Health Effects of Air Emissions from Confined Feeding Operations

This report is the last draft assembled by the Health Effects subgroup,not a final version. The report represents the significant efforts of the subgroup and is presented for information only. Some of the information or interpretation contained in the report does not reflect the opinions or decisions of the Health Effects subgroup or the Confined Feeding Operations project team. Therefore, this report did not achieve consensus and does not have the sanction of the CFO project team

A report to the CASA CFO Project Team from the Effects Subgroup

DRAFT for discussion purposes only

October 17, 2007

Acknowledgements

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Purpose of this Report

The purpose of the Effects Subgroup of the Confined Feeding Operations Project Team was to provide credible science based information that would enable the CFO Project Team to decide on a strategic plan to manage the emissions from CFOs in Alberta. This report provides a range of information for use by the Project Team and recommends how the CFO team can move forward to address health effects in particular.

Contents

Tables and Figures

NOTE: A section with acronyms and abbreviations may be needed.

Executive Summary

The Effects Subgroup of the CASA Confined Feeding Operations (CFO) Project Team was charged with gathering information for the team on the health effects of ammonia, hydrogen sulphide/TRS, volatile organic compounds, particulate matter, bioaerosols, and odour emissions from CFOs.

The Subgroup reviewed and assembled a great deal of information about the effects of these emissions on human, animal and ecological health. At a high level, the subgroup found health effects information on all the substances, but in a number of cases, there were limitations on the studies that have been done; for example, environmental monitoring and clinical assessments were not performed, and confounding aspects (such as the comparability of the study populations) were not considered or weighed.

The subgroup found it challenging to determine what the association is, if any, between CFO emissions and public health effects. Views differ, even among experts and in the literature, and trying to compare and assess the impacts of different emissions from operations with different kinds and numbers of livestock, and different climate, management practices and other conditions is very challenging. One way to simplify the association between health effects and CFO emissions is to focus on the proximity to CFOs, but even with this approach there are many contextual variables, acting together, that determine whether effects will occur.

The subgroup noted that there is little research on the health effects of animals in CFOs as these effects relate to air quality. Studies included in this report were conducted in both laboratory experiments and in a typical CFO. Findings within a CFO may not be as accurate due to the difficulty of isolating and measuring particular gases. Although little research has been conducted formally, agriculture producers have refined and improved their practices over time based on their knowledge, experience and daily observations. For example, when decreased growth rate is noticeable and may be due to air quality, producers have added technology, such as ventilation and management practices such as removing manure from the barn, to improve air quality. Quality care for the animals is crucial to the sustainability of the livestock industry.

Conclusions and Recommendations

Health Effects from CFO Emissions

Human health studies that look at the effects of emissions from CFOs are more numerous than those on animal health and ecological health.

Conclusion 1: The subgroup considered many studies and agreed by consensus that there are indeed health effects from CFO emissions. More specifically, the subgroup agreed that [The weight of evidence from research studies reviewed by the team demonstrates an association between CFO emissions and public health effects in surrounding communities.][1]

Gathering More Information on Health Effects from CFO Emissions

Although many information gaps exist on health effects from CFO emissions, the Effects Subgroup agreed that the CASA team was not the place to conduct studies on health effects. The Effects Subgroup agreed by consensus that:

[1] Subgroup members agreed to test the statement in square brackets with their stakeholders to determine the level of consensus.

Although there is value in further research on health effects from CFO emissions, neither CASA nor the CASA process should propose or finance scientific health effects research, as such studies are very consumptive of time and money, and such information is being gathered and addressed through other processes (e.g., universities and governments).

Ambient Air Quality Objectives for Emissions from CFOs

All substances considered by the subgroup are covered by ambient air quality objectives[2] except odour (see Odour section below). Alberta has AQOs for 44 substances, most of which do not have specific health effects linked to them; only total suspended particulates, sulphur dioxide, ozone, ethylene, and carbon monoxide are explicitly based on protecting either human or vegetation health.

[The Effects Subgroup concluded that more discussion is needed on if and how ambient air quality objectives apply to CFOs. The CFO team should discuss this and consider whether or not to make a recommendation to clarify this topic for Albertans.]

Ammonia Air Quality Objective

There is currently a 1-hour objective for ammonia in Alberta, and a 24-hour objective for ammonia has been proposed by a multi-stakeholder advisory committee to Alberta Environment, the Alberta Ambient Air Quality Objectives Stakeholder Advisory Committee (AAQOSAC), which wanted advice from the CASA CFO team. The Effects Subgroup believes that the science they reviewed supports a 24-hour ambient air quality objective for ammonia of 200 $\mu g/m^3$ as protective of human and vegetation health and encourages the CFO Project Team to endorse this proposed objective.

> **[Recommendation 1. The Effects Subgroup recommends that the CFO team provide formal endorsement to the Alberta Ambient Air Quality Objectives Stakeholder Advisory Committee to develop a 24-hour Alberta ambient air quality objective for ammonia.]**

Monitoring Air Quality Around CFOs

[The Effects Subgroup concluded that ambient monitoring around CFOs would be beneficial for providing more information on the potential for health effects from emissions, but for providing certainty to industry on the potential costs and benefits of reducing emissions.].

Odour from CFOs

Studies indicate that CFO odours do extend into surrounding areas at levels that may disrupt quality of life. **The subgroup agreed by consensus that odour from CFOs can have health effects**. However, there was no consensus on whether the effects are the result of a psychological conditioned response to the odour or whether there is a physiological basis for the effect. This issue is unclear in the scientific literature. More work would be needed to reach agreement on if and how psychosocial aspects affect thresholds, to determine which variable is being considered in terms of odour response (perception, recognition, complaint, irritant, annoyance). **The subgroup agreed that this area, along with others, represented an information gap.**

The subgroup also agreed by consensus that an ambient air quality objective is not the right mechanism to address odour. An odour management framework was suggested as one possible approach, and the subgroup is recommending that the CFO team consider the options below, as well as any other options that may be suggested. The Alberta Ambient Air Quality Objectives Stakeholder

[2] Although these are not necessarily based on health effects.

Advisory Committee (AAQOSAC) deferred developing an odour management framework for Alberta until the CFO team had had a chance to consider the question.

To address concerns related to odour, the Effects Subgroup proposes the following four options for consideration by the CFO Project Team:

- **a) The CFO Team could develop an odour management framework and agree by consensus to all the components, which could then be used by the AAQOSAC to develop a more comprehensive framework that includes other sectors.**
- **b) The CFO Team could decline to develop an odour management framework, in which case the AAQOSAC has indicated they will develop one for all sectors not just agriculture. If this option were pursued, it has been suggested that the stringency of the H_2S threshold may be negotiated at the CFO team.**
- **c) The CFO Team could recommend that an odour management framework be developed, but only make suggestions about what it should contain, and forward its suggestions to the AAQOSAC to consider in their work.**
- **d) In lieu of an odour management framework, the CFO Team could focus on other emissions that both contribute to odour and have direct health effects. The team may make recommendations on controlling or managing these emissions; e.g., how to achieve a lower H_2S ambient level.**

Specific Ideas on an Odour Management Framework to be considered by the CFO Team

An odour management framework could incorporate both qualitative (FIDOL)[3] and quantitative elements; quantitative elements would be based on Odour Units, a quantitative but subjective measurement of odour and of the concentration of individual odour compounds. Studies also suggest that total VOC concentration may be an excellent surrogate for assessing odour intensity, and is relatively simple and inexpensive to measure in the field.

[3] FIDOL stands for Frequency, Intensity, Duration, Offensiveness and Land use.

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1 Introduction

The CASA Board established the multi-stakeholder Confined Feeding Operations (CFO) Project Team in 2005. The goal of the team was to work within the CASA consensus process to develop a strategic plan to improve the management of air emissions from existing and future CFOs in Alberta and to improve relationships between stakeholders.

The team realized that the task was very large and decided to divide its work into four parts, each to be addressed by a subgroup. The Effects Subgroup was given the task of summarizing for the Project Team, the human health, animal health and/or ecological effects from the CFO emissions of ammonia, hydrogen sulphide/TRS, volatile organic compounds, particulate matter, bioaerosols, and odour. The team recognized the importance of greenhouse gas emissions from CFOs, but agreed that the team as a whole would look at these emissions when the subgroup had completed it work. The summary was to provide an overview of the information gathered, outstanding questions and information gaps. The Effects Subgroup was also asked to provide, if required, recommendations on how to fill those information gaps.

Members of the Effects Subgroup and their terms of reference appear in Appendix A of this document. This report is generally organized by substance, in the order in which the substances are listed in the terms of reference and noted above. The subgroup also agreed to include a section on Community Health Effects. All substance sections address human health, animal health and ecological effects, to the extent that such information is available, and most also contain information on ambient air quality objectives. The exception is odour, which focuses only on human health.

The subgroup compiled a great deal of information on these priority substances. This additional background may be useful to readers who want more details. It has been assembled as appendices in a companion document, which, with the exception of bioaerosols for which no separate appendix was prepared, corresponds to the order of the sections in this main report. For example, ammonia is section 2 of this report, and the associated appendices for ammonia begin with the letter B.

2 Ammonia (NH_3)

Ammonia is produced endogenously in the gastrointestinal tract, pancreas, and kidney during protein and nucleic acid metabolism. Endogenously formed NH_3 is metabolized primarily in the liver to urea. Urea and ammonium compounds are excreted through the kidneys in the urine (ATSDR 2002). The predominant pathway for the excretion or elimination of exogenous inhaled NH_3 is exhalation (WBK 2002).

Ammonia is produced by the aerobic and anaerobic microbial decomposition of nitrogen-containing organic matter in manure (EPA 2001). Nitrogen in manure is found in unabsorbed nutrients (e.g., proteins) and in urine as urea in mammals or uric acid in poultry. In the presence of water, urea and uric acid are easily hydrolyzed to form NH_3. NH_3 is highly water soluble and will accumulate in liquid, semi-solid or slurry manures but will rapidly volatize with drying. Sources of NH_3 include confinement buildings, open lots, manure stockpiles, anaerobic lagoons, and land applications of both wet and dry animal waste.

2.1 Health Effects

NH_3 is a highly water soluble gas and in water forms the alkaline and corrosive NH_4OH (ammonium hydroxide). As a result, NH_3 is irritating to the skin and mucous membranes of the body such as the eyes, throat, and lungs. At high concentrations NH_3 can cause severe burns of the skin and eye. At lower concentrations, NH_3 can cause wheezing, coughing, nasal and bronchial discharge, upper airway obstruction, bronchospasm and lung edema. The severity of effects depends on the level of exposure. Although NH_3 is primarily considered an upper airways irritant, it is efficiently scrubbed out by water of the mucous membranes. If adsorbed onto respirable dust, NH_3 can reach and damage the lower airways and lungs (WBK 2002).

Because NH_3 is rapidly metabolized by the body, it is a local rather than systemic irritant. Exposure effects are localized at the site of the air-body interface, such as at the surface of eye, skin or respiratory system.

Health effects can be reversible or irreversible depending on the exposure concentration and duration of exposure. Reversible effects include tearing and coughing. Irreversible effects, due to the corrosive action of NH_3 and resulting cell and tissue necrosis, include corneal scaring and chronic airway inflammation. Adverse effects are not likely to develop from chronic low level exposure because NH_3 is rapidly metabolized and excreted by the body.

The odour of NH_3 is described as sharp with the majority of reported thresholds ranging from 14 to 53 ppm (WBK 2002).

In a 1977 chamber study, Verbek (WBK 2002) reported eye and throat irritation among workers exposed for two hours to 50 ppm. Some workers were unable to tolerate 140 ppm for the full two-hour period. There was no measurable effect on pulmonary function up to 140 ppm among exposed workers. ATSDR (2002) reports that most persons can tolerate 250 ppm for 30–60 minutes.

Cole et al. (2000) identified NH_3 as one of the primary gases of interest to health researchers. Although the concentrations in confinement buildings are not usually high enough to be of occupational health concern, worker exposure limits were recognized as taking “into account

economic as well as health-based considerations (49, 50)" (Cole et al 2000, page 686). Most confinement worker exposure studies have found a correlation between one or more contaminants and worker lung function and/or respiratory symptoms (Cole et al 2000). Most correlations occurred with dusts, endotoxin and NH_3.

Cormier et al. in a 1977 study (WBK 2002) reported respiratory inflammation and symptoms and increased bronchial responsiveness in seven previously unexposed adults following two 5-hour exposure periods (eight days apart) in a swine confinement building. The mean exposure concentrations as determined by personal sampling over the two exposure periods were 10.1 mg/m^3 (14.5 ppm) and 12.4 mg/m^3 (17.8 ppm) NH_3 and 8.5 and 17 mg/m^3 for total dust (5 hour TWAs). A more intense inflammatory response observed in the previously unexposed subjects, compared to the chronically exposed farm workers, suggested either a healthy worker selection process or adaptation.

Chronic effects of ammonia exposure in humans include a reduction in pulmonary function, cough, phlegm, wheeze and dyspnea (WBK 2002). Chronic occupational exposure to levels of ammonia <25 ppm had little effect on pulmonary function in workers at some factories (ATSDR 2002). Studies of farmers exposed to NH_3 and other pollutants in livestock buildings suggest an association with increased respiratory symptoms such as bronchial reactivity and hyper-responsiveness, inflammation, cough, wheeze, shortness of breath and decreased lung function. The contribution of NH_3 to this association was unclear.

Exposure to concentrations > 5000 ppm (3,483 mg/m^3) is considered severely debilitating or lethal to animals and humans (WBK 2002).[4]

2.2 Animal Health Effects

In general, the health effects following chronic inhalation exposure of animals to ammonia include nasal irritation, lung inflammation, reduced olfactory acuity, and lethargy. Animal studies have also demonstrated reduced immune response and increased respiratory tract susceptibility to bacterial infection (WBK 2002).

As with humans, concentrations greater than 5000 ppm of ammonia will have a fatal effect on animals. Ammonia effects such as watering eyes and irritation in pigs can be observed at lower concentrations of 5 to 20 ppm (Barker 1996). Decreased growth rate is noticeable in pigs at about 50 ppm (Holland 2002; Murphy and Cargill 2004; Barker 1996). Chickens will show a decreased growth rate at about 75 to 100 ppm (Murphy and Cargill 2004). Poultry exposed to high levels of ammonia may show reductions in feed consumption, feed efficiency, weight gain and egg production (cited in Atia et al 2004).

The following table is taken from a report prepared for Alberta Environment "Assessment Report on Ammonia for Developing an Ambient Air Quality Guideline, Volume 1" by WBK & Associates Inc in March of 2002.

[4] Conversion factors for vapour at 25 °C and 101.3 kPa: 1 mg/m^3 = 1.44 ppm and 1 ppm = 0.707 mg/m^3.

Table 1. Summary of Effects in Animals Following Acute Ammonia Exposure

Effects Reported	Exposure Period	Air Concentration (ppm)	Species	Reference[a]
Respiratory irritation, decreased body weight and food uptake	1 wk, 24 hr/d	500	Rat	Richard et al, 1978
Lethargy	1 d 6hr/d	100	Rat	Tepper et al, 1985
Lethargy	1 d 6hr/d	100	Mouse	Trepper et al, 1985
Decreased respiratory rate, increased respiratory depth	1 d 3hr/d	50	Rabbit	Mayan and Merilan, 1972
No adverse effects	1 wk, 5d/wk	223	Rabbit	Coon et al, 1970
Temporary dyspnea and lacrimation	8 hr/d	1,105	Rabbit	Coon et al, 1970
No adverse effects	1 wk 5 d/wk	223	Dog	Coon et al, 1970
Temporary dyspnea and lacrimation	8 hr/d	1, 105	Dog	Coon et al, 1970
No adverse effects	3 d	10	Pig	Strombaugh et al, 1969
Oral, nasal and ocular irritation, coughing, decreased body weight and decreased food uptake	3 d	50	Pig	Strombaugh et al, 1969
No adverse effect on olfactory	1 d 45 min	40	Pig	Jones et al, 2001;2000
Increased neutrophil count of nasal mucosa and epithelial hyperplasia, functional disturbances of tracheal smooth-muscle contractions	6 d	25	Piglets	Urbain et al, 1996
Cell damage and inflammatory response of lung neutrophils, dose-dependent decrease in neutrophil recover rates	1 hr	50	Bovine lung neutrophils	Murata and Horino, 1999

[a] Cited in: WBK & Associates Inc report

2.3 Ecological Effects

The table below shows the effects of ammonia on a range of vegetation species.

Table 2. Effects of Ammonia on Vegetation Species

Parameter[a]	Species	NH_3 Concentration ($mg\ m^{-3}$)[b,c,d]	Duration	Effect[e,f]	Comments[g]
N content	*Agrostis capillaris*	0.05	8 mo	+++	OTC, shoots, roots
	Calluna vulgaris	0.1	38 w	+++	GHC[g]
	Deschampsia flexuosa	0.1	38 w	+++	GHC
	Pseudotsuga menziesii	0.18	13 w	+++	GHC, needle age 7 w
	Pseudotsuga menziesii	0.18	13 w	+[e]	GHC, needle age 1-2 years
	Pinus sylvestris	0.24	3 mo	+++	ICEC[g], current year needles
	Pinus sylvestris	0.24	3 mo	++[e]	ICEC, previous year needles
	Pseudotsuga menziesii	1.8	13 w	+++	ICEC, needle age 7 w
	Pseudotsuga menziesii	1.8	13 w	(+)[f]	ICEC, needle age 1 year
Arginine content	*Pinus sylvestris*	0.24	14 w	+++	ICEC, needle content
	Pinus sylvestris	n.d.[c]		+++	FO[g], in the vicinity of fur farms

Parameter[a]	Species	NH_3 Concentration ($mg\ m^{-3}$)[b,c,d]	Duration	Effect[e,f]	Comments[g]
Glutamine content	*Lycopersicon esculentum*	2.0	24 h	+++	ICEC, high irradiation
Chlorophyll a, b	*Pinus sylvestris*	0.24	14 w	++	ICEC, previous year needles
Photosynthetic rate, max. (P_{max})	*Populus euramericana*	0.1	6 w	+	ICEC, P_{max}
	Populus euramericana	0.1	8 w	+++	ICEC, P_{max}
Net photosynthesis (P_n)	*Pinus sylvestris*	0.24	3 mo	+	ICEC, PAR: 950 mol $m^{-2}s^{-1}$
Dark respiration	*Pinus sylvestris*	0.24	3 mo	+++	ICEC
Transpiration	*Pinus sylvestris*	0.24	3 mo	++	ICEC, in the dark
	Pinus sylvestris	0.24	3 mo	+++	ICEC, PAR: 950 mol $m^{-2}s^{-1}$
Water content	*Pinus sylvestris*	0.053	9 mo	-	OTC, after 2 w of desiccation
Water potential	*Pinus sylvestris*	0.105	9 mo	- -	OTC, after 2 w of desiccation
Erosion of the epicuticular wax layer	*Pinus sylvestris*	NH_3: 0.0 $_{SO2}$: 0.065	7 w	0	OTC
	Pinus sylvestris	NH_3: 0.10 SO2: 0.065	7 w	+++	OTC, smoothing of the waxes
	Pinus sylvestris	0.10	9 mo	0	OTC, current year needles
	Pseudotsuga menziesii	0.025	1 a	++	OTC, previous year needles
Nutrient content P	*Agrostis capillaries*	0.05	8 mo	+	OTC, shoots
P	*Agrostis capillaries*	0.05	8 mo	++	OTC, roots
K	*Agrostis capillaries*	0.05	8 mo	(-)	OTC, shoots
K	*Agrostis capillaries*	0.05	8 mo	- - -	OTC, roots
Ca, Mg	*Agrostis capillaries*	0.05	8 mo	+++	OTC, shoots, roots
Ca, Mg	*Pseudotsuga menziesii*	0.18	13 w	0	GHC, 7 w old needles
K	*Pseudotsuga menziesii*	0.18	13 w	- -	GHC, 7 w old needles
P	*Pseudotsuga menziesii*	0.18	13 w	-	GHC, 7 w old needles
K, P, Ca, Mg	*Pseudotsuga menziesii*	0.18	13 w	0	GHC, 1-2 year old needles
P, Ca, Mg, K	*Calluna vulgaris*	0.1	12 w	0	GHC, leaves
Mg, P, K	*Pinus sylvestris*	0.24	3 mo	0	ICEC, current and older needles
N/K ratio	*Pseudotsuga menziesii*	0.18	13 w	+++	ICEC, 7 w old needles
	Pseudotsuga menziesii	0.18	13 w	++	ICEC, previous year needles
N/Mg ratio	*Pseudotsuga menziesii*	0.18	13 w	+++	ICEC, 7 w old needles
	Pseudotsuga menziesii	0.18	13 w	(+)	ICEC, previous year needles
N/P ratio	*Pseudotsuga menziesii*	0.18	13 w	+++	ICEC, 7 w old needles
	Pseudotsuga menziesii	0.18	13 w	++	ICEC, previous year needles
Visible leaf injury	*Rhacomitrium lanuginosum*	0.03	23 d	+	ICEC, chlorotic leaves, very sensitive
	Rhacomitrium lanuginosum	0.06	17 d	+	ICEC, chlorotic leaves, very sensitive
	Campylopus flexuosus	0.12	23 d	+	ICEC, chlorotic leaves
	Campylopus flexuosus	0.12	17 d	+	ICEC, chlorotic leaves
	Campylopus flexuosus	0.12	14 d	+	ICEC, chlorotic leaves
	Hypnum jutlandicum	0.12	11 d	+	ICEC, chlorotic leaves
	Hypnum jutlandicum	0.12	14 d	+	ICEC, chlorotic leaves
	Hypnum jutlandicum	0.12	17 d	+	ICEC, chlorotic leaves

Parameter[a]	Species	NH_3 Concentration ($mg\ m^{-3}$)[b,c,d]	Duration	Effect[e,f]	Comments[g]
	Hypnum jutlandicum	0.12	23 d	+	ICEC, chlorotic leaves
	Rhacomitrium lanuginosum	0.12	14 d	+	ICEC, chlorotic leaves, very sensitive
	Campylopus flexuosus	0.24	11 d	0	ICEC
	Pleurozium schreberi	0.24	23 d	0	ICEC
	Pleurozium schreberi	0.24	17 d	0	ICEC
	Pleurozium schreberi	0.24	14 d	0	ICEC
	Pleurozium schreberi	0.24	11 d	0	ICEC
	Rhacomitrium lanuginosum	0.24	11 d	0	ICEC
	Pinus sylvestris	NH_3: 0.053 SO_2: 0.092	5 mo	++	OTC, synergism, NH_3 alone without effect
	Chamaecyparis columnaris var. *glauca*	0.15-0.30	60 d	+	GHC + FO, moderately sensitive
	Chamaecyparis lawsoniana	0.15-0.30	60 d	+	GHC + FO, moderately sensitive
	Cupressocyparis leylandii	< 0.15	60 d	+	GHC, sensitive
	Picea abies	< 0.15	60 d	+	GHC, sensitive
	Picea omorika	0.15-0.30	60 d	+	GHC, moderately sensitive
	Picea sitchensis	< 0.15	60 d	+	GHC, sensitive
	Pinus nigra var. *maritime*	0.15-0.30	60 d	+	GHC, moderately sensitive
	Pinus strobes	< 0.15	60 d	+	GHC, sensitive
	Pseudotsuga menziesii	0.15-0.30	60 d	+	GHC, moderately sensitive
	Taxus baccata	< 0.15	60 d	+	GHC, sensitive
	Taxus baccata 'Fastigiata'	0.15-0.30	60 d	+	GHC, moderately sensitive
	Pinus mugo var. *mughus*	> 0.3	60 d	+	GHC, insensitive
	Pinus nigra var. *nigra*	> 0.3	60 d	+	GHC, insensitive
	Pinus sylvestris	> 0.3	60 d	+	GHC, insensitive
	Taxus media 'Hicksii'	> 0.3	60 d	+	GHC, insensitive
	Thuja occidentalis	> 0.3	60 d	+	GHC, insensitive
	Tsuga canadensis 'Nana Compacta'	> 0.3	60 d	+	GHC, insensitive
	Brassica juncea	0.5-1.5	7 d	+	GHC, moderately sensitive
	Brassica oleracea (cauliflower, 3 cvs.)	< 0.5	7 d	+	GHC, sensitive
	Brassica oleracea (Brussels sprouts, cabbage, savoy)	0.5-1.5	7 d	+	GHC, moderately sensitive
	Brassica sinensis	0.5-1.5	7 d	+	GHC, moderately sensitive
	Capsicum annuum	0.5-1.5	7 d	+	GHC, moderately sensitive
	Chenopodium album	0.5-1.5	7 d	+	GHC, moderately sensitive
	Cucumis sativus	< 0.5	7 d	+	GHC, sensitive
	Fagopyrum esculentum	0.15-1.5	7 d	+	GHC, moderately sensitive
	Lactuca sativa	0.5-1.5	7 d	+	GHC, moderately sensitive
	Melilotus alba	0.5-1.5	7 d	+	GHC, moderately sensitive
	Nicotiana tabacum 'Bel W3'	0.5-1.5	7 d	+	GHC, moderately sensitive
	Phaseolus vulgaris	0.5-1.5	7 d	+	GHC, moderately sensitive
	Prunus laurocerasus 'Otto Luycken'	0.5-1.5	7 d	+	GHC, moderately sensitive
	Lycopersicum esculentum	< 0.5	7 d	+	GHC, sensitive

Parameter[a]	Species	NH_3 Concentration ($mg\ m^{-3}$)[b,c,d]	Duration	Effect[e,f]	Comments[g]
	Brassica oleracea var. *botryris* (cv. le Cerf)	0.6	16 d	+	GHC, plant age 75 d
	Brassica oleracea var. *botryris* (cv. VerbMech)	0.6	16 d	+	GHC, plant age 75 d
	Brassica pekinensis (cv. Granaat)	0.6	16 d	0	GHC plant age 75 d
	Lolium multiflorum (cv. Optima)	0.6	30 d	0	GHC, plant age 60 d
	Lycopersicon esculentum (cv. Money maker)	0.6	6 d	+	GHC, plant age 55 d
	Poa annua	0.6	30 d	0	GHC, plant age 60 d
	Brassica oleracea 'Rotkohl'	> 1.5	7 d	+	GHC + FO, insensitive
	Lolium multiflorum	> 1.5	7 d	+	GHC, insensitive
	Nepeta cataria	> 1.5	7 d	+	GHC, insensitive
	Poa annua	> 1.5	7 d	+	GHC, insensitive
	Populus euramericana	> 1.5	7 d	+	GHC, insensitive
	Pyrus comm. sat. 'Doyennée du com.'	> 1.5	7 d	+	GHC, insensitive
	Pyrus malus 'Golden Delicious'	> 1.5	7 d	+	GHC, insensitive
	Raphanus sativus	> 1.5	7 d	+	GHC, insensitive
	Rhododendron (2 cvs.)	> 1.5	7 d	+	GHC, insensitive
	Valerianella olitora	> 1.5	7 d	+	GHC, insensitive
	Cucumis sativus	2	30 d	[+++]	CFC, brownish discolouration
	Lactuca sativa var. *capitata* (cv. Attractie)	2.0	2 d	0	GHC, plant age 35 d
	Lactuca sativa var. *capitata* (cv. Attractie)	2.0	6 d	+	GHC, plant age 35 d
	Lactuca sativa var. *capitata* (cv. Attractie)	4.2	6 d	+	GHC, plant age 35 d
	Conifers	0.06[d]	60 d	++	FO, wintertime, close to a pig farm
[a]	Conifers	0.06[d]	53 d	0	FO, spring, close to a pig farm
[a]	Conifers	0.07[d]	50 d	0	OTC, wintertime
	Conifers	0.10[d]	53 d	0	OTC, spring
	Pseudotsuga menziesii	0.18	13 w	0	GHC, darker green colour
	Conifers	0.25[d]	50 d	+++	OTC, wintertime
[a]	Conifers	0.54[d]	53 d	0	OTC, spring
[a]	Conifers	0.69[d]	100 d	+	ICEC (8-12EC)
	Lycopersicon esculentum	2.0	24 h	+++	ICEC, dark
Flowering	*Amica Montana*	0.053	15 mo	- -	OTC
	Amica Montana	0.105	15 mo	- - -	OTC
	Petunia hybrida	2.42	14 d	- -	GHC, flowering reduced by 50%
Bud break	*Pinus sylvestris*	0.105	10 mo	- - -	OTC
Needle loss	*Picea abies*	2.3-3.9	1 w	[+]	GH, poultry farm exhaust
Deaf ear and grain development	*Avena sativa*	2.3-3.9	5 mo	[+]	GH, poultry farm exhaust
Dry weight	*Calluna vulgaris*	0.05-1.2	90 d	+++	OTC
	Agrostis capillaries	0.053	8 mo	+++	OTC, shoot, root

Parameter[a]	Species	NH_3 Concentration ($mg\ m^{-3}$)[b,c,d]	Duration	Effect[e,f]	Comments[g]
	Agrostis capillaries	0.053	8 mo	++	OTC, flowers
	Amica montana	0.053	8 mo	++	OTC, shoot, root
	Amica montana	0.053	8 mo	0	OTC, flowers
	Viola canina	0.053	8 mo	++	OTC, shoot, root or flowers
	Calluna vulgaris	0.1	38 w	+++	GHC
	Calluna vulgaris	0.1	4 w	0	GHC, shoot
	Calluna vulgaris	0.1	4 w	+	GHC, root
	Calluna vulgaris	0.1	9 w	++	GHC, shoot
	Calluna vulgaris	0.1	9 w	0	GHC, root
	Deschampsia flexuosa	0.1	4 w	++	GHC, shoot
	Deschampsia flexuosa	0.1	4 w	+++	GHC, root
	Deschampsia flexuosa	0.1	9 w	+++	GHC, shoot, root
	Deschampsia flexuosa	0.1	38 w	+++	GHC
	Amica Montana	0.105	15 mo	+++	OTC, shoot
	Agrostis capillaries	0.24	12 w	+++	ICEC, shoot, root
	Agrostis capillaries	0.24	12 w	+++	ICEC, total plant weight
	Antennaria dioica	0.24	12 w	+++	ICEC, shoot, root
	Antennaria dioica	0.24	12 w	+++	ICEC, total plant weight
	Calluna vulgaris	0.24	12 w	+++	ICEC, leaves, woody parts of the shoot
	Calluna vulgaris	0.24	12 w	+++	ICEC, shoot
	Calluna vulgaris	0.24	12 w	0	ICEC, root
	Deschampsia flexuosa	0.24	12 w	0	ICEC, shoot, root
	Pinus sylvestris	0.24	3 mo	+++	ICEC, previous year needles only
	Potentilla erecta	0.24	12 w	+++	ICEC, shoot, rhizome
	Potentilla erecta	0.24	12 w	0	ICEC, root
	Viola canina	0.24	12 w	+++	ICEC, shoot
	Viola canina	0.24	12 w	++	ICEC, root
	Lolium perenne	0.55	26 d	+++	GHC
	Brassica oleracea var. *botryris* (cv. le Cerf)	0.6	16 d	-	GHC, plant age 75 d
	Brassica pekinensis (cv. Granaat)	0.6	16 d	(-)	GHC, plant age 75 d
	Lolium multiflorum (cv. Optima)	0.6	30 d	+	GHC, plant age 60 d
	Lycopersicon esculentum (cv. Money maker)	0.6	3 d	(+)	GHC, plant age 55 d
	Lycopersicon esculentum (cv. Money maker)	0.6	6 d	(-)	GHC, plant age 55 d
	Poa annua	0.6	30 d	+++	GHC, plant age 60 d
	Lepidium sativum	1.08	14 d	- -	GHC
	Raphanus sativus	1.46	14 d	- -	GHC, hypocotyl weight
	Saintpaulia ionatha	1.46	14 d	-	GHC, no leaf injuries
	Petunia hybrida	1.66	14 d	-	GHC, leaf injuries
	Lactuca sativa var. *capitata* (cv. Attractie)	2.0	2 d	(0)	GHC, plant age 35 d
	Lactuca sativa var. *capitata* (cv. Attractie)	2.0	6 d	(++)	GHC, plant age 35 d

Parameter[a]	Species	NH_3 Concentration ($mg\ m^{-3}$)[b,c,d]	Duration	Effect[e,f]	Comments[g]
	Lactuca sativa var. *capitata* (cv. Attractie)	4.2	2 d	(+)	GHC, plant age 35 d
	Lactuca sativa var. *capitata* (cv. Attractie)	4.2	6 d	(-)	GHC, plant age 35 d
	Lactuca sativa var. *capitata*	2	14 d	[+]	CFC
	Phaseolus vulgaris	2.2	14 d	-	GHC, leaf injuries
	Saintpaulia ionatha	2.25	14 d	-	GHC, no leaf injuries
	Avena sativa	2.3-3.9	9 w	[+++]	GH, poultry farm exhausts
	Lolium perenne	2.3-3.9	5 mo	[++]	GH, poultry farm exhausts
	Picea abies	2.3-3.9	5 mo	[++]	GH, poultry, current year flushes
	Trifolium pratense	2.3-3.9	5 mo	[+++]	GH, poultry farm exhausts
	Trifolium pratense	2.3	14 d	- - -	GHC, leaf injuries
	Nicotiana tabacum Bel W3	2.37	14 d	-	GHC, leaf injuries
	Phaseolus vulgaris	3.31	14 d	-	GHC, leaf injuries, leaf littering
Cover	Bryophytes	0.053	24 mo	- - -	OTC
Tiller number	*Agrostis capillaries*	0.24	12 w	+++	ICEC
Growth	*Pinus sylvestris*	0.105	10 mo	- - -	OTC, apical shoot growth
Shoot/root ratio	*Calluna vulgaris*	0.05-1.2	90 d	+++	OTC
	Agrostis capillaries	0.053	8 mo	+++	OTC
	Arnica montana	0.053	8 mo	+	OTC
	Viola canina	0.053	8 mo	++	OTC
	Calluna vulgaris	0.1	38 w	+++	GHC
	Deschampsia flexuosa	0.1	38 w	++	GHC
	Potentilla erecta	0.24	12 w	++	ICEC
Needle/root ratio	*Pinus sylvestris*	0.24	3 mo	++	ICEC, no effect if old needles are concerned
Survival rate	*Calluna vulgaris*	0.053	8 mo	-	OTC, seedlings
	Calluna vulgaris	NH_3: 0.053 SO_2:0.092	8 mo	- - -	OTC, seedlings, synergistic effect
Frost hardiness -4, -7, -10EC	*Pinus sylvestris*	NH_3: 0.053 SO_2: 0.092	5 mo	- - -	OTC, synergistic effects
-10EC	*Pinus sylvestris*	0.105	5 mo	- -	OTC, less susceptible in winter
-10EC	*Pinus sylvestris*	0.15	21 w	- - -	OTC & ICEC
Mycorrhizal infection	*Pseudotsuga menziesii*	0.18	13 w	- - -	GHC, youngest roots
Larval development	*Calluna vulgaris*	0.107	12 mo	++	OTC, *Lochmaea suturalis*, at feeding with fumigated leaves
Interaction (NH_3 X SO_2): Erosion of the epicuticular wax layer	*Pinus sylvestris*	NH_3: 0.0 SO_2: 0.065	7 w	0	OTC
	Pinus sylvestris	NH_3: 0.10 SO_2: 0.065	7 w	+++	OTC, smoothing of the waxes
Interaction (NH_3 X SO_2): Visible injury	*Pinus sylvestris*	NH_3: 0.053 SO_2: 0.092	5 mo	++	OTC, synergism, NH_3 alone without effect
Interaction (NH_3 X SO_2): Survival rate	*Calluna vulgaris*	NH_3: 0.053 SO_2:0.092	8 mo	- - -	OTC, seedlings, synergistic effect
Interaction: (NH_3 X SO_2): Frost hardiness -4, -7, -10EC	*Pinus sylvestris*	NH_3: 0.053 SO_2: 0.092	5 mo	- - -	OTC, synergistic effects

Parameter[a]	Species	NH_3 Concentration ($mg\ m^{-3}$)[b,c,d]	Duration	Effect[e,f]	Comments[g]
Interaction (NH_3 X O_3): Visible injury biomass	*Phaseolus vulgaris*	NH_3: 0.03-0.06 O_3: 0-0.14	24d, 49d; 62 d 9 h,d	 +++ +++	OTC, Visible injury increased and biomass decreased with increasing O_3 levels. Biomass was stimulated at NH_3 006 mg
Interaction (NH_3 X O_3): Growth	*Pinus sylvestris*	NH_3: 0.02-0.16 O_3: 0-0.13	24h,d 15 mo 9 h,d	 ++	NH_3 stimulated growth, lowered needle water potential, increased drought stress O_3 ameliorated drought effect of NH_3; effect of NH_3 > O_3

[a] Injuries like needle tip necrosis, needle loss, etc.
[b] To convert µg m^{-3} to ppb, multiply by 1.44 or to convert mg m^{-3} to ppm, multiply by 1.44 (values are for 25 C at sea level or 760 mm Hg).
[c] +, increase 1-25% compared with controls; ++ increase 26-50% compared with controls; +++ increase > 50% compared with controls; -, decrease 1-25% compared with controls; - -, decrease 26-50% compared with controls; - - -, decrease > 50% compared with controls; 0, no difference to controls.
[d] ( ), effect statistically not significant; [ ], no statistical analysis of the data available.
[e] Given concentration is median, not mean.
[f] n.d., NH_y concentration not measured.
[g] ICEC, indoor controlled environment chambers; GHC, exposure chambers place in a greenhouse; GH, greenhouse fumigation; CFC, closed field chambers; OTC, open-top field chambers, FO, field observation.
Source: modified from Fangmeier et al. (1994).

2.4 Ambient Air Quality Objectives

The existing 1-hour ambient air quality objective (AQO) for NH_3 in Alberta is 1.4 mg/m^3 or 2 ppm. In 200x Alberta Environment convened a multistakeholder group, consisting of representatives from industry, the public, non-governmental organizations, and various government agencies and departments. Their task was to review and revise existing AQOs or create new ones. An NH_3 subgroup was formed in 2002 and made recommendations to the multistakeholder group for NH_3 AQOs in 2003. The recommendations were endorsed and Alberta Environment published notice of the proposed objectives in 2004: 1400 µg/m^3 1-hour average and 200 µg/m^3 24-hour average. The livestock industry and Alberta Agriculture expressed concern about the proposed objectives and the NH_3 subgroup, with new representations from agriculture, was reconvened in 2005. The subgroup completed its work the same year.

The 2002 NH_3 subgroup recommended a 1-hour and 24-hour AQO. A chronic exposure or annual average AQO was not considered necessary as NH_3 is rapidly detoxified and excreted in the body. The subgroup used health information from other jurisdictions to develop an AQO (see Table 3). The occupational study of Holness (1989) was used in the recommendation of the 200 µg/m^3 24-hour AQO. The work of Broderson et al. (1976), among others was considered supportive of the recommendation.

The 2005 NH_3 subgroup recommended maintaining the current 1-hour AQO of 1.4 mg/m^3 based on odour, the development of a 24-hour AQO for protection of public health and an annual average AQO for protection of vegetation. The subgroup was not able to reach consensus on the numeric values for the AQOs. However, the subgroup recommended using the Sustainable Resource and Environmental Management (SREM) approach to develop an AQO. The SREM approach is fundamentally a multi-stakeholder process to address and resolve complex environmental and land

use issues (Alberta 1999). In the context of NH_3, this process would entail consideration of the interaction of NH_3 in soil, water and air and in the context of the nitrogen cycle in AQO development.[5]

The Ammonia Subgroup decided that the 1-hour AQO will be retained and the NH_3 subgroup report will be passed on to the CASA CFO team for consideration. Any decision on a 24-hour or annual AQO will be deferred until the CASA CFO team has made its final recommendation. The proposed 200 $\mu g/m^3$ 24-hour average is considered to be protective of both human health and vegetation.

The subgroup agreed to reference Alberta Environment's report on ammonia; a full reference citation is needed.

[5] The nitrogen cycle and nitrogen cascade are described and illustrated in Appendix B-1 of the companion document to this report.

Table 3. Summary of Jurisdictional Protocols for Derivation NH_3 Health-Based Reference Concentrations

	EPA IRIS		Cal. EPA REL	ATSDR	ATSDR
Publication Date	July 28, 2003			Sept 2002	Sept 2004 Update
	RfC (Reference Concentration)		Chronic REL (Reference Exposure Level)	Chronic MRL (Minimal Risk Level)	Chronic MRL (Minimal Risk Level)
Critical Study/Effect	Broderson 1976 (increased susceptibility to respiratory infection and presence of nasal lesions)	Holness 1989 (no affects on lower lung function indicators)	Holness 1989 (no affects on lower lung function indicators)	Holness 1989 (no affects on lower lung function indicators)	Holness 1989 (no affects on lower lung function indicators)
LOAEL LOAELadj	17.4 mg/m3 17.4 mg/m3	-	-	-	-
LOAEL (HEC)	1.9 mg/m3	-	-	-	-
NOAEL (mg/m3)		6.4 8-hour average	6.4 (8-hour average) (assumed continuous at 10 m^3/8 hr d, 8 hrs/day, 5 days/wk for 12 years	8.8 (8-hr average) (based on a high range average rather than overall average)	6.5 (8-hour average), no significant alterations in lung function in chronically exposed workers
NOAELadj (mg/m3)		2.3 24-hour average continuous (10m3/20m3 x 5d/7d)	2.3 (10/20x5d/7d)	2.2 24-hour average continuous (8.4 hr/24 hr x 5d/7d)	1.5 24-hour average continuous (8 hr/24 hr x 5d/7d)
NOAEL (HEC)		-	-		-
Interspecies Extrapolation		-	-	-	-
Humans Variability		10x	10x	10x	10x
Subchronic to Chronic		-	1x	-	
Modifying or Uncertainty Factor		3x (proximity of animal LOAEL to human NOAEL, lack of reproductive/development studies & chronic exposure studies)	-	-	3x (lack of reproductive and developmental studies)
Health Based Criteria		100 ug/m3 24-hr average	200 ug/m3 lifetime time average (annual)	200 ug/m3 24 hr average	70 ug/m3 24 hr average

Detailed Information - Jurisdictional Protocols for Derivation NH_3 Health-Based Reference Concentrations

	EPA IRIS		Cal. EPA REL	ATSDR	ATSDR
Publication Date	July 28, 2003			Sept 2002	Sept 2004 Update
	RfC (Reference Concentration)		Chronic REL (Reference Exposure Level)	Chronic MRL (Minimal Risk Level)	Chronic MRL (Minimal Risk Level)
Definition	As estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure of the human population including sensitive subgroups that is likely to be without appreciable risk of deleterious effects during a lifetime. Chronic: Repeated exposure by the oral, dermal, or inhalation route for more than approximately 10% of the life span in humans (more than approximately 90 days to 2 years in typically used laboratory animal species).		A chronic REL is an airborne level that would pose no significant health risk to individuals exposed to that level over a lifetime. RELs are based solely on health considerations and designed to protect the most sensitive individuals in the population by the inclusion of margins of safety.	MRL is an estimate of the daily exposure to a substance that is likely to be without appreciable risk of adverse noncancer health effects over the specified duration of exposure Chronic (365 days and longer) exposure durations	MRL is an estimate of the daily exposure to a substance that is likely to be without appreciable risk of adverse noncancer health effects over the specified duration of exposure Chronic (365 days and longer) exposure durations
Critical Study / Effect	Broderson 1976 Exposed rats to 0, 17.4, 35, 106 and 177 mg/m3 continuously for 6 days and then inoculated with Mycoplasma and monitored for infection. Increased respiratory infection and lesions down to 17.4 mg/m3. Effects were rhinitis, otitis media, tracheitis and pneumonia. Control was NH3 exposure with no inoculation. In addition, microscopic lesions (epithelial thickening & hyperplasia) were higher in nasal passages, tracheas and lungs than controls.	Holness 1989 52 exposed workers and 31 control office workers in a soda ash plant. Average years of work at plant = 12 years. Single work shift airborne NH3 measurement for each worker. Lung function tests (FVC, FEV1.0, FEF50&75) at the beginning and end of work week showed no stat. difference to controls, although workers reported respiratory aggravation, eye irritation and skin problems.	Holness 1989	Holness 1989	Holness 1989
LOAEL LOAELadj	17.4 mg/m3 17.4 mg/m3	-	-	-	-
LOAEL (HEC)	1.9 mg/m3	-	-	-	-
NOAEL (mg/m3)		6.4 8-hour average	6.4 8-hour average (assumed continuous at 10 m^3/8 hr d, 8 hrs/day, 5 days/wk for 12 years	8.8 8-hr average (based on a high range average rather than overall average)	6.5 8-hour average), no significant alterations in lung function in chronically exposed workers
NOAELadj (mg/m3)		2.3 24-hour average continuous (10m3/20m3 x 5d/7d)	2.3 (10/20x5d/7d)	2.2 24-hour average continuous (8.4 hr/24 hr x 5d/7d)	1.5 24-hour average continuous (8 hr/24 hr x 5d/7d)

	EPA IRIS		Cal. EPA REL	ATSDR	ATSDR
NOAEL (HEC)		-	-		-
Interspecies Extrapolation		-	-	-	-
Humans Variability		10x	10x	10x	10x
Subchronic to Chronic		-	1x	-	
Modifying or Uncertainty Factor		3x (proximity of animal LOAEL to human NOAEL, lack of reproductive/development studies & chronic exposure studies)	-	-	3x (lack of reproductive and developmental studies)
Health Based Criteria		100 ug/m3 24-hr average	200 ug/m3 lifetime time average (annual)	200 ug/m 24 hr average	70 ug/m3 24 hr average

	EPA IRIS	Cal. EPA REL	ATSDR	ATSDR
Additional Comments and Supporting Studies	Schoeb 1982 also observed increased Mycoplasma infection in rats exposed at 71 mg/m3 (the only test concentration versus the control). Study also showed that all the NH3 was absorbed in the upper airways, indicating that the infection was secondary to NH3 absorption in the nose and not a direct effect of ammonia on the lung itself. Pulmonary function tests used by Holness were not considered sensitive indicator of NH3 exposure because NH3 does not penetrate deep into the lungs.. Holness did not assess the upper airways or nasal passages. The low prevalence of atopy in workers vs. controls suggests that some worker selection bias may have occurred. In addition, the finding of a rat $NOAEL_{HEC}$ of 1.9 mg/m3 similar to Holness NOAEL of 2.3 mg/m3, may be attributable to the ability of NH3 to more likely affect the upper respiratory tract than the lower lungs. The Holness study provides a less sensitive endpoint than Brodersons in that the Broderson study assessed upper respiratory tract effects.	Broderson (1976) study on rats provides a LOAEL of 17.4 mg/m3, increased susceptibility to respiratory infection Other Supporting Studies: Ferguson (1977), groups of healthy adults exposed for 5 days to varying NH3 levels and durations: 0, 17.8, 35.5 and 71 mg/m3 for 2, 4 or 6 hrs/day. One group was exposed to 35 mg/m3, 6/hrs/day, 6 wks. Adaptation tot initial irritation observed with no adverse effect (lung tests). Coon (1970), rats and guinea pigs exposed continuously to 40 to 470 mg/m3. Rats showed nasal discharge @ 262 mg/m3, 90 days, suggesting a $NOAEL_{subchronic}$ of 127 mg/3 for upper respiratory tract irritation. Most of the rats died at 455 mg/m3. Guinea pigs unaffected to 35 mg/3 for 114 days. Anderson (1964), guinea pigs to 35 mg/m3 continuously for 6 wks, observed pulmonary oedema (equiv. to REL of 10 ug/m3).	Supported by farmers and livestock confinement building studies showing increased respiratory symptoms (inflammation, cough, wheezing, shortness of breath, bronchial hyperresponsiveness) and decrease lung function FEV1.0, MEF 50&75 and MMEF in farmers exposed to 1.6 to 14.6 mg/m3 (Choudat 1994, Cormier 2000, Donham 1995, Heederik 1990, Reynolds 1996, Vogelzang 1997, 2000). Farmers likely exposed to other respiratory hazards such as endotoxins. Two Saudi Arabi studies of Fertilizer Plant workers showed a stat. significant association between exposure to NH3 and respiratory symptoms including bronchial asthma (Ballal 1998). But, no continuous exposure levels could be calculated because the # of days worked per week was not provided.	Supported by farmers and livestock confinement building studies showing increased respiratory symptoms (inflammation, cough, wheezing, shortness of breath, bronchial hyperresponsiveness) and decrease lung function FEV1.0, MEF50&75 and MMEF in farmers exposed to 1.6 to 14.6 mg/m3 (Choudat 1994, Cormier 2000, Donham 1995, Heederik 1990, Reynolds 1996, Vogelzang 1997, 2000). Farmers likely exposed to other respiratory hazards such as dust & endotoxins. Saudi Arabi study of Fertilizer Plant workers who showed a stat. significant association between exposure to NH3 and respiratory symptoms including bronchial asthma (Ballal 1998). The range of exposures in the 2 plants was 0.02 to 130 mg/m3, the geometric means were less than the OEL of 18 mg/m3. Insufficient data with which to derive an MRL.

Conversion Factor: 1 ppm NH3 = 0.0707 mg/m3. 1 mg/m3=1.414 ppm

FEV1.0 sec.= 1 second forced expiratory volume
FVC = forced vital capacity
MEF50 &75=the maximum expiratory flow rates
MMEF=Maximal mid-expiratory flow rate.
FEF50&75 = forced expiratory flow rate at 59 & 75% of vital capacity.

California EPA:

The California EPA (Section 3.3.2,Chronic REL methodology document, 2000) acknowledges the US EPA protocol of applying an adjustment or uncertainty factor of between 1 to 10 times for using subchronic studies (i.e., for exposure that are <10% of average life-span) in deriving chronic RfCs. Because of the subjectivity involved in applying a specific adjustment, the California EPA tightened the application criteria:

- 10 x adjustment for exposure periods <8% of expected lifetime
- 3 x adjustment for exposures periods 8-12%.
- 1x adjustment for exposures periods >12%.

2.5 NH_3 and CFOs

The U.S. National Research Council (NRC 2003) ranked NH_3 emissions from animal feeding operations and ability to cause local effects (i.e., at the property line or the nearest dwelling) as minor. The primary effect of concern was contribution to atmospheric haze on a regional, national and global scale. NH_3 released into the atmosphere can react with sulphuric and nitric acids to form $PM_{2.5}$, ammonium sulphate and nitrate aerosols.

Reynolds et al. (1997)[6] measured single day NH_3 levels 60 m downwind of a number of large swine production facilities. 6-hour average ($\pm$standard deviation) NH_3 levels were for the large facility (4,000 head no lagoon) 177 ($\pm$45) µg/m^3, for the medium facility (4000 head with lagoons) 61 ($\pm$64) µg/m^3, for the small facility (2000 head with lagoons) 151 ($\pm$113) µg/m^3 and for small conventional (50 head with no lagoon) 98 ($\pm$133) µg/m^3. The downwind range was 7 to 325 µg/m^3. The upwind controls were all < 3 µg/m^3 6-hour average. N=5 for each location.

McGinn et al. (2003)[7] measured NH_3 levels around a Lethbridge area cattle feedlot. All measurements in this study were on feedlot property. McGinn et al. measured ammonia concentrations at fixed monitoring stations (using annular denuder tubes) at 3 meters east of feedlot pens and at 100 and 200 meters further east for 6,000, 12,000 and 25,000 head feedlots between May 22 and August 3, 1999. Of all the 2- to 3-day sampling periods, only the 8 presented here and in the paper were associated with continuous westerly winds and hence may represent near worst-case measurement. Respective 2- to 3-day average and maximum concentrations at the edge of the feedlots during these 8 periods are shown in the table below. Additional downwind measurements producing 2-day averages at 3, 100 and 200 meters distance of the 12,000 and 25,000 feedlots are also presented (n= 4 to 5 for each feedlot). Although the 100 and 200 meter samples were collected on feedlot property in this study, these distances should also be considered as representative of possible offsite location for Alberta feedlots.

The average and maximum wind speeds during the eight monitoring periods were 2.4 and 4.3 m/s (8.6 and 15.5 km/hr). The higher NH_3 concentration for the 12,000 head feedlot compared to the 25,000 lot may be due to differences in animal density. Animal densities for the 6,000, 12,000 and 25,000 head feedlots were 20, 13.3 and 25.6 m^2 per animal, respectively (McGinn et al 2003).

[6] Cited in Ammonia Subgroup 2005
[7] Cited in Ammonia Subgroup 2005

Table 4. 2- to 3- Day Average NH_3 Concentrations (µg/m^3) around a Lethbridge Area Feedlot

Monitoring Period	Distance from Feedlot	2 to 3 day average NH_3 (µg/m^3)	Feedlot head		
			6,000	12,000	25,000
8 periods (2-3 days each)	3 meters	Average◊	130	813	459
		Maximum◊	459	1,488	1,050
2 days (n=4 to 5 for each feedlot)	3 meters	Average□*	-	~590	~510
		Maximum□*	-	~880	~650
	100 meters	Average□*	-	~250	~280
		Maximum□*	-	~360	~450
	200 meters	Average□*	-	~90	~170
		Maximum□*	-	~170	~290

Source: McGinn et al 2003, in Ammonia Subgroup 2005

◊ 2-3 day averages

□ 2-day averages

*Numerical values estimated from Figure 2A in McGinn *et al.* (2003)

McGinn (2003) also measured NH_3 decay over distance. At 200 meters distant, NH_3 concentrations were reduced to between 65 and 82% of levels measured at the edge of the feedlot on two test days.

Atia et al (2004), Arogo (2001) and Krupa (2002)[8] reviewed ammonia decay and concluded that the lifetime of NH_3 in the atmosphere is short, between 2.8 hours and 5 days depending on the presence of moisture, co-pollutants and dry/wet deposition rates. Fifty and 70% reductions in airborne NH_3 have been measured at 0.6 and 4 km from sources, respectively. The presence of moisture and co-pollutants such as SO_2 and NO_2 promote the formation of ammonium (NH_4^+) and ammonium salts. Harper et al. (2005) showed that ammonia levels downwind of a swine facility fell close to normal background within a few hundred meters of the site in both winter and summer.

In addition to occupational risks, CFO researchers have raised concerns about respiratory health issues in residents near animal confinement facilities (WBK 2002).

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3 Hydrogen Sulphide (H_2S) and Reduced Sulphur Compounds

H_2S and other Reduced Sulphur Compounds (RSCs) are also discussed in the sections on Odours and VOCs. The reader is advised to read these two sections for information on H_2S and RSC generation and emissions, and on health related to odour. RSCs identified in these two sections include propyl, methyl, butyl, dimethyl mercaptans, H_2S, dimethyl disulphide and dimethlytrisulphide.

3.1 Health Effects

The U.S. National Research Council (NRC 2003) rated H_2S emissions from CFOs as a significant quality-of-human-life concern at the local scale, at the property line or the nearest dwelling.

H_2S and RSCs are microbially produced by the anaerobic decomposition of organic matter. At low concentrations, H_2S disrupts cellular energy production processes (i.e., inhibits ATP synthesis via mitochondrial oxidative phosphorylation). At low concentrations H_2S is primarily an eye and respiratory tract irritant. At high concentrations, it is neurotoxic and as concentration or exposure time increases, H_2S exposure effects are more serious and include eye damage, lung edema, unconsciousness and death. Paradoxically many people lose their ability to smell H_2S at 100 ppm and greater (Michigan 2006).

3.2 Animal Health Effects

High levels of H_2S can negatively affect animal health. Reports show that at 20 ppm the animal will start to show stress such as reduced feed intake and fear of light. At 200 ppm, pigs demonstrate pulmonary edema and breathing problems. Concentrations greater than 1000 ppm would result in death (Murphy and Cargill 2004).

Information on the effects of low concentrations of H_2S on animals is limited. Experimental studies have been conducted on animals such as cats, dogs, guinea pigs and rabbits. These studies found effects such as local irritation of eyes after many hours of exposure at about 100 ppm; eye and mucous membrane irritation occurs in one hour at 215 ppm, and systemic effects in less than one hour at 530 ppm. Death will result at concentrations of 1000 ppm for several hours, and in 15 minutes at concentrations of 2000 ppm (WHO 2000).

In 2006, the Western Interprovincial Scientific Studies Association (WISSA) completed a study of effects of oil and gas industry emissions on beef cattle. The study found that, within the range of exposures measured during the study, an increased frequency of calves receiving treatment was associated with exposure to H_2S. Also, further exposure of the calves to H_2S in their first month of life was associated with a 2% increase in the number of calves that received treatment after the first month, before they were turned out onto summer pasture.

3.3 Ecological Effects

Reduced sulphur compounds also have an effect on vegetation. Plants exposed to atmospheric sulphur-containing compounds may respond in a number of ways (Noggle et al. 1986):

- the sulphur may act as a fertilizer, enhancing growth and yield;
- there may be no response by the plant;

- biochemical and/or physiological changes may be triggered that can range from easily measured effects to those that are not detectable;
- visible injury may occur, which may or may not be accompanied by a decrease in growth and/or yield; and/or
- the plant's growth and/or yield may decrease.

The effects of reduced sulphur compounds on vegetation were most intensively studied from the 1980s through to the early 1990s (e.g., Kord et al. 1993b; de Kok et al. 1997; Chen and Paull 1998). Studies have focused on the effects of H_2S on plant growth and physiology, with a few publications reporting the impacts of other RSC (e.g., Ren et al. 1996; Obenland et al. 1998).

The type and extent of injury to plants by sulphur compounds depends on genetic, physiological and environmental factors; the specific conditions of exposure; the rate of entry of the pollutant into the plant; and the capacity of physiological processes within the plant to prevent the accumulation of toxic compounds (Noggle et al. 1986). The rate of entry or uptake depends on the physiology of the plant and environmental factors.

Taylor et al. (1983) compared the relative fluxes of SO_2, H_2S, COS, CS and methyl mercaptan (CH_3SH) into bush bean and soybean. The ranking of the five gases for internal flux via the stomata was $SO_2 > H_2S > COS > CS_2 > CH_3SH$. The greater the flux, the greater the potential for toxic effect. The authors suggest that this trend implies potential ranking for the relative toxicity of the compounds examined, although biological effects were not directly measured.

The effects of RSC may be differentially expressed among horticultural, agricultural and forest species, with different economic consequences. Effects on appearance of leaves and other plant parts would result in a negative economic effect on horticultural species (e.g., lettuce), and may have no economic consequences for agricultural or forest species. Effects on yield are the key concern for agricultural species, while effects on plant growth are the primary concern for forest species. For this reason, the effects of each of the RSC are assessed for each of these three plant groups in the following review and assessment.

The National Research Council of Canada (NRCC 1981) listed a variety of biochemical changes that occur in plants as a result of exposure to H_2S, including decrease in sugar, starch and chlorophyll levels, and stimulation or depression of several enzymatic activities and inhibition of NADH oxidation by mitochondria. It was suggested that these biochemical impacts occur due to the influence of H_2S on enzyme inhibition.

For more details on the effects of reduced sulphur compounds on vegetation, see Appendix C-1.

[Note: The text on ecological effects was adapted from the material provided by Laura, entitled "Effects of Reduced Sulphur Compounds on Vegetation," a section in the Assessment Report on Reduced Sulphur Compounds for Developing Ambient Air Quality Objectives. The remaining sections of that document contain additional details on specific chemicals and plants, and the text has been inserted as Appendix C-1.]

3.4 Ambient Air Quality Objectives

The table below shows the recommended objectives proposed in 2004 by the Alberta Environment RSC Subgroup to the Alberta Environment Ambient Air Quality Objective Working Group. On April 7 and June 7, 2004, the AQO Working Group deferred a decision on endorsement of the AQO recommendations pending a resolution on the development of a provincial Odour Management Framework.

Table 5. Current and Recommended AENV TRS Subgroup Ambient Air Quality Objectives

Reduced Sulphur Compound	**Subgroup Proposed ppb (µg/m^3)**		**Current AQO ppb (µg/m^3)**	
	1-hour	**24-hour**	**1-hour**	**24-hour**
Carbon Disulphide (CS_2)	10 (30) odour	-	10 (30) odour	-
Hydrogen Sulphide (H_2S)	5 (7) or 10 (14) odour	3 (4) health	10 (14) odour	3 (4) -
-Total Reduced Sulphur Compound (TRS)	10 (13)	3 (4) (consider)	-	-
Consider Development of a Qualitative Odour Management Framework	√	√	-	-

The Subgroup recommendations for H_2S were based on the multi-jurisdictional summary shown in Table 6. The recommended AQOs for carbon disulphide (CS_2) and TRS were linked to H_2S. Jurisdictionally the reference concentrations, or "no effect" levels for humans, for CS_2 are 0.3 ppm (0.8 mg/m^3) 24-hour average from ATSDR (1996), and 0.5 ppm (0.7 mg/m^3) 24-hour average EPA IRIS (IRIS 1995) for chronic exposures. Both reference concentrations for CS_2 are based on protecting against neurological effects as the most sensitive or critical adverse effect of exposure. The RSC Subgroup concluded that the toxicologies for methyl mercaptan, dimethyl sulphide, dimethyl disulphide, and carbonyl sulphide were not sufficiently developed to permit the development of an AQO (AENV SO_2 2004).

1 Table 6. Summary of Jurisdictional Protocols for Derivation of H_2S Health-Based Reference Concentrations

	EPA IRIS	Cal. EPA REL	Cal. EPA REL	ATSDR	ATSDR	ATSDR	WHO (CICAD)	WHO (CICAD)	Strickland	Alberta Health & Wellness
Publication Date	July 28, 2003	March 1999		2004	2004	July 1999	2003	2003	2002	2002
	Chronic RfC (Reference Concentration)	Acute REL (Reference Exposure Level)	Chronic REL (Reference Exposure Level)	Acute MRL (Minimal Risk Level)	Intermediate MRL (Minimal Risk Level)	Chronic MRL (Minimal Risk Level)	Short Term Tolerable concentration	Medium Term Tolerable concentration	ARE (Acute Reference Conc.)	Short-term exposure
Definition	As estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure of the human population including sensitive subgroups that is likely to be without appreciable risk of deleterious effects during a lifetime. Chronic: Repeated exposure by the oral, dermal, or inhalation route for more than approximately 10% of the life span in humans (more than approximately 90 days to 2 years in typically used laboratory animal species).	The level at or below which no adverse effects are expected. Exposure above this level may produce mild irritation of the eyes, nose, or throat, or may result in other mild adverse physiological changes. For most individuals, these symptoms do not persist after exposure ceases. This level is generally used as the Reference Exposure Level (REL).[1]	A chronic REL is an airborne level that would pose no significant health risk to individuals exposed to that level over a lifetime. RELs are based solely on health considerations and designed to protect the most sensitive individuals in the population by the inclusion of margins of safety.	MRL is an estimate of the daily exposure to a substance that is likely to be without appreciable risk of adverse noncancer health effects over the specified duration of exposure Acute (<14 days)	MRL is an estimate of the daily exposure to a substance that is likely to be without appreciable risk of adverse noncancer health effects over the specified duration of exposure Intermediate (>14-364 days)	MRL is an estimate of the daily exposure to a substance that is likely to be without appreciable risk of adverse noncancer health effects over the specified duration of exposure. Chronic (365 days and longer) exposure durations	Exposure durations of 1 to 14 days.	Exposure durations of up to 90 days.	For assessing health risk to the public for sensitive health effects due to single continuous exposures up to 24 hours duration	The review focused on: 1. health effects following short-term exposure which includes both acute and subacute exposures (few hours to a few days). 2. low dose of H_2S (up to 100 ppm). 3. inhalation exposure.
Critical Study / Effect	Brenneman et al. 2000 – nasal lesions (olfactory neurons) in rats. Exposed 6 hr/d, 7 d/wk for 10 wks. CIIT 1983 study previously used by EPA for RfC derivation.	Cal. State 1969, CARB 1984, Reynolds 1985, Amoore 1985. Odour threshold, headache and nausea considered.	CIIT 1983, nasal inflammation mice exposed 6 hr/d, 5 d/wk, 90 days.	Jappinen et al 1990.	Brenneman 2000 Rats, 6 hr/d, 7 d/wk, 10 wks to 0, 10, 30 & 80 ppm. Nasal lesions, inflammation & neuronal loss at 30 and 80 ppm. .	none	Jappinen et al 1990.	Brenneman 2000. Rats, nasal lesions: 0, 14, 42, 100 mg/m3 for 6 hr/d, 7 d/wk, 10 wks. Increased olfactory neuron loss and basal cell hyperplasia.	Categorical regression analysis of multiple studies, including Bhambhani, Jappinen, Skrajny, Hannah and Roth, Dorman (human & animals)	Jappinen et al 1990; Bhambhani, Y. and M Singh 1991

	EPA IRIS	Cal. EPA REL	Cal. EPA REL	ATSDR	ATSDR	ATSDR	WHO (CICAD)	WHO (CICAD)	Strickland	Alberta Health & Wellness
LOAEL		0012-0.069 ppm (geo mean 0.03 ppm), 1 hr assumed.	80 ppm	2 ppm (2 of 10 mild asthmatic adult volunteers showed changes in airway resistance >30% indicating airway obstruction. Results not statistically signif.	30 ppm	none	2 ppm (2.8 mg/m3) @ 30 min. 2 of 10 mild asthmatic adult volunteers showed changes in airway resistance >30% indicating airway obstruction. 3 reported headaches. Results not statistically signif.		Point of Departure = 95% LCL of the 10% probability of mild adverse respiratory effects (functional/ physiological impairment) [This is not a LOAEL]	2 ppm (2.8 mg/m3) @ 30 mins. 2 of 10 mild asthmatic adult volunteers showed changes in airway resistance >30% indicating airway obstruction. 3 reported headaches. Results not statistically significant. LOAEL = 2.0 ppm •Single exposure for 16 minutes @ 0, 0.5, 2.0, and 5.0 ppm. LOAEL = 5.0 ppm
LOAEL→ NOAEL Adjustment		-	-	3x	-		10x			
NOAEL (mg/m^3)	13.9 (10 ppm) 6 hr average	≤0.01 ppm (assumed)	30.5 ppm 6 hr average	0.7 ppm	10 ppm			14 mg/m3		Single exposure for 16 minutes @ 0, 0.5, 2.0, and 5.0 ppm. NOAEL = 2.0 ppm (?)
NOAELadj (mg/m^3)	3.48 (24 hr average)		5.4 ppm 24 hr average		2.5 ppm 24 hr			3.5 mg/m3 24 hour average		
NOAEL (HEC)	0.64		0.85 ppm		0.46 ppm			0.63 mg/m3		
Interspecies Extrapolation	3x	1	3x	-	3x			3x	(only human data used)	
Humans Variability	10x	1	10x	3x	10x		3x	10x	10x	
Subchronic to Chronic	10x		3x							
Modifying Factor	1x									
Health Based Criteria	1 ppb (2 ug/m^3) Annual average	30 ppb (42 ug/m^3) 1 hr average	8 ppb (10 ug/m^3) lifetime	200 ppb 24 hr average	20 ppb 24 hr average		71 ppb (100 ug/m^3) 24 hr average	14 ppb (20 ug/m^3) 24 hr average	ug/m^3 (ppb) 1hr 500 (360) 4 hr 300 (220) 8 hr, 200 (140)	

	EPA IRIS	Cal. EPA REL	Cal. EPA REL	ATSDR	ATSDR	ATSDR	WHO (CICAD)	WHO (CICAD)	Strickland	Alberta Health & Wellness
Additional Comments	LOAEL developmental (altered cerebellar Purkinje cells) 28 mg /m3 (Hannah & Roth 1999) and NOAEL (altered neurotransmitters in brains of in utero rats & pups) 28 mg/m3 (Skrajny 1992) are close to above Brenneman NOAEL and the RfC provides some assurance of protection for these other effects as well. Dorman (2000) examined fertility & developmental effects on rats exposed between 10 – 80 ppm 6 hr/d, 7 d/wk for 2 wks prior to & during gestation. No reproductive or developmental affects noted on a variety of parameters.	Cal. Panel of 16 persons: odour threshold 0.012-0.069 ppm (geo mean 0.029) with odour threshold set at 0.03 ppm. Amoore reported threshold of 0.0081 ppm and that 40% pop. would find 0.03 ppm objectionable. CARB: linked odour with headache and nausea. REL may be to be revised in future NOTE: The NOAEL was not used to derive the REL, therefore it assumes a certain proportion of the population will respond.	Odour threshold 10 ug/m3 (geom. mean) (Amoore 1985). 68% population expected to have threshold 2.5-40 ug/m3. Amoore (1985) review of lab. and sociological studies was that typically a factor of 3 above odour threshold for odour recognition. 5x above threshold produces annoyance (ie aesthetic, behavioural, and physiological response (ie nausea & headache)). Amoore predicted that at 10 ug/m3, 5% population annoyance response. At REL, most would detect but not adversely respond (believed to provide	Bhambhani (1996) No significant blood or physiol. in female volunteers at 5 ppm 30 minutes, 50% aerobic maximum. Males exhibited compromised aerobic metabolism (signif. Changes in lactate and activities of lactate dehydrogenase and cytochrome oxidase. Bhambhani 1991 no resp. or CV effects in exercising male volunteers down to 0.5 ppm, 16 minutes. Spolyar 1951. Respiratory distress in 2 workers exposed to 40 ppm for <25	Hannah & Roth altered Purkinje cell architecture & growth in offspring of pregnant rats exposed to 20 ppm, 7 hr/day 5th gestational day to day 21 postpartum. Skrajny 1992 showed changes in serotonin & norepinephrine levels in frontal cortex of rat offspring at 20 ppm. NOAELs not identified. CIIT 1983, similar design to above. Signf. wt. reductions at 80 ppm and in brain weights. No evidence of nasal histopathology.		The LOAEL was not adjusted to a 24 hr aver because for single exposures to high conc. of H2S, the response is concentration rather than time dependent (Guidotti 1996). Bhambhani (1991, 96, 97) reported metabolic effects volunteers @ 7 mg/m3 (30 mins) – oral inhalation only.	Respiratory, neurological and ocular effects are considered sensitive endpoints (with respiratory as major target). Sensitive populations identified as those with compromised respiratory function (eg asthmatics, elderly, children with compromised lung function).		

	EPA IRIS	Cal. EPA REL	Cal. EPA REL	ATSDR	ATSDR	ATSDR	WHO (CICAD)	WHO (CICAD)	Strickland	Alberta Health & Wellness
			reasonable protection against annoyance). Caution: other RSC have odour thresholds as much as 100x lower than H2S. Odour complaints poorly correlated to H2S at RSC sites.	mins. Lopez (1987), animals studies showed altered cell respiration at 10 ppm 40 minutes. Khan 1990 & 91 Significant dose response relationship at 50, 200 and 400 ppm (altered rat microsomal cytochrome & succinate oxidase activity at 200 and 400ppm, but not 50 ppm.			WHO ambient air quality guideline is 150 ug/m3 24 hr average, eye irritation. To avoid odour annoyance, 7 ug/m3 30 minute average. Although odour annoyance cannot be regarded as an adverse health effect in a strict sense, it does affect the quality of life. Therefore, odour threshold levels have been indicated where relevant and used as a basis for separate guideline values. The problems of irritation (for example, of the skin) and headache were also considered as possible problems of annoyance. It was agreed that headache should be regarded as a health endpoint and not merely as a matter of annoyance.[2] Defn: annoyance = acceptability and annoyance, where the *nuisance threshold level* is defined as the concentration at which not more than a small proportion of the population (less than 5%) experiences annoyance for a small part of the time (less than 2%); since annoyance will be influenced by a number of psychological and socioeconomic factors, a nuisance threshold level cannot be defined on the basis of concentration alone.		

1 OEHHA's Acute RELs are intended to be compared to the modeled one-hour maximum (or multi-hour as noted for specific reproductive/developmental toxicants) concentrations used in the hazard index approach to risk assessment OEHHA recommends that these acute RELs be used to evaluate exposures that occur no more frequently than every two weeks in a given year. The two-week interval was chosen because in most acute toxicology experiments two weeks is the duration of time an animal is observed for signs of adverse outcome following exposure (Cal. EPA, 1999).

2 WHO 2000. Ambient Air Quality Guidelines. Part 1, Chapter 2, Page 8.

Conversion Factor: 1 ppm H_2S= 1.4 mg/m^3. 1 mg/m^3=0.71 ppm

The California EPA (Section 3.3.2,Chronic REL methodology document, 2000) acknowledges the US EPA protocol of applying an adjustment or uncertainty factor of between 1 to 10 times for using subchronic studies (i.e., for exposure that are <10% of average life-span) in deriving chronic RfCs. Because of the subjectivity involved in applying a specific adjustment, the California EPA tightened the application criteria:

- 10 x adjustment for exposure periods <8% of expected lifetime
- 3 x adjustment for exposures periods 8-12%.
- 1x adjustment for exposures periods >12%.

Reference: California Environmental Protection Agency, Air Toxics Hot Spots Program Risk Assessment Guidelines Part I The Determination of Acute Reference Exposure Levels for Airborne Toxicants, http://www.oehha.ca.gov/air/pdf/acuterel.pdf, Air Toxicology and Epidemiology Section, Office of Environmental Health Hazard Assessment, 1999 pp 71.

3.5 H_2S and Reduced Sulphur Compounds and CFOs

Alberta Environment (2002) reported the following RSC emissions from anaerobically stored manure from a 1999 study by Clanton and Morey.

Table 7. Measurement Results of RSC in Air Samples at Manure Storage Site

RSC	No. of Samples	Minimum (ppb)	Maximum (ppb)	Mean (ppb)
Carbonyl sulphide	36	2.9	35.1	10.9
Methyl mercaptan	13	1.9	26.9	8.5
Dimethyl sulphide	8	2.2	44.4	8.6
Carbon disulphide	49	1.9	405	32.3
Dimethyl disulphide	7	1.2	6.5	2.7
Hydrogen sulphide	48	4	2820	445

Source: Clanton and Morey 1999 from AENV 2002

The reported odour thresholds for these compounds (AENV 2002) are:

- methyl mercaptan 1.6 ppb (3.2 µg/m^3)
- dimethyl sulphide 1 to 63 ppb (2.5 µg/m^3 to 160 µg/m^3)
- carbon disulphide, 16 ppb (50 µg/m^3)
- dimethyl disulphide 0.8 - 4 ppb (3 - 16 µg/m^3)
- hydrogen sulphide 20-35 ppb (30-50 µg/m^3)
- ethyl mercaptan 1 ppb (2.5 µg/m^3)
- propyl mercaptan 0.75 ppb (2.3 µg/m^3)
- n-butyl mercaptan. 0.1 to 1 ppb (0.2 to 2 µg/m^3)

Odour thresholds for H_2S of 5 ppb (7 µg/m^3) (Nagy 1992) and 2 ppb (3 µg/m^3) (Auvermann 2002), 8 ppb (Michigan 2006), and a range of 0.5 to 30 ppb (ATSDR 2006) have also been reported. The AENV 1-hour Ambient Air Quality Objective for H_2S of 10 ppb (14 µg/m^3) is based on the odour threshold.

References

Missing references: ATSDR 2006; Auvermann 2002; Michigan 2006; Nagy 1992; NRC 2003
Plus all the ones noted under Ecological Effects.

AENV 2002. Assessment Report On Reduced Sulphur Compounds For Developing An Ambient Air Quality Guideline. AMEC Earth & Environmental Limited and University of Calgary.

AENV SO_2 2004. Summary Report Reduced Sulphur Compounds (RSC) Subgroup. AENV Ambient Air Quality Objective Working Group, SO_2 Subgroup. January 2004.

ATSDR 1996. Toxicological Profile for Carbon Disulfide. Agency for Toxic Substances Disease Registry. U.S. Public Health Service. August 1996.

IRIS 1995. Carbon Disulfide. Integrated Risk Information System. U.S. EPA. http://www.epa.gov/iris/subst/0217.htm. Accessed March 30 2007.

4 Volatile Organic Compounds (VOCs)

Volatile organic compounds (VOCs) are organic compounds that volatilize into the air easily at room temperature (NRC 2002). Like hydrogen sulphide, total reduced sulphur compounds, and ammonia, VOCs can have an effect themselves, and they can contribute to an odour combination that can have an effect. Both odour intensity and total VOCs can be measured.[9] The U.S. National Research Council (NRC 2003) ranked VOC emissions from animal feeding operations and their ability to cause local effects (i.e., at the property line or the nearest dwelling) as minor. The primary effect of concern was on quality of life.

4.1 Health Effects

Michigan (2006) and NRC (2002) note that studies by Wing and Wolf (2000), Thu et al (1997) and Schiffman et al. (1995) provide evidence that VOC effects can include mood disturbance and headache, excessive coughing, burning eyes, and diarrhea as compared to a control group. However, NRC suggests caution in interpreting these studies because environmental data, including VOC levels, were not determined. The causal factors for these measured effects are uncertain, although the effects, even in studies where the confounders were considered, remained.

A study by Beck et al (2007) discusses total VOCs (TVOCs) on farms and TVOC health effects. They draw some useful conclusions, noting, for example, "Generally it was likely that the concentrations of VOCs were too low to have health effects on their own. On the other hand, the VOC concentrations were in a multifactor range in which health effects could occur depending on the interaction with other exposure factors."

Schiffman (1998; in Johnston and Weibel 2006) identified four ways by which VOCs can adversely affect humans.

- VOCs can irritate eyes, nose, throat and cause headaches and drowsiness.
- VOCs can produce reversible or irreversible effects in organs and tissues (beyond simple irritation).
- VOCs can affect neuro-chemical activity which can impair mood and performance.
- Odours can trigger memories that can affect cognitive function, altering one's emotional state and mood.

4.2 Animal Health Effects

None noted.

[9] See the section on Odour for more information on this aspect.

4.3 Ecological Effects

The following information on the impacts of various VOCs is derived from assessment reports for ambient air quality objectives effects on vegetation.

2-Ethylhexanol

Little is known about the direct effects of VOCs on plants. A literature search resulted in the identification of only one research article on the effect of 2-ethylhexanol in liquid media on algae. Nothing has been reported for effects on terrestrial vegetation.

Ethylbenzene

No air studies

Isopropanol

No air studies.

Toluene

Chlorosis and growth inhibition in plants may occur at toluene air concentrations of <6,000 mg/m^3 (CEPA 1992).

Xylene

Barley exposed to 20,000 mg/m^3 of xylene vapour for four hours displayed 80% injury of leaves within 24 hours (Currier 1951; Currier and Peoples 1954).

4.4 Ambient Air Quality Objectives

California developed an acute inhalation reference exposure level (REL) for phenol of 5.8 mg/m^3 (1.5 ppm) 1-hour average based on preventing irritation of the eyes, nose and throat, based on controlled adult exposure study (OEHHA 1999). The odour threshold is 150 $\mu g/m^3$ (40 ppb). The acute REL is a concentration that is not likely to cause adverse effects in a human population, including sensitive subgroups, exposed for a period of one hour (Cal EPA 1999).

The California chronic inhalation REL for cresol of 600 $\mu g/m^3$ (100 ppb) is based on preventing neurotoxic effects, such as salivation, rapid respiration, hypoactivity, urination and tremors (OEHHA 2000). The chronic REL is based on a laboratory animal study. For phenol the chronic REL is 200 $\mu g/m^3$ (50 ppb) and is based on preventing toxic effects in the nervous, digestive and circulatory systems and, kidney. The critical effects of concern were muscle tremor, neurological impairment and elevated blood serum liver enzymes.

The U.S. Agency Toxic Substances and Disease Registry (ATSDR) Minimal Risk Level or MRL for phenol is 77 $\mu g/m^3$ (20 ppb) 24-hour average for exposures greater than 1 year (ATSDR 2006). Phenol is a recognized respiratory irritant. MRLs are estimates of the daily concentration that the public, including vulnerables, can be exposed to without adverse effect. The odour threshold is reported as 40 ppb.

Ontario has developed ambient air quality criteria and standards for some VOCs, as indicated in the table below.

Table 8. The Ontario Ambient Air Quality Criteria and Standards for Substances of Interest

Substance	Standards (µg/m^3)		Criteria (µg/m^3)			
	½-hour	24-hour	½-hour Point of Impingement	24-hour	1-hour	10-minute
Acetic acid	2,500 (odour)	-	-	2,500 (to be updated)	-	-
Benzyl alcohol	-	-	2,460	880	-	-
iso-Butanol	-	-	1,940 (odour)	-	15,000 (health)	2,640
n-Butanol	-	-	2,278 (odour)	-	15,000 (health)	3,100
Phenol	100 (health)	30 (health)	-	-	-	-
Propionic acid	-	-	100 (odour)	-	100 (to be updated)	-

Source: OME 2005

4.5 VOCs and CFOs

VOCs and volatile fatty acids (VFAs) emitted from concentrated animal feeding operations are a mixture of various organic acids, esters, alcohols, aldehydes, ketones, halogenates, amines, and hydrocarbons (EPA 2001). Odorous VOCs emitted from CFOs include the volatile acids (acetic, propionic, formic, butyric, and valeric), indole, phenols, volatile amines, methyl mercaptan, and skatole (EPA 2001). Of these compounds, only phenol and cresol have well-developed toxicologies; methyl mercaptan is discussed in the section on H_2S and Reduced Sulphur Compounds.

Emissions characteristics depend on the type and age of the animals, type and quality of feeds, and operational practices. The manure characteristics will depend on the preceding variables and will influence the emissions characteristics of the manure. Relevant operational variables include how the manure is collected, stored, and land applied.

The concentration and quality of VOC emissions depends on the incomplete and anaerobic decomposition of waste organic matter (Michigan 2006). VOC emissions are insignificant both during aerobic conditions and anaerobically when methanogenic bacteria are not inhibited (see also Zahn 2001, Odour Chapter). Under balanced anaerobic conditions, organic wastes are converted to simple organic acids and VOCs that are metabolized to methane and carbon dioxide by methanogenic bacteria. However, inhibition of methanogenic bacteria increases the formation and volatilization of VOCs. For more information on management mechanisms to control emissions from CFOs, see Appendix D-1.

The table below provides more information on VOCs in livestock wastes.

Table 9. Volatile compounds identified in livestock wastes with documented Acute or Chronic Inhalation Toxicity Values

VOLATILE COMPOUNDS IDENTIFIED IN LIVESTOCK WASTES WITH DOCUMENTED ACUTE OR CHRONIC INHALATION TOXICITY VALUES

Compound	Acute Toxicity Values			Chronic Toxicity Values				
				Cancer		Non-cancer		
	Toxicity Value ($\mu g/m^3$)	Toxic Endpoint	Source	Toxicity Value ($\mu g/m^3$)	Source	Toxicity Value ($\mu g/m^3$)	Toxic Endpoint	Source
Acetaldehyde	---	---	---	5	MPCA IHRV[1]	9	Upper respiratory system	USEPA RfC
Acrolein	0.19	Irritant - eye	Cal-OEHHA REL [2]	---	---	0.02	Upper respiratory system	USEPA RfC
Benzene	1,000	Developmental	MPCA IHRV	1.3	MPCA IHRV	60	Nervous system; blood; developmental	Cal-OEHHA REL
2-Butanone (methyl ethyl ketone)	10,000	Irritant - eye and respiratory system	MPCA IHRV	---	---	1,000	Developmental	USEPA RfC
Carbon disulfide	6,000	Developmental	MPCA IHRV	---	---	700	Nervous system	MPCA IHRV
Chloroform	100	Developmental	MPCA IHRV	0.4	USEPA	300	Liver; kidney; developmental	Cal-OEHHA REL
Formaldehyde	94	Irritant - eye and respiratory system	MPCA IHRV	0.8	MPCA IHRV	3	Respiratory system; eyes	Cal-OEHHA REL
Hexane	---	---	---	---	---	2,000	Nervous system; upper respiratory system	MPCA IHRV
Methanol	25,000	Central nervous system	MPCA IHRV	---	---	4,000	Developmental	Cal-OEHHA REL
2-Methoxyethanol (ethylene glycol methyl ether)	90	Developmental	MPCA IHRV	---	---	20	Reproductive	USEPA RfC
Naphthalene	---	---	---	---	---	3	Upper respiratory system	MPCA IHRV
Phenol	5,800	Irritant - eye and respiratory system	MPCA IHRV	---	---	200	Liver; cardiovascular; kidney; nervous system	Cal-OEHHA REL
Tetrachloroethylene (perchloroethylene)	20,000	Irritant - eye and respiratory system; central nervous system	MPCA IHRV	17	USEPA	35	Liver and kidney	Cal-OEHHA REL
Toluene	37,000	Irritant - eye and respiratory system; central nervous system	MPCA IHRV	---	---	400	Nervous/upper respiratory system	MPCA IHRV
Triethylamine	2,800	Irritant - eye; transient corneal edema	MPCA IHRV	---	---	7	Upper respiratory system	USEPA RfC
Xylenes	22,000	Irritant - eye and respiratory system; central nervous system	MPCA IHRV	---	---	700	Nervous/upper respiratory systems	Cal-OEHHA REL

Notes:
1 The Inhalation Health Risk Values (IHRVs) shown are from a draft document (MPCA-MDH, 2000), subject to final review and approval.
2 The California Office of Environmental Health Hazard Assessment (OEHHA), the group tasked with reviewing and updating the list of "Proposition 65" chemicals, developed the Reference Exposure Levels (RELs) shown above.

Source: Minnesota 2001

Researchers have suggested that between 100 and 330 different VOCs and VFAs are generated depending on the type of animals and the practices found at each concentrated animal feeding operation (Michigan 2006). The major constituents that have been qualitatively identified include organic sulphides and disulphides, C_4 to C_7 aldehydes, trimethylamine, C_4 amines, quinoline, dimethylpyrazine, and C_3 to C_6 organic acids. Minor constituents include C_4 to C_7 alcohols, ketones, aliphatic hydrocarbons, and aromatic compounds (NRC 2002). Some may irritate the skin, eye, nose, and throat on contact and the mucous membranes if inhaled. VOCs can also be precursors to O_3 and $PM_{2.5}$. Some studies (NRC 2002) have found that the 27 most prevalent VOCs could be classified as phenols, indoles, alkanes, amines, fatty acids, and sulphur-containing compounds (Michigan 2006).

As measured by their odour thresholds, phenol, cresol and their relatives, which are described as having a medicinal odour quality, are among the strongest odorants associated with livestock manure (Auvermann 2002). Alcohols, ketones and aldehydes are regarded as "sweet or pungent" in odour quality while reduced sulphur compounds such as mercaptans and H_2S are regarded as having "rotten" or "rotten egg" odours. Amine compounds produce odours characterized as "fishy or pungent," and are also prominent odorants. Reduced sulphur compounds and amines are by-products of protein decomposition.

A more detailed description of VOCs from confined feeding operations and their relationship to odour is included in Appendix D-1. A comprehensive list of CFO VOCs is presented in Appendix D-2.

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Note: Are the two Zahn references really JA Zahn and JL Zahn? If so, that distinction should be made in the text. If they are the same person, then please use a) and b) to distinguish them here and in the citation.

5 Particulate Matter (PM)

Airborne particulate matter (PM) can be broadly classified into two categories based on size: fine and coarse. Fine PM, or $PM_{2.5,}$ refers to airborne particulates that are 2.5 µm or less in diameter. Coarse PM, or $PM_{10\text{-}2.5}$, are particulates with a diameter between 2.5 and 10 µm. Another commonly cited PM category is PM_{10}, which includes all particles that are 10 µm or less in diameter. Although fine and coarse PM are mutually exclusive, PM_{10} includes the $PM_{2.5}$ and $PM_{10\text{-}2.5}$ subfractions.

PM does not have a specific chemical composition but can be further characterized based on the originating source and biological effect. $PM_{2.5}$ is primarily a result of carbon-based combustion processes such as forest fires, wood burning fireplaces and stoves, natural gas furnaces, vehicle engines, and boilers. $PM_{2.5}$ formation also results from the condensation or reaction of other combustion or high temperature by-products, such as vehicle exhaust, smelting and metals manufacturing.

$PM_{10\text{-}2.5}$ is generally produced by mechanical processes such as crushing or impact, and includes wind erosion and resuspension from natural sources such as windblown dust of geological origin, including road dust and agricultural practices, sea salt particulates, vegetation-derived particulates including seeds, pollens, spores, leaf waxes and resins, mining, and quarry operations. $PM_{10\text{-}2.5}$ is also produced from forest fires.

5.1 Health Effects

Inhaled $PM_{2.5}$ can penetrate deep into the lungs at the level of the alveolus or the lung terminus. The alveoli are where oxygen exchange occurs between the air and the blood, with the circulatory system distributing the oxygenated blood throughout the body. In contrast, $PM_{10\text{-}2.5}$ is preferentially deposited in the upper airways – the nose, throat and tracheobronchial area. Whereas the effects of $PM_{10\text{-}2.5}$ may be generally restricted to the nose and upper airways, the involvement of the circulatory system with $PM_{2.5}$ provides for distribution of $PM_{2.5}$ constituents to organs and tissues throughout the body and thus, for the manifestation of a more varied toxic response.

Some studies suggest that exposure to both PM_{10} and $PM_{2.5}$ may be associated with increased mortality from cardio-respiratory diseases and increased morbidity due to increased hospitalization for cardio-respiratory diseases, decreased lung function in children and asthmatic adults, and chronic effects including reduced lung function and capacity in children and increased development of chronic bronchitis and asthma in some adults (BC Environment 2003).

The preferential deposition of $PM_{10\text{-}2.5}$ in the upper airways is associated with irritation and inflammation of the upper respiratory tract, including aggravation of asthma. The presence of particulates of biological origin in $PM_{10\text{-}2.5}$ may predispose sensitized individuals to an allergic response, independent of asthma.

Exposure to $PM_{2.5}$ may be associated with decreased lung function as well as increases in:

- Mortality for respiratory and cardiovascular causes,
- Hospital admissions and emergency room visits for respiratory and cardiovascular reasons,
- Pneumonia and aggravation of chronic obstructive pulmonary disease,
- Aggravation of asthma, and

- Symptom presentation for cough association with lower and upper respiratory effects.

Studies supporting the above $PM_{2.5}$ health effects and concerns include human epidemiological studies, although animal studies have also contributed. The vast majority of the epidemiological studies have focused on PM originating from urban or metropolitan areas around the world, including Canada and the U.S. In rural areas, the applicability of $PM_{2.5}$ ambient air quality criteria should be applied cautiously, as $PM_{2.5}$ in rural areas is not as dominated by combustion particulates or high-temperature processes as it is in urban and metropolitan areas. However, the Alberta PM and Ozone Management Framework (see section 5.4) applies throughout Alberta. The Framework provides a mechanism for backing out (i.e., subtracting) non-anthropogenic PM for determining compliance to the various triggers levels, which are 15, 20 and 30 µg/m^3 24-hour average. In addition, consideration should be given to the application of ambient air quality guidelines for coarse ($PM_{10-2.5}$) or inhalable PM_{10} fractions in rural areas, as the coarse fraction is dominated by road dust, agricultural sources, wind blown dust and bioaerosols including pollen and spores. The inhalable PM_{10} ambient air quality guideline of B.C. (50 µg/m^3 24-hour average) may provide guidance.

In a 2004 non-occupational study, healthy and mildly asthmatic subjects were exposed to NH_3 and endotoxin for three 30-minute periods over three weeks. No significant change in lung function was detected among healthy volunteers at the end of the exposure regime. In asthmatics, a significant transient decrease in lung function and increased bronchial hyperactivity was induced by grain dust alone. The duration of lung function decrements were increased by two times for co-exposure to NH_3 and grain dust versus grain dust alone. NH_3 at 16 to 25 ppm had no effect on mild asthmatics.

5.1.1 Occupational Exposure

Occupational exposure to organic dusts are associated with asthma, rhinitis, bronchitis, hypersensitivity pneumonitis (HP) and organic dust toxic syndrome (ODTS). ODTS is associated with flu-like symptoms including fever, chills, headache, cough, breathing difficulty, muscle aches and nausea. The development of ODTS is associated with a single and intense exposure to bacteria and moulds (New York City Department of Health 2002). Co-exposure to bacterial or fungal spores and endotoxins may be synergistic in the development of ODTS (Kirkhorn et al. 2000). Unlike ODTS, HP is an allergic reaction to bacterial and fungal allergens (Minnesota 2001) and can result in permanent lung damage. HP is typically associated with repeated heavy exposures in agricultural settings (New York City Department of Health 2002).

High dust levels associated with floor feeding of hogs, indoor feed grinding and the use of high moisture corns has been associated nasal irritation, coughing, wheezing, and dyspnea in workers. Working with liquid manure was also associated with these symptoms. Epidemiologic studies of swine facility workers have documented increased morning phlegm, coughing, scratchy throat, burning eyes, wheezing, shortness of breath, and chronic bronchitis, compared to individuals not working in these facilities (Cole et al. 2000).

The Michigan Department of Environmental Quality (2006) reported a strong and consistent dose-response relationship for CFO workers exposed to PM and endotoxin, with adverse respiratory effects and compromised lung function. Similarly, exposure studies in poultry workers have demonstrated a strong correlation between NH_3 and PM and respiratory effects. NH_3 is believed to damage the cilia providing muco-ciliary clearance in the upper airways resulting in increased

respiratory susceptibility to irritation and other effects. In addition to NH_3, other gases such as H_2S may adsorb onto dust particles and cause nasal and respiratory irritation (Minnesota 2001).

Michigan (2006) cites the work of Donham et al. and Vogelzang et al. on swine and poultry workers published in 2000. Based on associations between exposure and lung function in swine workers, a workplace threshold value of 5.4 mg NH_3/m^3 and 2.8 mg/m^3 inhalable (PM_{10}) organic dusts as work shift averages were recommended for confinement buildings. For poultry workers, the following work shift averages were associated with significant pulmonary function decrements: 2.4 mg/m^3 total dust, 0.16 mg/m^3 respirable ($PM_{2.5}$) dust, 614 endotoxin units/m^3 (EU/m^3) total endotoxin, 0.35 EU/m^3 respirable endotoxin, and 12 ppm ammonia.

A cross-sectional study of swine workers by Zejda in 1993 (Cole 2000) found that workers using dust masks had a lower prevalence of chronic respiratory symptoms. In addition, workers had better lung function than those who did not wear masks. However, if workers began wearing masks because they were already experiencing symptoms, the lung function test results were comparable to workers who did not wear masks.

Ontario (1997) in a 1993 compilation of studies from Australia, Finland, Denmark, Sweden, Scotland, the U.S. and Canada found high levels of occupational respiratory illness in farm workers (see the table below). For instance, 1 to 2 dairy producers in 10 has chronic bronchitis, 1 in 20 has asthma and 1 in 18 will develop Farmer's Lung. Ontario estimated that 0.5 to 1% of dairy farm workers have or will experience reduced pulmonary capacity reduction over their farming career. Suspected agents are airborne dusts originating from mouldy hay, straw and grain. Pig farmers were identified at higher risk and suffer from bronchitis, occupational asthma and from the organic dust toxic syndrome. Poultry building operators were considered at risk because of exposures to high levels of airborne dust and endotoxins. Ontario recommends farm workers wear respiratory protection such as a face mask or positive pressure respirators, especially during feeding and animal handling.

Table 10. Statistics on the potential respiratory problems for farm workers (percent ranges)

Suspected Conditions	Dairy	Pork	Poultry	Known Symptoms
Bronchitis - Acute	N/A	70-90 %	15-25 %	Cough, phlegm, tightness of chest shortness of breath, wheeze
Bronchitis - Chronic	10-20 %	15-30 %	8-15 %	Cough, phlegm, tightness of chest shortness of breath, wheeze
Occupational Asthma	4-7 %	20-30 %	5-10 %	Tightness of chest, shortness of breath, wheeze
Organic Dust Toxic Syndrome (ODTS) - Acute or Chronic	N/A	20-30 %	N/A	Febrile episodes, headaches, muscle aches, flu-like illness, shortness of breath
Farmer's Lung	2-10 %	N/A	N/A	Same as ODTS

Source: Ontario 1997

Confinement worker exposures to airborne dusts originating from antimicrobial-containing feeds, contaminated animal wastes, and contaminated animal tissues can result in the transfer or development of antimicrobial-resistant bacteria in animals and workers (Cole 2000). Levy (1978) reported the emergence of tetracycline-resistant bacteria in poultry within 36 hours of the introduction of a tetracycline-containing feed. Tetracycline resistant bacteria developed in workers 4 to 6 months later. Similarly, Marshall (1990) reported the isolation of a resistant strain of *E. coli* in a worker following injection of the strain into swine. Ontario (OME 2005) has an ambient air quality criterion for penicillin of 0.1 mg/m^3 24-hour average based on health.

Epidemiologic studies (Cole 2000) have generally shown that farmers and abattoir workers have higher incidences of antimicrobial-resistant bacteria than other workers. Nijsten (1994) found that pig farmers had the highest prevalence of antimicrobial resistance in fecal isolates compared to slaughterhouse workers and suburban residents from the same geographic area. Similarly, a Japanese study found a higher prevalence of antimicrobial resistance in pig breeders and slaughterhouse workers than in urban controls. The patterns of resistance in the swine and slaughterhouse workers suggested the transfer of bacterial genetic material between the animals and workers.

More symptoms of chronic bronchitis and asthma and more missed work days are reported by swine confinement workers than controls (Cole 2000). Documented symptoms include wheezing, coughing, sinusitis, fever, chest tightness, nasal irritation, phlegm, throat irritation, and sneezing.

Most confinement worker exposure studies have found a correlation between one or more contaminants and worker lung function and/or respiratory symptoms (Cole 2000). Most correlations occurred with dusts, endotoxin and NH_3.

Healthy, non-smoking and previously unexposed volunteers developed a variety of symptoms after several hours of exposure to swine dust in a CFO. Symptoms included cough, nasal stuffiness, moderate chills, headaches, muscle pain, mental fatigue, malaise, and nausea (Cole 2000).

5.2 Animal Health Effects

The effects of dust on animals depend on the particle size; larger particles (20 µm) can be trapped in the nasal cavity while those less than 10 µm may travel to the trachea and large bronchi in pigs (Murphy and Cargill, 2004). The effects of dust on animals are difficult to quantify because of the different sizes, what the dust carries with it and where the dust is from. Feed is a major source of dust, however most feed particles are between 10 and 100 µm and will have little effect on respiratory health. Dust levels vary depending on ventilations and air movement, humidity, management of manure, and even seasonal variations. Reduced performance and depressed growth rates were noticeable in pigs exposed to 5.2 or 9.9 mg/m^3 of dust (Murphy and Cargill 2004). Research by Holland (2002) also shows a reduced growth rate in pigs from higher levels of particulate matter.

5.3 Ecological Effects

Nothing noted

5.4 Ambient Air Quality Objectives and Guidelines

The Federal-Provincial Working Group on Air Quality Objectives and Guidelines identified reference levels of 25 µg/m^3 (24-hour average) for PM_{10} and 15 µg/m^3 (24-hour average) for $PM_{2.5}$ (Health Canada 1999). These reference levels represent estimates of the lowest ambient PM levels at which statistically significant increases in health effects have been demonstrated. However, they are not to be interpreted as effects thresholds, nor are they intended to be used as air quality management targets. The reason that the reference concentrations are not to be used as health effects thresholds is because linearity is continued below the reference concentrations but with the loss of statistical significance. Evidence suggests that health effects occur below the reference concentrations (Devlin et al. 2003; Lipsett et al. 2006; Timonen et al. 2005).

5.4.1 $PM_{2.5}$

The CCME Canada Wide Standard (CWS) for $PM_{2.5}$ is 30 µg/m^3 24-hour, which includes considerations of public health protection balanced against economic and technological factors. The CWS was developed in consultation with Health Canada's assessment of $PM_{2.5}$ toxicology and public health protection. Compliance with the CWS is determined by the annual 98th percentile averaged over three years.

The CWS recognizes that health effects can occur below 30 µg/m^3 24 hour average and that the CWS should not be used as a level that can be polluted up to. Importantly, the CWS incorporates two important principles, Keeping Clean Areas Clean and Continuous Improvement; that is, $PM_{2.5}$ levels in relatively undeveloped areas should not be allowed to deteriorate, and continuous improvement should be the goal in other areas, achieved in part through the incorporation of Best Available Economically Feasible Technology (BAEFT) to reduce emissions. These principles are also recognized in the Alberta PM and Ozone Management Framework (CASA 2003) developed through a CASA multi-stakeholder, consensus-based process involving various levels of government, including Alberta Environment, municipalities, health regions, industry, and non-government organizations.

Although Alberta does not currently have an ambient air quality objective for $PM_{2.5}$, an objective is in the development stage. Alberta's PM and Ozone Management Framework is a guidance document on identifying and managing sources of $PM_{2.5}$ in a geographic area such that the principles of the CWS are adhered to. The Framework describes three triggers, two of which are of particular interest: the Management Trigger and the Exceedance Trigger. Above the Management Trigger (that is, above 20 µg/m^3 but below 30 µg/m^3 24 hour) emission and source reduction strategies are required to prevent further deterioration in ambient air quality and to prevent levels from reaching 30 µg/m^3. Above 30 µg/m^3 24 hour – the Exceedence Trigger – actions are required to bring ambient levels down to below 30 µg/m^3 and into the management zone.

The Alberta PM and Ozone Management Framework, using the Canada Wide Standard, recommends the following health-effects based levels:

1. 15 µg/m^3 24-hour, the threshold for initiating surveillance or baseline monitoring; consisting of trend analysis of ambient PM, source identification and characterization and, if possible, emissions management in accord with CI and KCAC provisions of the CWS and Framework.
2. 20 µg/m^3 24-hour, threshold for initiating pollution reduction strategies to prevent further deterioration in air quality. Management strategies are diverse and include targeted emissions

reductions in associated with air quality trend analysis and forecasting, the identification of contributing sources, and the incorporation of BAEFT.

3. 30 µg/m^3 24-hour, level not to be exceeded. Mandatory development and application of an air quality and emissions management plan.

5.4.2 $PM_{10-2.5}$ and PM_{10}

Alberta does not have an ambient air quality objective for $PM_{10-2.5}$ or PM_{10}. The British Columbia ambient air quality objective for PM_{10} is 50 µg/m^3 24-hour average, but BC has no objective for $PM_{2.5}$. The BC government cites studies suggesting that exposure to both PM_{10} and $PM_{2.5}$ may be associated with increased mortality from cardio-respiratory diseases and increased morbidity due to increased hospitalization for cardio-respiratory diseases, decreased lung function in children and asthmatic adults, and chronic effects including reduced lung function and capacity in children and increased development of chronic bronchitis and asthma in some adults (BC Environment 2003). The U.S. EPA (2006) has proposed a $PM_{10-2.5}$ national ambient air quality standard of 70 µg/m^3 24-hour based on increased morbidity and mortality effects. These effects include increased hospital admissions for respiratory symptoms and heart disease, decreased lung function, and increased premature death.

Health Canada (2001) cites evidence supporting the role of $PM_{10-2.5}$ in the development and expression of upper airway symptoms from epidemiological studies of respiratory symptoms, lung function and hospital admissions. Clinical studies have sometimes found effects and other times not (CCME 2004). The evidence for increased mortality from $PM_{10-2.5}$ exposure was considered equivocal. Health Canada continues to consider the need for and development of an ambient air quality numeric for $PM_{10-2.5}$. Knowledge gaps requiring resolution include source–receptor relationships, pathways of exposure, clarity on specific health effects, and the availability and effectiveness of mitigation measures.

5.5 PM and CFOs

The U.S. National Research Council (NRC 2003) identified animal feeding operations as a source of both PM_{10} and $PM_{2.5}$ on a local geographic scale, at the property line or nearest dwelling. The primary effects of concern for $PM_{2.5}$ were health and haze and, for PM_{10}, haze.

Primary PM sources from CFOs include feed, bedding materials, dry manure, unpaved soil surfaces, animal dander, poultry feathers, animal activity, animal housing buildings and exhaust fans, mineral and organic material from soil, manure, and water droplets generated by high-pressure liquid sprays (US EPA 2001; NRC 2003). Other important variables include the amount of mechanical and animal activity of the soil and manure including the moisture content and in-situ size fractionations. Potential PM emission sources for confinement operations are the confinement building, dry manure storage, and land application sites. The relative significance of each source depends on three interrelated factors: 1) the type of animal being raised, 2) the design of the confinement facility being utilized, and 3) the method of manure handling (U.S. EPA 2001). Secondary PM sources, that is, gases converted to aerosols by later atmospheric reaction into $PM_{2.5}$, include NH_3, NO, and H_2S (NRC 2003).

PM produced from livestock housing operations can contain viable particles (bioaerosols), such as pathogenic bacteria, and viruses, and endotoxins, which are capable of lodging in the respiratory

system and have the potential to cause detrimental, and sometimes serious, respiratory effects (Roumeliotis and Heyst 2006).

The size distribution of PM associated with CFOs is not well developed, but limited information suggests that the $PM_{2.5}$ fraction is minor (EPA 2001). Particle size distribution data was found only for beef feedlots. In one study, ambient measurements of PM_{10} and $PM_{2.5}$ were taken downwind (15 to 61 metres) of three cattle feedlots in the Southern Great Plains (Sweeten et al 1998). In this study, PM_{10} was measured as 20 to 40% of TSP and $PM_{2.5}$ was 5 percent of TSP.

Although CFO dust contains both inorganic and organic fractions, the organic fraction is the most significant and of greatest health concern (Minnesota 2001). The composition of the PM organic fraction from CFOs varies and can include animal dander, fungi and other allergens, endotoxins, mycotoxins, (1→3)-ß-*D*-glucan and fugitive dusts consisting of particles of feathers, manure, feed grains, dried forage, and silage (US EPA 2001; Minnesota 2001). The inorganic fraction consists of local soils components and may include silicates, carbonates and crystalline silica (Michigan 2006).

More information on monitoring and managing PM in CFOs can be found in Appendix E-1.

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6 Bioaerosols

Bioaerosols are a mixture of organic dust (proteins and polycarbonates) and consist of numerous biological compounds including cell debris, viruses, pathogenic and non-pathogenic live or dead bacteria and fungi, and bacterial organisms; by-products of microorganisms such as endotoxin, mycotoxins, peptidoglycans, glucans (beta-glucan), pollen, plant fibre, animal dander and spores, and other organic compounds (Hauswirth and Sundy 2004; Seedorf 2004). Other air pollutants such as ozone, tobacco smoke, chemical products, inorganic dust, and diesel particulates mingle with bioaerosols and contribute to the biological activity of these exposures (Hauswirth and Sundy 2004). Thus, bioaerosols are characterized by a range of biological properties including infectivity, allergenicity, toxicity and pharmacological or similar effects (Seedorf 2004).

6.1 Health Effects

In a regional context, there is increasing concern that bioaerosol emissions may also be noxious agents that affect people and farm animals living in the vicinity of animal enterprises, because abiotic and biotic particles may travel over relatively great distances (Seedorf 2004; Green et al 2006).

Bioaerosols can adversely affect human health by inhalation, skin and/or eye contact or ingestion. Because exposures occur to complex mixtures of pathogens, toxins, allergens and chemicals, a wide range of health effects are associated with exposure to bioaerosols, including infectious diseases, acute toxic effects, allergies and cancer (Douwes et al 2003). It must be emphasized that both pathogenic organisms and allergens in bioaerosols can be detrimental to health. Respiratory symptoms and lung function impairment are the most widely studied and probably among the most important health effects associated with bioaerosols. The clinical expression of respiratory effects or airway disease is likely to be influenced by a combination of the components of bioaerosols and the dose and timing of the exposure, as well as intrinsic differences in the host response to bioaerosols, such as genetics or immunological reactivity of the individual (Hauswirth and Sundy 2004).

Endotoxin, a component of the cell wall of gram negative bacteria with strong pro-inflammatory properties, is the most widely studied bioaerosol in relation to airway disease. Endotoxins are ubiquitous in the general environment and are present in house dust (Heedrik et al 2006). Very high exposures to endotoxins occur in livestock farming (Heedrik et al 2006). Endotoxins are abundant in manure and have been most consistently associated with a range of respiratory health effects in both the occupational and residential indoor environment, including impairment in lung function and the onset of allergy and asthma (Douwes et al 2003). When endotoxin is inhaled it can potentially cause chronic respiratory symptoms (cough, phlegm production and wheezing), pulmonary impairment, malaise, and fever (Cole et al 2000).

Despite the recognition of the importance of bioaerosol exposure on human health, the precise role of biological agents in the development and aggravation of symptoms and disease is only poorly understood (Douwes et al 2003). It is not clear (with the exception of specific pathogens, and a few individual components such as bacterial endotoxin and specific allergens) which specific components primarily account for the presumed health effects. Dose-response relationships have not been described and knowledge about threshold values (with the exception of a few agents) is not available. Pathogenic micro-organisms may be hazardous at extremely low levels, while other organisms may only become important health hazards at concentrations orders of magnitude higher (Douwes et al 2003).

6.1.1 Occupational Health Effects

Most studies exploring adverse health effects from bioaerosol exposure have been conducted for agricultural workers. Care must be taken to avoid drawing conclusions about the nature or extent of neighbourhood human health effects using only occupational health data. However, studying the workers can contribute to the understanding of potential health effects in CFO neighbours. (See the particulate matter section of this report for more information on occupational exposures.)

Respiratory symptoms (and impairment of lung function) in workers have been found to be associated with total and respirable dust concentrations, endotoxin in the dust, and ammonia (NH_3) measured in the air of the barns. It has been known for some time that working in hog confinement facilities causes chronic or intermittent lower respiratory tract symptoms in approximately one-third of workers. These respiratory symptoms consist of cough with or without production of phlegm, chest tightness, wheezing, and shortness of breath with heavy exertion. Respiratory health effects, including symptoms of pulmonary disease and lung function test result abnormalities, have been described. Depending on the constellation of symptoms displayed and the results of pulmonary function testing, the worker may suffer from chronic bronchitis, the asthma-like syndrome, or exacerbation of pre-existing asthma. It is said that exacerbation of underlying asthma can also occur secondary to hog barn exposures, although the context of this problem is not well documented. Rarely, a true allergy of hogs develops in the process; this hog allergy can be associated with allergic asthma. Nasal symptoms are also common in swine confinement workers; up to 74% of workers have been described as reporting nasal stuffiness, sinusitis symptoms and other nasal complaints (Essen et al 2005).

6.2 Animal Health Effects

Some examples of bioaerosols in a pig barn may be spores, fungi, bacteria and fragments such as endotoxins, volatile fatty acids and mycotoxins. Bioaerosols in a CFO may come from bedding or manure. The effect of bioaerosols on animals is unclear because of variations in CFO management, construction and the difficulty in quantifying amounts. A current hypothesis is that chronic exposure to pigs of certain bioaerosols will reduce growth rates, and may lead to impaired disease resistance (Murphy and Cargill, 2004).

6.3 Ecological Effects

None noted

6.4 Bioaerosols and CFOs

The CFO environment is rich in microbial life. Bioaerosols are produced from livestock feed, bedding material, the animals themselves, and their faeces (Seedorf 2004). The cell debris and microbial organisms become aerosolized to form bioaerosols originating from animal respiration, skin, fur, feathers, dander and manure. Thus, bioaerosols are emitted from CFOs in significant amounts and in varying compositions.

It has been well documented that the air within swine CFOs is highly contaminated with bacteria, yeasts and moulds. Mean total bacterial concentrations range from 10^4 colony-forming units/m^3

(CFU/m^3) to 10^7 CFU/m^3 (Chapin 2004). Potential human pathogens detected have included: *Enterococcus, Staphylococcus, Pseudomonas, Bacillus, Listeria, and Escherichia coli* (Chapin 2005).

In poultry and swine CFOs, gram-positive bacteria are present in the greatest concentration with *Enterococcus* accounting for 68–96% of total bacteria, as reported by Clark in 1983 (Cole et al 2000). Total bacteria typically include 7–53% gram-negative bacteria with 12–40% of the gram-negative bacteria being adsorbed to respirable particulates. Gram negative bacteria are very susceptible to inactivation by oxygen and thus may be considered non viable. Evidence suggests that viruses remain more viable on bioaerosols.

Green et al (2006) found that *Staphyococcus aureus* were the dominant species found in swine CFOs, accounting for 67% of the organisms recovered.

Antibiotic-resistant bacteria including high level multi-drug resistant *Enterococcus*, coagulase-negative staphylococci, and viridans group streptococci have also been detected in the air of swine CFOs (Chapin et al 2005).

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7 Odour

7.1 What is Odour?

It is important to understand the difference between odour and odourant. Odour is defined as "an organoleptic attribute perceptible by the olfactory organ on sniffing certain volatile substances." Odourants are "substances which stimulate the human olfactory system so that an odour is perceived" (Nimmermark 2004).

Odours can be characterized by concentration, intensity, persistence, hedonic tone and character. The odour concentration is a multiple of the concentration at the detection levels, measured with the help of a panel using an olfactometer diluting the odourous sample. The odour intensity refers to the perceived strength of the odour sensation. Odour intensity is often based on the intensity of a reference gas. An example of intensity scaling is intensity categories from "no odour" and "very faint" up to "very strong." The pleasantness of odour is described by the hedonic tone. On a hedonic tone scale, +5 is extremely pleasant and -5 extremely unpleasant. The character or quality of an odour describes in words what an odour smells, like for instance, earthy, floral, fruity, etc. (Nimmermark 2004).

In terms of odours from confined feeding operations, the distinction should be made between odours and gases. The term "odour" actually refers to the complex combination of gases, vapours, and dust that result from both the feed method, animal living arrangements and the anaerobic decomposition of manure (Tyndall and Colletti 2000).

7.2 Health Effects of Odour

Individual complaint and response to odour may be multifactorial, involving a combination of conditioned (learned) and stress-related responses, innate odour aversion (genetic and evolutionary adaptation), pheromonal response, non irritant-based physiological response and a physiologically-based irritant response.

Five distinct thresholds can be used to define a response to odour:

- odour perception
- odour recognition
- odour complaint
- odour annoyance
- odour sensory irritation.

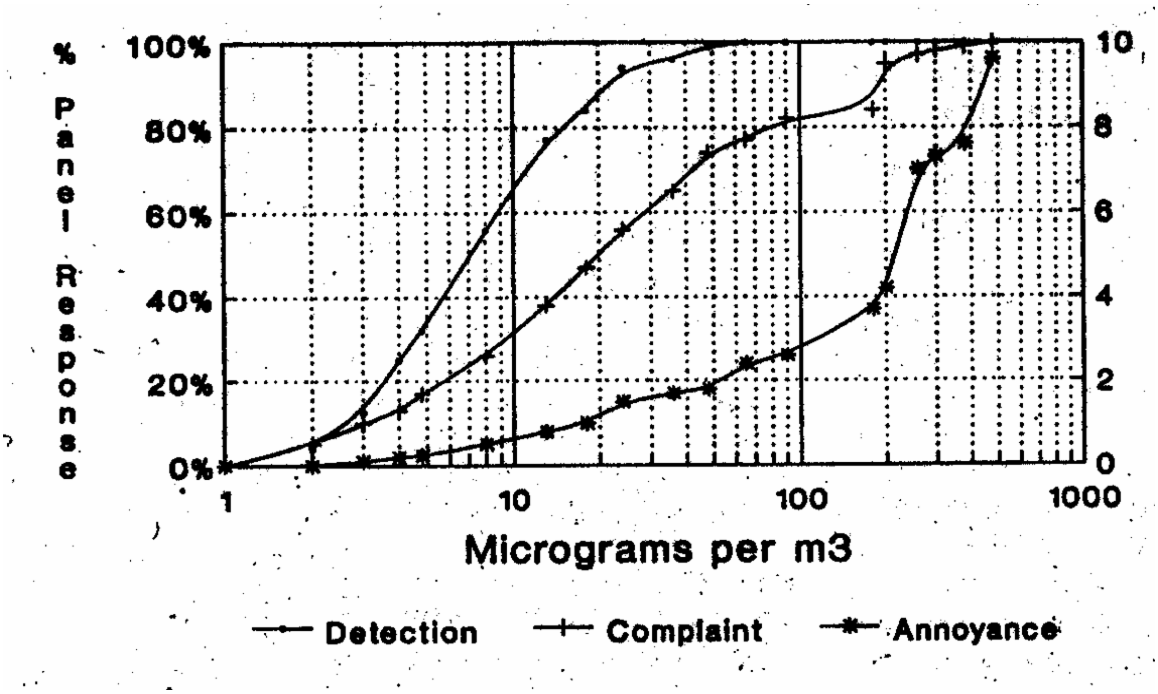
At the odour threshold, there is the initial and subtle perception of a previously unrecognized odour. At the recognition level, the unique quality of the odour can be distinguished (e.g., the distinct rotten-egg odour would identify the presence of hydrogen sulphide gas). At the complaint level, the odour is perceived as unacceptable and affecting the quality of life but there is no symptom manifestation; for instance, there may be a complaint that the odour is affecting the enjoyment of an outdoor barbeque. The annoyance level is where susceptible individuals perceive the odour to be unacceptable by overtly affecting the quality of life and due to symptom presentation such as headache, nausea, dizziness. At this stage, research is unclear as to whether symptom manifestation is associated with a conditioned or stress-related response, innate odour aversion, or a non-irritant based physiology.

However, it is only at the sensory irritation level that identified irritant-based physiological mechanisms are involved. These physiological mechanisms are intrinsic to the toxicological properties of a chemical substance and are independent of odour (Shusterman 1992; Schiffman et al 2000; Schiffman and Williams 2005).

Investigations of odour complaints from individuals (such as H_2S or CFO odours), need to clearly distinguish between odour perception, complaint, annoyance and irritation and may be guided by the qualitative nature of the odour complaint (e.g., odour frequency, intensity, duration and offensiveness, type of land use) and/or quantitative information.

H_2S is one example of an odourous chemical found in CFOs. As shown in figure 1, the median response for H_2S odour perception (threshold) is 7 µg/m^3 (5 ppb), for complaints 20 µg/m^3 (14 ppb) and for annoyance 200µg/m^3 (140 ppb). These thresholds are based on nose-only exposures and therefore may underestimate threshold concentrations. Shusterman (Auvermann 2002) reported the following for H_2S: 2 ppb odour threshold, 4 ppb recognition threshold, 100 ppb annoyance threshold and 2,000 ppb (2 ppm) sensory irritation threshold.

Figure 1. Human Panel Response Demonstrating H_2S Odour Perception, Complaint and Annoyance. (H_2S in µg/m^3 of air)



Source Nagy 1991

The perception of odour occurs at receptors in the olfactory epithelium in the top of the nasal cavity. Activated odour receptors transmit neural pulses via the olfactory nerve (i.e., first cranial nerve) to the olfactory bulb and the brain where odour perception that can be described as, for example, fruity, floral, earth, fishy or fecal, occurs (Shusterman 2002; Schiffman 1998; Cole et al 2000). Odour signals are processed in two different areas of the brain: the limbic system and front cortex. The limbic system provides an emotional-memory context to odour perception and can also influence function of the hypothalamus and pituitary glands – the main hormonal control centers of the body.

The frontal cortex is where odour sensations are consciously compared to other sensations and previous experiences (McGinley and McGinley 1999).

At higher chemical concentrations, stimulation of free nerve endings in the nose, throat, eyes and lungs cause sensory irritation. Activation of the free nerve endings in the upper and lower respiratory system, including the eye,[10] produce sensory sensations or pungency variously described as irritation, tingling, burning, stinging, scratching, piquancy, prickling, freshness, and itching. Sensory irritants can affect the lung, reducing respiratory volume and causing inflammation. People with preexisting respiratory problems may be particularly vulnerable to the adverse effects of irritants, and can experience an increase in nasal resistance, respiration rates, and heart rates (Schiffman and Williams 2005).

When irritant compounds come in contact with the lower and upper airway, many systemic responses can occur, including altered respiratory rate, reduced respiratory volume, increased duration of expiration, contraction of the larynx and bronchi and increased bronchial tone, increased nasal secretion, inflammation, and nasal airflow resistance, eye tearing, alterations in body movements, peripheral vasoconstriction and increased blood pressure and sneezing (Schiffman 2005).

Odours can also affect mood and stress depending on whether an odour is perceived as pleasant or unpleasant (Cole et al 2000). Mood impairment and stress have also been associated with the development of coronary artery disease, chronic hypertension, and structural changes of the heart. (Nimmermark 2004) and affect the immune system and hippocampal damage (Schiffman 1998).

Both innate physiological responses and learned responses may contribute to the impairment of mood (Schiffman 1998). Conversely, depressed persons may be more likely to complain about unpleasant odours. Odours that are perceived positively or pleasantly have been reported to contribute to improved emotional and physical health.

The role of social conditioning and psychological bias in odour response merits consideration. Schiffman (1998) cites a large number of studies as demonstrating the role of social conditioning and cognitive bias in odour perception and response. A review of all these studies is beyond the scope and resources of this review. However, three studies were selected for review based on their purporting to demonstrate social condition and cognitive bias. The studies were selected from a review by Schiffman (1998) prior to obtaining the original research articles. These studies (Dalton 1996, Knasko 1990, and Shusterman 1988) are reviewed below.

Schiffman (1998) cites Dalton 1996 and 1997 as demonstrating how cognitive bias about the health effects of an odour can either positively or negatively modulate odour perception. In the 1996 study on the perception of odour intensity (Dalton 1996), three exposure groups were given different information about an odourant, isobornyl acetate, to which they were going to be exposed. The positive group was advised that the odour was a natural extract used by aromatherapists. The negative group was advised that the odourant was an industrial chemical with purported adverse health effects with prolonged exposure. The neutral group was advised that the odourant was a substance commonly used and approved for olfactory experiments. All groups were exposed to a steady state concentration of the odourant for 20 minutes. The positive group showed normal adaptation over the test period, reporting decreased odour intensity over time (adaptation is a normal

[10] This occurs via at least one of four cranial nerves in these areas such as the trigeminal (cranial nerve V) and vagus (cranial nerve X) nerves.

response to odour). The negative group perceived odour intensity as increasing after 10 minutes. The results of the neutral group were between the positive and negative groups.

Dalton reports that a health symptom assessment was not a formal part of the study, but the 13 individuals in the negative bias group spontaneously reported headache, lethargy, dizziness, or irritation, compared to two in the neutral group and none in the positive group. However, symptoms were not rigorously assessed in this study. The presence of symptoms in the other groups was not rigorously assessed. In addition, the study is unclear as to airborne concentrations of isobornyl acetate in the study. All that can be said is that the odours used in determining odour thresholds originated from 26 dilutions of isobornyl acetate in mineral oil ranging from 5 mM (0.1% vol/vol) to 1.4 µM ($3x10^{-8}$ % vol/vol), a difference of 3,500 times. The actual airborne concentration over the experimental 20 minute exposure period for determining odour intensity is not clear. Other than originating from 50% isobornyl acetate in mineral oil, and the experimenters rating the odour as moderate or 20 on a scale reaching 50 for maximum. The odour intensity rating of 20 or moderate is also what the test subjects generally rated the intensity as. It is also unclear whether or not irritant concentrations were reached in the protocol. Odour threshold and toxicological information is lacking for this compound.

A similar 1990 study by Knasko et al. was also cited by Schiffman (Schiffman 1998) as showing that people's cognitive expectations about odour and irritation can influence sensory perception. Subjects were negatively or positively biased about an odour and then placed in a room containing the odour when in fact there was no odourant present. People who were biased with the suggestion of the presence of a malodour reported more negative mood and more symptoms of discomfort than persons with the suggestion that the feigned odour was pleasant. However a closer look at this study (Knasko 1990) is less definitive regarding the conclusions. The number of symptoms experienced by the subjects were binned into four categories: those reporting 0-1, 2-3, 4-5 or 6-7 symptoms during a session. The number of subjects reporting 6-7 symptoms was larger for the "unpleasant group" versus the pleasant and neutral (i.e., 8 versus 2 and 1). However, 12 and 14 individuals in the pleasant and neutral group reported 4-5 symptoms, whereas only 5 individuals did in the unpleasant group. The situation reversed in the 2-3 symptom group and reversed again in the 0-1 symptom group. This study is not as clear in its findings and conclusions as the beginning of the paragraph suggests.

Schiffman (1998) cites the work of Shusterman (1988) as demonstrating the role of conditioning or learned associations in odour response. Shusterman (1988) reported two cases where workplace exposure to transient odours, phosphine or phenol-formaldehyde, were initially tolerated. However, following an acute overexposure accident that produced irritation such as burning sensation, shortness of breath, and headache, subsequent workplace odour that were previously tolerated without concern, elicited panic and hyperventilation in the subjects. Shusterman (1988) considered the response an adaptive or protective psychophysiological response. One of the subjects was able to learn to become desensitized to workplace odours using breathing exercises, while the other subject found alternative employment at a drycleaner's, where dry-cleaning odours (perchloroethylene) were tolerated.

Schiffman (1998) suggests that the above studies by Dalton, Knasko and Shusterman provide evidence that cognitive bias of odours can influence symptom presentation. However, the two studies by Dalton and Knasko cannot be described as robust in their findings or conclusions. The Knasko symptom classification results are highly variable and the Dalton symptom findings were not rigorously determined. Few studies examine the influence of cognitive bias in the context of the

odour, complaint and annoyance thresholds. There is little evidence to show if or how perception, complaint, annoyance, or irritation thresholds are, or are not, affected by cognitive bias or social conditioning. These studies suggest that cognitive bias may influence a subject's response around the perception threshold, when response is more malleable. However, that malleability may decrease as concentration and odour intensity increase.

A study by Prah et al (1998) provides insight into the relationship between VOC odour, irritation and cognitive processes. Study subjects consisted of 100 young non-smoking females with an average age of 25.1 years with no history of allergy, pulmonary disease, chemical sensitivity and no serious illness. The authors state that women were selected because they tend to report symptoms at a higher rate than men. In addition, all subjects received a physical exam, skin allergy tests, pregnancy test, and personality assessment. Subjects were exposed to a single VOC or to mixtures of VOCs for six hours and their symptomatic and sensory responses determined through questionnaire. As with the Dalton (1996), the subjects were 'neutrally' biased by assurance that the chemical were commonly found in indoor air and at levels below OH&S standards.

Subjects were exposed to a single VOC or mixture of VOC where the total concentration in all exposure scenarios equaled 24 mg/m^3. Subjects were exposed to one of six equimolar concentrations equivalent to 24 mg/m^3 toluene, control, m-xylene, n-butyl acetate, m-xylene plus n-butyl acetate, a mixture of 21 chemicals including n-butyl acetate and m-xylene, and to the same mixture of chemicals without n-butyl acetate and m-xylene (19 chemicals).

Subjects were divided into six exposure groups of 15 to 20 subjects per group and were exposed only once. The exposure regimes were as follows:

- n-butyl acetate alone @ 17.7 mg/m^3
- m-xylene alone @ 29.95 mg/m^3
- equimolar parts n-butyl acetate and m-xylene @ 24.4 mg/m^3
- all 21 VOCs as shown in Table 12 @ 23.73 mg/m^3
- 19 VOCs minus n-butyl acetate and m-xylene.
- clean air control

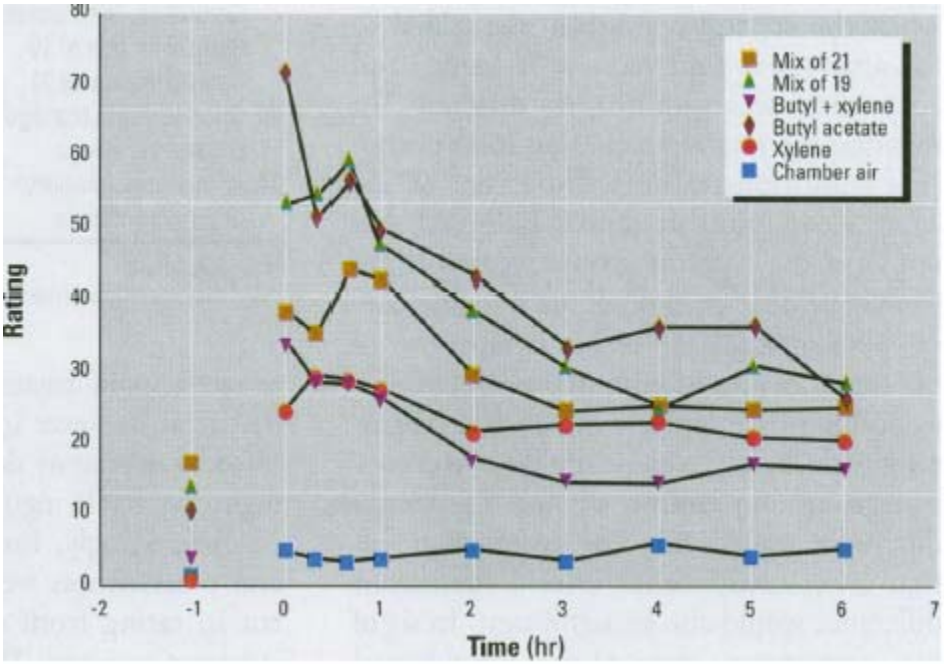
The odour thresholds for the various chemicals were considered similar: m-xylene 1.1 ppm (4.79 mg/m^3) and butyl acetate 0.39 ppm (1.86 mg/m^3). Odour thresholds for the remaining chemical ranged from 0.016 mg/m^3 for apinene to 234 mg/m^3 for hexane.

The results indicated that there was no difference in reporting of symptoms or sensory response between the chemical exposure regimes. When the control group was added, some variables, primarily odour intensity and nasal irritation, attained significance.

However, there are some interesting observations that provide insight into the interaction between cognitive processes, odour perception and irritation. All exposure regimes resulted in eye and throat irritation (Figure 2), although to different degrees. As shown in the figure, partial adaptation to nasal irritation, and also to odour intensity (data not included), occurred as exposure time progressed. By the end of the 6-hour exposure, nasal irritation levels were ~40% of initial levels, with the exception of xylene. In contrast to nasal irritation, both ocular and throat irritation showed no significant changes, or adaptation, with time. No other health related effects were observed (e.g., cough, chest tightness, dry throat, stuffy nose, skin rash, sneezing, pain, dry skin or neurological impairments such as cognitive dysfunction, memory loss, depression, tension or dizziness).

The authors write that adaptation to irritation was observed in the trigeminally innervated nasal but not at the ocular to pharyngeal mucosas. “It is likely that trigeminal adaptation”, the authors write, “can be influenced by perceived toxicity of exposure in a similar manner to that reported by Dalton et al. (29)” (page 743). Trigeminal adaptation explains why even though all the subjects reported nasal, ocular and throat irritation; adaptation only occurred for nasal irritation. The authors felt that the presence of adaptation may be attributable to the absence of stress, the subjects were considered ‘neutrally’ biased. Yet the authors also felt that the lack of adaptation may be a consequence of cognitive processes as occurs in a sick building (where stress or tension may be engender non-adaptation). The authors write that failure to show trigeminal or olfactory adaptation may be an adaptive response to a stressful situation. Contrary to this, olfactory adaptation was observed in this study, Figure 3. The authors, in attempting to explain the two conflicting observations on adaptation have contradicted themselves. In conclusion, it would appear that some symptoms or tissues or organs are malleable, and some are not, to cognitive processes as evidenced by nasal adaptation and the lack of adaptations for ocular and throat irritation. As can be seen in the figure, reported irritation initially rises then declines as the exposure progresses. Baseline data were obtained at the -1 time point.

Figure 2. Adaptation to nasal irritation from chemical exposures



Source: Prah et al. 1998.

Table 11. Composition and Concentrations of the 21-VOC Mixture (6.7 ppm, 24 mg/m^3)

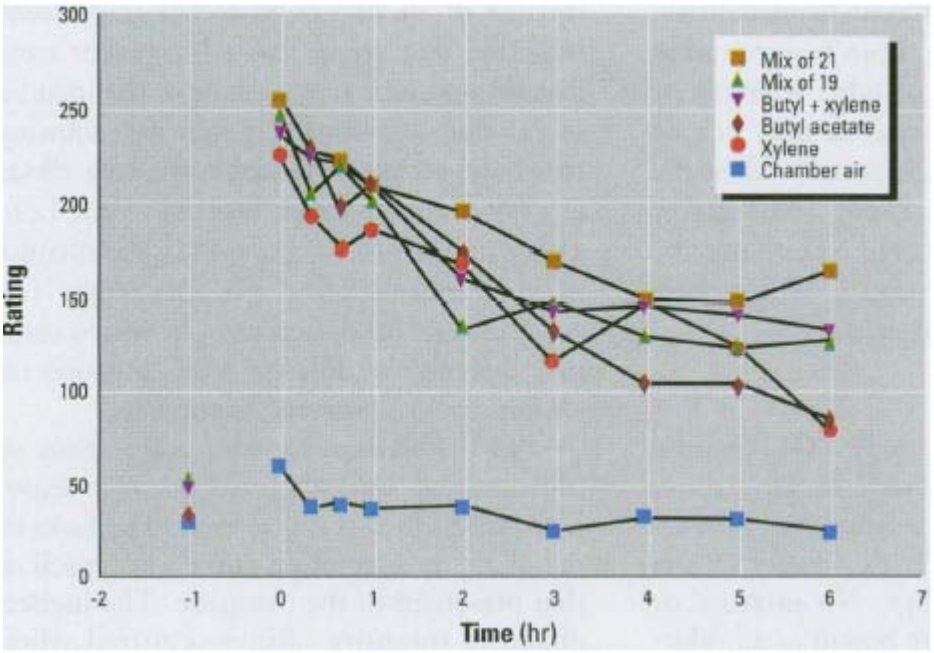
Composition of the Mixture at 6.7 ppm (24 mg/m^3)

Chemical	PEL (ppm/mg/m^3)	21 VOCs (mg/m^3)
1. *n*-Butyl acetate	150/71	7.750
2. *m*-Xylene	100/435	7.750
3. *n*-Butanol	100/300	0.775
4. *n*-Decane	No TLV	0.775
5. 1-Decene	No TLV	0.775
6. Ethylbenzene	100/435	0.775
7. Ethoxyethylacetate	100/540	0.775
8. *n*-Hexanal	No TLV	0.775
9. *n*-Hexane	500/1800	0.775
10. *n*-Nonane	200	0.775
11. α-Pinene	No TLV	0.775
12. 2-Butanone	200/590	0.078
13. Cyclohexane	300/1050	0.078
14. 3-Methyl-2-butanone	200	0.078
15. 4-Methyl-2-pentanone	100/410	0.078
16. *n*-Pentanal	50	0.078
17. Isopropanol	400/980	0.078
18. *n*-Propylbenzene	No TLV	0.078
19. 1,2,4-Trimethylbenzene	25/120	0.078
20. *n*-Undecane	No TLV	0.078
21. 1-Octene	No TLV	0.008

Abbreviations: PEL, permissible exposure limit; VOC, volatile organic compound; TLV, threshold limit value.

Source: Prah et al. 1998.

Figure 3. Olfactory adaptation to the various exposure conditions



Source: Prah et al. 1998.

Differences between the control and exposure conditions were significant, but the differences between the exposure conditions were not significant in magnitude or rate of adaptation. Baseline data were obtained at the -1 time point.

Schiffman (1998) cites studies that suggest that the nervous system can trigger an inflammatory response in the nose and respiratory tract, implying that learned response can be transmitted to the nose to produce symptoms. Significantly however, the odour concentration involved in the study were around the perception threshold and therefore subject to cognitive bias.

7.2.1 When is an odour effect a health effect?

The Effects Subgroup agreed that odour does cause health effects, but the linkage between odour and health is complicated, in particular, the question of whether odours affect health through a physiological or psychological mechanism. Research exists to support both points of view. There is also concern around the level at which effects occur. At certain levels, there is a true toxicological effect; e.g., at 10 ppm, H_2S clearly has a health effect, and the physiological effects are related to the substance as an irritant; in other words, the H_2S itself has a toxicological effect that is not due to odour. At lower levels (e.g., 10 ppb), some would argue there is an odour effect and it's psychological, not physiological. In these two situations, there could well be different requirements and expectations for action, and different management approaches, depending on whether the effect is psychological or physiological.

Are exposures to odours below irritant thresholds cause for public health concern? The argument may be made that non-physiologically based odour responses are not a *bona fide* public health

concern. Alternatively, the WHO broad definition of health may used to exclude forming such a conclusion.

The WHO defined health as, “a state of complete physical, mental and social well-being, and not merely the absence of disease or infirmity” and “the extent to which an individual or a group is able, on the one hand, to realize aspirations and to satisfy needs, and on the other, to change or cope with the environment.” In Canada, provincial, territorial and federal governments (Health Canada 2004) have endorsed this broad definition of health. Well-being is defined by Webster’s dictionary as, “the state of being happy, healthy, or prosperous.” Frankish (1996) provided a definition of health which coincides with the second half of the WHO definition, and perhaps of well-being, “the capacity of people to adapt to, respond to, or control life’s challenges and changes.”

A broad definition of health that encompasses health as both physical and psychosocial well being in the context of adaptive capacity transcends the question of whether odour elicits a public response via physiological or non-physiological mechanisms. New Zealand (2003) says that an odour is considered offensive or objectionable if it causes an adverse effect at or beyond the property line of the originating source. An adverse effect includes a continuum from noxious, dangerous, offensive or objectionable. Thus an odour at high concentrations may have discrete physiologically based adverse effects as captured under the definition of dangerous or noxious. At low concentrations, the adverse effects that manifest are captured by the definitions of offensive and objectionable, and occur at concentrations far less than would cause cellular or tissue damage or harm.

New Zealand (2002) defines “offensiveness” as: ...*giving or meant to give offence... disgusting, foul smelling, nauseous, repulsive..*, and “objectionable” as *open to objection, unpleasant, offensive* (page 19). New Zealand also makes the case that the finding of an offensive or objectionable odour must be reasonable, that is, “by an ordinary person who is representative of the community at large and neither hypersensitive nor insensitive, deciding whether the activity is disgusting, nauseous, repulsive or otherwise objectionable.”(page 19). Further, the finding of an objectionable or offensive odour cannot be determined by one person or government investigator unless an adverse effect is demonstrated.

New Zealand (2002) further says that offensive or objectionable odours can occur at very low concentrations, far less than the concentration that would cause cellular or tissue damage or harm. Direct health effects associated with high concentrations include skin, eye or nose irritation. Effects that contribute to a reduced quality of life are nausea, headache, retching, difficulty breathing, frustration, annoyance, depression, stress, tearfulness, reduced appetite and embarrassment in front of visitors.

New Zealand (2002) cites a 1992 study by Schusterman that confirmed community odour effects that extended beyond aesthetic nuisance and annoyance effects to include headache, nausea, sleep disturbance, and eye and throat irritation at measured or modeled exposure concentrations well below those expected from physiologically based toxic effects.

Cavalini (1994) investigated the relationship between objective exposure to odourant concentrations emitted by several industrial plants and odour annoyance and subjective health complaints. The study concluded that long-term averaged exposure was related to odour annoyance and the effects of long-term low level exposure are similar to temporary high exposure.

The issue of odours and linkage to health effects is complex. Donham et al. 2007 reported on the consensus achieved among environmental scientists from North America and Europe at a scientific conference and workshop held in March 2004 in Iowa City, Iowa. The focus of the conference was on major environmental health issues associated with concentrated animal feeding operations and on sustaining the health of rural communities. The recommendations from workshops scientists suggested additional research should be conducted to further delineate the mechanisms of effects and impacts on susceptible subgroups which include psychophysiologic impacts of malodour; impacts of malodour on mental health and quality of life; and respiratory impacts of bioaerosol mixture, especially among asthmatics, children and the elderly. The workgroup agreed that the World Health Organisation's definition of health, "a state of complete physical, mental and social well being and not merely the absence of disease or infirmity," applies to rural communities. Donham et al. cited various studies where neighbours of large scale CFO's experience excessive respiratory symptoms, increased levels of mood disorders, anxiety, depression and sleep disturbances attributed to exposures to malodourous compounds (Thu et al. 1997; Wing and Wolf 2000; Campagna et al. 2004; Schiffman et al. 1995, 2000).

7.3 CFO Odours and Health

The U.S. National Research Council (NRC 2003) ranked odour emissions from animal feeding operations and ability to cause local effects (i.e., at the property line or the nearest dwelling) as major. The primary effect of concern was on quality of life.

Schiffman in a 1995 study (Minnesota 2001; Cole et al 2000) of 44 persons living near a swine feed operation in North Carolina found increased psychological tension, depression, anger, fatigue and confusion and decreased vigor compared to controls ($P<0.0001$). Persons exposed to the odours also had more total mood disturbance ($P<0.0001$). In a 1995 study of another swine confinement facility, Thu (Minnesota 2001) found statistically significant increased physical and mental effects in residents within 3.2 kms of a swine facility. These included the following symptom clusters: (a) respiratory inflammation or airway hyperreactivity (e.g., wheeze and cough) associated with exposure to air pollution, chronic agricultural dust, endotoxins and smoking; (b) nausea, dizziness, weakness and fainting associated with endotoxin exposure; (c) headaches and plugged ears (25% of swine workers have chronic sinusitis); (d) runny nose, scratchy throat, and burning eyes associated with exposure to irritant gases such as ammonia. There was no evidence of increased psychological symptoms such as depression or anxiety. Environmental monitoring was not performed.

One limitation of the above studies is that they did not include environmental or exposure monitoring, i.e., measurements of levels of exposures to specific chemicals such as airborne gases, vapours, or particulates. Some of the studies did not mention odour, but may have assumed odour was a factor due to the radius within which the study subjects resided. In addition, the studies were limited to swine operations. The findings with respect to swine should not be applied to poultry, cattle and dairy operations without some contextual analysis. In terms of the health outcomes for the swine studies, exposure to toxic levels of airborne gases, vapours or particulates (rather than the odours) cannot be ruled out as a cause for the observed health effects. Whether the effect is due to odours or some specific constituent(s), these studies suggest that there may be concern for public health, whether as a quality of life, annoyance or physiological effect. Some studies were adjusted for demographic variables.

Two studies, one by Zahn (2001) and one by McGinn (2003) are of interest with respect to measuring and monitoring odour. McGinn's work was not a health study but may be of particular interest as it was done in Alberta. Zahn's study, although not done in Alberta, is particularly relevant because it provides background to McGinn.

Zahn (2001) observed excellent correlation between odour intensity and total VOC concentration at swine feed-to-finish production facilities. Emissions from 29 swine manure management systems were sampled in Iowa, Oklahoma and North Carolina during August and September 1997. Manure management systems were categorized into 4 types, types 1 to 4, based on the total sulphur and phosphorous content of the liquid manure effluent, figure w, A. The 4 type categories also captured the difference in kind of the manure management systems: Type 1 deep pits, Type 2 concrete lined basins, Type 3 lagoons, and Type 4 photosynthetic lagoons.

Odour intensity was measured by a trained panel using olfactometers and the odours were rated as neutral (a rating of 3 out of 10), unpleasant (6.5/10) and unbearable (10/10). Odour intensities according to manure management type were highly correlated, $r^2 = 0.88$, to total VOC concentrations (see Figure w, in Appendix F-1). Total VOCs and odour samples were collected or measured above the manure management systems.

McGinn (2003) investigated emissions, including odours, from cattle feedlots near Lethbridge. Odour intensities were determined for samples of air collected up to 800 m downwind of cattle feedlots with averaging times of 5 minutes to 4 hours.

Details on both the McGinn and Zahn studies can be found in Appendix F-2. Appendix G-1 describes measurement and management mechanisms related to odour.

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8 Community Health Effects

Several epidemiological studies have investigated differential reporting of adverse symptoms and effects for communities and residents near CFOs. Among many others, the team was made aware of a study done in the Brooks area of southeast Alberta in 1999 to look at the links between air quality and local health. Some of the studies that have focused on community exposures to confined feeding operations are noted in this section. Although these studies looked at community health, they were not done in the context of the priority substances that were the focus of this CASA project. They were done without monitoring for specific compounds and thus cannot be linked to these substances. Nevertheless, these studies do add to the weight of evidence about potential effects of airborne substances on those who live near CFOs.

8.1 Health and Well Being

Donham et al. (2007) reported on the consensus achieved among environmental scientists from North America and Europe at a scientific conference and workshop held in March 2004 in Iowa City, Iowa addressing major environmental health issues associated with concentrated animal feeding operations and on sustaining the health of rural communities. The workgroup agreed that the World Health Organization's definition of health, "a state of complete physical, mental and social well being and not merely the absence of disease or infirmity," applies to rural communities. Donham et al. cite some studies (Thu et al. 1997; Wing and Wolf 2000; Campagna et al. 2004; Schiffman et al. 1995, 2000) where neighbours of large scale CFOs experienced excessive respiratory symptoms, increased levels of mood disorders, anxiety, depression and sleep disturbances attributed to exposures to malodourous compounds. The recommendations from workshop scientists suggested additional research should be conducted to further delineate the mechanisms of effects and impacts on susceptible subgroups, including psychophysiologic impacts of malodour; impacts of malodour on mental health and quality of life; and respiratory impacts of bioaerosol mixtures, especially among asthmatics, children and the elderly.

A study by Wing and Wolf (2000) reported on health symptoms and quality of life among residents living near a swine CFO in North Carolina who were less able to open their windows or enjoy the outdoors. The study involved 155 individuals each in three rural communities: one with no livestock facilities within 3.2 kms; one within 3.2 kms of a dairy facility; and another within 3.2 kms of a swine CFO. Those living within 3.2 kms of the swine CFO reported significantly greater frequency of headaches, runny nose, sore throat, coughing, burning eyes, and diarrhoea than the other two groups. The frequencies were adjusted for age, sex, smoking status and employment (i.e., working at home).[11]

Michigan (2006) cites a study by Thu et al. that examined the physical and psychological health data from 18 residents living within two miles of a swine containment facility. Those living near a swine operation experienced increased rates of respiratory problems, eye irritation, nausea, weakness and chest tightness. However, the study did not suggest increased rates of anxiety and depression. Schiffman et al. (1995) recorded the psychological effect of odours from a swine facility on 44 volunteers. Compared to matched controls, exposure subjects reported more tension, depression, anger, fatigue, confusion and less vigor.

[11] For more details on the Wing and Wolf study, see the section and appendices on VOCs in this report.

Heederik (2007) reports a 2005 German study by Radon that found residents living within 500 meters of CFOs experienced significantly increased prevalence of self-reported wheezing and decreased respiratory function (FEV_1), compared to urban residents. In a subsequent 2007 study, Radon observed increased prevalence of self reported asthma symptoms and nasal allergies with increased odour complaint for non-CFO working residents, from four towns, living within 500 m of high density CFOs (Radon et al. 2007). The number of animals was a predictor of self-reported wheeze and decreased lung function (measured as FEV1), but not allergic rhinitis or sensitization. Increased wheeze and FEV_1 were demonstrated at > 8 to10 animal houses located within 500 meters of homes. The number of animal houses within 500 meters ranged from 0 to 20. The number of animals within the animal houses was not known, but animal densities in the four towns, ranging in area from 42 to 113 km^2, were as follows:

Cattle, 23 to 420 head/km^2
Pigs, 310 to 1,300 head/km^2
Chickens, 5,100 to 19,000 head/km^2
Turkeys, 143 to 5,700 head/km^2.

Although a direct comparison to Alberta is not possible, McGinn (2003) provided data on the number of animals in the Lethbridge Northern Irrigation District, encompassing 71 km^2, from a 1995 study by McCarley. McGinn says this area is considered one of the most concentrated beef feedlot areas in Canada. Based on this 1995 study, animal densities in this area were:

Beef Cattle, 4,100 head/km^2
Dairy Cattle, 177 head/km^2
Pigs, 89 head/km^2
Chickens, 7,000 head/km^2

Radon et al. (2007) recognized that farming practices in North America and Europe were different.

8.1.1 Allergy and Asthma

A 2005 epidemiological study by Merchant (Michigan 2006) examined asthma in Iowa children and found a high prevalence of asthma health outcomes among children living on farms that raise swine (44.1% prevalence, $p = 0.03$) and among children living on farms that raise swine and add antibiotics to feed (55.8%, $p = 0.013$), despite lower rates of hypersensitivity and allergy and significantly lower exposure to household tobacco smoke. Children not raised on farms had a prevalence of 33.6% ($p = 0.19$) and those living on farms that did not raise swine (the reference group) had a prevalence of 26.2%. As is often the case with epidemiological studies of this nature, there was no measurement of, and thus no possible correlation to, exposures to dusts, VOCs, gases, odours or bioaerosols.

Heederik (2007) also documents many studies of elevated exposures among farm workers that support the expectation of increased asthma prevalence and increased asthma morbidity and mortality. Animal farmers have been observed to have the highest risk for asthma compared to farm workers not involved with animals. In animal production workers, asthma was often seen with increased endotoxin exposure in the absence of allergic or atopic sensitivity. A 2005 study of Dutch pig farmers by Portengen found a decreased prevalence of atopic sensitization with increasing endotoxin exposure.

The possible development of asthma and allergy in the public residing in communities adjacent to CFOs is controversial. A recent review by Heederik (2007) provides an excellent overview of the

numerous studies on this subject. The 'hygiene hypothesis' postulates that low level exposures to bioaerosols, including infection, in early life may protect against the development of allergic asthma and allergic sensitivities. However, such exposures may also promote the development of nonallergic asthma. In addition, high exposures may promote the development of asthma and allergies. Bioaerosol exposures in farm or livestock settings must always consider endotoxins, fungi, gram-positive and gram-negative bacteria, bacterial DNA, storage mites, and allergens from crops, feeds and animals. None of these components are experienced in isolation.

As cited by Heederik, the scientific literature generally supports the 'hygiene hypothesis', but with some qualification. Contrary to the above study by Merchant (Michigan 2006), a number of studies have shown a low prevalence of atopy (i.e., hereditary predisposition to allergy including hay fever, skin and asthma), hay fever and to a lesser degree asthma in the children and adolescents of farming families. Contact with livestock in the first year of life was identified as a factor for reduced risk.

Heederik reports lower frequencies of asthma and hay fever are generally observed in children with contact to livestock. At school age, the amount of endotoxin at home was inversely related to the occurrence of allergic asthma, hay fever and sensitization (Waser et al. 2004). In addition, Waser found higher levels of endotoxins in the homes of farming versus non-farming families, associated with regular animal contact by children.

Similarly in a study of urban and rural children in India, higher levels of endotoxin in home indoor dust and regular animal contact (Vedanthan et al. 2006) was associated with lower prevalence of self reported asthma symptoms and allergic sensitization. In an urban and rural study in Estonia, allergic sensitization was more common in urban and suburban areas than in rural (Raukas-Kivioja et al. 2007). In addition, living in an urban or suburban area before the age of five significantly increased the risk of allergic sensitization.

Danov and Guilbert (2007) report that a number of studies have demonstrated that children on traditional farms with regular contact with livestock were less likely to develop allergic sensitization and asthma. Many studies have shown that the alternative approach, allergen avoidance in children did not result in reduced allergic sensitisation or asthma compared to controls. Overall, in reviewing the literature on asthma and allergic sensitization, Danov and Guilbert concluded that environmental exposures in infancy or early childhood were associated with a reduce prevalence of asthma. But, studies have not always demonstrated a significant difference in objective measurements of lung function or bronchial-hyper-responsiveness.

However, some study findings conflict with the expectations of the 'hygiene hypothesis', reporting a positive association between endotoxin exposure and child asthma. Braun-Fahrländer in a 1999 study (Heederik 2007) showed that allergic asthma and allergy in children were lower with increasing exposure to lipopolysaccharides, but with an increased prevalence of nonatopic (i.e., non allergic) wheeze, an asthma indicator. Lipopolysaccarides, or endotoxins, are constituents of the cell wall of gram-negative bacteria are inflammatory but not allergenic. Similarly, Shirakawa in a 1997 study (Heederik 2007) of infants with familial predisposition to asthma or allergy found that indoor endotoxin exposure early in life was associated with increased risk of wheeze.

Radon (2007) cautions that the lower prevalence of respiratory allergies among subjects with farm-animal contact in early infancy were mainly conducted with traditional farming, and may not apply to intensive or confined feeding operations.

For adults and children with pre-existing allergic asthma, bioaerosols exposure may exacerbate symptoms of asthma (e.g., wheeze), which is not unexpected. Thorne in a 2005 study (Heederik 2007) found that endotoxin exposure was a significant risk factor for aggravation of asthma and increased medication use. However, unexpectedly this risk remained regardless of whether the study subjects were allergic or not.

In a review of risk factors for allergy and asthma development, Kaiser (2004) concluded that there is general support to suggest that early exposure to endotoxin and/or living on a farm protects against the development of allergic sensitization. Studies have generally shown that contact with livestock reduced the risk of atopic or allergic asthma in children and adolescents, with endotoxin exposure possibly being a determining factor. In conclusion, bioaerosols exposure, and infection, early in life may provide a protective effect against the development of allergies and allergic asthma. In terms of nonallergic or nonatopic asthma, the evidence suggests the possible causal role of endotoxins in adulthood.

8.1.2 Antibiotic Resistance

The increase in antibiotic resistance in pathogenic bacteria, traced to the use and overuse of antibiotics, is an increasing medical concern. Evidence is increasing that antibiotic resistant bacteria originating on farms is a public health concern (Heilig et al. 2002).

Estimates of the overall antibiotic use in the United States are variable (Gilchrist et al. 2007). In 2001 the Union of Concerned Scientists estimated that 87% of antibiotics were for animal use and 13% for human medical use. Levy in 1998 estimated that 40% of antibiotics were for animals. The Animal Health Institute, representing pharmaceutical manufacturers, estimated only 13% of antibiotics were for animal production (Thorne 2007). The Institute of Occupational Medicine concluded that substantial efforts must be made to decrease the inappropriate overuse of antibiotics in animals (NAS 2003 as cited by Gilchrist et al. 2007).

Feeding antibiotics to swine and cattle has been associated with the development of antibiotic resistance in these animals. Compared with the poultry industry, where antibiotic use is minimal, bacterial resistance in poultry bacteria has remained constant (Gibbs et al 2004).

Zahn in a 2001 study (Gilchrist et al 2007) compared the number of bacteria resistant to tylosin in the exhaust air of swine CFOs using and not using the antibiotic tylosin in feed. Tylosin-resistant bacteria were three times higher in the exhaust of buildings using the antibiotic.

Gibbs et al. (2004) found resistant bacterial forms from inside and 25 m downwind of swine confinement facilities, indicating that resistant organisms being produced in and released from these facilities could cause a potential health hazard. The study consistently found levels of total microorganisms above 10^3 CFU/m^3 of air, a level used as indicator of possible human health concern, inside and downwind of the confinement facilities in the presence of animals.

The development of resistant bacteria was examined following the introduction of tetracycline-containing feed in a poultry farm study by Levy in 1976 (Gilchrist et al 2007). No bacterial resistance was found in animals or humans prior to the addition of the tetracycline in the feed. Within five months of introduction, 31% of the farm workers and 7% of the neighbours had intestinal bacteria with tetracycline-resistance.

8.1.3 Zoonoses

Zoonoses, the transmission of illnesses from animals to humans, potentially arise from the transportation, handling, processing and rendering of animal and animal products including manure handling (Gilchrist et al 2007). Exposure pathways include air, water, consumption and handling of meat and animal contact.

Appendix G-1 describes measurement and management mechanisms related to community health effects and bioaerosols.

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Appendix A-1: Subgroup Members and Terms of Reference

Ann Baran	Southern Alberta Group for the Environment
Laura Blair	Alberta Environment
Kerra Chomlak	CASA
Deb Mooney	Alberta Health and Wellness
Usha Mulukutla	Calgary Health Region
Carrie Selin	Intensive Livestock Working Group
Dennis Stefani	Calgary Health Region
Karina Thomas	Alberta Health and Wellness
Ross Warner	SERLO
Brenda Woo	Health Canada

CFO – Effects Subgroup Terms of Reference
24 November 2006

Tasks

1. Review existing information pertaining to the effects of Ammonia, Hydrogen Sulphide/TRS, Volatile Organic Compounds, Particulate Matter, Bioaerosols, on human, animal and ecosystem health.
2. Review existing information regarding human health effects from odour and greenhouse gas.
3. Provide summary information from tasks 1 and 2 to the CFO project team
4. Identify gaps in information and understanding regarding the effects of priority substances and odour.
5. Provide workplan and budget updates to the CFO Team on a regular bases.
6. Provide a final report and, if required, recommendations to the CFO Project Team.

Deliverable

To summarize, for the CFO Project Team, the human health, animal health and/or ecological effects from the CFO emissions of Ammonia, Hydrogen Sulphide/TRS, Volatile Organic Compounds, Particulate Matter, Bioaerosols, odour and GHG. This summary will provide an overview of the information gathered, outstanding questions and information gaps. The Effects Subgroup will also provide, if required, recommendations on how to fill those information gaps.

Current Membership:

Calgary Health Region
SERLO
Health Canada
Intensive Livestock Working Group
Alberta Health and Wellness

Desired Membership Additions

Alberta Environment – Ecological Effects Expert

Environment Canada – Ecological Effects Expert

Purpose:

To provide credible science based information that will enable the CFO Project Team decision on strategic plan to manage the emissions from CFOs in Alberta.

Principles:

1. The information provided by the subgroup to the CFO Project Team will be credible and science based.
2. The subgroup will work by consensus on process issues but is not seeking consensus on information.
3. The subgroup will make technical decisions regarding the management of their work.