if you start underwriting for this topic, will be those people who claim to be unexposed to tobacco smoke and who actually are exposed to tobacco smoke.

Maybe they just don't know it or didn't want to admit it. These studies estimated that it was about 20% of the group that was being studied. After they adjusted for the truly exposed group, they found that environmental tobacco smoke increased the risk by 54%. So, we've got a relative cancer risk rate here of 1.54 as compared to the truly unexposed.

Let's get back to the issue that I referred to earlier, that cotinine can be used to estimate the exposure, but it is not directly proportional to the risk that these individuals are exposed to. The cancer risk is coming from some of the carcinogens that we saw earlier. I don't think nicotine is a carcinogen, is it?

Here's a point that I think really brings this home. I pulled together some of the previous data and we look at cotinine levels in urine compared to the relative risk rate for different groups. The environmental tobacco smoke group is going to show an average cotinine level of 7.7 nanograms per milliliter. They're going to have a relative risk rate of 1.54. Smokers show cotinine levels of 1,390 in the urine, compared to a relative risk rate of 1.8 for cancer. The cotinine levels are not proportional to the additional cancer risk that's coming from these studies.

Let's consider heart disease risks. This is another area where it has been shown that environmental tobacco smoke has some implications. There are a number of compounds that are included here that damage the heart's of active smokers. These compounds are also found in ETS. Many of these studies in the past have been based on spouses of smokers. The more recent one that's referred to, which we'll get to in a bit, tracked 37,000 nurses through a period of ten years and studies workplace exposure, residential exposure, and social exposure.

Let's talk about the total estimated deaths from passive smoking per year. We just looked at the cancer risk and it is not the largest anticipated or projected cause of death from passive smoking. There are 12,000 deaths per year from other cancers, 3,700 from lung cancer, and 37,000 from heart disease. Heart disease is the primary projected cause of death from exposure to passive smoke.

You've probably heard about the article that was referred to a few times already, and it's very timely. On May 20, *USA Today* had an article about this study of 37,000 nurses who were tracked through a ten-year period of time, which estimated the level of relative risk that these nurses experienced from exposure to secondary smoke. An important point about this study is that it included a fairly large population—37,000 nurses. Some of the previous research included smaller groups of people and produced relative risk rates and other results that jump around a bit. That made the results of those studies less credible, which made that data easier to attack. This study uses a larger group of people and tracks them over a ten-year

period of time. The other thing, as I indicated earlier, is that the study covered more than just the residential exposure. The study was focused on heart disease and also considers workplace exposure and social exposure to secondary smoke, so this is fairly important research at this time.

The study shows that women regularly exposed to secondary smoke had a 91% greater risk of heart disease than those not exposed. This is also a greater relative risk rate than some of the other studies have shown. This is a fairly high increase in risk for being exposed to secondary smoke. That 91% was for regular exposure. Occasional exposure resulted in a 58% greater risk.

I want to go into some of the calculations. In this 1993 article that I wrote, we went through and looked at the change in the rates for a term insurance policy if you were to divide things up into smoke-free, nonsmoker, and smoker. That's what you'd have to do if you were dealing with this issue in the insurance environment. You're going to have to segregate the smoke-free group and put them into a separate bucket, and then deal with the secondary-smoke-exposed group and put them into a new bucket. And then you'd still have the smokers, which are where they are now. There are marketing implications. There are pricing implications. The cost of testing may or may not add to what you're experiencing now. But how many cases do you test? This is something that could be dealt with through the application for the most part. And then testing could be used only for larger policy sizes.

I'm going to stop now and ask for any questions. Also if Mr. Reynolds wants to offer his opposing argument, we're ready to hear him.

From the Floor: Yes, I have a question. I'm wondering about these studies, and to what extent they compensate for confounding factors, such as alcohol, diet, and lifestyle. Would any member of the panel have comments on that?

Mr. Harris: Yes, I can. I've looked at some of the writings on confounding factors. They have tried to factor those out. I can give you the names of some of the studies that I've looked at. Actually, many of the studies that we've run into have come from the U.K. and Canada. Some of the more usable studies have been outside of the U.S. With the exception of the 1986 *Academy of Science Study* and the EPA report, there seems to be a dearth of information in the U.S. in some of these areas, and we've looked overseas to come up with some of the data. Confounding factors were looked at. You'd have to read the studies to see exactly what they did with it.

From the Floor: Any comments on the recent enforceability of the nonsmoker statement in the contract?

Mr. Mast: That's an excellent question because it probably varies from company to company in terms of what they're willing to try to contest. It probably would take a very large claim to really try to clarify whether a preexisting condition existed and whether someone misrepresented and created fraud by not disclosing the smoking history.

From the Floor: Do you know of any companies where the denial has been upheld on the basis of misstatement?

Mr. Mast: Oh, yes.

From the Floor: It is?

Mr. Mast: Yes. Very much so. I don't know how many. I've read about several personally. One very famous one occurred in the state of New York about ten years or so ago, which was very significant and involved a very significant amount of insurance. But some companies say that they'll just adjust the claim payment by the difference in premium they would have charged had that person been properly classified as a smoker versus premiums charged on a nonsmoker basis. To me, that's almost giving it away. There's no disincentive for people to misrepresent.

From the Floor: I'm wondering whether other pollutants should be considered in underwriting.

Mr. Harris: Well, it's one more factor that can be taken into account. I guess there are other pollutants, as Jess indicated. Do you start testing for pollution exposure? Auto exhaust fumes exposure? Dietary habits? I think with some of the more select policies, pollution is not taken into account, but diet and exercise are taken into account. There's also family history. Jess can perhaps address some of those. But one of the things that we discussed when we were interviewed by these people from *Money* Magazine is that there are, to my knowledge, no insurance companies that are taking exposure to secondary smoke into account in underwriting any of their policies. Is anybody here doing it? Is anybody's company doing it, to their knowledge? Does anybody know of anybody who's doing it? No. As we saw from some of the data here, you've got possibly half the risk from exposure to secondary smoke that you have from active smoking. That's a fairly sizable risk to not take into account in your underwriting process.

From the Floor: What about additional health care costs?

Mr. Harris: Morbidity. I haven't focused on that. I indicated that we had the one study, which was done by Milliman and Robertson in conjunction with a large

corporation, where we tracked their population and found that smokers had 31% higher health care costs than nonsmokers. I am not aware of any studies on the impact of secondary smoke on health care costs. The study that I discussed on heart disease looks at the relative risk rate for heart disease. I've extrapolated it to mortality. If you looked at heart disease by disease state, that same relative risk rate would apply to the cost by disease state, if you want to get into health insurance.

From the Floor: This is more a second-hand smoke question than a general smoking question. Dr. Lee, can you just give a brief overview of the half life of cotinine in serum versus urine versus saliva.

Dr. Lee: Generally speaking, whatever is reflected in the saliva is about the same as in blood, because when a person smokes and it goes into the saliva, very soon that is reflected in the blood because there's such a close transition between saliva and blood. There was some data earlier that showed saliva and urine and blood tests. The saliva and blood levels were just about the same. For an active smoker, it goes from saliva to the blood and then to the urine. So, the urine can show levels up to as much as 48 hours later. We usually say 24–48 hours. A very heavy smoker could keep it in the urine for even longer than that.

From the Floor: And the cotinine—for testing purposes, will remain in those media for comparable times?

Dr. Lee: Right. Usually if a person is a regular smoker it never gets down to zero before it starts on the upward swing again.