

FINAL

CARBON MONOXIDE

Background

It is our belief that the Surgeon General's Report may indict carbon monoxide (CO) as the substance in cigarette smoke responsible for the development of cardiovascular disease.

Claims about the alleged health effects of CO as found in cigarette smoke appear to be based on the 1967 research by Astrup and his co-workers. Because of this study which was reported widely in the literature from 1967 until 1974,¹ CO was assigned by some the role of a major atherogenic (causing atherosclerosis) agent in tobacco smoke. After two studies found only negligible effects of CO exposure on the accumulation of cholesterol in the aorta of other animals, Astrup repeated his earlier experiments. This time the results showed no influence of CO exposure on the development of atherosclerosis in rabbits. The researchers wrote,

"Using the same criteria for intimal damage as in earlier morphological studies, no histotoxic effect on intimal/subintimal morphology of coronary arteries or the aorta could be demonstrated, when light-microscopic evaluation was performed blindly."

In other words, in 1978 and again in 1980, Astrup and his co-workers reported that CO was not responsible for changes in the walls of the coronary arteries and aorta of rabbits.² Despite these important retractions, Astrup's

earlier indictment of CO is still cited by those who contend that CO in cigarette smoke is the causal agent for the development of coronary heart disease.

Other scientists have questioned the role of CO in tobacco smoke in the causation of cardiovascular disease. Even a German scientist with anti-smoking views has reviewed the literature on CO and heart disease and concluded:

"To summarize the chronic effects of CO on the development of arteriosclerosis, one can say that in numerous experiments CO has no effect in normocholesterolemic animals."³

It must be pointed out when discussing CO that people are exposed to CO whether or not they smoke. Man-made sources for CO include automobile exhaust fumes, industrial emissions and gas stoves. Carbon monoxide is also a natural product of body metabolism. It has been estimated that without any exposure to environmental carbon monoxide, the blood contains from 0.2% to 1.0% carboxyhemoglobin (the compound formed by CO and red blood pigment)⁴ Thus, people are exposed to carbon monoxide from various sources in normal, everyday environments.

Some people are exposed to relatively high amounts of CO in their work and living environments. For example, tunnel workers and motor vehicle examiners are known to be exposed to high levels on a regular basis. Yet data on these occupational groups do not support claims that CO causes

heart disease. In fact, workers were found to have no higher rates of cardiovascular disease than the general public.⁵

Those who claim that CO in tobacco smoke causes heart disease often cite the studies of well-known anti-smoking scientists like Aronow. However, it is important to note that Aronow's experiments with angina patients and his conclusions about the effects of CO have been questioned by numerous scientists.

That the relationship of CO to heart disease has not been established was admitted by Dr. Ernst Wynder in his statement to the National Commission on Smoking and Public Policy:

"...I attended a meeting in Germany just recently... There were half a dozen experts who were interested in myocardial infarction and the relative significance of nicotine and carbon monoxide in the [development] of myocardial infarction and arteriosclerosis. I drew a diagram and asked each one to put up the relative significance of CO and nicotine, both to arteriosclerosis and to sudden death. And every one of those six people gave me a different answer... which clearly indicated we hadn't proved this particular issue."⁶

The final claim that we believe could appear in the Report is the allegation that elevated COHb levels in pregnant women is responsible for low birth-weight babies. Low birth-weight is a complex problem. Thus, a simplistic focus on smoking alone is inappropriate and scientifically

unsound. The Director of a large Public Health Service Study compared birth weights of children born to women before and after they began to smoke.⁷ Dr. Jacob Yerushalmy demonstrated that smoking women tend to have smaller infants even before they took up the habit. He concluded that the crucial factor in the tendency to low birth-weight babies was "the smoker, not the smoking." This suggests that numerous biological and social factors can affect the weight of a newborn baby, and smoking or elevated COHb levels may or may not be one of them.

It should be emphasized that with CO as with the other issues contained in this year's Surgeon General's Report, politics is likely to win out over science insofar as the content of the report is concerned. There is conflicting evidence on the possible effects of CO and/or elevated COHb levels in smokers. Although there should be a call for research in order to investigate and substantiate any claims involving CO, we believe the report will probably cite only those studies that can support its position. We have presented evidence to support the other side.

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