1995

Chloroform exposure and dose determination associated with competitive swimmers during a two-hour swim practice

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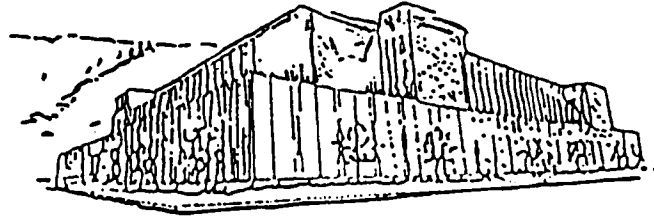*The University of Montana*

CHLOROFORM EXPOSURE AND DOSE DETERMINATION
ASSOCIATED WITH COMPETITIVE SWIMMERS DURING
A TWO-HOUR SWIM PRACTICE

by:

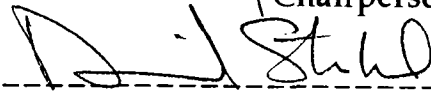David C. Berkoff

B.A., Harvard University, 1989

Presented in partial fulfillment of the requirements
for the degree of
Master of Science

Department of Environmental Studies
University of Montana
Fall, 1995

Approved by:

Chairperson

Graduate Dean

12-8-95

Date

UMI®

Dissertation Publishing

ProQuest®

Chloroform Exposure and Dose Determination Associated with Competitive Swimmers during a Two-Hour Swim Practice (49 pp.)

Director: Dr. Brent C. Ruby

One male and one female subject were submitted to a swimming protocol (10x100yd, 22x250yd) in which exhaled breath samples were collected using a Summa® single breath container system. Sampling occurred prior to, during exercise (120 minutes) at select intervals and post-exercise in a chloroform-free environment (180 minutes). Exercise heart rate and minute ventilation (V_E) were recorded periodically to assess exercise intensity. Samples were analyzed for chloroform as previously described by Winberry et. al. (1988) and was detected in all breath samples. Exercise HR ranged from 129-146 and 149-182 bpm for the male and female subject, respectively. Exercise VO_2 averaged 1.5 and 1.8 $L \cdot min^{-1}$ for the male and female subject, respectively. Results demonstrated a near 100 fold increase in exhaled breath [chloroform] within 60 minutes of exercise and exposure to pool water. Breath concentrations increased from 3.18 to 371.73 $\mu g/m3$ and 3.46 to 339.91 $\mu g/m3$ for the male and female subject, respectively. Pool air [chloroform] ranged from 145.28 and 147.64 $\mu g/m3$ during the entire exposure period. Although chloroform removal was rapid (approximately 10 fold within 90 minutes recovery), values did not return to pre-exercise levels within 180 minutes. Evidence showed that both inhalation and dermal routes of exposure were equally responsible for total body burden. Using $V_{E\,BTPS}$ ($L \cdot min^{-1}$), total exposure time, an EPA efficiency coefficient for uptake (0.7), and ambient chloroform concentration, the estimated total dose of exposure was 1429 μg and 1532 μg chloroform for the male and female subject, respectively (using an equation developed by Jo et. al., 1990). Although the estimated dose is below EPA standards, the potential for health risk may be seen for those athletes exposed to pool water for extreme periods of time in pools with poor air ventilation and high pool water chloroform concentrations.

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Chapter I

Introduction

The wide-spread use of chlorine in public drinking water systems has removed the threat of many water-borne diseases. Countless millions of people have been saved from premature death and debilitating diseases due to relatively microbe-free drinking water. The use of chlorine spread quickly to the purification of pool water. However, public drinking water and pool water are not completely safe. The discovery of chloroform and other trihalomethanes (THM) by Rook, and Bellar, respectively in 1974 in water supplies, and subsequently in pool water by Beech et. al., (1980) raised concerns about the possible health effects associated with long-term exposure. While drinking water systems and pool environments are different, their chemistry is somewhat similar. Organic materials combine with free chlorine to form elevated levels of THM.

Many harmful chlorinated organics are found as chlorination by-products. Because of its volatility, quantity, ability to be absorbed, and status as a class B2 carcinogen, chloroform is a chief health concern. Long-term use of chlorinated drinking water has shown associations of colorectal and bladder cancers (Morris et. al., 1992) and birth-related health problems such as cleft palate, miscarriage and low birth-weight (Bove et. al., 1992).

Some concern has focused on drinking water systems concerning the efficacy of the United States Environmental Protection Agency's (EPA) Safe Drinking Water Act standard for THM of 100 ppb. Showering has been found to be a significant contributor to chloroform exposure. Dermal and inhalation routes are thought to be equally significant contributors to total chloroform exposure (Jo et. al., 1990).

Unlike public tap water, pool water has no health-related standards under the EPA's guidance. Aggazzotti et. al., (1993) found concentrations of chloroform in the expired breath of swimmers ranging from 13.9 to 311 $\mu g/m^3$. Exercise intensity, respiration and environmental air chloroform concentrations have a direct effect on chloroform breath concentrations (Aggazzotti et. al., 1990, Lévesque et. al., 1994).

A person swimming one hour three times per week may have the equivalent exposure to chloroform equal to two times or more the EPA standard for drinking water (Weisel and Shepard 1994). Accordingly, assuming a linear relationship exists between exposure and adverse health effects, Weisel and Shepard (1994) estimated a two-fold increase in possible long-term health problems. A study by Wilson (1995) suggested levels of exposure to chloroform in pools may vary greatly due to differences in water and environmental air concentrations of chloroform. While some studies have attempted to measure chloroform exposure of the average recreational swimmer, no studies have completely focused on the potential chloroform burden of competitive swimmers in a more typical and longer exposure period.

Significance of the Problem

The average competitive swimmer swims for two hours per day, five days per week. A swimmers' dedication to their sport can see them spend up to thirty hours per week in the pool for five to fifteen years. During this time water is agitated, skin temperature is increased, and respiration and heart rate are elevated, all of which have been found to be significant predictors of increased blood and breath chloroform levels (Aggazzotti et. al., 1990; Aggazzotti et. al., 1993; Lévesque et. al., 1994). If exposure to chloroform is

significant during a ten minute shower (Jo et. al., 1990), or over a one hour swim, will this trend of increasing exposure and adverse health effects continue on a linear curve?

Over ten million people in the United States swim recreationally or competitively on a regular basis. As a competitive swimmer who has participated in two Olympic Games, the sport of swimming has given me a unique and rich lifetime of experiences. However, my time spent in the pool is a cause for some personal concern. Almost three full years of my twenty-nine alive have been spent submerged with an elevated heart rate in chlorinated water. What does that mean for the long-term future of my health and for the future of my aquatic colleagues?

Statement of the Problem

The purpose of this study was to measure the effects of a two-hour swimming practice in a chlorinated pool environment on chloroform concentrations in expired air samples of two subjects, a 23 year-old male and a 22 year-old female.

Expected Results

(1) Male subject would show significant increases in chloroform concentrations in sampled expired air over time during exposure to the pool environment.

(2) Chloroform concentrations in the expired air of female subject were expected to increase significantly during exercise as compared to the pre-exercise sample.

Justification: Studies by Aggazzotti et. al., (1990, 1993) and Lévesque et. al., (1994) found that blood plasma chloroform levels as well as alveolar air chloroform concentrations samples in swimmers exposed to chlorinated pools increased with level of activity, agitation of water, and environmental chloroform concentration.

(3) Increased ventilation was theorized to result in a rapid rate of chloroform uptake followed by another rise due to a later dermal exposure route.

Justification: Weisel and Shepard (1994) measured chloroform removal rates in one subject exposed to one hour of swimming on five separate occasions. Chloroform was found to leave the body mainly via a two-part curve over time consistent with separate uptake through dermal and inhalation pathways. Inhalation was said to be the more rapid for both uptake and removal, while the dermal pathway was the more gradual.

(4) During post-exercise sampling, it was hypothesized that chloroform removal would follow a two-peaked curve representative of inhalation and dermal routes of intake during exercise.

Justification: According to data produced by Weisel and Shepard (1994), chloroform absorbed dermally is eliminated more slowly, as compared to chloroform absorbed through inhalation.

Descriptive Assumptions

Assumption 1: It was believed that at some point during the simulated two-hour practice, an equilibrium would be reached in both male and female subjects, where the uptake of chloroform through all routes of exposure would equal the removal of chloroform through expired air.

<u>Assumption 2:</u> It was thought that chloroform breath concentrations would be related to minute ventilation. Raymer et. al., (1993) suggested variability in VOC breath concentrations can occur even when ventilation rates are similar. However, monitoring of ventilation and heart rate can be effective in helping determine total dose of exposure to a compound (Raymer et. al., 1993).

<u>Scope and Limitations</u>

This study was limited to two single subject case studies (one female and one male.) Due to differences in age, gender, fitness level, minute-ventilation and heart rate, uptake of chloroform into body systems may vary to some degree. Furthermore the use of a single subject case study design removed the possibilities for inferential statistical analysis and thus limited this study to descriptive statistics.

This study does offer a general idea of the nature of chloroform uptake and removal in healthy, young adult male and female competitive swimmers. However, results do not show the mean and range from a representative sample of competitive swimmers.

Chapter II
Review of Literature

Introduction

Water-borne diseases derived from public drinking water systems rarely pose a problem in the United States. Over 200 million people obtain their tap water from chlorinated public systems, or more than 80% of the country's population. While water-borne diseases are still a cause for concern in many other countries, the implementation of chlorination systems at the turn of the century in this country virtually eliminated diseases such as cholera, typhoid, and polio (Miller et. al., 1978).

However, many potentially harmful halogenated organics are formed in the chlorination of water supplies. Dissolved organic materials combine with free chlorine to form chloroform, a trihalomethane (THM). The formation of THMs, including chloroform, was first noted by Rook, and Bellar, et. al., in 1974. THMs include dibromochloromethane, bromodichloromethane (a group 2B carcinogen), bromoform (a suggested 2B carcinogen), and chloroform.

Under the Safe Drinking Water Act of 1974 (SDWA), water supplies are regulated and protected from potential contamination by harmful pollutants. While the SDWA standard for THM, including chloroform, in drinking water is 1.0 parts per million (ppm) (the EPA has recently proposed a .25 ppm standard), standards do not apply to chlorinated pool waters. In fact, no safety standards exist for chloroform and other common chlorination disinfection by-products in pool environments. Microbial sterility rather than chemical purity is the goal in pools.

However, much of the same chemistry present in the production of chlorinated tap water exists in swimming pools. Chlorine is generally present in pools at higher concentrations than in tap water. These standards vary from state to state and even from county to county within states. Generally, local health standards require chlorine or a similar disinfecting agent to be present in pools at 1.0 to 3.0 ppm.

Although previously not perceived as a problem, organic material is common in pools. Hair, blood, skin, saliva, and other sources of organic material are sufficient to cause the formation of many halogenated organic compounds (Beech et. al., 1980). Studies have shown that trihalomethanes (THM) are present in surprising concentrations in and around indoor and outdoor pools. THM levels averaged 125 parts per billion (ppb) for freshwater pools and 657 ppb for saline pools (Beech et. al., 1980). Other halogenated organics are present in chlorinated water. Trichloroacetic acid and dichloroacetic acid for example, may be present in chlorinated water at up to ten times the concentration of THM (Gabler, 1988). However, less is known of the possible adverse health effects of these chemicals.

Although many mutagenic, teratogenic, and carcinogenic compounds can be found in chlorinated waters, chloroform and its related forms represent the greatest health concerns due to their volatility, relative quantity in chlorinated waters, rapid uptake in exposed subjects, and classification as a Class B2 carcinogen by the EPA. It is for these reasons many recent experimentations and health studies have focused on THM compounds.

Non-Human Experimentation

Bellar (1974) found concentrations of THM in drinking water ranging from 37 to 150 PPB, and concluded that although chloroform was not known

to have any acute effects upon humans, chronic studies should be undertaken to determine whether associations between THM and adverse health problems occur (Bellar 1974).

As a result of the Rook and Bellar studies, experimental tests have been administered upon animals to determine acute and sub-chronic effects. One study found that rats developed kidney epithelial tumors with low dose and high dose exposure to chloroform over a 78-week period (Page and Saffiotti 1976). The same study found that after 92 weeks of exposure to chloroform, mice developed hepatocellular carcinoma at both low and high doses, and nodular hyperplasia in the livers of male mice at low doses when the carcinoma was not present (Page and Saffiotti 1976).

A 90-day sub-acute study of the effects of chloroform on rats and mice found that liver fat concentrations increased in rats, and that hemorrhaging of the lungs in mice, as well as liver and spleen damage in mice, occurred at significantly higher rates following exposure (Jorgensen and Rushbrook 1980).

Other experimental data shows adverse teratogenic and embryotoxic effects on female rats. Studies have indicated that chloroform exposed rats had impaired ability to maintain pregnancies, and their offspring exhibited increased incidence of cleft palate, low birth weight, and decreased ossification of bones (Murray et. al., 1979). Another study suggested that chloroform caused low conception rates and was significantly embryotoxic, causing birth defects and low birth weight (Schwetz et. al., 1974).

Human Epidemiological Studies

Much of what has been reviewed above tells us about the by-products of water chlorination and the acute effects they have on animals, but it does not reveal what human reactions to exposures might be. Often the symptoms of a

disease caused by exposure can take twenty to forty years to surface in humans. Epidemiological studies can tell us more about what the risks of a certain exposure may be on human populations, but the causality of the result can almost never be proven.

Since the discovery of THM's in drinking water, epidemiological studies have searched for associations of negative health effects with the use of chlorinated water supplies. Based on results of experimental animals studies, the original focus of these human epidemiological studies was for types of cancers, but more recently studies have focused on reproductive problems.

Cancer Mortality

Using a logistical regression statistical technique, Cantor et. al., (1978) concluded that people who have been drinking chlorinated surface water for a long period of time have an elevated risk of developing bladder and brain cancer in both sexes, and non-Hodgkin's lymphoma and kidney cancer in men. Urinary tract and gastrointestinal cancers were associated with the reuse of water supplies in the United Kingdom (Beresford 1983). Another study concluded that pancreatic cancer was associated with the level of THM in drinking waters (Carlo et. al., 1980). Ijsselmuiden et. al., (1992) also found a positive association with pancreatic cancer and chlorinated drinking water use in caucasian Washington, D.C. area residents. Bowel, liver, and bladder cancers in St. Louis were associated positively with THM production in drinking water (Marienfeld et. al., 1986).

Alvanja et. al., (1978) found positive associations for colon, bladder, esophageal, lung, pancreas, and rectal cancer deaths and chlorinated water use in New York using a chi square statistical test. Young et. al., (1981) also found associations for colon cancer, but marginal associations for brain, and stomach

cancer fatalities for women in Wisconsin. The Wilkins and Comstock (1981) cohort study found minimal correlations of bladder cancer in men and liver cancer in women in Maryland. Gottlieb et. al., (1982) found positive associations for rectal and breast cancer fatalities in Louisiana women. Cragle et. al., (1985) saw positive associations of colon cancer and duration of exposure to chlorinated tap water in North Carolina. Zeirler et. al., (1988) saw positive associations between long-term exposure to chlorinated tap water and bladder cancer mortality.

Some studies found limited or no correlation between chlorinated tap water and cancer rates. Brenniman et. al., (1980) found a marginal correlation between colon and rectal cancer and use of chlorinated ground water sources. Lawrence et. al., (1984) found no association between chloroform exposure and colorectal cancer incidence. Zeirler et. al., (1986) found statistically insignificant correlations between use of chlorinated waters and bladder cancer. And Young et. al., (1987) found no association between exposure to trihalomethanes and colon cancer.

Perhaps the best review of human epidemiological cancer studies is by Morris et. al., (1992) who used a meta-analysis technique to determine the statistical significance of previous studies. This assessment found positive associations between chlorination and bladder and rectal cancers.

Birth Related Disorders

The most recent epidemiological studies have shown a correlation between certain birth-related problems and chlorinated drinking water. Congenital defects such as cleft palate, low birth weight, and miscarriages were found to be positively associated with the long-term use of chlorinated drinking waters in Suburban New Jersey (Bove et. al., 1992a,b). The risk of

premature birth and low birth weight was ten to thirty percent higher among women using chlorinated water; while cleft palate and cleft lip occurrence were three and one half times higher. When pregnant women were exposed to water containing THM levels at 80 ppb (the EPA safe drinking water standard is 1.0 ppm), neural tube defects increased thirty-one percent (Bove et. al., 1992a,b). An increase in central nervous system defects was associated with 80 PPB THM or more in water sources as well.

A separate study concluded that chloroform levels in drinking water correlated positively with intrauterine growth retardation, but not with low birthweight and prematurity in women in Iowa (Kramer et. al., 1991).

Acute Responses to Pool Water Exposures

While no data exists on the long-term health effects of continued and long-duration exposure to pool environments, some experimental work has been completed on acute responses to exposures to pools.

Many acute reactions to exposure to chlorinated pools are common to recreational and competitive swimmers. Brominated pools seem to cause a higher incidence of dermatoses in users (Rycroft and Penny 1983). Stress-induced asthma is now a common diagnosis for many respiratory disturbances in swimmers (Penny 1983). As a result, one acute study comparing a group of competitive swimmers with a control group looked for differences in allergic responses, sensitization to aeroallergens, and imbalances in the immune system. Eleven of the fourteen swimmers had cases of respiratory conjunctivitis, rhinitis, rhinoconjunctivitis, laryngitis, or bronchitis (Zwick et. al., 1990). Sensitization to aeroallergens was found in eleven of the fourteen swimmers using a radioalleroabsorbency test, as compared to only five controls. Seven of the swimmers had immune

imbalances in T-cell, B-cell, or killer cells,, while only two controls showed imbalances.

Shower and Pool Water Exposure to Chloroform

Much focus has been given to uptake of chloroform and other volatile organics (VOC) from exposure to tap water through alternate routes of exposure, namely dermal and inhalation. The original EPA assumption in determining a Safe Drinking Water Act standard for chloroform was that ingestion of water contributed the sole source of chloroform exposure (two liters per day for a lifetime in a 70kg person). However, recent experimentation found that even a daily ten-minute shower has been found to significantly increase exposure to chloroform (Jo et. al., 1990).

Brown et. al., 1984, calculated that skin permeability for uptake of chloroform in showers was 0.6 to 1.0 cm/hr, finding that dermal uptake contributed a significant amount of chloroform to the total assumed body burden. Brown found 64 % of chloroform exposure can be attributed to skin absorption. Jo et. al., (1990) concluded that dermal and inhalation uptake of chloroform during a ten-minute shower were approximately equal; the combined inhalation and dermal uptake contributing about the equivalent of drinking 1.2 liters of tap chlorinated water. Gabler et. al., (1988) estimated that inhalation of shower vapor constituted the equivalent chloroform exposure of drinking two liters of tap water.

Swimming in pool water has been shown to contribute significantly to chloroform exposure. Lahl et. al., (1981) estimated that adults are exposed to a 50μg dose of chloroform (0.7μg/kg) through inhalation alone in a 30 minute swim. Aggazzotti et. al., (1993) found that alveolar air concentrations were affected by chloroform air concentration, level of physical exertion, and age of

subject. Younger subjects were found to have the highest concentrations of breath chloroform. Lévesque et. al., (1994) found elevated breath chloroform in swimmers to be related to pool water and air concentrations.

Weisel and Shepard (1994) found that inhalation and dermal routes of exposure both significantly contributed to body burden, with inhalation possibly being the more rapid route, and dermal uptake being the more gradual. It was estimated that the chloroform dose for persons who swim one hour three times per week is 2-times higher than from drinking or showering alone, concluding that adverse health effects associated with chloroform exposure may be at least 2-times higher in swimmers than non-swimmers.

Chapter III

Methodology

Subject Selection

The earliest water chlorination-related health projects examined the potential health risks associated with long-term exposure to chloroform in drinking water. Another significant source of chloroform exposure was later found to be exposure to chlorinated pool environments (Beech et. al., 1980). Competitive swimmers assume perhaps the greatest risk of long-term chloroform exposure of all bathers due to the duration of exposure and elevated heart and respiration rates.

For these reasons, two competitive swimmers with at least five years of competitive experience were selected. Both were in good health, were non-smokers, and accustomed to vigorous swimming exercise.

The first subject was a 70 kg, twenty-three year-old male triathlete and Master's swimmer with ten years swimming experience. The second subject was a 65 kg, twenty-three year-old female with more than fifteen years of competitive swimming experience.

Briefing Subjects on the Study

Subjects were given information regarding the history of chloroform and health concerns related to human exposure, read and signed an informed consent form approved by the Internal Review Board of the University of Montana. Subjects were allowed and encouraged to review recent studies contained in the References section of this thesis.

Subjects were informed as to the nature of the simulated practice and the effort level desired during exercise. Subjects were instructed as to the use of the single breath canisters (SBC's), and the collection of heart rate and minute

ventilation data. Post-exercise removal from the pool area and sampling was also explained.

Sampling System

The SUMMA® one liter evacuated stainless steel SBC sampling method is an effective system in determining concentration of volatile organics in exhaled breath (Boise, Idaho) (Pleil and Lindstrom 1994). Sampling of exhaled breath is a non-intrusive and effective way to determine level of exposure (Pleil and Lindstrom 1994), as single breath canisters allow for the quick and relatively easy collection of exhaled air.

Subjects can easily give samples in the seated position by inserting a five centimeter Teflon tube attached to the single breath container, opening the canister valve, and exhaling (see Appendix VI). One hand holds the base of the canister while the other operates the valve. After the canister is filled the subject can then close the valve. Two canisters were reserved in this study for the mastering of the sampling technique.

Heart and Ventilation Rate Analysis

Ventilation rate has been found as important in helping to predict exposure levels of volatile organic compounds (Raymer et. al., 1993). Increased respiration rate is found to lead to increased levels of exposure to many VOC's including chloroform (Raymer et. al., 1993; Aggazzotti et. al., 1993). Rough estimates of exposure level have been made by knowing ventilation rates and air concentrations of contaminants (Weisel and Shepard 1994; Lévesque et. al., 1994; Wilson 1995).

Consequently, thirty-second samples of expired air were collected during the exercise protocol immediately following swimming intervals at 25, 50, 75,

110 minutes. A Hans-Rudolph non-rebreathing mouthpiece was connected to a gas collection bag using low-resistance tubing and a three-way flow valve. Samples were immediately transported to the Human Performance Laboratory and analyzed. Expiatory volumes (VE) were expressed as body temperature pressure saturated (BTPS) and standard temperature pressure dry (STPD) after measurement in a gas flow meter (Parkinson Cowan Industrial Products, London, England).

Monitoring of heart rate to determine level of exertion during exposure tests has been used by Raymer et. al., (1993). Heart rate was continuously recorded after each swimming interval using a chest strap heart rate monitor (Polar, Port Washington, New York) to maintain a consistent level of exertion during the two-hour swimming session.

Experimental Design

This experiment represents two case studies relating a typical competitive swimming practice and the subsequent intake and loss of chloroform in two individual subjects.

Two sets of data were collected over a five-hour period. Two hours were dedicated to swimming a scheduled practice and sampling of exhaled breath during exercise. Three-hours of post-exercise sampling were conducted away from the pool area in a chloroform-free room in an adjacent building to examine the nature of chloroform removal from the body.

A. Environmental Sampling

The University of Montana Pool is a seven lane, 25 yard, deck-level gutter system swimming facility, ranging depth from one meter to almost four meters. Pool temperature is maintained at 84 degrees Fahrenheit. The

pH and gas chlorine concentrations are maintained via computer at 7.4 and 1.5 PPM respectively.

Sampling of environmental air at the University of Montana Pool was conducted at one, 60, and 120 minutes during the exposure period via grab samples in single breath canisters at a height of approximately six inches above the water surface. Swimmer level air samples were also taken with a personal whole air sampler (PWAS) integrated sampling system during the exposure period (Whitaker et. al., 1995).

Sampling of environmental air was conducted at the University of Montana Human Performance Laboratory in McGill Hall. Although this area was assumed to be chloroform-free, SBC grab samples of pool air were taken at 90 minutes and 180 minutes. A PWAS integrated air sample was taken for approximately three hours during the post-exposure period.

A single environmental air SBC grab sample was taken at one minute after the exposure period outside of the pool as subjects were in transit to the Human Performance Laboratory.

B. Exercise Sampling

Subjects gave expired air samples prior to the exposure and exercise portion of this study. Both subjects were then asked to complete a prescribed swimming workout. The first 10 minute period of 5x100 yard swims on 2 minutes simulated a typical warm-up. The following 110 minutes, subjects completed 22x250 yards swims on 5 minutes. During the exposure period, the male subject was sampled for expired air at selected intervals for two hours immediately following the interval distance (Figure 3.1.). The female subject gave expired air samples at 65 and 120 minutes only. This was due to a limited number of SBC containers.

Time --->

*   *   *

Δ_Δ_Δ_Δ_Δ_Δ__Δ__Δ___Δ_____Δ_____Δ_____Δ_____Δ
0 2 4 6 8 10 15 20   30   45   60   90   120

Δ-5x100 yds on 2:00-Δ---------------------------22x250 yards on 5:00----------------------------Δ

Swimming Interval
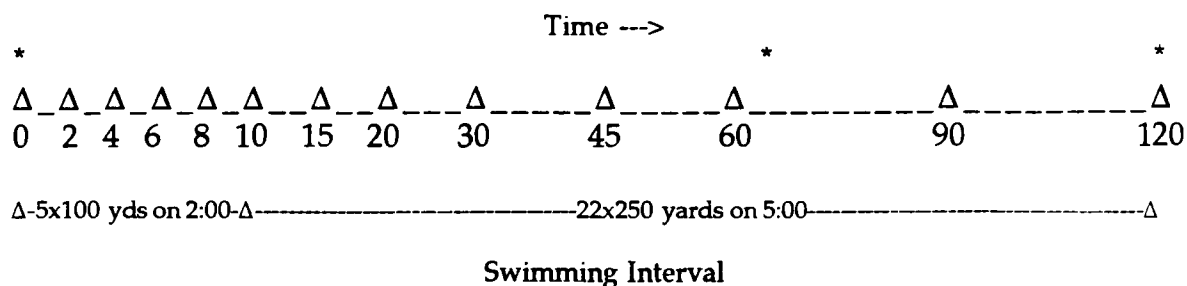
Figure 3.1. ExposureSampling. Δ designates male subject, * designates female subject.

C. Post-Exercise Sampling

Upon completion of the two-hour exposure period, both subjects were removed from the pool environment and moved to the Human Performance laboratory for post-exposure sampling. No shower was taken after the swim period as showering has been noted as a potential route of exposure to additive chloroform (Jo et. al., 1990). Expired air samples were taken at one minute and two minutes after removal from the pool environment at an outdoor location en route to the laboratory. The remainder of the samples were taken at the Human Performance laboratory (Figure 3.2.).

Time --->

Δ_Δ_Δ__Δ__Δ___Δ___Δ___Δ___Δ___Δ___Δ___Δ___Δ___Δ
1 2 4   8 10   15   30   45   60   75   90 105 120 150 180
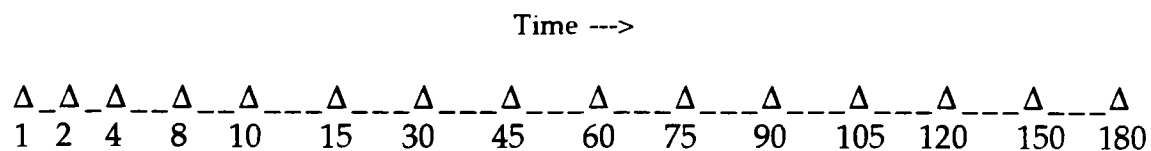
Figure 3.2. Post Exercise sampling over a three hour period.

Analysis of Samples

Because of the ability to transport and store the SBC's, immediate analysis of samples was not essential to the efficacy of results. Therefore, samples were taken to the Atmospheric Research and Exposure Assessment Laboratory at the U.S. Environmental Protection Agency in Research Triangle Park, North Carolina for analysis.

Samples were analyzed for chloroform using EPA Method TO-14 protocol as implemented by Graseby Nutech for the Model 3550A Cryogenic Concentrator (Graseby Nutech. Smyrna, GA) (Winberry et. al., 1988). Data was subsequently compiled by Andrew Lindstrom and Joachim Pleil at the U.S. EPA and returned.

Chapter IV

Results and Discussion

Environmental Data

Water samples collected at the University of Montana pool site were found to contain 68 and 73 $\mu g/l$ of chloroform. These results are consistent with past literature (Beech et. al., 1980 found 100 ppb TTHM).

SBC grab samples of swimmer-level air were taken at 1, 60, and 120 minutes during the exposure portion of the experiment (Table 4.1). Although the lower concentration may have been dependent upon less water agitation at the time of sampling, the one minute sample was noted to have a weak vacuum during opening and its corresponding result was inconsistent with the other two samples. The samples from 60 minutes and 120 minutes showed results of 145.28 and 145.37 $\mu g/m^3$ chloroform, respectively. The PWAS integrated sampler continuously sampled environmental air for almost two hours showing a result of 147.64 $\mu g/m^3$ of chloroform in the pool area.

Table 4.1. Pool Air Chloroform

Time	Chloroform ($\mu g/m^3$)
1:00	36.63*
60:00	145.28
120:00	145.37
PWAS (continuous)	147.64

*canister noted for weak vacuum during sampling

Post-exposure air samples were taken at 1 minute in an outdoor area leading to the Human Performance Laboratory, and at 90 minutes and 180 minutes in the Human Performance Laboratory. The one minute post-exposure sample was 2.86 $\mu g/m^3$. The 90 minute and 180 minute post exposure samples showed 2.20 and 1.79 $\mu g/m^3$ of chloroform respectively. The PWAS integrated sampler showed a chloroform concentration of 3.96 $\mu g/m^3$ during 174 minutes in the lab area. All post-exercise environmental air results suggest very little potential for impact on expired breath chloroform data.

Male Subject

Samples of expired breath were taken on the intervals as described in Chapter III. Pre-exposure sampling of the male subject showed very little chloroform in expired air (3.18 $\mu g/m^3$). The female subject had a similar chloroform breath concentration (3.46 $\mu g/m^3$). This value is slightly higher than the general exposure to environmental chloroform data value of 1.3 $\mu g/m^3$ as found by the EPA in a study of over 800,000 people (Pellizzari et. al., 1981, 1987a, 1987b). Although subjects were asked not to swim, shower, or ingest any potential chloroform containing food items in the 24 hours prior to thesis study, elevated breath concentrations in the expired breath of the

in the expired breath of the male subject increased immediately following the commencement of swimming (Graph 4.1). Chloroform concentrations increased in an almost linear manner until 60 minutes at which breath concentrations began to peak. Chloroform in expired air reached a peak concentration of 371.73 $\mu g/m^3$ at 90 minutes and dropped rapidly to 257.01 at 120 minutes (Graph 4.1). This anomaly may have occurred due to the corresponding decrease in minute ventilation, or a poor sample (Graph 4.3).
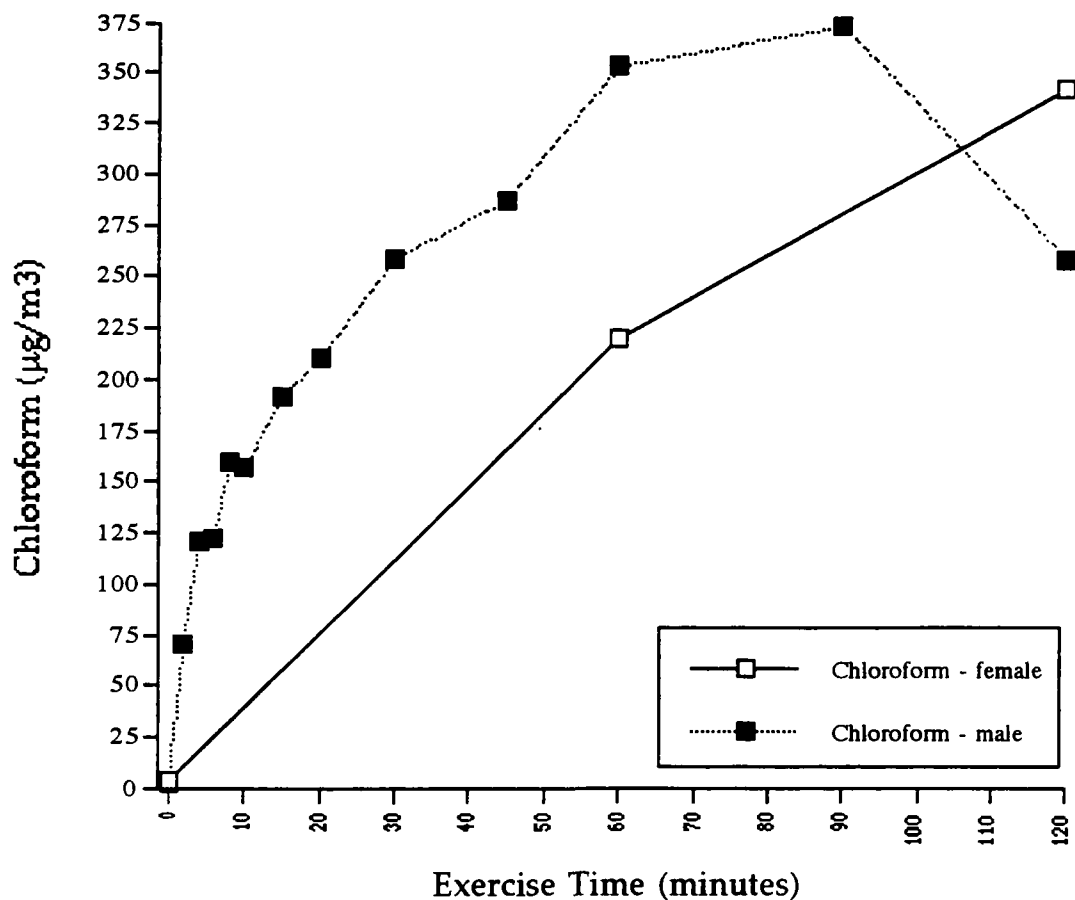
Graph 4.1. Exposure Period (both subjects).

Post-exposure sampling began at one minute after removal from the pool environment. Breath chloroform concentrations dropped rapidly from 257.01 $\mu g/m^3$ at 120 minutes of exposure to 215.02 $\mu g/m^3$ at one minute to 67.42 $\mu g/m^3$ over a period of 45 minutes post-exposure. Breath chloroform concentrations then fell more gradually to 28.35 $\mu g/m^3$ at 150 minutes (Graph 4.2.). The sample collected at 180 minutes after exposure was contaminated in route to the EPA. While breath chloroform levels dropped rapidly at first and then more slowly after 45 minutes, the corresponding data did not support the Weisel and Shepard (1994) two-peaked elimination curve model

suggesting a separate elimination of inhalation and dermally absorbed chloroform.
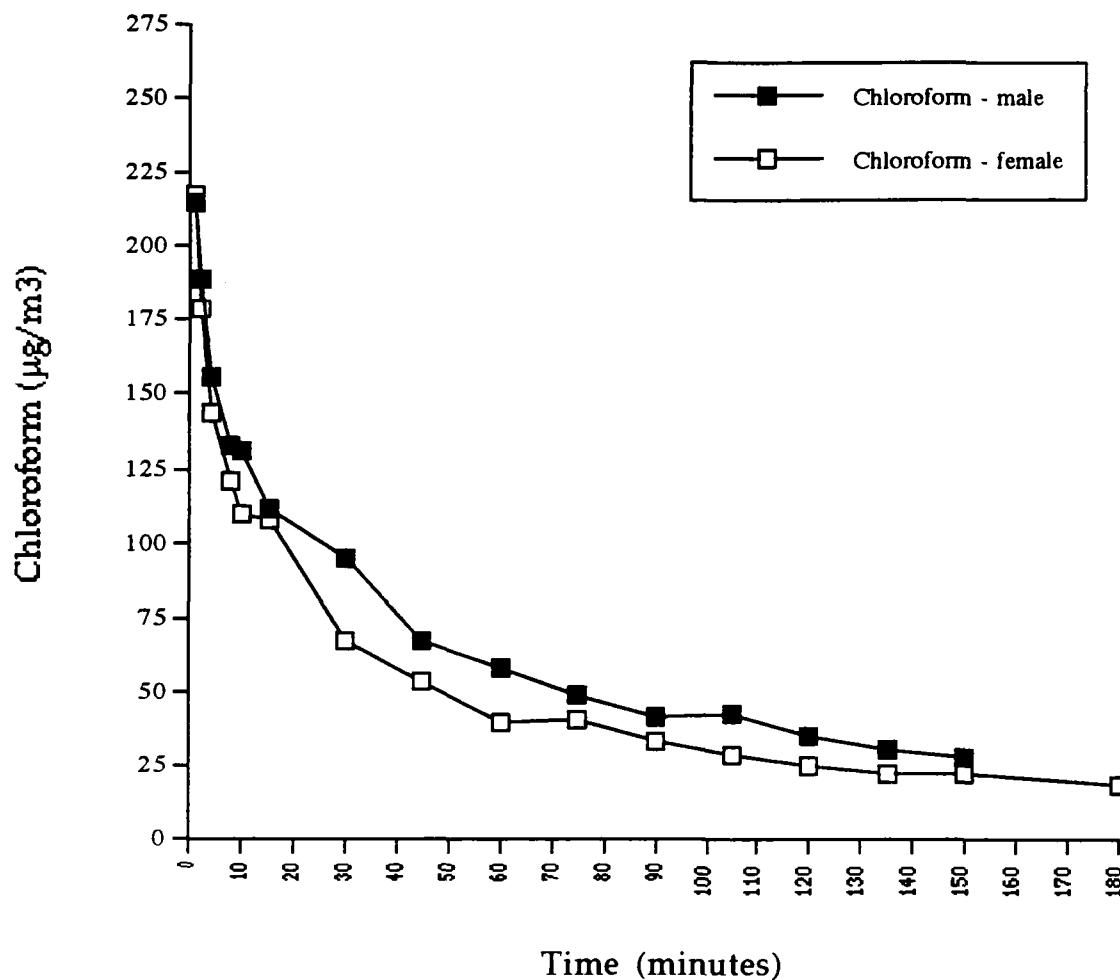
Graph 4.2. Post-Exposure Breath Chloroform (both subjects).

These data strongly suggest that both the dermal and inhalation routes of exposure are at least equal in their contribution to the total exposure burden. This supports assertions by Jo et. al., 1990 and Brown et. al., 1984 that dermal route of exposure to chloroform may contribute up to 64% of the total chloroform body burden, and refutes suggestions by Wilson (1995) and

Levesque et. al., (1994), that the dermal pathway contributed less than inhalation.

Deck-level pool air chloroform concentrations ranged from 145 to 147 $\mu g/m^3$ or less than twice the highest concentrations found in the expired breath of the male subject. Even if some chloroform were to accumulate within the subject's body, dermal absorption would contribute at least half the total burden as chloroform breath concentrations dropped very rapidly only after one minute during the post-exposure period in a chloroform-free environment. This suggests that the "pooling" of chloroform in the blood is slight, and both the dermal and inhalation routes are of similar value. While increases in minute ventilation during exercise enlarge the potential for uptake of chloroform during exposure, it has been suggested that skin permeability also increases due to elevated body surface temperature during the same period, perhaps to an even greater magnitude than inhalation (Aggazzotti et. al., 1993). A subsequent study may be designed to determine the actual contribution of dermal absorption. Subjects could be tethered and supplied chloroform-free air while swimming and compared to tethered subjects who breathed pool air.

Heartrate (HR), VO2, and ventilation (VE in BTPS and STPD) data were taken throughout the exposure period as a descriptive value and to maintain a steady-state level of exercise intensity. The HR for the male subject ranged from 129 beats per minute to 146 beats per minute immediately after exercise intervals (Graph 4.3, Appendix IV).

The VE data were taken at approximately 25, 50, 75, and 110 minutes, immediately following the corresponding exercise interval. VE ranged from 47.14 to 58.4 liters/minute BTPS, with an average value of 52.96 liters/minute, or from 38.19 to 46.09 liters/minute STPD, with an average

value of 41.79 liters/minute. The slight decrease in ventilation rates during the latter part of the two-hour exercise period may be attributed to a slightly lower exercise intensity due to longer intervals.

VO2 was analyzed at the same intervals as VE. VO2 for the male subject ranged from 1.6 to 1.93 liters/minute (Graph 4.5., Appendix VI).
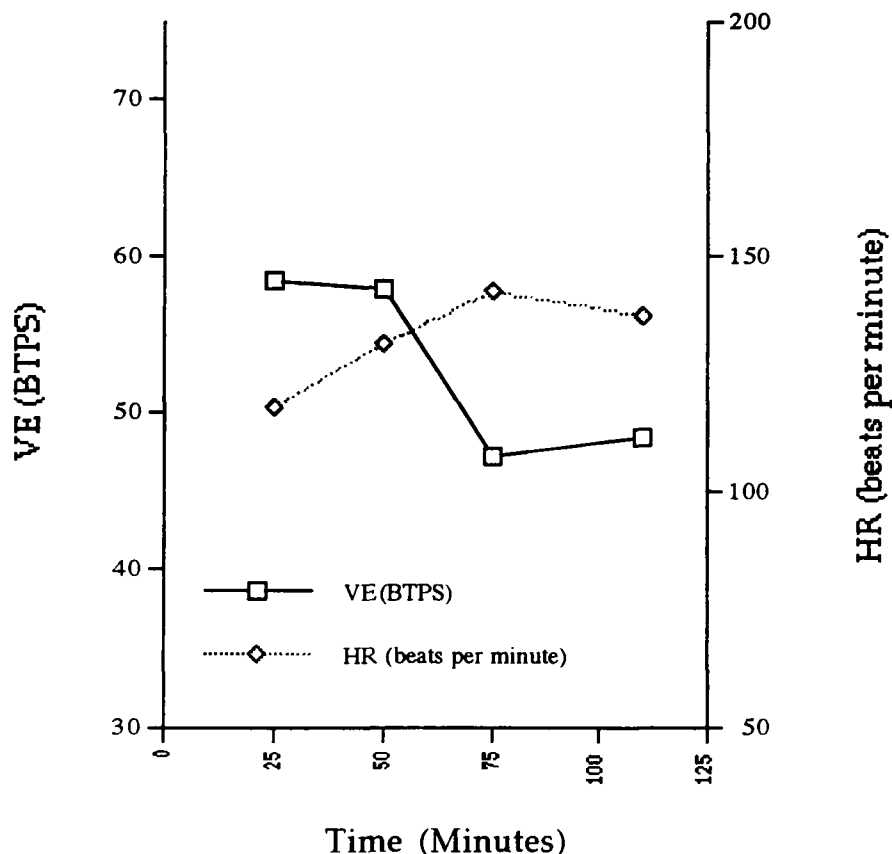
Graph 4.3. VE and HR of the male subject at selected intervals during exercise.

Female Subject

Results of the female subject generally corroborate those results found with the male subject. Samples of expired breath were taken on the intervals described in Chapter III. While extensive sampling during exercise was conducted with the male subject, only two samples were taken during

exercise with the female subject. Chloroform concentration for the female subject pre-exposure sample was 3.46 μg/m^3. A sample at 65 minutes showed that breath chloroform concentrations had increased to 220.35 μg/m^3. Breath chloroform concentrations increased at 2 hours to 339.91 μg/m^3 (Graph 4.1.).

Post-exposure sampling for chloroform followed the same schedule as the male subject. Breath concentrations of chloroform dropped rapidly from 217.29 μg/m^3 at one minute to 67.32 μg/m^3 at 30 minutes, and then more gradually to 18.90 μg/m^3 at 180 minutes. Again, while breath chloroform concentration did drop rapidly at first and then more slowly after 30 minutes, no double-peaked curve occurred representing a separate elimination of chloroform from a faster inhalation and latter dermal pathway in the model graph (4.2.).

Data suggests that uptake and elimination of chloroform were very rapid and not dependent upon route of exposure. Data from expired breath of the female subject also supports past assertions by Jo et. al., 1990 and Brown et. al., 1984, that the dermal route of exposure contributes at least half the total exposure burden.

The HR, VO2, and VE data were taken during the exposure period as shown in Chapter III. The heartrate of the female subject ranged from 149 to 182 beats per minute immediately following exercise intervals. Differences in heartrate from the male and female subject may have been due to relative physical condition or individual differences in exercise intensity. The VE data was collected at approximately 25, 50, 75, and 110 minutes. VE ranged from 58.24 to 63.8 liters/minute BTPS, with an average of 61.13 liters/minute, or 45.95 to 50.34 liters/minute with an average of 48.23 liters/minute STPD (Graph 4.4.).

VO2 for the female subject ranged from 1.94 to 2.26 L/ minute (Graph 4.5., Appendix VI). Heartrate ranged from 149 to 182 beats/minute with an average of 164 beats/minute (Appendix IV).
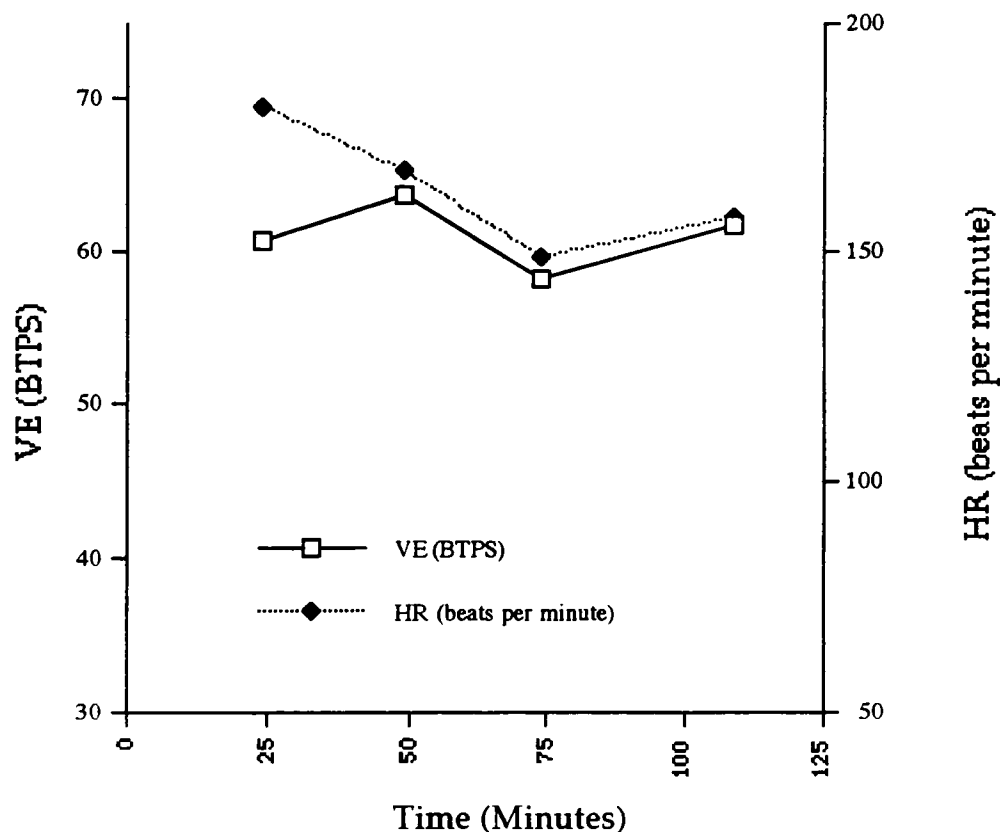
Graph 4.4. VE and HR data (female subject).

Quality Assurance

Greater than 13% of samples were tested in replicate with a mean relative difference of 5.3% (range 1.1 - 15.7%) for chloroform. A four-point calibration curve within the full range of exhaled breath values (0 - 400 $\mu g/m^3$) showed excellent linearity with an $r^2 > 0.999$. The limit of quantification (LOQ) chloroform was established using a signal to noise ratio criterion of 5 to 1, leading to an approximate LOQ of 1.7 $\mu g/m^3$. Analysis of

both field blanks showed chloroform concentrations below their respective LOQ's.
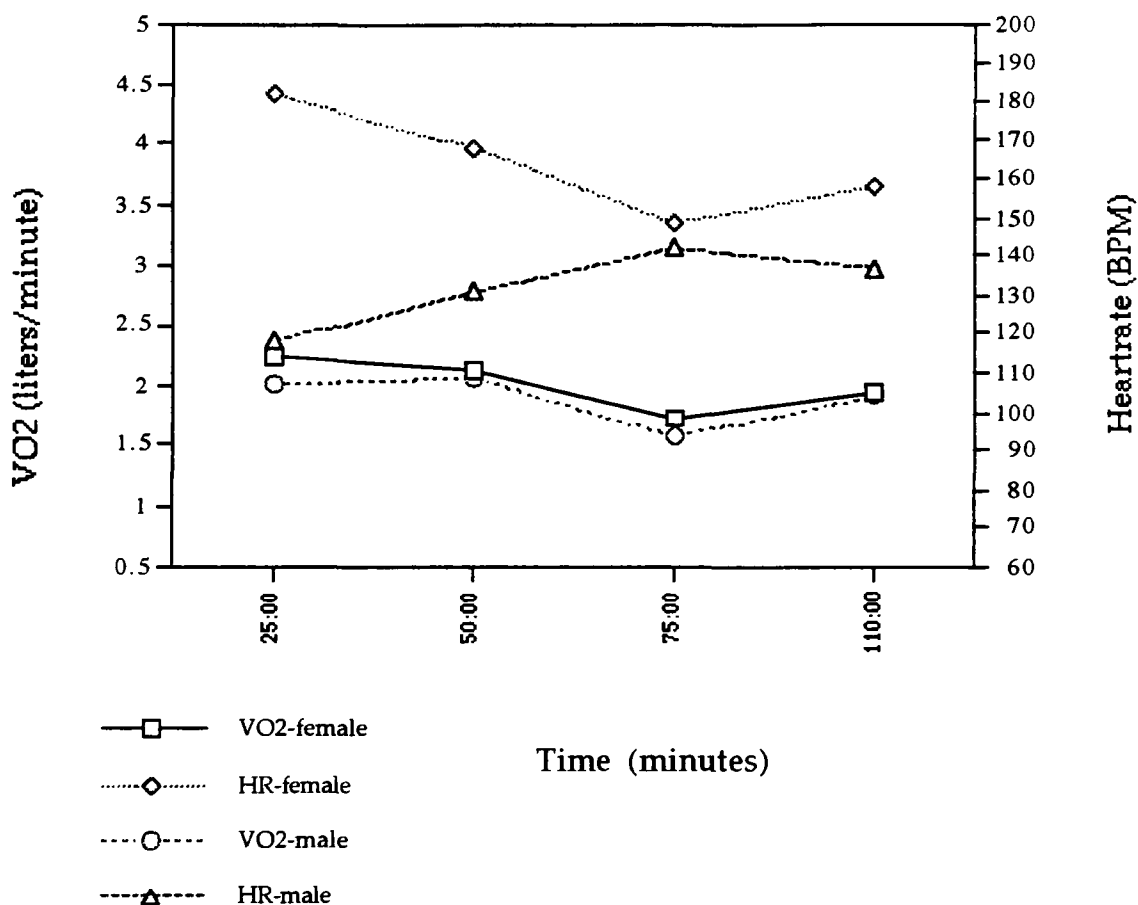
Graph 4.5. VO2 and HR (male and female subjects).

Quantification of Dose

Models to determine dose of chloroform exposure have been used by Jo, et. al., 1990 in showers, and by Lévesque et. al., 1994; Lahl et. al., 1981; Jo et. al., 1994; Wilson (1994); and Beech (1980) in swimming pools, albeit with large

discrepancies. Exactly how much of the total chloroform burden comes from the dermal, inhalation, and other incidental routes is also of some disagreement. Subsequently, estimates of total dose for swimmers exposed to chlorinated pools and the relative quantification of distribution of this burden from different pathways has ranged widely.

Lahl et. al., 1981 estimated a dose of 500 μg for a 70 kg person while assuming inhalation as the major route of exposure and ingestion of water as a secondary source. Beech estimated a worst-case dose of 2820 μg chloroform for a child during a three-hour swim looking at inhalation, dermal, incidental, and buccal and sublingual regions. Jo et. Al., 1994 found a dose of 82.7 μg for an adult swimming for one hour where 55.8 μg of the total dose arose from dermal absorption. Lévesque et. al., 1994 studied swimmers supplied chloroform-free air and suggested 77% of the total burden arose from inhalation while only 24% originated from dermal absorption for a total dose of 65 μg/kg or 4550 μg for a 70 kg person. Wilson (1994) used only inhalation and incidental pathways for determination for a dose of almost 70 μg/ hour (Table 4.3).

For the determination of dose for this study, the following equation was used based on a publication by Jo et. al., 1990 which examined chloroform burden in showering (Table 4.2). The use of the Jo et. al., 1990, equation was justified due to similar estimates in this study as to the relative contribution from dermal and inhalation exposure. The dose from inhalation and dermal absorption was assumed to be equal, as data from this study strongly suggested that dermal contribution to total chloroform burden was at least equal to, if not greater than, inhalation contribution to dose.

Inhalation dose (D_i) was calculated using an absorption efficiency (E) of .77 (US EPA standard, 1980), the pool air chloroform concentration (C) of 146 $\mu g/m^3$, ventilation rate in $m^3/min.$ (VE), and total time of exposure (T).

Table 4.2. Inhalation Dose Estimate.

$$D_i = E \times C \times T \times VE$$

or

$$D_i \text{ female } = 824.67 \ \mu g \ \text{(BTPS)}$$

$$D_i \text{ male } = 714.45 \ \mu g \ \text{(BTPS)}$$

or

$$D_i \text{ female } = 650.54 \ \mu g \ \text{(STPD)}$$

$$D_i \text{ male } = 563.76 \ \mu g \ \text{(STPD)}$$

As explained earlier, the exposure data in this study suggests that the total chloroform dose is at least twice that of inhalation alone. Estimated dose may actually be somewhat higher. Therefore by simply multiplying the calculated inhalation dose from above by two, the estimated dose for the 65 kg female subject would be equal to or greater than 1531.52 μg and 1428.9 μg (calculated using BTPS) for the 70 kg male subject.

These calculations fall somewhere between the Lahl et. al., (1981) and Beech (1980) calculations for chloroform exposure in swimming pools, but are well below the estimate of Lévesque et. al., (1994) (see Table 4.3.). Differences in estimates from past authors may be related to pool temperature, pool facility ventilation efficiency, duration of exposure, pool water and air

chloroform concentrations, HR and VE, and the assumed contribution of different pathways of exposure.

Although these calculations for dose are only rough estimates, it can be inferred that some swimmers may be at an even greater health risk. Many elite athletes find themselves in pool water at elevated heart and ventilation rates for up to six hours per day, seven days per week. This may mean a daily dose three times greater than was estimated as a daily dose for the two subjects in this study.

Potential Health Risks

Wilson (1994) estimated cancer risk associated with chloroform exposure in swimming pools. His estimated dose of $0.92\mu g/kg/day$ was more than 100 times below the EPA maximum acceptable cancer risk of 1.0 E-04 (1/10,000) for chloroform (ASTDR, 1992). Wilson (1995) estimated that values in his study would have to be 100 times greater to create an unacceptable cancer risk according to EPA standards. In the study by Levesque et. al., (1994), where chloroform breath concentration were found to be 50 times greater than the Wilson (1995) study, an unacceptable cancer risk could arise when swimmers are exposed to the pool water for more than two hours. For example, using a linear equation, if swimmers were exposed to the pool environment described in Levesque et. al., (1994), the chloroform dose for a 70 kg person would be 18,000 μg per day.

The estimated doses of 825 and 714 $\mu g/hr$ (Table 4.2.) for the female and male subjects in this study, respectively, are also far below the EPA unacceptable cancer risk level (ASTDR, 1992). The potential for unacceptable cancer risks may arise when swimmers are in pool water periods over six hours per day. Pool exposure coupled with exposures associated with

showering, food products, and water chlorinated water consumption may create a much higher exposure level than is examined in this study. For example, if a person were to be exposed per day to 1500 μg through two hours of swimming, plus another 100 μg through food sources, 250 μg through water ingestion, and 100 μg of chloroform through showering, the total chloroform dose is closer to 2000μg/day. The dose of exposure may approach the EPA unacceptable cancer risk level if swimmers were to triple this daily dose. It should be noted, however, that the EPA standard for chloroform is a 1/10,000. This does not mean that there is no risk when daily doses are below the EPA standard, as oher carcinogens are given a 1/1,000,000 unacceptable risk of cancer incidence.

Table 4.3. Comparison of Dose Estimates.

Author(s)	Finding	Pathway Contribution	Dose (μg/2hr swim)
Wilson (1995)	.92 μg/kg/hr	No dermal burden	128.8
Jo et. al., (1994)	82.7 μg/hr	67% dermal burden	163.4
Weisel and Shepard (1994)	estimate	N/A	200
Lahl et. al., (1981)	estimate of 500 μg/hr	N/A	1000
Berkoff (1995)	20.4-23.6 μg/kg/2hr	at least 50% dermal	1429-1532
Beech et. al., (1980)	estimate 2820 μg/3hr	N/A	1880
Levesque et. al., (1995)	65 μg/kg/hr	77% inhal/24% derm	9100

Chapter V

Conclusions

The purpose of this study was to test subjects exposed to a chlorinated pool environment for chloroform during a typical two-hour swimming practice. Exhaled breath samples were collected during and after exercise and assessed for chloroform.

1. After exposure to pool water, the expired breath samples of both subjects showed a rapid increase of chloroform concentrations over a two-hour period.

2. Assuming that breath chloroform accurately reflects blood chloroform, the dermal contribution to the total exposure burden was estimated to be equal to or greater than the inhalation contribution alone.

3. The chloroform elimination data from both subjects suggests that chloroform is absorbed, both dermally and through inhalation, rapidly and immediately after the commencement of exposure. There was no evidence in this study that inhalation of chloroform was more rapid than the uptake of chloroform from dermal absorption.

4. An estimate of dose associated with a two-hour swim in this pool was 20.41 μg/kg for the male subject, and 23.56 μg/kg for the female subject. This estimate does not represent an unacceptable cancer risk as prescribed by EPA standards (ASTDR, 1992), although that risk will increase with increased duration of exposure.

While the potential for risks to long-term health associated with swimming pool exposures in this study suggest no problems according to the EPA standard of 1.0 E-04 for chloroform, this study only represents two case studies over a two-hour period. Many factors could change these results. Swimmers often endure longer exposures both daily and over a lifetime, and pools vary with respect to chlorine concentration, water temperature, air ventilation capacities, and number of bathers. Further studies should be conducted examining the nature of chloroform exposure and calculation of dose.

Associations of cancers and chlorinated drinking water ingestion suggest that persons who are exposed to chloroform in pool water are also at a similar and perhaps increased risk. Studies should be conducted examining the relationship between swimming over a number of years and rates of certain disease types. These kinds of epidemiological studies will help determine whether health risks ultimately arise from swimming in chlorinated pools.

It is likely that EPA standards for cancer risk are far too conservative in assessing risk associated with pool water exposure. Because increases in cancer risk are noted in long-term exposure to chlorinated drinking water (Morris et. al., 1986), it would seem logical that greater dose concentrations as seen in pool water exposure would result in increased cancer risk even if the values found in this and other studies falls below the EPA standard.

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Appendix I

Participant Information

Thank you for agreeing to particpate. This study will examine the nature of the body's uptake and elimination of chloroform, a quick-forming by-product of pool water chlorination and known carcinogen, during a typical swiming practice situation.

You will be asked to complete a two-hour swimming workout (6000 yards) where heart rate, minute-ventilation, and exhaled breath samples for chloroform detection will be collected. After the swimming session, you will move quickly to the Health and Human Performance Laboratory for three hours of post-exposure sampling.

Project staff will assist you and coordinate all sampling procedures. If you have any questions or concerns during the study, please ask any staff member. You will provided results after they are compiled in the coming months.

Things to remember:

1. Be at the UM Health and Human Performance Lab at 5:40 AM. Do NOT go the the Grizzly Pool. You will be giving one exhaled breath sample prior to exposure to the pool environment.

2. Bring a suit, towel, goggles, and a bag with your street clothes to the lab. You will not be able to go to the lockerrooms to change.

3. Please eat before the swim session. You will not be permitted to eat until the sampling is finished at 11:00 AM.

4. Bottled water will be provided.

5. Do NOT swim or shower in the 24-Hours prior to April 19.

6. Bring a book or homework for the three-hour post-exposure sampling time. There won't be much else to do but sit.

Appendix II

An Assessment of Chloroform Exposure
Associated with Competitive Swimmers
During a Two-Hour Swim Practice

Principle Investigator: David Berkoff, EVST

Coinvestigator: Dr. Brent Ruby, HHP

Introduction

Over the past few years many health-related studies have focused upon the effects of chloroform on the human body as a result of exposure to chlorinated tap water. Chloroform, a class B carcinogen, is a natural by-product of water chlorination. Organic materials present in water combine with free chlorine to produce chloroform. Although not the only chlorinated organic produced in the chlorination process, chloroform is the most suspect health concern due to its relatively high concentrations and volatility.

Bove, et al. (1992), found that long-term exposure to chlorinated tap water sources produced an increased occurrence of birth-related defects. Many other studies have shown increased incidences of bladder and colorectal cancers associated with chlorinated tap water use (Morris et al. 1992).

Recently, studies have shown that chloroform is absorbed through the skin and lung tissue during showers in equal or greater quantity than is thought to be absorbed through the consumption of water alone (Jo et al. 1990). It was this discovery that prompted the U.S. Environmental Protection Agency to rethink its safety standards for chloroform in municipal water sources.

While drinking water has produced the most attention in the effort to improve safety standards, pool water chlorination goes virtually unchecked. In pool environments, microbial safety and not chemical safety is of greatest concern. However, pool chlorination is very similar because chloroform levels are produced at levels equal to or above that of chlorinated drinking water (Beech et al. 1980).

To further this concern, swimmers are often exposed to chloroform for several hours at a time, while the typical tap water exposure lasts only several minutes. Swimmers also often have elevated skin temperatures, heart rates, and breathing rates. All of these physiological factors may contribute to the significant intake of chloroform. Weisel and Shepard (1994), measured exhaled breath chloroform concentrations and conservatively estimated that swimmers exposed to pool water for one hour three times per week were being exposed to chloroform more than three times that of people using tap

water for drinking purposes alone. For many elite swimmers, exposure in pools may reach twenty-five hours per week.

While immediate health-related problems associated with swimming in chlorinated water are not significant, the long-term health effects due to chloroform exposure could be large.

As a competitive swimmer who spent almost three entire years of my twenty-eight year life submerged in chlorinated pools, it is of great concern to me and my colleagues to determine the level and significance of exposure that swimmers have to chloroform in pool environments.

The purpose of this study is to examine the nature of chloroform uptake in two competitive swimmers over time by sampling exhaled air during a simulated two-hour swim practice. In addition, the nature of chloroform elimination from the body will also be measured in order to estimate the average dose of exposure.

Benefits

This study will provide important data as to the nature of chloroform uptake a competitive or recreational swimmer may experience during exposure to a chlorinated swimming pool. From such data an assessment of levels of exposure to chloroform can be made. Because millions of people worldwide swim regularly, the results of this study could have far-reaching value.

Beside the knowledge and increased awareness of potential hazards from swimming in chlorinated pools, there will be no direct benefit to the subjects.

Methods

Two subjects will be recruited for proficiency in swimming and current physical fitness. Subjects will be University of Montana students ranging in age from 19 to 24 years and will have at least five years experience in competitive swimming. All subsequent testing will be conducted at the University of Montana Grizzly Pool and Health and Human Performance Laboratory (HHP lab).

After being assessed as physically fit to participate with the study, subjects will be asked to participate in a single experimental trial in which they will swim at an easy to moderate level of physical exertion for two hours (7100 yards) during which they will be monitored for heart rate and minute-ventilation. Because the experimental time of swim and distance are typical of many competitive swimming practices, chloroform levels in the expired air samples of subjects should roughly represent that of most swimmers who are exposed to chlorinated pools for extended periods of time. After completion of the swim, subjects will be asked to move to the chloroform-free HHP lab and give non-intrusive exhaled breath samples at set time intervals for three hours. Three hours is necessary for sampling of exhaled breath in order to see substantial removal of chloroform from subjects via

different routes of exposure and body compartments: inhalation, dermal, and deep tissue penetration.

Each sample will be taken by using a one liter evacuated stainless steel SUMMA® canister. Canisters are hand held, easily opened and closed, and have a gentle pull as to simulate passive exhaled breath and should cause no discomfort to the subjects (see diagram). Canisters will be pre-marked as to limit confusion during sampling.

Samples will be shipped to the Atmospheric and Exposures Laboratory at the U.S. Environmental Protection Agency in Research Triangle Park in North Carolina for analysis by gas chromatograph/ mass spectrometry.

Results will be compiled by David Berkoff and an explanation of results and copy of any subsequent presentations or publications will be given to the subjects.

Risks

No risks and minimal discomforts are anticipated during the exercise portion of this study. Any risks and discomforts associated with exercise will be minimized by recruiting highly trained subjects.

Use of non-intrusive SUMMA® containers will cause no discomfort. These sampling devices are small, lightweight and use is easily mastered after one attempt.

Subjects will be asked to not eat during or after the study time of five hours. Ingestion of foods has been noted as a source of chloroform and this could invalidate any data collected. Subjects will be given chlorine-free water to drink during the post-exercise sampling period if desired.

Confidentiality

All results and data from this study will remain confidential. Presented and published results will not include information regarding the two subjects and will be coded in order to protect individual confidentiality.

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Appendix III

<u>Written Consent and Disclaimer</u>

Chloroform is a natural by-product of the water chlorination process. However, long-term exposure to chloroform has been linked to several adverse health problems including cancer and birth defects. This study involves the measuring of chloroform uptake during swimming in a chlorinated pool and its subsequent elimination from the body via exhaled breath. A determination of average dose may be found using an elimination curve. Results from this study may help determine what level of exposure competitive swimmers experience during their careers and what health risks may be associated with competitive swimming.

As a participant, you will be asked to give several exhaled breath samples during a two-hour swimming period and a three-hour post-exposure period using stainless steel single breath canisters. Samples are taken easily by inserting a teflon tube into one's mouth, turning a release valve and exhaling easily. Once the canister vacuum stops, the valve can be closed again. No discomfort is known to be associated with the use of sampling canisters.

The two-hour exercise period will involve 7100 yards of swimming with intermittent sampling. Participants will be asked to be in proper physical condition to be able to adequately handle the swimming portion of the study. As a result of improper physical readiness, the subject may experience fatigue or may fail to complete the swimming portion of the study.

Subjects will be asked to wear a heartrate monitor and submit to ventilation rate tests during the swimming portion of the study. Subjects will experience no discomfort during the ventilation or heartrate tests.

Individuals trained in exercise physiology, CPR, first aid, and safety training will conduct the tests. If you should experience discomfort during the test, you may stop participation immediately. Any questions should be directed to David Berkoff, Project Director at 549-8431, or to Dr. Brent Ruby, Director of the Human Performance Laboratory in McGill Hall Room 121.

"In the event that you are injured as a result of this research you should individually seek appropriate medical treatment. If the injury is caused by the negligence of the University or any of its employees, you may be entitled to reimbursement or compensation pursuant to the Comprehensive State Insurance Plan established by the Department of Administration under the authority of M.C.A., Title 2, Chapter 9. In the event of a claim for such injury, further information may be obtained from the University's Claim Representative or University Legal Counsel."

I have read the above statements, and thoroughly understand all of the risks involved in participation with this study. I authorize David C. Berkoff and all assistants to administer and conduct this study as safely as possible and with minimum discomfort.

Signature of participant_____Date_____

Project Director_____Date_____

Appendix IV

Heart Rate Data (bpm)

Interval (minutes)	Male Subject	Female Subject
10	131	172
15	139	170
20	130	177
25	118	182
30	140	175
35	141	179
40	135	161
45	137	176
50	131	168
55	137	170
60	129	164
65	133	159
70	139	161
75	142	149
80	140	155
85	143	167
90	138	163
95	141	160
100	142	170
105	146	163
110	137	158
115	135	161
120	130	160

Appendix V

Minute Ventilation Data

Male Subject (BTPS)

Time (min.)	23:00	48:00	73:00	108:00
L/minute	58.4	57.9	47.1	48.4

Female Subject (BTPS)

Time (min.)	24:00	49:00	74:00	109:00
L/minute	60.7	63.8	58.2	61.8

Male Subject (STPD)

Time (min.)	23:00	48:00	73:00	108:00
L/minute	46.1	45.9	38.2	39.5

Female Subject (STPD)

Time (min.)	24:00	49:00	74:00	109:00
L/minute	47.8	50.3	45.9	48.7

Appendix VI

VO2 Data (L/minute)

Male Subject

Time (min.)	23:00	48:00	73:00	108:00
VO2 (L/min.)	2.01	2.07	1.57	1.92

Female Subject

Time (min.)	24:00	49:00	74:00	109:00
VO2 (L/min.)	2.24	2.13	1.73	1.96

Disposable Teflon
Mouthpiece →

30 cm

1 Liter Volume
SUMMA® Canister
and Valve →
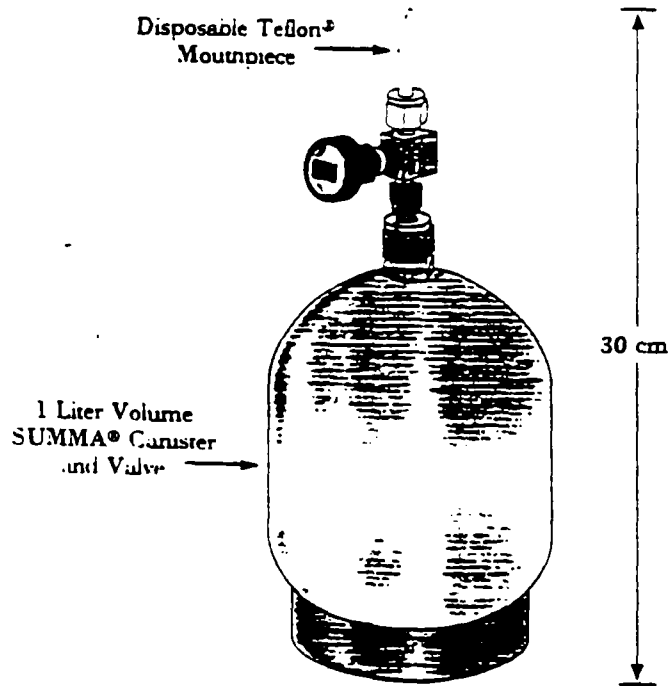
Summa® SBC with disposable teflon mouthpiece (From: Pleil and Lindstrom, 1994).

Subject using SUMMA® SBC. (From: Pleil and Lindstrom, 1994).