Attachment B

# PVC AND FIRES: RESEARCH QUANTIFIES THE POTENTIAL RISK TO FIREFIGHTERS

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# PVC AND FIRES: RESEARCH QUANTIFIES THE POTENTIAL RISK TO FIREFIGHTERS

## INTRODUCTION

The largest single use of PVC is in construction applications. For this reason, firefighters and others have rightfully had special concerns about the way that vinyl products perform in a fire -- specifically, about the combustion byproducts that may be generated.

When vinyl products burn a number of toxic combustion products are produced. These products include carbon monoxide, hydrogen chloride, smoke and other materials. Amongst these materials, carbon monoxide has received a great deal of attention because of its widely recognized role in the cause of most fire fatalities. However, hydrogen chloride has also received considerable attention because of its irritant properties and its unique occurrence as a product of PVC combustion. Claims have been made that PVC is unusually toxic due to the generation of hydrogen chloride during combustion.

Until the mid-1980s, most understanding of hydrogen chloride toxicity was based on responses observed in rodents. The only information on its effects on humans was limited to a few studies from around the turn-of-the-century, using concentrations that were too low to fully characterize its effects. However, the fact that there were numerous anatomical and

physiological differences between rodents and higher mammals suggested that human response to hydrogen chloride might be different. Furthermore, studies sponsored by the Federal Aviation Administration suggested that some rodent species are not good predictors of human response to hydrogen chloride.

In 1983, the Vinyl Institute commissioned Southwest Research Institute to conduct a series of studies in baboons that would: 1) determine the concentrations of hydrogen chloride likely to cause immediate and delayed health effects in humans; and 2) assess the usefulness of rodents as models for predicting the potential health effects of hydrogen chloride on humans.

These studies show real difference in the ways rodents and baboons react to hydrogen chloride. They also suggest that humans can survive much higher levels of hydrogen chloride than previously assumed and that the potential for delayed effects on lung function is less than expected.

These results have significant implications for firefighters. First, they indicate that PVC products present no unusual fire hazard either to firefighters or fire victims. Second, they indicate that PVC combustion byproducts -- namely, hydrogen chloride -- generally require no extraordinary precautions or protective equipment. Specifically, normal precautions should be taken, such as the use of self-contained breathing apparatus, to protect against toxic products of combustion which are common concern in all fires.

## EARLY STUDIES

Several early studies conducted on hydrogen chloride have provided the framework for the more recent work. Our only direct information on the toxicity of hydrogen chloride in humans comes from studies of low level exposure to hydrogen chloride conducted around the turn-of-the century (Matt, 1889; Lehman, 1908). These studies show that man will not willingly expose himself to hydrogen chloride concentrations of 250 parts per million for more than five minutes. However, beyond this fact, there has not been enough information available in humans to provide a complete understanding of the potential hazards of hydrogen chloride. So, there has been a lot of speculation in the literature about the levels of hydrogen chloride that might be lethal to humans. Although these statements give the appearance of fact, they are not supported by any hard data.

## SPECIES DIFFERENCES IN LETHALITY

Most of the earlier studies of the toxic effects of high levels of hydrogen chloride used rodents such as mice, rats and guinea pigs. This is largely because rodents are inexpensive and easy to handle. However, these studies found differences in the way that these animals respond to hydrogen chloride. For example, Darmer, et al., (1974) studied the effect of high levels of hydrogen chloride on rats and mice. Comparing their lowest lethal doses, the smallest amount of hydrogen chloride causing death, he showed that mice were three to 10 times more sensitive than rats. Higgins, et al., (1972) also reported similar differences. In these investigations, mice were approximately four to 10 times more sensitive based on lethal effects.

Although there was evidence, such as in the studies mentioned above, which showed that the toxicity of hydrogen chloride varied from species to species, more credence was given

to the more severe effects noted in mice. In their chapter on "Characteristics of Irritant Gases," Henderson and Haggard (1943) said that 1,000 to 2,000 parts per million hydrogen chloride was dangerous to humans for even short exposure periods. These values were very close to the lowest lethal dose reported for mice. Unfortunately, many who have read Henderson and Haggard's review, have failed to notice that the values of 1,000 to 2,000 parts per million are just guesses. There are no actual data to support these values. It is particularly perplexing that these numbers have been, and still are being, treated as fact.

Others, in guessing about the toxicity of hydrogen chloride, have noticed that a lot of hydrogen chloride gets removed from the air in the nose and upper respiratory passages before it can get breathed into the lungs. This provides a measure of protection for the lungs. Rats and mice breathe solely through their noses, never through their mouths. However, humans and other primates such as baboons, when faced with irritating substances like hydrogen chloride, bypass their nasal passages and breathe through their mouths. So, some have theorized that lung damage from hydrogen chloride would be much more severe in humans than in rodents (Alarie, 1981). Fortunately, a number of recent studies have provided us with a better appreciation of the importance of species differences and its relevance to our understanding of the toxicity of hydrogen chloride to man.

## RECENT STUDIES

In 1984, Kaplan, et al., reported on the effects of hydrogen chloride in both rodents and baboons. These studies, sponsored by the Federal Aviation Association (FAA), rated the ability of a number of combustion gases, including hydrogen chloride, to impair escape. The FAA's purpose in these studies was to determine whether hydrogen chloride and other fire gases would impair a passengers ability to escape from a burning airplane. This work was the first

evaluation of the effects of high concentrations of hydrogen chloride in baboons. Baboons were used because of their biological similarity to man.

Contrary to reports of hydrogen chloride's ability to impair escape ability in rodents, the FAA found that five-minute exposures of baboons to 17,290 ppm hydrogen chloride did not cause such effects. Furthermore, the baboons where able to survive these much higher levels of exposure to hydrogen chloride than had been anticipated.

Other investigators (Kaplan, 1984; Kaplan, et al., 1988; and Hartzell, et al., 1988) have also found that primates are much less sensitive to hydrogen chloride than previously believed. Kaplan (1984) reported that baboons can survive exposure to 30,000 ppm and 15,000 ppm hydrogen chloride for five and 10 minutes, respectively.

In 1988, Kaplan, et al., studied the pulmonary effects of hydrogen chloride in baboons under the sponsorship of the Vinyl Institute. In these investigations, groups of three lightly anesthetized baboons were exposed (head only) to target concentrations of 0, 500, 5,000, or 10,000 ppm hydrogen chloride for 15 minutes. These concentrations were selected to enable comparison of results with data reported in rodent studies in the literature. Additionally, the two higher target concentrations are considerably higher than the maximum concentration of 3000 ppm measured in large-scale fire tests (Beitel, et al., 1986). The effects of the exposure on lung function and structure of the baboons was followed for up to a year. The baboon was chosen because of its similarities to man and its common use in studying human respiratory disease (Johanson, et al., 1982; Collins and Jones, 1978; Patra, et al., 1986).

## SHORT-TERM EFFECTS ON BABOONS

Baboons exposed to hydrogen chloride for 15 minutes, appeared to be an increase in the rate and depth of breathing, although the change in depth of breathing was not statistically significant. Additionally, the overall amount of air breathed in was also increased. However, there was considerable variability among animals in these responses. An increase in breathing rate has also been reported in humans inhaling low concentrations of sulfur acid mist, although breathing became shallower, in this case, resulting in a decrease in the overall amount of air breathed in (Amdur, et al., 1952).

Hydrogen chloride also had an effect on the acidity, oxygen and carbon dioxide content of the blood of the baboons after 15 minutes exposure. The acidity of the blood stayed about the same. However, the carbon dioxide and oxygen levels in the blood increased and decreased, respectively. The decrease in blood oxygen may be what triggered the increased breathing of the animals exposed to hydrogen chloride. This is a normal physiological response to low blood oxygen.

The cause of the low blood oxygen was not determined. However, it may have been due either to the constriction of a small airways or to the accumulations of fluid in the lungs. However, observations and x-rays taken within an hour after exposure, did not show any signs of fluid accumulation. This finding is in contrast to the observation of lung fluid accumulation in rodents exposed to high concentrations of hydrogen chloride by some investigators (Machle, et al., 1942; Darmer, et al., 1974). The short and relatively wide airways and other aspects of the anatomy of the lungs of rodents, may make them more prone to fluid accumulation in the lung.

## LONG-TERM EFFECTS ON BABOONS

Only the short-term effects of exposure to hydrogen chloride have been described so far. However, the long-term effects of hydrogen chloride exposure were also investigated by examination of these same baboons one year later for any persistent effects. The results of these examinations indicated that there was no long-term effect on the acidity, oxygen and carbon dioxide levels in the blood of the baboons exposed at even the highest level. Additionally, visual observations of the baboons and the results of tests of lung function indicate that long-term effects of hydrogen chloride one year after exposure were mainly limited to the 10,000 ppm-exposed animals. In this group, inhalation of the very high concentrations of hydrogen chloride caused severe injury to the lining of the nasal passages, with the subsequent development of nasal obstruction and continuous mouth breathing by the animals. There were significant differences in various indicators of lung function at one year after exposure between the 10,000 ppm hydrogen chloride-exposed and control animals. Significant differences in lung function were also evident in some of the other exposure groups at various times, but these differences occurred in inconsistent pattern. The importance of these differences is difficult to assess. However, the limited alterations of lung function and the absence of significant lung tissue damage, except at the highest hydrogen chloride concentration, suggest that the moist tissues of the mouth and nasal passages and upper respiratory tract effectively trap inhaled hydrogen chloride. Thus, it appears that hydrogen chloride's ability to penetrate into the lower portions of the lung is minimal.

Another way to assess the effects of hydrogen chloride is to assess its effect on the normal breathing response of baboons exposed to carbon dioxide. This was also done with the baboons in this study. Normally, there is an increase in the rate, depth and the overall amount of air breathed in by baboons which have been exposed to carbon dioxide. This is a normal

response designed to get more oxygen into animals starved for oxygen because of breathing carbon dioxide. Impairment of this response could imply that the hydrogen chloride produced some type of disturbance of the breathing control mechanism of the lungs.

The results, however, indicated that exposure to hydrogen chloride did not have any significant long-term effects on the breathing control system of the baboons, except at the highest concentration. Some differences in response between treated and untreated baboons were evident at lower hydrogen chloride concentrations at other time points. However, many of these differences were due to an increased rather than a decreased response and may not be due to hydrogen chloride. As will be seen, rodents and baboons exposed to hydrogen chloride differ in their response to carbon dioxide.

Based on the overall results of this study, very high concentrations of hydrogen chloride, of about 10,000 parts per million, can have certain long-term effects. However, baboons and presumably other primates, including man, can survive short exposures at these concentrations. This tolerance to hydrogen chloride was also observed in a previous study in which baboons survived a five-minute exposure to concentrations of from 190 to 11,400 ppm of hydrogen chloride (Kaplan, et al., 1985). In another study (Hinderer and Kaplan, 1986), an exposure of approximately 30,000 ppm for five minutes, 15,000 ppm for 10 minutes, or 10,000 ppm for 15 minutes did not result in lethality of any of the animals. However, these results are not consistent with statements that 1,000 to 2,000 parts per million hydrogen chloride is lethal to humans at short exposure periods (Henderson and Haggard, 1943; Alarie, 1985). Nor, are these results consistent with claims that mice are less sensitive than humans to hydrogen chloride or that lethal levels of exposure in mice should be divided by a certain factor for extrapolation to humans (Alarie, 1985; Kennah, et al., 1987).

## EFFECTS ON RODENTS

The preceding studies strongly suggest that mice, in particular, are not good models for predicting the toxicity of irritant gases such as hydrogen chloride in man. However, the use of baboons, the most appropriate model for man, has certain limitations, such as expense. Consequently, it would be advantageous if a rodent species were available that was an acceptable model. This is why The Vinyl Institute also sponsored a series of studies comparing the effects of hydrogen chloride in the mouse, rat, and guinea pig. This study is also important because of recent attempts to directly use rodent toxicity data for regulation of building and finishing materials.

This study was not completed, as designed, with the mouse, because of the low survival at 500 and 2,500 ppm of hydrogen chloride. This confirms the findings of other studies (Darmer, et al., 1974; Kaplan, et al., 1989) that the mouse is much more sensitive than the rat to the lethal effects of hydrogen chloride. The results of this study also indicate that the guinea pig is more sensitive than the rat to the acute lethal effects of hydrogen chloride. Comparing the results of the preceding study in baboons with these results, shows the sensitivity of the rat is more like the baboon than either mice or guinea pigs. However, the baboon appears much less sensitive, in this respect, to hydrogen chloride exposure than even the rat.

The changes in the breathing patterns of the mouse, rat and guinea pig in response to hydrogen chloride were similar. The breathing rate decreased accompanied by increase in the depth of breathing. However, the size of these changes differed widely in the three species. In the mouse, the overall amount of air breathed within a given amount of time was virtually unchanged by exposures at 500 or 2,500 parts per million. In the rat, exposure to 4,200 parts per million hydrogen chloride resulted in a marked decrease in the overall amount per unit

time of air they breathed. In contrast, the amount of air breathed by guinea pigs was increased by exposure to hydrogen chloride. The response of the baboons, as mentioned previously, was an across-the-board increase in all three parameters. This again shows a difference in the way that baboons and rodents respond to hydrogen chloride.

In both the rat and the guinea pig, exposure to hydrogen chloride caused a decrease in the acidity of the blood during the exposure. In the rat, 4,200 ppm hydrogen chloride appeared to cause an increase in blood carbon dioxide and a transient decrease in blood oxygen values. Exposure to 500 or 4,200 ppm hydrogen chloride appeared to have little effect on the blood carbon dioxide or oxygen values of the guinea pig. In comparison, as mentioned previously, the baboon did not exhibit any significant decrease in acidity, but did experience effects on carbon dioxide and oxygen levels which were similar to the rat. Again, none of the responses in the rodents model that of the baboon exactly.

Exposure to low concentrations of approximately 500 ppm hydrogen chloride did not cause any significant tissue damage in either the mouse or the guinea pig. However, severe damage to the respiratory tract and deaths during or shortly after exposure occurred in mice exposed to a higher concentration of 2,500 ppm hydrogen chloride. Exposure of guinea pigs at the higher concentration of 4,200 ppm hydrogen chloride also caused severe respiratory tract damage and deaths following exposure, and, possibly, residual damage to the lungs at three months following exposure. Exposure of rats to 4,200 ppm hydrogen chloride resulted in minimal damage to the respiratory tract at three months post-exposure. In comparison, microscopic examinations of baboons at one year after exposure, showed minimal tissue damage. Rodents that died during or following exposure to hydrogen chloride, exhibited severe damage to the respiratory tract, including fluid in the lungs of some animals. Similar data are not available for the baboon because there were no mortalities. However, clinical observations,

measures of carbon dioxide and oxygen in the blood, and lung function tests suggest that damage to the respiratory tract of the baboon was less severe than in the rodent. As stated before, it is possible that anatomical differences in the respiratory tracts of rodents and primates are responsible for the apparent difference in the effects of hydrogen chloride on these two species.

In rats, exposure to 4,200 ppm hydrogen chloride caused a somewhat reduced response in the rate of breathing of animals exposed to carbon dioxide at three days post-exposure, but not a three months after hydrogen chloride exposure. However, no effect on the overall amount of air breathed in per minute by rats was exhibited at either time period. Again, observing the effects of hydrogen chloride on the response to carbon dioxide is a way to measure the measure its effects on the breathing control system of the lungs in rodents. Normally, just as for baboons, there is an increase in the rate, depth and the overall amount of air breathed by rodents which have been exposed to carbon dioxide. This data suggests that hydrogen chloride caused some sort of short-term, but not long-term disturbance of the breathing control mechanism of the lungs of rats.

In guinea pigs, exposure to hydrogen chloride did not appear to affect the increased response in rate or depth of breathing in response to carbon dioxide exposure. However, the overall amount of air breathed in response to carbon dioxide exposure appeared to be reduced in the guinea pigs at three days and three months following exposure to 500 or 4,200 ppm hydrogen chloride.

The decreased breathing rate observed in the mouse at day three and the decrease in the overall amount of air breathed in by guinea pigs at day three and three months post exposure to hydrogen chloride contrasts with the response of the baboon. Hydrogen chloride,

at concentrations similar to what these rodents were exposed, did not appear to have any effect on the response of the baboons to carbon dioxide.

Comparison of the effects of hydrogen chloride in rodents with those observed in the baboon reveals several important differences between the two species. These differences indicate serious limitations in the use of data from the mouse, the rat, or the guinea pig to predict the toxic hazard of irritant combustion atmospheres in humans.

## Summary and Conclusion

The purpose of this overview has been to provide you with some of the latest scientific information on hydrogen chloride toxicity based on research sponsored by The Vinyl Institute. However, the real question is what all of this means for firefighters who may be exposed to hydrogen chloride during the course of their work. First, it is clear that hydrogen chloride can be lethal provided a person is exposed to a sufficient amount for a long enough time. However, estimates of the toxicity of HCL based on rodent studies have given the false impression that HCL is a "supertoxicant". The research conducted by the Vinyl Institute strongly suggests that this is not the case for hydrogen chloride.

One way to get an accurate perspective of the toxicity of hydrogen chloride is to compare its lethal exposure dose with that of some of the most important toxic gases present in fires. The lethal exposure dose is the minimum amount of a material required to kill test animals. The larger the lethal exposure dose, the less toxic the material. Available data show that the lethal exposure doses of carbon monoxide and hydrogen chloride to be similar, ranging from 112,000 ppm min to 192,000 ppm min (Hartzell, et al., 1985; Hartzell, et al., 1987). In

contrast, the lethal exposure dose of acrolein, a product of wood combustion, is 2,500-5,000 ppm min (Kaplan, et al., 1985). The peak concentrations of these gases have been measured in two studies involving firefighters equipped with monitoring devices (Burgess, W. A. , et al., 1979; Grand, A. F., et al., 1981). The peak level measured for carbon monoxide was 7,450 ppm, in contrast the peak concentration for hydrogen chloride was 280 ppm. It is interesting to compare the time that one would have to breathe each of these gases at these peak levels in order to reach its respective lethal exposure dose. In the case of carbon monoxide and acrolein, this occurs in thirty minutes, but for hydrogen chloride this point is not reached until 8.9 hours. This is important because it supports the conclusion of other studies that have shown that carbon monoxide, a product of combustion of any organic material, is the principal killer in fires. Carbon monoxide combines with hemoglobin in the blood and deprives the tissues of oxygen. Severe deprivation of oxygen leads to death.

Obviously, any unnecessary exposure to hydrogen chloride, carbon monoxide or any toxic materials should be avoided as much as possible. Consequently, as in all fire situations, the firefighter must wear self-contained-breathing apparatus throughout the incident, including overhaul.

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