Appendix C Chemical Mixtures Exposure

Appendix C

Chemical Mixtures Exposure

General comments about chemical mixtures

Environmental chemical research has mainly centered on toxicity testing and mechanistic studies of single chemicals. This research lead to a better understanding of the interactions of exposure and susceptibility. However, ATSDR recognizes that humans are often exposed to multiple chemicals. Knowledge based on individual chemical exposure and toxicity is often a limiting factor in the human health assessment process. While interactions among some chemicals have been demonstrated at high concentrations, interactions at low environmental levels have not been scientifically demonstrated. Predicting whether chemicals will act in a potentiating, additive, synergistic, antagonistic, or independent manner at environmental concentrations or in the workplace has limitations.

Chemicals mixtures are found in the air we breathe, the food we eat, and the water we drink. With over 80,000 existing chemicals and 2,000 more being added each year, people are exposed to thousands of chemicals in different combinations every day in the home, the environment, and the workplace. Some of these chemicals have similar mechanisms of action or affect the same organ or tissue, so interactions between these chemicals are possible.

Chemical mixtures may contain two or three chemicals of a similar class or more complex mixtures may contain hundreds of chemicals from different classes. These chemicals may express different levels of toxicity and different modes of action. Changes in one chemical caused by another may alter the resultant toxicity from predicted values. Though changes in toxicity have sometimes been described for simple mixtures, understanding the interactions of complex mixtures has not been achieved.

Individual testing of the endless number of potential combinations is virtually impossible. Even if cost were not considered, the number of animals required to perform statistically relevant toxicity tests with multiple doses for multiple exposure periods would be prohibitive. An experiment investigating three chemicals at five different dose levels at only one time point after exposure, would require 125 treatment groups and 750 animals, if only six animals are included in each treatment group. Therefore, it is unlikely that questions concerning chemical mixtures will be answered through traditional animal research in the near future.

Interactions between chemicals can be potentiating, additive, synergistic, antagonistic; or there may be no interaction and thus, independent. ATSDR evaluates the potential for chemical mixtures on a site-by-site basis. ATSDR assumes that chemicals act independently if they have different modes of action, but additively if the modes of action are the same or effects are on the same target organ, unless there is evidence of interaction between the chemicals. For non-cancer effects, ATSDR assumes that a threshold exists for health effects. For cancer effects, ATSDR assumes that a threshold may not exist for genotoxic chemicals.

ATSDR's approach for the assessment of exposure to chemical mixtures included

- (1) Evaluating cumulative exposures by summing the individual risks for each carcinogen in the absence of compelling evidence supporting a greater than or less than additive model. This method of addressing cumulative risks has been externally peer-reviewed and found to be appropriate and relevant [55]. Under this response addition model, the predicted response to the mixture would be simply additive. This model assumes the contaminants act independently. For past exposures to the maximum level estimated by ATSDR air dispersion modeling, the cumulative risk from individual chemical exposures is considered to be a *low* increase in the risk for developing cancer. Almost all of the estimated risk was due to 1,3-butadiene, benzene, and formaldehyde exposure. A cumulative risk of 4E-04 was estimated by summing individual risks (Table C-1). Actual risks would likely be considerably less than this estimate due to the conservative nature of the assessment using a worst-case emissions scenario and continuous lifetime exposure to maximum average annual concentrations, and assuming additive toxicity.
- (2) Evaluating the evidence for potential interactions among the contaminants.

ATSDR investigated several approaches to evaluate interaction and concluded that scientific information was insufficient to compare the chemical mixture as a whole mixture, a similar mixture, or a component mixture. Epidemiological evidence of interaction involving 1,3-butadiene and benzene is inconclusive but recent evidence suggests independent action [56]. Evidence of interaction between formaldehyde and benzene or 1,3-butadiene was not located and formaldehyde appears to exert toxic effects by a different mode-of-action.

In assessing air emissions for potential interactions of chemical mixtures, ATSDR considered potential effects from the co-exposure of benzene and 1,3-butadiene. They were selected because these chemicals:

- represented the greatest estimated risk, considering quantity and toxicity,
- included the same organ system (bone marrow) as a target for carcinogenic effects, and
- epidemiological investigations of workers have reported confounding exposures to chemical mixtures.

Potential interactions between benzene and 1,3-butadiene have not been studied. Occupational exposure to high levels of benzene or 1,3-butadiene have been associated with the development of leukemia [57–59]. The metabolism of benzene and 1,3-butadiene appears to be similar in laboratory animals, with both chemicals metabolized primarily in the liver by the P450 family of enzymes (principally by the P450 isozyme 2E1 at these concentrations) [60–63]. Like benzene, 1,3-butadiene is metabolized to reactive metabolites but the precise mechanism is unknown [64]. Evidence indicates that the same metabolites detected in laboratory animals will be formed in humans, although the rates may be different [65]. Which metabolite(s) is responsible for the

causation of cancer is still uncertain. Differences in measured concentration levels in mice and rats do not explain the differences in cancer in these species. All three metabolites are mutagenic *in vivo* and *in vitro*. Based on the overall evidence from human, animal, and mutagenicity investigations, EPA concludes 1,3-butadiene to be a known human carcinogen [29].

Benzene is a known human carcinogen in humans while 1,3-butadiene shows clear evidence of carcinogenicity in animals and more recent evidence suggests stronger carcinogenic potential in humans [56]. While occupational exposure to high concentrations of benzene is known to increase the risk for developing non-lymphocytic leukemia, high doses of 1,3-butadiene have been associated with cancers at multiple sites in laboratory animals, including hematopoietic cancers such as lymphocytic leukemia [66]. Epidemiological studies suggest that co-exposure to 1,3-butadiene, styrene, and benzene may be associated with leukemia whereas exposure to 1,3-butadiene alone may be associated with lymphosarcomas [29]. Evidence of an association with high doses of 1,3-butadiene and leukemia in occupational studies is often confounded by co-exposure to other chemicals. The strongest evidence for the carcinogenicity of 1,3-butadiene in humans has occurred during co-exposure to styrene and benzene [56].

Occupational studies are evaluated for the relevance of the effect and the chemical mixture. Relevance is evaluated by assessing the temporality, strength of association, consistency, specificity, dose response, and biological plausibility. The recent University of Alabama-Birmingham (UAB) study was found to be particularly relevant to exposures of 1,3-butadiene, styrene, and benzene [56]. The UAB study investigated styrene and benzene exposures as well as 1,3-butadiene and concluded that the observed associations of leukemia with 1,3-butadiene exposure were not due to confounding exposures to the other chemicals. The authors conclude that exposures to 1,3-butadiene alone were associated with leukemia mortality. The dose-response analysis generated by the authors was used by ATSDR to compare to potential exposures around Kelly AFB. ATSDR compared the highest potential exposure period at Kelly AFB (before 1970) to the dose response of the UAB study.

The highest potential exposure period to benzene and 1,3-butadiene at Kelly would have occurred before 1970 based on operational information, type of jet fuel use, and air dispersion modeling of estimated emissions. Levels of 1,3-butadiene in the community are estimated to have been 20 µg/m³ (9 parts per billion [ppb]). The majority of housing in the communities was started in the 1950s which would equate to a maximum cumulative dosage of 180 ppb-years, assuming a 20-year exposure (1950–1970). Exposures after that time would be much less compared to the time period before 1970 because operations were significantly less (82,000 takeoff and landings/year compared to 330,000/year. See Appendix B). Exposures after 1970 resulted in a cumulative exposure dose of 54 ppb-years for the period 1970–1994. Kelly AFB changed from JP-4 jet fuel to JP-8 jet fuel in 1994. JP-4 jet fuel contained at least 100 times the benzene concentration measured in JP-8 jet fuel [3].

In the UAB cohort, the median cumulative exposure to 1,3-butadiene, styrene, and benzene was 11,200, 7,400, and 2,900 ppb-years, respectively. Among those dying of leukemia, the median cumulative exposure to 1,3-butadiene was 36,400 ppb-years, 200 times greater than the maximum

estimated annual average exposure at Kelly AFB (180 ppb-years). The UAB cohort consisted of workers, generally considered the healthiest segment of the general population.

Benzene is a known human carcinogen with the bone marrow as the primary target organ. Exposures to high concentrations of benzene have been associated with the development of leukemia, primarily acute non-lymphocytic leukemia (ANLL). Levels above 40 ppm-years are considered to increase the risk for developing leukemia in occupational exposures [27]. Occupational exposure (8 hours/day, 5 days/week, 50 weeks/year) to benzene at 40 ppm-years would be mathematically equivalent to a lifetime environmental exposure (76 years) of 120 ppb (384 μ g/m³). The estimated community exposure to past levels of benzene of 20 μ g/m³ for 20 years is equivalent to a lifetime exposure to 6 μ g/m³, or 64 times less than the lowest level of concern reported in epidemiological studies of occupational exposures. The estimated community exposure was also five times less than the level ATSDR considers as no apparent health hazard (32 μ g/m³) [54].

Occupational studies have reported an association with benzene or 1,3-butadiene exposure and leukemia mortality in workers. Exposure to levels of benzene and 1,3-butadiene estimated with limited data and air dispersion modeling to have been present in the community prior to 1970 would not be expected to result in leukemia mortality in healthy individuals. Susceptible members of the community may be at greater risk for developing hematopoietic perturbations than workers because

- These occupational studies were performed on workers with daily 8-hour exposures who died of leukemia. The potential health effects these same levels might have to more susceptible members of the general population, continually exposed to lower concentrations, is uncertain.
- These occupational studies do not identify the types of exposures which may have resulted in developing disease, as some individuals may be exposed to higher concentrations for shorter periods of time than others.
- These occupational studies reported mortality (death) from leukemia. It is not known if other workers developed disease or incurred reduced quality of life as a result of exposure.
- Scientific studies have not been performed on potential health effects from exposure to a chemical mixture of 1,3-butadiene and benzene.

Although increased risks for leukemia have been found in medical workers and other professionals exposed to formaldehyde, studies in industrial workers, who are thought to have higher exposures, have shown inconsistent associations [21, 22]. Some scientists have concluded that there is little likelihood that formaldehyde can induce toxicity at sites remote from the respiratory tract [67].

Summary

Estimated levels of past air exposures to benzene, 1,3-butadiene, and formaldehyde:

- are above some comparison values which are levels that ATSDR considers "safe," even to more sensitive populations. Exceeding a comparison value does *not* indicate that health effects would be likely, but indicates additional investigation may be warranted.
- are below levels associated with worker mortality from leukemia.

Cumulative estimated risks for past air exposures to benzene, 1,3-butadiene, and formaldehyde:

- are based on the addition of estimates of individual contaminant risks as interactions have not been demonstrated [56].
- results in an estimated low increase in the risk for developing cancer.

Epidemiological evidence indicates that:

- exposure to high levels of either benzene or 1,3-butadiene is associated with leukemia mortality in workers, but at levels much higher than estimates of past exposures of either contaminant at Kelly AFB.
- exposure to high levels of formaldehyde has been associated with leukemia in medical professionals and embalmers, but results of exposure in industrial workers has not been consistent.

ATSDR concludes that additional investigation is warranted because:

- 1. The community has been exposed to chemicals which have been associated with cancer in workers.
- 2. Confidence in the representativeness and comprehensiveness of the data is very low because most of the sampling and analytical data provided by Kelly AFB were collected before regulatory agencies began reviewing data. Exposure scenarios are also uncertain.
- 3. Health outcome data indicate that a biologically plausible health outcome, leukemia, was elevated (statistically significant) between 1990–1994 in three ZIP Codes, two of which were downwind and the third was off-base military housing.
- 4. Potential cumulative effects of chemical mixtures like 1,3-butadiene and benzene are unknown.

Table C-1. Estimated cumulative cancer risks from benzene, 1,3-butadiene, and formaldehyde exposure to stationary and aircraft historical air emissions.

Chemical	Scenario	Estimated Cancer Risk ^d			
1,3-butadiene	B52 human data ^a	4E-5			
benzene	B52 human data ^b	7E-05			
formaldehyde	B52 animal data ^c	3E-04			
Total Estimated Cumulative Risk 4E-04					

- a Cancer Slope Factor (4.3E-6/µg/m³) derived from human data [External Review Draft Health Risk Assessment of 1,3-Butadiene. US EPA. NCEA-W-0267. January 1998. National Center for Environmental Assessment. Office of Research and Development. Washington, DC.]
- b Cancer Slope Factor (7.8E-06/μg/m³) derived from human data [IRIS].
- c Cancer Slope Factor (0.000013/µg/m³) from animal data [IRIS]. No human data available.
- d All risk estimates are based on continuous 20 year exposures before 1973 and from 1973 to 1994 to estimated maximum annual average concentrations during each era. Level of operations was assumed to be 336,000 before 1973 and 112,000 1973 to 1994. Risks were summed for both eras.

Appendix D
Response to Comments from External Peer Review

Response to Comments from External Peer Review

1. Does the public health consultation adequately describe the nature and extent of contamination?

Reviewer 1

Comment: The consultation describes the contamination about as well as can be expected. Of course, we would all wish for better data. However, given the overall circumstances, the consultation does an excellent job.

Response: No response needed.

Reviewer 2

Comment: In Background, the authors explained the importance of past air emissions and considered contaminations in this consultation. Also, they described contaminants from industrial and aircraft emissions in pages 9 and 10. The characteristics, emissions, and known adverse health effects of those contaminants are explained in detail in Appendix B. This document adequately described the nature and extent of possible past contamination by activities at Kelly AFB.

Response: No response needed.

Reviewer 3

Comment: More description in the text including criteria or standard levels is needed. Measured data results would also be desirable. Discussion of impacts could be expanded. *Response:* The target audience for this document is the community. So as not to detract from the intended message to be communicated in the text, ATSDR puts as much of the detail as possible in appendixes.

2. Does the public health consultation adequately describe the existence of potential pathways of human exposure?

Reviewer 1

Comment: As with the previous question, the pathways of human exposure are fraught with uncertainty in this particular assessment. However, the consultation does an excellent job of describing the major concerns for the potential pathways.

Response: No response needed.

Reviewer 2

Comment: The authors described the potential exposure pathway elements in Table 1 in which only direct airborne exposures are described. Because toxic chemicals and organic carbons (both gaseous and particulate) can adhere to airborne particles and accumulate on them, the deposited and resuspended particles can act as an airborne vector for these compounds. This represents a potential exposure to residents downwind. Even though this indirect exposure seems to be beyond the scope of this consultation, it is worth noting it in the document.

Response: ATSDR agrees that deposition and resuspension may represent a pathway of concern. ATSDR chose to conservatively address the concern attributed to the potential

pathway by assuming the inhalation exposure represented 100% of the exposure from air emissions, not reducing the exposure to account for deposition. Direct 100% inhalation exposure would be a greater exposure than a portion of the exposure from inhalation and a portion of exposure from chemicals deposited.

Reviewer 3

Comment: Discussion of potential pathways could be expanded. Sources are defined well, but exposure is not directly addressed.

Response: This document is one of several documents prepared to assess environmental exposures at Kelly AFB. The potential pathways were discussed in the Phase I document. This document focused on past air emissions.

3. Are all relevant environmental and toxicological data (i.e., hazard identification, exposure assessment) being appropriately used?

Reviewer 1

Comment: I am not aware of all the potential sources of environmental data for this particular consultation. However, it appears to be a reasonable collection of data (albeit from a highly uncertain history), and the data appear to support reasonable conclusions. More specifically, when conservative assumptions about the data lead to the conclusion of "no significant risk," this is an appropriate use of data. This is a classic screening approach that is "good enough" to answer most of the questions being posed.

Response: No response needed.

Reviewer 2

Comment: The authors utilized industrial emission data, specific aircraft emission data, and detailed aircraft operational information supplied by Kelly AFB (Appendix B). The hexavalent chromium emission data, jet fuel misting information, and incineration emission data were not available. The model-estimated concentrations were compared with ATSDR chronic non-cancer data, worker exposure data, ATSDR cancer comparison data, and estimated cancer risk data. In pages 7 and 8, the authors clearly explained their selection of contaminants. All environmental and toxicological data were appropriately used for the consultation.

Response: No response needed.

Reviewer 3

Comment: Yes, this has been done but detail could be added.

Response: The target audience for this document is the community. As such the level of detail is deemed appropriate.

4. Does the public health consultation accurately and clearly communicate the health threat posed by the site?

Reviewer 1

Comment: The consultation does a good job in this area. However, the ATSDR should be prepared to answer the following questions.

Have other groups made estimates of the risks at Kelly Air Force Base? If so, how do they compare with this report?

Are there estimates of the risks at other Air Force Bases? If so, how do they compare with this report?

Response: Kelly AFB through the RCRA and Superfund programs have completed risk assessments on individual operable units and solid waste management units. These risk assessments, however, do not cover past air emissions.

The U.S. Army Center for Health Promotion and Preventive Medicine Environmental Health Risk Assessment Program (CHPPM), in response to our report, modeled past aircraft emissions using the Federal Aviation Administration's EDMS model [49]. ATSDR has recently been given a draft document. The report includes modeled ambient air concentrations from aircraft emissions but does not include calculations of cancer risk. CHPPM's predicted air concentrations from B52 emissions are within 10% of ATSDR predictions. The B52 was used as a worst case (largest emitter) to determine if further evaluation was necessary.

The CHPPM also predicted air concentrations from a "more realistic" fleet of aircraft which was not available to ATSDR at the time the work on this report was initiated. The results of this scenario and its assumptions need to be evaluated further.

ATSDR will consider the results of the CHPPM report when it becomes final.

ATSDR does not know of any other risk estimates from past air emissions from other Air Force bases.

Reviewer 2

Comment: The reviewer cannot judge the accuracy, but the authors clearly described and considered possible health threat posed by Kelly AFB in Appendix C. Also, they reported information on potentially susceptible populations (page 13).

Response: No response needed.

Reviewer 3

Comment: The threat is accurately described, but more text is required to clearly communicate the threat. Detail should not only appear in tables, but should be discussed. **Response:** ATSDR also provides fact sheets with presentations or availability sessions to clearly communicate messages and provide health education.

5. Are the conclusions and recommendations appropriate in view of the site's condition as described in the public health consultation?

Reviewer 1

Comment: Yes, overall they seem reasonable. However, see question #7 for further comments in this area.

Response: No response needed.

Reviewer 2

Comment: The authors' conclusions for the individual contaminations from stationary sources and aircraft sources are appropriate. Their recommendations of further studies on elevated leukemia outcomes and on-base exposure are relevant.

Response: No response needed.

Reviewer 3

Comment: Yes, I agree with the recommendations and conclusions.

Response: No response needed.

6. Given the available information, are the methods used suitable for determining the range of historic ambient air concentrations from aircraft emissions?

Reviewer 1

Comment: They are suitable given the fundamental uncertainty of the data. However, it may be useful for the reader to see a summary of all the assumptions that make ISCST a conservative model for this consultation. The assumptions and conservative nature of this model will be important in interpreting the results.

Response: The assumptions and methodology of the ISC modeling are presented in the discussion of Aircraft Emissions which includes 7 to 8 pages of text. A short summary of these assumptions were added as Table A-1.

Reviewer 2

Comment: All available data and operation information were utilized to estimate downwind concentrations in this consultation. The available air transport models were suitable used. While the authors mentioned ISCST model for the estimation of aircraft emissions, they did not clarify which model they used for the stationary emissions. From the reference (Rodgers et al., J.Exp.Anal.Env. Epi. 9, 535, 1999), the reviewer assumes simple Gaussian dispersion model was used. These steady state plume models (Gaussian model and ISCST model) make an important simplifying assumption, namely that there is no vertical variation in either the wind speed or turbulence intensity. They only consider the standard deviation of Gaussian distribution as dispersion parameters. This drawback often results in overestimations of gaseous or particulate pollutant concentrations (Winges KD USEPA/910/988/202/R, 1990; Kim, E. and Larson, T.V. Atmospheric Environment 35, 3509–3519). It can be one of the possible reason for high estimated concentrations in Tables B-1 and B-8. It looks to the reviewer that Figures B2 and B3 are identical, and Figures B4 and B5 are identical, even though the emissions are different between Butadiene and Benzene. It needs some clarification.

Response: The ISCST3 model was also used for the stationary sources. Appendix B was modified to include this information. The ISCST3 model uses a Gaussian distribution and the dispersion parameters of Pasquill-Gifford. The ISC model includes a variation of wind velocity (EPA 1995; EPA-454/B-95-003a available at

http://www.epa.gov/scram001/tt22.htm#isc) with height. The Fugitive Dust Model (FDM) described by Winges (1990) does not. As the reviewer points out, neither ISC nor the FDM vary settling velocity of particulates with height. For the modeling, ATSDR assumed all emissions were gaseous. ATSDR acknowledges the uncertainty of this assumption but it

presents a worst-case or highest exposure concentration scenario for the modeling of metals and organics.

ATSDR also acknowledges the inherent uncertainty in the ISCST3 model. Using a Gaussian-plume model, Rodgers et al. (J.Exp.Anal.Env. Epi. 9, 535, 1999), identify an uncertainty of approximately a factor of 2 in flat terrain (i.e., modeled concentrations range from ½ to 2 times the actual values). This range of uncertainty is relevant in this case because the terrain at Kelly AFB is flat. The Section titled "How did ATSDR evaluate past emissions at Kelly AFB" and Appendix B were modified to include this description of uncertainty.

We are aware that Figures B2 and B3 (predicted air concentrations of butadiene and benzene, from B52 emissions) and Figures B4 and B5 (predicted air concentrations of butadiene and benzene from F16 emissions) appear the same. This occurred because the emission rates of 1,3-butadiene and benzene are similar for the significant mode of operation. For the B52, modes contributing the predominant risk are startup, shutdown, and taxi. The engine setting during these modes is idle. The total emissions for the time the plane is using an idle engine setting are 2544 g/plane benzene and 2534 g/plane butadiene.

For the F16, the mode contributing the predominant risk is engine check and takeoff as seen by the higher concentrations at the ends of the runway. The engine check emissions are 204 g/plane benzene and 173 g/plane butadiene. The takeoff emissions are 305 g/plane benzene and 322 g/plane butadiene.

Reviewer 3

Comment: It would appear so, but more detail and better organization of presented data is needed.

Response: The target audience for this document is the community. As such the level of detail is deemed appropriate.

7. ATSDR identifies a range of risk estimates (identified in Table B-8) for potential past exposures to benzene and butadiene by utilizing the B52 aircraft as a worst-case and the F16 aircraft as a best case emissions scenario. From a public health perspective and considering the uncertainty, would modeling each individual aircraft emissions (more than 50 different types of aircraft) change the conclusions and recommendations?

Reviewer 1

Comment: There is no need to consider further data when the worst-case scenario leads to the conclusion that a public health concern is "unlikely." Lower exposures would yield the same conclusion, and the assumptions about the best and worst case seem very reasonable. This is screening assessment at its best.

The more provocative conclusion -- #3 on page 14 – is that the exposures "may have resulted in an increased risk for developing cancer." Given the uncertainties throughout this consultation (not just with the ISCST modeling), it may be more accurate to say that "the data are inconclusive with regard to cancer risks and follow-up is needed." Both statements may be true, but it is a strategic decision on which statement to use. The former statement

may evoke needless fear from the public. In my view, the later statement cannot be seen as overly optimistic, particularly since it calls for follow-up. However, the investigators who are closest to the community will make the most appropriate judgment on this issue.

In either case, follow-up with the Health Outcome Data Evaluation Health Consultation is still very appropriate.

Response: No response needed.

Reviewer 2

Comment: It does not seem to the reviewer that the conclusion and recommendations would change by modeling each aircraft emission. The number of operations was not specified for each individual aircraft. Therefore, the estimated concentrations downwind would be between the worst and the best case concentrations, if all model inputs are the same except source strength.

Response: No response needed.

Reviewer 3

Comment: Yes, the levels would decrease substantially.

Response: No response needed.

8. Are there any other comments that you would like to make about the health consultation?

Reviewer 1

Comment: No comment.

Reviewer 2

Comment: None.

Reviewer 3

Comment: Yes. Please see attached pages.

Response: ATSDR has responded to each comment in the following section.

9. Are there any comments on ATSDR's peer review process?

Reviewer 1

Comment: No comment.

Reviewer 2

Comment: None.

Reviewer 3

Comment: No.

10. Are there any other comments?

Reviewer 1

Comment: No comment.

Reviewer 2

Comment: Typo:

pp.72, please correct 'Figure B-8' to Figure B-9'.

pp. 40, 11th line of 4th paragraph, please delete 'and' in An individual's actual risk and may ...'

Response: These corrections were made.

Reviewer 3
Comment: No.

Reviewer 3 comments to question 8.

General Comments:

Comment: The main body of the text should include more detail on methodology, data, and maps. The reader finds considerable detail but often it is hidden in the footnotes of tables or is only included in the appendices. Conclusions seem to be valid, but a better presentation would lead to more reader confidence.

Response: ATSDR has adopted the present format on advice from health educators and risk communicators because the target audience of the health assessment is the general public, not the scientific community. Detail, whenever possible, is relegated to appendixes, footnotes, etc., to avoid distraction from the intended messages to the general public, but still included for the scientific readers.

Specific Comments:

Comment: Page 2, 1st Paragraph, Line 4: The sentence that begins "In an attempt" is slightly awkward and should be two sentences.

Response: The sentence has been restructured.

Comment: Page 3, 1st Finding: The text does not seem to agree with Table 1. Perhaps it should state that analysis of hexavalent chromium before 1980 was not possible. As stated, it seems to imply that hexavalent chromium from stationary sources did have adverse health effects.

Response: ATSDR has clarified the message.

Comment: Page 6, 1st Paragraph, Line 7: A conclusion of the study is presented in the background section with no support. Reasons should be given.

Response: The conclusion refers to current air emissions and the assessment and reasons for that conclusion are contained in that document.

Comment: Page 6, 1st Paragraph, Line 9: The reference to past air emissions (before 1995) is not supported. Earlier in the report (Table 1) past was also given as 1980. 1 think the text is

trying to tell me that only pre-1995 emissions were evaluated due to the change to JP-8, but I cannot be sure.

Response: The text states that pre-1995 air emissions were evaluated because of the use of JP-4 jet fuel before 1995. That point in time was necessarily used for all emissions even though chromium emissions changed in 1980. ATSDR initiated an investigation in 1996 and published a public health assessment (August 1999) addressing emissions from 1995 through base closure (2001).

Comment: Page 7, 2nd Paragraph, Line 1: I think the first sentence should be qualified by including "...at Kelly AFB." since this statement does not apply to the general literature. The next sentence stating distant past also has the same problem.

Response: This section applies to the general literature. For example, EPA's Air Toxics Monitoring Program began in the 1980s. ATSDR has modified the sentence to clarify.

Comment: Page 7, 4th Paragraph, Line 3: The sentence that begins on this line is confusing. It should be known if the chemicals were present or not.

Response: It is not known at what level the chemicals were present.

Comment: Page 9, 1st Paragraph, Line 6: Why include such a long listing of chemicals if it is not complete?

Response: This listing identifies the chemicals for which data was provided.

Comment: Page 9, 1st Paragraph, Line 8: If most chemicals did not exceed health-based comparison values the important information is which ones did. This should be reworded or stated. Then the following sentence says no chemicals exceeded the noncancer comparison values while the next sentence said two did. A clean up is needed in this paragraph.

Response: Health-based comparison values includes both cancer and noncancer comparison values. The text continues to specify that *noncancer* comparison values were *not* exceeded and two *cancer* comparison values were exceeded.

Comment: Page 9, 2nd Paragraph: This conclusion in bold font does not seem to agree with the previous paragraph.

Response: Exceeding a comparison value does not constitute a public health hazard, but identifies chemicals for which further evaluation is indicated.

Comment: Page 10, 1st Paragraph: The details on how much risk is involved and details should really be included in the main text and not only in the appendix.

Response: See general comments response.

Comment: Page 10, 2nd Paragraph: Again the conclusion in bold font does not seem to agree with the previous paragraph.

Response: Exceeding a comparison value does not constitute a public health hazard, but identifies chemicals for which further evaluation is indicated.

Comment: Page 12, 1st Paragraph: What health outcomes were further evaluated and why? This is important information.

Response: The information is given in following paragraphs.

Comment: Page 12, 5th Paragraph: The maps should be presented in the main body of the text and would really help the reader.

Response: Because there may be multiple references to the same maps, ATSDR elects to locate maps in one place to avoid duplication.

Comment: Page 12, 6th Paragraph: If results are available, why not include them here? **Response:** Results are not yet available.

Comment: Page 13: Nice discussion, but what is needed is a summary paragraph of how these issues directly apply to this project.

Response: While the information may be relevant to this project, its direct relevance remains to be determined by followup activities and is best presented by those investigators. For example, the association of acute nonlymphocytic leukemia in children and parental occupational exposures to benzene may have relevance to the ZIP Code containing off-base housing and reporting elevated leukemia outcomes. Further investigation would be needed and the information is best presented in its entirety at that time. A summary statement has been added.

Comment: Page 17, 2nd Paragraph, Line 4: Site topography and geometry are also key inputs. **Response**: "Site topography and geometry" have been added.

Comment: Page 17, 2nd Set of Bullet Items, 1st Bullet: "....24 hours a day for any time period..." is a little confusing.

Response: The text was clarified by stating that the models can be used to estimate a substance's concentration for different time periods for which both emissions and meteorological data exist and that the ISCST model used in this report generates an hourly model result. The hourly results can then be compiled to generate maximum and average values over different time periods.

Comment: Page 18, 1st Paragraph: If the concentrations were measured, they did exist. I think what you may be trying to say is that these results are not applicable to all times or can be used for other nearby locations.

Response: The interpretation is as intended and no response is needed.

Comment: Page 18, Last Paragraph, Line 5: The sentence ending on this line should include "...... at the modeled locations." This is true because further downwind longer half-lives do make a difference.

Response: The text specifies that the point is near the base perimeter and that downwind concentrations will increase and thus "do make a difference." The text was changed to indicate that the fixed point off base was at the modeled location at the base perimeter.

Comment: Page 19, Figure A-1: Why does the last data point (~1500 minutes) does the concentration go down?

Response: The last data point was in error. The concentration was incorrectly entered as $0.00117 \, \mu g/m^3$ and the correct value is $0.00122 \, \mu g/m^3$. ATSDR also ran the model with half-lives of 280 minutes and 2,160 minutes to fill in the time between 60 and 500 minutes and to extend the end

of the curve. These additional points confirm the conclusion that the concentration becomes stable at the location of concern as the half-life approached 3 to 4 hours (180–240 minutes). Figure A-1 has been corrected and revised. The geographic location is in the Kelly Gardens community immediately north of and adjacent to Kelly AFB. The location in geographic coordinates is 641,600 meters west and 4,173,700 meters north in statewide grid, Texas South Central Zone, North American Datum of 1983.

Comment: Page 19, 1st Paragraph, Line 8: Should this be a factor be less than 50? 1 don't get this number from Figure A-2.

Response: The factor of 50 was the result of the change in the model parameters (release height, downwash, and rural versus urban dispersion) and the decrease in concentration as a function of distance within 300 meters of the source. Because this was not apparent, the number has been changed to 3 to represent the change in model parameters only and a factor of about 50 depending on the receptors location inside the base boundaries.

Comment: Page 19: This would also be the place to introduce which models were used, what inputs, were worst case or typical weather values used, etc.

Response: ATSDR added this information.

Comment: Page 19, Caption for Figure A-2: Some parameters would have an effect. Those in the evaluation shown in the Figure perhaps have no effect.

Response: The caption for Figure A-2 has been changed to:

"Figure A-2. Input Parameter Comparison. Selection of model parameters shown in the Figure have no effect on off-base concentrations of contaminants, but may have significant effects upon on-base concentrations."

Comment: Page 21, Last Paragraph: Since hexavalent chromium is a solid, while the other pollutants are a gas, you may wish to mention modeling assumptions here, such as settling. **Response**: ATSDR added text to describe the assumption that all chemicals including metals were assumed to be in the gas form.

Comment: Page 22, 2nd Paragraph: More description of details would really help the reader. The levels that are used for comparisons (both criteria and measured) would indicate support for the statements made. The details do come out after a very careful review of Table B-1, but such important statements used in the paragraph should be supported in the text.

Response: See response to general comment.

Comment: Page 24, 1st Paragraph: Just a thought, but could the efficiency of the scrubber used for control be included and possibly allow better interpretation of impacts before 1980? **Response:** Scrubber efficiency was considered but ATSDR determined that the uncertainty was too large to evaluate further. The uncertainty exists because the operation of the scrubbers in Building 301 changed in 1980 and four other plating shops existed prior to 1980.

The text states that scrubbers were installed in 1980 in Building 301. Additional information obtained by ATSDR shows that the scrubbers were installed when the building was constructed in 1977 with stack sampling tests in 1980. The text was clarified with this information (Kelly

AFB 2001). The scrubbers on Building 301 were originally designed to operate in a wet mode. However, a memorandum indicates that insufficient deionized water was available to operate the units so decisions were made to operate the units in a dry mode (Backlund 1995). The stack tests were completed in 1980 in a dry mode. The actual efficiencies prior to 1980 are not known. Emissions can be estimated from plating operations by knowing the level of plating operations but these data has not been identified.

Four other plating shops existed prior to 1980. The most significant one is the operation in Buildings 258/259 which began operation in 1942 and shutdown in 1977 (Kelly AFB 2001). The Air Force considered the information on past emissions from Buildings 258/259 incomplete and of low confidence because the buildings were demolished prior to this inquiry, there is missing data, lack of confidence in personnel interviews, and lack of construction drawings (EARTTECH 2000a, 2000b). As a result of these uncertainties, ATSDR did not evaluate impact before 1980.

References

Backlund 1995. Memorandum for Information from SA_ALC/EMC, Department of the Airforce, Headquarters San Antonio Air Logistics Center (AFMC), Kelly Air Force Base, Texas (R.J. Backland, P.E.) To SA-ALC/EMC (D.S. Guadarrama, P.E.), Subject Shutdown of Wet Scrubber Mode at Bldg 301 and Stack Sampling).

Kelly AFB 2001. Point Paper for Chromium Emission Data from Historical Plating Operations, Kelly AFB, Draft, June 26, 2001.

EARTHTECH 2000a. Final Report. Historical Air Emissions Estimate. Kelly AFB, TX. EARTH TECH, Inc. San Antonio, TX. March 27, 2000.

EARTHTECH 2000b. Addendum to the Historical Air Emissions Estimate Report, March 20, 2000. EARTH TECH, Inc. August 28, 2000. Transmitted by Charles Williams (Kelly AFB) on December 20, 2000

Comment: Page 25, 3rd Paragraph: The model used is included, but an earlier introduction would be better for reader understanding. Also, how the model was used should be mentioned. Details are given later but the fact a volume approach using ISCST with estimated positions rather than using EDMS should be discussed.

Response: The following changes, shown in italics with accompanying text were made to address this comment.

Page 8 – First Paragraph

Data on JP-4 jet fuel speciation acquired by ATSDR and operational data provided by Kelly AFB were used to conduct an air dispersion model of aircraft emissions. A worst-case jet fuel emissions scenario was used for modeling aircraft emissions. *The Industrial Source Complex air dispersion model was used (ISCST3, see Appendix B for details).*

Page 25. Model Inputs The Industrial Source Complex-Short Term (ISCST) model was used to perform the air modeling. To use this model, information on the source of pollutants, ambient meteorology, and information on receptor locations must be entered into the model. The model simulates the movement of the pollutants in the atmosphere and calculates a concentration at the given receptor locations. *The emissions were treated as a series of volume sources behind the aircraft (see page 32 for details)*.

Bottom of Page 33 and Top of Page 34.

Forty-eight volume sources were used to represent taxiway emissions. Fourteen were used to represent takeoffs. Thirty were used to represent climbout. Eighty were used to represent approach. These sources represent aircraft movement at approximately 3-second intervals. Sources in each category were spaced according to their respective speed during that mode.

The commenter suggests that the EDMS model should be discussed in conjunction with the use of the ISCST model with volume sources. The ISCST model was used for modeling jet aircraft emissions using volume sources. The size and location of these volume sources were estimated and discussed in Appendix B. The use of the EDMS model at the time the modeling for this report was developed (March 2001 through June 2001) was not an option. In March 2001, version 3 was the current version of the EDMS model. EDMS version 4 was released in May 2001. The EDMS models (Versions 3 and 4) were developed for criteria air pollutants plus hydrocarbons (carbon monoxide, nitrogen oxides, sulphur oxides, hydrocarbons, and suspended particles). The EDMS V3 was not easily adaptable for other emission factors and chemicals including hazardous air pollutants that were the subject of this study. EDMS currently was not designed to perform air toxic analyses for aviation sources, but could have been supplemented with other air toxic methodology and models ([Federal Register: October 13, 1999 (Volume 64, Number 197)] [Notices] [Page 55525–55595]). The EDMS V4 model now has the flexibility to import the emission factors for new aircraft and additional chemicals. The EDMS V3 did not.

EDMS V3 used two models called PAL2 and CALINE3 that simulated aircraft emissions as line sources. CALINE3 is a line source model and assumes that a zone (volume) containing the line source exists with the zone. The size of the zone is a function of line width and an initial vertical dispersion. The contaminants in this zone then undergo vertical and horizontal dispersion using a steady-state Gaussian model (Benson 1979). PAL2 calculates a line source by integration of point sources (EPA 1978). The location accuracy of the points and lines representing the planes and the relative accuracy when compared to ATSDR's volumes sources is not known.

EDMS V4 is a significant revision of EDMS V3. EDMS V4 uses EPA's AERMOD air dispersion model. AERMOD in EDMS uses areas sources for aircraft taxiing, aircraft queuing, aircraft accelerating on the runway, aircraft after takeoff and during the landing approach. The area source was selected, as opposed to using a series of volume sources based on recommendations from the American Meteorological Society/EPA Regulatory Model Improvement Committee (AERMIC) (CSSI Inc. 2002). A comparison of the EDMS V4 model with ATSDR's approach is possible because the U.S. Army Center for Health Promotion and Preventive Medicine (CHPPM), in the fall of 2003, used the EDMS V4 to evaluate ATSDR's results. Using ATSDR's assumption of 336,000 operations of the B52H and the emission rates

identified in ATSDR's report, CHPPM's result at the point of maximum impact were within 10% of ATSDR's result (personal communication, Les Pilcher, US Army Center for Health Promotion and Preventive Medicine, December 19, 2003, [49]. This indicates that the different models using the same assumptions have good agreement.

References

Benson, Paul 1979. Abridged version of "CALINE3 - A Versatile Dispersion Model for Predicting Air Pollutant Levels Near Highways and Arterial Streets-Interim Report" Report Number FHWA/CA/TL-79/23, Nov. 1979. Paul E. Benson, Office of Transportation Laboratory, California Department of Transportation. Abridged Version by Computer Sciences Corporation [http://www.epa.gov/scram001/userg/regmod/caline3.pdf].

CSSI, Inc. 2002. Emissions and Dispersion Modeling System (EDMS) Reference Manual. Prepared for U.S. Department of Transportation, Federal Aviation Administration, Washington, D.C. Document Number FAA-AEE-01-01

US Environmental Protection Agency (EPA). 1978. User's guide for PAL, A guassian-plume algorithm for point, area, and line sources. Research Triangle Park (NC): Office of Research and Development, Environmental Sciences Research Laboratory, February. Report No. EPA-600/4-78-013.

Comment: Page 26, 5th Paragraph: Listing of all these aircraft would seem to be better in a table. *Response:* The list was revised as a table.

Comment: Page 26, 5th Paragraph: If only the B52H and F16 are to be used, why include that emission data exist for all of these other aircraft were found?

Response: The list of aircraft with available emissions data was provided to demonstrate the process ATSDR went through to reconstruct past exposures. The list more clearly demonstrates the advantages and disadvantages of the assumption to only use the B52-H and the F16. It also provides readers with knowledge of the data that is available. No change was made in the text.

Comment: Page 27, Table B-2: The numbers look too high. Could a B52H emit 113 kilograms of hydrocarbons during the taxi-out? This is true of Table B-3 as well. Also, if Touch & Go are not going to be used, why include?

Response: ATSDR checked the source document and it indeed lists 113 kilograms of hydrocarbons (HCs) during the taxi-out (Seitchek 1985). This number does seem high. It was derived based on time-in-mode, engine setting, and HC emission rate. The power setting for taxiout is idle which has the highest HC emissions rate. For the TF33-3 engine, the rate is 84 g HC/kg fuel. The fuel rate is 0.11 kg/s so the HC emission rate is 9.24 g/s. For 113 kilograms, the time-in-mode for taxi-out would need to be 3.4 hours which seems very unrealistic. ATSDR checked the KC-135A from this reference for taxi-out and came up with 11.5 hours which is even more unrealistic. ATSDR suspects a systematic error in Table A of Seitchek (1985). It's possible that the units for the table are kilograms and not metric tons. A note was added to Table B-2 about this possible error.

Because the values in Table B-2 were only used for a comparison among planes and not used in the emissions modeling, the error in Seitchek (1985) does not change our results. The hydrocarbon rates used in the modeling was 94 g HC/kg fuel and 0.14 kg/s of fuel (Spicer et al 1988). These values are similar to Seitchek (1985). The times-in-mode used in the ATSDR modeling was 9 minutes for taxi-out (Naugle et al 1975) for a total of 7.1 kg HC released during taxi-out.

References

Aircraft Emissions Characterization." C.W. Spicer, M.W. Holdren, S.E. Miller, D.L. Smith, R.N. Smith, D.P. Hughes. Final Report, March 1988, Engineering and Services Laboratory, Air Force Engineering & Services Center, Tyndall Air Force Base, ESL-TR-87-63.

USAF Aircraft Engine Emissions Estimator, Glenn D. Seitchek, ESL-TR-85-14, November 1985.

USAF Aircraft Pollution Emission Factors and Landing and Takeoff (LTO), Dennis Naugle, et al, AD/A-006 239 (February 1975)

Comment: Page 29, Equation at Top of Page: Derivation of this formula should be discussed. I get different answers when using moles for mass conversion.

Response: The equation is

The equation is

(1)
$$\% wt \frac{HAP}{HC} = \left(\frac{[HAP]}{[HC]}\right) \times \left(\frac{Number of C_{HC}}{Number of C_{HAP}}\right) \times \left(\frac{MW_{HAP}}{MW_{HC}}\right)$$

where:

[HAP] = concentration of organic compound in ppm_vC

[HC] = concentration of total hydrocarbons in ppm_vC

Number of C_{HC} = Number of carbon molecules, 9.3 is used for HC*

 $Number of C_{HAP} = Number of carbon molecules in the HAP$

 MW_{HAP} = Molecular Weight of the HAP]

 $MW_{HC} = Molecular$ weight of the total hydrocarbons = 130*

*Douglas, Everett, Naval Aviation Depot, Naval Air Station, San Diego, California. Email record of personal communication regarding information about converting units and data on the number of carbons and molecular weights of total hydrocarbons in jet fuel, February 12, 2001.

The units of concentration in this formula require ppm_vC. Moles should not to be used in this formula as it is based on a volume per volume basis. The formula was taken from AESO (1999) and the text will be referenced accordingly. There was a typographically error and OC was changed to HAP and the subscript "v" was added to indicate that it is based on volume not mass.

The derivation of the formula is based on two equations:

$$(2) ppm_{v} = \frac{ppm_{v}}{\#C}$$

where ppm_v = parts per million by volume ppmC = parts per million carbon #C = number of carbons in the molecule

and the ideal gas law,

$$(3) PV = nRT,$$

which is used to convert ppm_v to a mass basis.

Where P = pressure of the gas

V =the volume it occupies

T = its temperature (in Kelvin)

n = number of moles of gas present

R = universal gas constant

First, using the ideal gas law, the volume of 1 mole of air (V) is calculated.

(4)
$$V = R\left(\frac{L - atm}{{}^{o}K - Mole}\right) \times \frac{T({}^{o}K)}{1atm} = \frac{L}{mole}$$

At standard temperature and pressure (273°K and 1 atm). The volume is

(5)
$$V_{STP} = 0.8206 \left(\frac{L - atm}{{}^{o}K - Mole} \right) \times \frac{273^{o}K}{1atm} = 22.4 \frac{L}{mole}$$

ppm_v is defined as

$$(6) ppm_{v} = \frac{\mu L_{HAP}}{L_{air}}$$

where V_{air} is the total volume of air, V_{HAP} is the volume of air occupied by the HAP Using dimensional analysis and equations 5 and 6.

(7)
$$ppm_{v} = \frac{\mu L_{HAP}}{L_{air}} \times \frac{10^{3}}{1m^{3}_{air}} \times \frac{1L_{HAP}}{10^{6} \mu L_{HAP}} \times \frac{mole}{22.4L_{HAP}} \times MW \frac{g}{mole} \times \frac{10^{3} mg}{g}$$

or

(8)
$$ppm_{v} = \left(\frac{MW_{HAP}}{22.4L_{HAP}}\right) \frac{mg}{m^{3}}$$

Combining with equation 1 and solving for mass

(9) concentration in mg/m³ =
$$\frac{ppm_v C_{HAP}}{\# C_{HAP}} \times \left(\frac{MW_{HAP}}{22.4L_{HAP}}\right) \frac{mg}{m^3_{air}}$$

To obtain a weight ratio of HAP to hydrocarbon (HC)

(10)
$$\% wt \left(\frac{HAP}{HC}\right) = \frac{\frac{ppm_v}{\#C_{HAP}} \times \left(\frac{MW_{HAP}}{22.4 L_{HAP}}\right) \frac{mg}{m_{air}^3}}{\frac{ppm_v C_{HC}}{\#C_{HC}} \times \left(\frac{MW_{HC}}{22.4 L_{bC}}\right) \frac{mg}{m_{air}^3}}$$

Simplifying and rearranging brings us back to equation 1.

(11)
$$\% wt \left(\frac{HAP}{HC}\right) = \frac{\frac{ppm_v C_{HAP}}{\# C_{HAP}} \times (MW_{HAP})}{\frac{ppm_v C_{HC}}{\# C_{HC}} \times (MW_{HC})} = \frac{ppm_v C_{HAP} \times (MW_{HAP}) \times \# C_{HC}}{ppm_v C_{HC} \times (MW_{HC}) \times \# C_{HAP}}$$

Note that this equation is independent of temperature and pressure.

References

Aircraft Environmental Support Office (AESO), 1999. Toxic Organic Contaminants in the Exhaust of Gas Turbine Engines for JP-5 and JP-8 Fuel: Draft. San Diego (CA): U.S. Navy, Aircraft Environmental Support Office Naval Support Depot-North Island. AESO Report No. 12-90, Revision B. February.

Comment: Page 30, Continuation of Table: Headings should be included at the top of the page. **Response**: The heading was added.

Comment: Page 33, 2nd Paragraph: Do you mean Touch & Go operations were divided equally among takeoffs and landings?

Response: Touch and go operations were not specified in the number of annual operations. Since the annual operations most likely did include touch and go operations, ATSDR took the most conservative approach (highest emissions) and assumed that the unknown number of touch and go operations was a takeoff or a landing but not both. This means that the 336,000 operations were divided into 168,000 takeoffs and 168,000 landings. This text was added to the report for clarity.

Comment: Page 33, 9th Paragraph: "..... from about 480 meters." Does this mean to the ground, around this height, or something else?

Response: This meant that source release heights for approach varied from about 480 to 0 meters above ground. The text was modified for clarification.

Comment: Page 33, 11th Paragraph-. 46 minutes seems like a very long taxi time.

Response: The taxi time is the total time for taxi during takeoff and taxi during landing (see Table B-4)and includes time for startup (20 minutes), outbound taxi (9 minutes), inbound taxi (12 minutes), and idle at shutdown (4.8 minutes). This data was obtained from USAF Aircraft Pollution Emission Factors and Landing and Takeoff (LTO), Dennis Naugle, et al, AD/A-006 239 (February 1975). The text was clarified accordingly.

References

USAF Aircraft Pollution Emission Factors and Landing and Takeoff (LTO), Dennis Naugle, et al, AD/A-006 239 (February 1975)

Comment: Page 34: I expected information on the F- 16 to also be presented instead of just the B52H.

Response: This page does include information on the F16.

Comment: Page 34, 2nd Paragraph: It would be good to tell the reader what the level where health effects begin is and from what reference in this paragraph.

Response: See response to general comment and Figures B-6 and B-7.

Comment: Page 44, Figure B-7: The public may have the wrong idea of what is toxic when outdoor levels at Kelly AFB are reported to be above those in a smoked-fill bar. Some description in the text may help.

Response: ATSDR objectively presents information reported in the scientific literature to give a complete perspective.

Comment: Page 46: It is confusing to have two Appendix Bs. Perhaps B1 and B2?

Response: There is only one Appendix B, containing two attachments.

Comment: Page 55, 1st Paragraph, Line 3: The words "...near Kelly AFB." after the word "...susceptibility...." may be called for.

Response: This is a general statement not specific to Kelly AFB.

Comment: Page 55, 5th Paragraph: This paragraph should be included in main body of text. *Response:* ATSDR prefers that these general methodological statements remain with other like statements in an appendix than inserted into a discussion of findings in the main body of text.

Comment: Page 56, 1st Paragraph: How risk factors were developed is not completely

described. More detail would be helpful.

Response: More detail was added to the tables and text.

Comment: Page 59, 4th Bullet Item: Is there a low to moderate increase in risk or a low to moderate risk?

Response: There is a low to moderate increase in estimated risk over the background risk.

Comment: Page 60, Table C-1: That 6 people in 1000 would be at cancer risk is quite high. This needs more discussion in the text as does even 3 people in 1000.

Response: Clarification has been added to text.

Comment: Page 64, Reference 40: A much more recent reference exists.

Response: ATSDR agrees that a more recent reference exists; however, the data came from

Reference 40 because it was the document that was available at the time.

Comment: Page 66, 4th Paragraph: It would be good to report measured values.

Response: The text in this paragraph referred to concentrations reported from the air toxics monitor located about 10 to 15 miles northeast of Kelly AFB at 254 Seale Road, San Antonio, Texas. The concentrations are shown below. These values are annual maximum numbers. Detection levels were used when a compound was not detected. This table and the information about the monitor was not included in the revised report (response to peer review comments) because this data was provided for clarification purposes only and do not impact the results and conclusions on past air emissions.

	Bu	Butadiene		enzene	
Year	mg/m	Risk	mg/ m³	Risk	Total Risk
1994	0.17	4.73E-05	2.15	0	0.0000641
1995	0.71*	1.98E-04	1.63	1.27E-05	2.11E-04
1996	0.71*	1.98E-04	1.38	1.08E-05	2.09E-04
1997	0.71*	1.98E-04	1.76	1.37E-05	2.12E-04
1998	0.71*	1.98E-04	1.46	1.14E-05	2.09E-04
1999	0.74	2.07E-04	1.42	0	2.18E-04
2000	0.10	2.93E-05	1.09	0	3.78E-05
2001	0.11	3.15E-05	1.57	1.22E-05	0.0000437
Average	0.49	0	1.56	0	0.000151

Inhalation unit risk used for butadiene = $0.00028 \text{ (mg/m}^3)^{-1}$ Inhalation unit risk used for benzene = $0.000078 \text{ (mg/m}^3)^{-1}$

Comment: Page 67, 4th Paragraph: What was the logic for only using a 9 hour half-life? This doesn't appear to follow from the table above the paragraph.

Response: ATSDR concurs that the table and text are not clear. 1,3-butadiene is estimated to have a short atmospheric lifetime because of its reactivity. The actual lifetime depends upon the conditions at the time of release. The primary removal mechanisms are through chemical reactions with hydroxyl radicals and ozone. Therefore, factors influencing 1,3-butadiene's atmospheric lifetime, such as the time of day, sunlight intensity, temperature, etc., also include those affecting the availability of hydroxyl radicals and ozone (EPA 1993). The literature reports different half-lives and in many cases, do not specify controlling factors that would influence butadiene degradation. The Table was an attempt to show the half-lives as a function of a single factor.

For the modeling, ATSDR used a report by the California Air Resources Board that stated "[a]tmospheric half-lives of 1 to 9 hours are expected." (CARB 1997). This range was

^{*} Detection level used

reasonable to evaluate as 1 hour was near the lower end reported. Nine hours was reasonable to use as a higher value because it is in the range of the higher values. Higher half-lives would not significantly change the concentrations near the base where the population of interest resides because the travel of air emissions time is much faster than 9 hours (Figure A-1 demonstrated this for hexavalent chromium). The model was run with no degradation as a worst case.

ATSDR clarified this in the text and merged this discussion with text in Appendix B under a new section called *Sensitivity Analysis*.

References

EPA 1993. Motor Vehicle-Related Air Toxics Study, April 1993. Technical Support Branch, Emission Planning and Strategies Division, Office of Mobile Sources, Office of Air and Radiation, U.S. Environmental Protection Agency, Washington D.C.

CARB 1997. Toxic Air Contaminant Identification List Summaries, September 1997. Stationary Source Division, Substance Evaluation Section, California Air Resources Board, California Environmental Protection Agency, Sacramento, CA.

Comment: Page 69, Table B-8: Again, high risk values are reported and probably need to be discussed more.

Response: Clarification has been added to text.

References

- 1. ATSDR Public Health Assessment. 1999. Kelly AFB. U.S. Health and Human Services. Atlanta, Georgia. August 28.
- 2. Letter from Congressman Frank Tejeda to Dr. David Satcher, ATSDR. April 8, 1996.
- 3. Smith LB, Zelnick S. 1998. Benzene Exposure Study During Aircraft Fuel Tank Entry. International Conference on the Environmental Health and Safety of Jet Fuel. April. San Antonio, Texas.
- 4. Comment by USAF during public comment period. 1999. ATSDR Public Health Assessment. Kelly AFB. Final Report. Response to Comments. To be released Fall 2004.
- 5. Hansen H, De Rosa CT, Pohl H, Fay M, and Mumtaz MM. 1998. Public Health Challenges Posed by Chemical Mixtures. Environ Health Perspect. 106(6):1271–1280.
- 6. ATSDR. Cancer Policy Framework. January 1993.
- 7. Rogers, Felix, J., George G. Killough, Shirley J. Thompson, Cherly L. Addy, Robert E. McKeown, and David J. Cowen. 1999. Estimating environmental exposures to sulfur dioxide from multiple industrial sources for a case-control study, Journal of Exposure Analysis and Environmental Epidemiology. 9(6): 535–545.
- 8. Final Report. Historical Air Emissions Estimate. Kelly AFB, TX. EARTH TECH, Inc. San Antonio, TX. March 27, 2000.
- 9. Update to the Historical Air Emissions Estimate Report for Kelly AFB, March 27, 2000. EARTH TECH, Inc. June 6, 2000.
- Addendum to the Historical Air Emissions Estimate Report, March 20, 2000.
 EARTH TECH, Inc. August 28, 2000. Transmitted by Charles Williams (Kelly AFB) on December 20, 2000
- 11. Memo from CH2MHill to Capt. Brian Fitzgerald. Bldg. 348 Incinerator Background. June 1. 2000.
- 12. ATSDR. AROA. Telephone conversation. David Fowler (ATSDR) with Captain Brian Fitzgerald, Kelly AFB. June, 2000.
- 13. 76th Medical Operations Squadron. Potential Health Hazard Assessment of Fugitive Vapor Emissions from 1500 Area Fuel Tanks. Kelly Air Force Base. March 23, 1995.
- 14. Kelly Air Force Base Draft Programmatic Environmental Impact Statement.

 Disposal of Kelly Air Force Base, Texas. Prepared by U.S. Air Force and Science Applications International Corporation, January 1997.
- 15. Aircraft Noise in the Vicinity of Kelly Air Force Base, San Antonio Texas (Updated Report), March 1994. Prepared for the U.S. Department of Justice by Harris, Miller, & Hanson, Inc., Lexington, MA. HMMH Report No. 292610-B.
- 16. Spicer DW, Holdren MW, Smith DL, Miller SE, and Hughes DP. 1990. Aircraft Emissions Characterization, F101 and F110 Engines. Final Report, March. Engineering and Services Laboratory, Air Force Engineering and Services Center, Tyndall Air Force Base, ESL-TR-89-13.

- 17. Consultative Letter. Availability of Information Related to Unburned Fuel and Oil Misting Emissions From Aircraft Takeoffs and Landings. Air Force Institute for Environmental, Safety and Occupational Health Risk Analysis, Brooks AFB, Texas. AFIERA-RS-BR-CL-2001-0011. February 13, 2001.
- 18. Anderson, Bruce, Senior Research Scientist, Atmospheric Sciences Division, NASA Langley Research Center. Email record of personal communication regarding jet engine exhaust, February 1, 2001.
- 19. ATSDR. Toxicological Profile for Benzene. U.S. Department of Health and Human Services. September 1997.
- 20. ATSDR. Toxicological Profile for 1,3-Butadiene. U.S. Department of Health and Human Services. July 1992.
- 21. Hauptmann M, Lubin JH, Stewart PA, Hayes RB, and Blair A. Mortality from lymphohematopoietic malignancies among workers in formaldehyde industries. J Nat Cancer Institute, 2003; 95(21):1615–23.
- 22. Walrath J and Fraumeni JF Jr. Cancer and other causes of death among embalmers. Cancer Research, 1984; 44:4638–41.
- 23. Casanova-Schmitz M, Starr TB, and Heck H D'A. Differentiation between metabolic incorporation and covalent binding in the labeling of macromolecules in the rat nasal mucosa and bone marrow by inhaled [14C]-and [3H]formaldehyde. Tox Appl Pharm, 1984; 76:26–44.
- 24. Shaham J, Bomstein Y, Gurvich R, Rashkovsky M, and Kaufman Z. DNA-protein crosslinks and p53 protein expression in relation to occupational exposure to formaldehyde. Occup Environ Med 2003; 60:403–09.
- 25. ATSDR. Health Outcome Data Evaluation Health Consultation. Released for Public Comment. Division of Health Studies. Atlanta, GA. June, 2001.
- 26. Chemical and Radiation Leukemogenesis in Humans and Rodents and the Value of Rodent Models for Assessing Risks of Lymphohematopoietic Cancers. 1997. EPA National Center for Environmental Assessment. Office of Research and Development. Washington DC. EPA/600/R-97/090.
- 27. Carcinogenic Effects of Benzene: An Update. U.S. Environmental Protection Agency. National Center for Environmental Assessment. Washington DC. April 1998. EPA/600/P-97/001F.
- 28. Daugherty ML. Toxicity Summary for Benzene. 1992. Oak Ridge National Laboratory, Oak Ridge, Tennessee. September.
- 29. Health Risk Assessment of 1,3-Butadiene. National Center for Environmental Assessment. US. Environmental Protection Agency. Washington DC. External Review Draft. January 1998. NCEA-W-0267.
- 30. Melnick RL and Huff J. 1992. 1,3-butadiene: toxicity and carcinogenicity in laboratory animals and in humans. N.I.O.E.H. Sciences, ed., Reviews of Environmental Contamination and Toxicology, New York. Springer-Verlag, p 111–114.
- 31. The Training Manual on Pediatric Environmental Health. Accessed on 12/05/01. http://www.cehn.org/cehn/trainingmanual/manual-front.html
- 32. Buckley JD, Robison LL, Swotinsky R, et al. 1989. Occupational exposures of parents of children with acute nonlymphocytic leukemia; a report from the childrens' cancer study group. Cancer Res 49:4030–4037.

- 33. Cancer. Principles and Practice of Oncology. Fourth Edition. Edited by DeVita VT Jr., Hellman S, Rosenberg SA. J.B. Lippincott Co., Philadelphia. 1993.
- 34. Alexander FE, Patheal SL, Biondi A, Brandalise S, Cabera M-E, et al. Transplacental chemical exosure and risk of infant leukemia with MLL gene fusion. Cancer Res, 2001; 61:2542–6.
- 35. Rosvold EA, Mcglynn KA, Lustbader ED, and Buetow KH. 1995. Identification of an NAD(P)H:quinone oxidoreductase polymorphism and its association with lung cancer and smoking. Pharmacogenetics. 5:199–206.
- 36. Seaton, MJ, Schlosser PM, Bond JA, and Medinsky MA. 1994. Benzene metabolism by human liver microsomes in relation to cytochrome P450 2E1 activity. Carcinogenesis. 15:1799–1806.
- 37. ATSDR. Public Health Assessment. Final Report. Kelly AFB. To be released in 2004.
- 38. ATSDR Toxicological Profile for Methylene Chloride. September 2000. U.S. Department of Health and Human Services. Atlanta, Georgia.
- 39. ATSDR Toxicological Profile for Tetrachloroethylene. September 1997. U.S. Department of Health and Human Services. Atlanta, Georgia.
- 40. Joint Use Supplemental Environmental Impact Statement. Kelly Air Force Base. Prepared by URS Corporation. August 2000.
- 41. Kelly AFB. Official Pictorial History Book. Http://137.242.1.211/Historybook.htm [accessed 2/2/2001
- 42. U.S. Navy Fuels Comparison Chart. U.S. Naval Air Station, Fallon, NV.
- 43. USAF Aircraft Engine Emissions Estimator, Glenn D. Seitchek, ESL-TR-85-14, November 1985.
- 44. USAF Aircraft Pollution Emission Factors and Landing and Takeoff (LTO), Dennis Naugle, et al. AD/A-006239 (February 1975).
- 45. Joint Use Supplemental Environmental Impact Statement. Kelly Air Force Base. San Antonio, TX. August 2000.
- 46. Gale Research, 1980. Climates of the States. Volume 2: Ohio-Wyoming, Second Edition, Gale Research Company, Detroit, MI.
- 47. EDMS (The Emissions and Dispersion Modeling System) Federal Aviation Administration, Office of Environment and Energy, Washington, D.C., 1988.
- 48. USAF Aircraft Takeoff Length Distances and Climbout Profiles, D.F. Merricucci, October, 1974.
- 49. Aircraft Emission Estimate and Air Dispersion Analysis of Past Emissions-DRAFT, Kelly Air Force Base, November 6, 2003. U.S. Army Center for Health Promotion and Preventive Medicine Environmental Health Risk Assessment Program.
- 50. CARB 1997. Toxic Air Contaminant Identification List Summaries, September 1997. Stationary Source Division, Substance Evaluation Section, California Air Resources Board, California Environmental Protection Agency, Sacramento, CA.
- 51. Blair A, Saracci R, Stewart PA, et al. 1990. Epidemiologic evidence on the relationship between formaldehyde exposure and cancer. Scand J Work Environ Health 16:381–393.
- 52. Collins JJ, Acquavella JF, Esmen NA. 1997. An updated meta-analysis of formaldehyde exposure and upper respiratory tract cancers. J Occup Environ Med

- 39:639-651.
- 53. ATSDR Toxicological Profile for Formaldehyde. July 1999. U.S. Department of Health and Human Services. Atlanta, Georgia.
- 54. ATSDR Draft Guidelines for Assessment of Benzene in Air. 2000. Division of Health Assessment and Consultation.
- 55. ATSDR. External Peer Review Comments to Kelly AFB Public Health Assessment. August, 1999.
- 56. Macaluso M, Larson R, Delzell E, Sathiakumar N, Hobinga M, Julian J, Muir D, and Cole P. 1996. Leukemia and cumulative exposure to butadiene, styrene and benzene among workers in the synthetic rubber industry. Toxicology 113: 190–202.
- 57. Ott MG, Townsend JC, Fishbeck WA, et al. 1978. Mortality among workers occupationally exposed to benzene. Arch Environ Health 33:3–10.
- 58. Rinsky RA, Young RJ, Smith AB. 1981. Leukemia in benzene workers. Am J Ind Med 2:217–245.
- 59. Delzell E, Sathiakumar N, Hovinga M, Macaluso M, Julian J, Larson R, Cole P, and Muir DC. 1996. A follow-up study of synthetic rubber workers. Toxicology. 113(1–3):182–9.
- 60. Casarett and Doull's Toxicology. 2001. Sixth Edition. McGraw-Hill, New York. Ed: Klaassen.
- 61. Duescher RJ and Elfarra AA. 1994. Human liver microsomes are efficient catalysts of 1,3-butadiene oxidation: evidence for major roles by cytochromes P450 2A6 and 2E1. Arch Biochem Biophys. 311:342–349.
- 62. Valentine RL, Lee SS, Seaton MJ, Asgharian B, Farris F, Corton JC, Gonzalez FJ, and Medinsky MA. 1996. Reduction of benzene metabolism and toxicity in mice that lack CYP2E1 expression. Toxicol. Appl. Pharmacol. 141:205–213.
- 63. Norppa H and Sorsa M. 1993. Genetic toxicity of 1,3-butadiene and styrene: International Agency for Research on Cancer. Sci Publ. p.185–193.
- 64. ECETOC Working Group. 1993. 1,3-Butadiene Criteria Document. National Institutes of Health. TA:ECETOC Special Report PG:110. IP:VI:4.
- 65. Bond JA, Csanady GA, Leavens T, and Medinsky MA. 1993. Research strategy for assessing target tissue dosimetry of 1,3-butadiene in laboratory animals and humans. International Agency for Research on Cancer. Sci Publ, p. 45–55.5
- 66. National Toxicology Program. 1993. NTP technical report on the toxicology and carcinogenesis studies of 1,3-butadiene: (CAS no 106-99-0) in B6C3F1 mice (inhalation studies). National Toxicology Program, p 391.
- 67. Heck Jd'A, Casanova M, Starr TB. Formaldehyde toxicity new understanding. Toxicology 1990:20(6):397–426.