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EXPOSURE OF POULTRY FARM WORKERS TO AMMONIA, PARTICULATE MATTER AND MICROORGANISMS IN THE POTCHEFSTROOM DISTRICT, SOUTH AFRICA

by

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LIST OF ABBREVIATIONS

ACGIH	American Conference of Governmental Industrial Hygienists
AIHA	American Industrial Hygiene Association
ANCOVA	Analysis of covariance
APF	Assigned protection factor
ATS	American Thoracic Society
CAFO	Concentrated animal feeding operation
CDC	Centers for disease control
CEIL	Ceiling level
cfu	Colony forming units
CI	Confidence interval
COPD	Chronic obstructive pulmonary disease
ERPG	Emergency response planning guidelines
ERS	European Respiratory Society
FEV_1	Forced expiratory volume in one second
FLAGH	Farm labour and general health
FVC	Forced vital capacity
GEV_1	Geforseerde ekspiratoriese volume in een sekonde
GOLD	Global Initiative for Chronic Obstructive Lung Disease
GVK	Geforseerde ekspiratoriese kapasiteit
HIV	Human Immunodeficiency virus
HP	Hypersensitivity pneumonitis
MMOA	Mining Medical and Other health care professionals Association
MSHA	Mine Safety and Health Administration
NHLBI	National Heart, Lung and Blood Institute
NIOSH	National Institute for Occupational Safety and Health
NPL	National priority list
ODTS	Organic dust toxic syndrome
OESSM	Occupational exposure sampling strategy manual
OR	Odds ratio
OSHA	Occupational Safety and Health Administration
PEL	Permissible exposure limit
PM	Particulate matter
PPE	Personal protective equipment
ppm	Parts per million

RH	Relative humidity
SAIOH	Southern African Institute of Occupational Hygiene
SASOHN	South African Society of Occupational Health Nursing Practitioners
SASOM	South African Society of Occupational Medicine
SD	Standard deviation
STEL	Short term exposure limit
TB	Tuberculosis
TBG	Tydsbeswaarde gemiddelde
T_d	Dry bulb temperature
TLV	Threshold limit value
T_w	Wet bulb temperature
TWA	Time weighted average
WEEL	Workplace environmental exposure level guide
WHO	World Health Organisation

AFRIKAANSE TITEL: Blootstelling van werkers op hoenderplase aan ammoniak, stof en mikroörganismes in die Potchefstroom distrik, Suid-Afrika.

OPSOMMING

Motivering: Daar is baie meer navorsing gedoen oor respiratoriese gevare in die mynbedryf en ander swaar industrieë as in die hoenderbedryf, daarom is daar relatief min epidemiologiese data beskikbaar wat pulmonale infeksies in die konteks van die landbou omgewing aanspreek, veral vir die Suid-Afrikaanse populasie. Hoenderhuisstof was oor die algemeen gesien as onaktief, wat beteken dat dit nie baie negatiewe effekte op die longe van mense het nie. Nuwe navorsing toon egter dat hoenderhuisstof nie as onaktief gesien kan word nie, want dit is hoofsaaklik organiese stof wat bakterieë en ander biologies aktiewe substanse bevat. Verskeie gepubliseerde navorsingsartikels dokumenteer dat die wettige en aanbevole blootstellingslimiete vir die landbou omgewing te hoog is vir hoë digtheid diere voedingsoperasies. Daar vind 'n vermenging van biologies aktiewe agente plaas wat sinergisties kan werk om sodoende respiratoriese effekte tot gevolg te hê by baie laer vlakke van blootstelling. Die meeste van die blootstellingslimiete wat tans in Suid-Afrika toegepas word, is oorgeneem vanaf internasionale limiete en aanbevelings. Geografie, klimaat en graad van industrialisasie speel 'n baie groot rol in die lugkwaliteit van die landbou omgewing, daarom kan die relevansie van die buitelandse blootstellingslimiete bevraagteken word.

Doel: Om vas te stel of daar 'n korrelasie is tussen blootstelling aan hoenderhuisstof en longfunksie van hoenderplaas-werkers in die Potchefstroom distrik van Suid-Afrika. Verder, ook om vas te stel of die wetlike limiete wat tans in Suid-Afrika gebruik word vir ammoniak en stof blootstelling, voldoende beskerming bied vir hoenderplaas werkers wat blootgestel word aan biologies aktiewe stof.

Metodologie: Die betrokke studie was 'n deursnit, waarnemings-loodsstudie. Daar is 'n populasie van vyftig kontrakwerkers wat gemoeid is met verwydering van hoendermis uit hoenderhokke in die Potchefstroom distrik geïdentifiseer, negentien van die vyftig was ewekansig gekies vir deelname aan hierdie studie. Blootstelling aan totale- en respireerbare stof is bepaal deur middel van persoonlike monitering vir die volle duur van die tydsbeswaarde gemiddelde periode (8 uur TBG). Area monitering is gedoen vir ammoniak en bio-aërosole in die hoenderhuise van drie spesifiek afgebakende areas rondom Potchefstroom, weersomstandighede is ook in

ag geneem. Longfunksietoetse (spirometrie) is uitgevoer voor en na elke werkskof. Vraelyste is voltooi met die hulp van ondervraers om beroeps- en blootstellingsgeskiedenis te ondersoek en om moontlike simptome van organiese stofblootstelling te identifiseer.

Resultate en gevolgtrekkings: Die gemiddelde totale- en respireerbare stof konsentrasies voldoen aan die wetlike limiete van OSHA (Occupational Safety and Health Administration), NIOSH (National Institute for Occupational Safety and Health) en die Regulasies vir gevaarlike chemiese substansies van 1995, maar vyf-en-vyftig persent van die gemete totale stof en al die gemete respireerbare stof konsentrasies het die waardes oorskry wat Donham vir menslike gesondheid voorstel. Die spirometrie waardes van die proefpersone is normaal; daar is geen statisties betekenisvolle verskil tussen die gemiddelde basislyn GEV_1/GVK (verhouding van die geforseerde ekspiratoriese volume in een sekonde oor die geforseerde vitale kapasiteit) en die voorspelde GEV_1/GVK nie. Die resultate toon geen betekenisvolle veranderinge in enige van die gemete veranderlikes oor die verloop van die werkskof nie en daar is ook geen betekenisvolle korrelasies gevind tussen die stof konsentrasies en enige van die spirometer opnames nie. Dit kan saamgevat word dat die beroepsblootstelling aan ammoniak, stof en mikroörganismes op die hoenderplase in die Potchefstroom distrik, Suid-Afrika, geen negatiewe effekte op die werkers se longfunksie het nie en dat die wetlike blootstellingslimiete wat tans in Suid-Afrika gebruik word, voldoende beskerming aan die werkers bied in die korttermyn.

Sleutelwoorde: beroepsblootstelling, stof, ammoniak, mikroörganismes, longfunksie, hoenderbedryf, landbou, Suid-Afrika.

ENGLISH TITLE: Exposure of poultry farm workers to ammonia, particulate matter and microorganisms in the Potchefstroom district, South Africa.

SUMMARY

Motivation: The investigation of agricultural respiratory hazards has lagged behind the investigation of hazards in mining and other heavy industries. Relatively few epidemiological data are available addressing pulmonary infections in the context of the agricultural work environment, especially for the South African population. Poultry house dust was generally considered nuisance or inert, meaning it has little adverse effect on human lungs. New research shows that because poultry house dust is largely organic and contain bacteria and other bioactive substances, it cannot be considered inert. Several published research manuscripts document that the legal and recommended exposure limits for the toxic substances found in the agricultural environment are to high for concentrated animal feeding operations (CAFO's). In CAFO's there is a mixture of biologically active agents that can work synergistic to produce respiratory and systemic effects at much lower levels. Most of the current legal exposure limits used in South Africa are adopted from international limits and guidelines. Because of the influence of geography, climate and degree of industrialisation on the agricultural air quality, the relevance of the foreign exposure limits is questionable.

Aim: To determine if there is a correlation between occupational exposure to poultry farm dust and the lung function of poultry farm workers in the Potchefstroom district, South Africa. Also to determine if the current legal exposure limits used for ammonia and particulate matter (PM) in South Africa, offer adequate worker protection for poultry farm workers exposed to biologically active dust.

Methodology: This was an observational, cross-sectional pilot study. A target population of fifty contract workers concerned with the removal and disposal of poultry manure were identified in the Potchefstroom district and a random sample of nineteen was drawn for participation in this study. Exposure to total and respirable dust were determined by means of personal sampling for the full duration of the time-averaging period (8-hour TWA). Area monitoring for ammonia and bio-aerosols were done in poultry houses in three specific demarcated areas around Potchefstroom, and weather conditions were taken into account. Lung function tests (spirometry)

were conducted before and after each work shift. Interviewer administered questionnaires were used to assess occupational and exposure histories and to detect symptoms of organic dust exposure.

Results and conclusions: The mean total- and respirable dust concentrations complied with the legal limits of OSHA, NIOSH and the Regulations for hazardous chemical substances of 1995. However, fifty five percent of the measured total dust concentrations and all of the respirable dust measurements exceeded Donham's recommended values for human health. The spirometric values of the subjects were normal; there was no statistical difference between the mean baseline FEV_1/FVC and the mean predicted FEV_1/FVC. Results also show no statistically significant cross shift changes in any of the measured variables and there is no significant correlation of the measured dust concentrations to any of the spirometric measurements. It can be concluded that occupational exposure to ammonia, particulate matter and microorganisms on poultry farms in the Potchefsroom district, South Africa, do not have any adverse effects on the workers' lung function and the workers are adequately protected in the short term, by the legal limits that are currently used in South Africa.

Keywords: occupational exposure, particulate matter, ammonia, microorganisms, pulmonary function, poultry industry, agriculture, South Africa.

PREFACE

This dissertation is presented for the partial completion of the M.Sc. degree in Occupational Hygiene at the North-West University, Potchefstroom. It was decided to use the article format for the purpose of this study. Therefore, Chapter 2 is a manuscript in the form of an article. The article will be submitted for publication to the accredited journal, Occupational Health Southern Africa. Although the appropriate and relevant literature background is discussed in the manuscript, Chapter 1 also gives an additional, more elaborate literature background. In the manuscript the project leader and assistant project leader are named as co-authors. The main and first author was however responsible for most stages of the manuscript, including literature searches, the collection of data, interpretation of results and writing of the article. The co-authors, therefore, acted in their roles as project leader and assistant project leader. All co-authors gave consent that the article could be used in this dissertation. In Chapter 3 a summary of the main findings is provided, confounders are discussed, conclusions are drawn and recommendations are made. The relevant references are provided according to the authors' instructions provided by the journal, Occupational Health Southern Africa. For the purpose of uniformity, the same style of reference was used throughout this dissertation.

AUTHORS' CONTRIBUTIONS

The contribution of each of the researchers involved in this study is given in the following table:

Name	Role in the study
Miss AC de Jager (Honns. B.Sc) (Physiologist)	Responsible for literature searches, collection of data, analysis of data (microbiological and gravimetrical methods), initial statistical analysis, interpretation of results, planning, design, and writing of the manuscript.
Mr MN van Aarde (M.Sc.) (Occupational hygienist, physiologist)	Promoter. Supervised the writing of the manuscript, collection of data, as well as initial planning and design of the manuscript.
Prof JM van Rooyen (D.Sc.) (Physiologist)	Co-promoter. Supervised the writing of the manuscript as well as the initial planning and design of the manuscript, and gave valid scientific input.
Miss IM Palmer (Honns. B.Sc.) (Physiologist)	Assistance in data collection and microbiological analysis.

The following is a statement of the co-authors confirming their individual role in the study and giving their permission that the manuscript may form part of this dissertation.

I declare that I have approved the above-mentioned manuscript, that my role in the study, as indicated above, is representative of my actual contribution and that I hereby give my consent that it may be published as part of the M.Sc. dissertation of Miss AC de Jager.

Mr MN van Aarde

Prof JM van Rooyen

Miss IM Palmer

CHAPTER 1

INTRODUCTION AND LITERATURE OVERVIEW

GENERAL INTRODUCTION

This project is an expansion of the FLAGH-project (Farm Labour And General Health) of the North-West University. It is mainly concerned with the exposure of poultry farm workers to ammonia, particulate matter and microorganisms.

As a result of the widely held images of urban pollution and the persistence of an "agrarian myth" that associates life on the farm with healthful joys, a fundamental reality is ignored: *Agriculture can be a dangerous occupation.*

Agriculture has been defined by the World Health Organization (WHO) as all forms of activity connecting with growing, harvesting, and primarily processing of all kinds of crops; with breeding, raising and caring for animals; and with tending gardens and nurseries[1]. Respiratory diseases associated with agriculture were one of the first-recognised occupational hazards. As early as 1555, Danish Bishop Olaus Magnus warned about the dangers of inhaling grain dust in his book "Historia de Gentibus Seprentrionalibus" and Ramazzini again noted the risk in 1700 in his seminal work *De Morbis Artificum* [2,3]. Despite this early recognition of respiratory hazards in agriculture, it has only been in the 20th century that this problem has been carefully studied and documented. In general, the investigation of agricultural respiratory hazards has lagged behind the investigation of hazards in mining and other heavy industries. These agricultural hazards, however, are of serious concern[4]. While agriculture may be spoken of as a single industry, it is extremely diverse with substantial respiratory hazards occurring from organic and inorganic dust, chemicals, gases, and infectious agents. This is different from many industries posing the threat of occupational respiratory diseases, which can be characterised by a common method of production and one or two predominant respiratory hazards or categories of hazards – e.g.: asbestos, polycyclic aromatic hydrocarbons or silica. The nature of agricultural practice also varies with climate, geography, and other determinants of the commodities grown, and with the degree of industrialisation of the country or region. The permutations of potential exposures are virtually infinite[4].

Relatively few epidemiological data are available addressing pulmonary infections in the context of the agricultural work environment, especially for the South African population. Agricultural workers may fall victim to the normal range of respiratory infections experienced by persons from the general population, yet there are many

infectious conditions for which agricultural work represents a clear risk factor because of unique exposures[5].

The atmosphere in poultry houses, particularly where ventilation is limited, can adversely affect human health. The acceptable level of airborne dust and gasses often exceeds limits imposed elsewhere. To further worsen the problem, organic dust particles abundant in poultry houses can interact with human physiological systems. In addition, ammonia can absorb onto dust particles, which allows it to bypass some of the control systems in the respiratory tract and find its way into the alveoli of the lungs[5].

This raises the following questions:

1) Is there a correlation between the amount of occupational exposure to poultry farm dust and the lung function of the poultry farm workers?

2) Do the legal exposure limits for ammonia and particulate matter in South Africa offer adequate worker protection for poultry farm workers exposed to biologically active dust?

The ultimate goal of this study is to communicate an understanding of respiratory disease in agricultural populations, poultry farming in particular.

LITERATURE OVERVIEW

1. THE AGRICULTURAL WORK FORCE

The World Health Organization (WHO) defined an *agricultural worker* in 1962 as any person engaged either permanently or temporarily, irrespective of legal status, in activities related to agriculture[1].

Agriculture continues to play a fundamental role in the economy and daily existence of the populations of developing countries. The relative size of the agricultural workforce is substantially greater in developing countries than in the industrialised nations[6]. Across South Africa, a wide variety of agriculture can be found. Agricultural production in South Africa has almost doubled in the past 30 years although production varies from year to year due to erratic weather[7].

Unlike many other occupations, farmers often continue to work well beyond 65 years of age, and the farm is often both residence and worksite. Even family members not directly engaged in the farm work may be incidentally exposed to respiratory hazards (the risk of active workers is even higher). Thus farming, uniquely among industries, may result in extensive exposure to workers' family members, increasing the population at risk by several-fold[4].

There is also a widening gap between health care services offered in rural and urban areas. Primary health care, emergency medical services, public health measures, and health related social services are generally less available in rural areas. Agricultural respiratory diseases must be considered in the context of rural health care delivery, as well as characteristics of the particular population[4].

2. ORGANIC DUST

The University of Iowa's Environmental Health Sciences Research Centre states that it is organic dust that accounts for the most common exposure leading to agricultural respiratory disease, and that virtually everybody who works in agriculture gets exposed to some organic dust[8]. Airborne and settled particulate matter of biologic origin is often referred to collectively, in the field of occupational hygiene, as organic dust. The term is broadly defined as dust with very heterogeneous composition[9]. Organic dust exposures may vary qualitatively as well as quantitatively from one occupation to another. Poultry house dust contains feed and fecal particles, feather barbules, skin debris, fungal fragments, and spores, bacteria and bacterial fragments, viruses and particles of litter. Such dust was generally considered nuisance or inert, meaning it has little adverse effect on the lungs. New research shows that because poultry house dusts are largely organic and contain bacteria and other bioactive substances, it cannot be considered inert[10].

The particle size of dust is measured in microns (μ). Collaboration between international organisations has produced agreement on the definition of health-related aerosol fractions. The sizes are termed *inhalable* fraction, *thoracic* fraction and *respirable* fraction[11].

- *The inhalable fraction*, which includes the thoracic and respirable fractions, is defined as the mass fraction of total airborne particles that are inhaled through the nose and/or mouth.

- *The thoracic fraction*, which includes respirable fraction, is defined as the mass fraction that penetrates the respiratory system beyond the larynx.

- *The respirable fraction* is defined as the mass fraction that penetrates to the unciliated airways of the lung, known as the alveolar region, where gas exchange takes place[11].

Dust concentrations in poultry houses usually vary between 0.02 and 81 mg/m^3 for inhalable dust and between 0.01 and 6.5 mg/m^3 for respirable dust.

Factors that affect dust concentrations in poultry houses include:

- Class of animal
- Animal activity levels
- Choice of bedding materials
- Cleanliness of the buildings
- Temperature
- Relative humidity
- Ventilation rate
- Stocking density
- Feeding method[12].

Poultry barns in Canada have much greater concentrations of respirable dust during winter than during summer. Keeping respirable dust to an acceptable level in the winter months is difficult to achieve. Active animals propel dust into the air at such rates that it cannot be removed rapidly enough by the ventilation systems. Summer levels of respirable dust are usually lower because of high ventilation rates[5].

When fine dust enters the respiratory system, the human body considers it to be foreign material that should be defended against. The main effects of dust on health are an inflammatory response (chronic irritation) or a toxic response[13].

3. BIOLOGICAL AGENTS

Biological agents are usually defined as agents from plant or animal matter or from microorganisms. The effects of exposure to biological agents can range from contagious infectious diseases, to acute toxic effects, allergies and also cancer.

3.1 PATHOGENS

An infection involves more than simple colonisation. Infection means that an organism is present in the host and is replicating with the result that the host develops sub clinical or clinical disease[14]. Disease-causing microorganisms are referred to as pathogens. Diseases that can be transmitted from animals to humans are referred to as zoonotic diseases. Pathogens from poultry manure can be transmitted to humans via air or fecel-oral transmission.

Air transmission

Airborne dust particles or droplets of water may contain bacteria or viruses, which can be inhaled by humans.

Fecel-oral transmission

Humans can ingest manure pathogens by consuming contaminated drinking water, swimming in contaminated surface water and by failing to wash their hands after handling infected poultry or manure[12].

Some of the pathogens with an influence on human respiratory health are *Pseudomonas aeruginosa, Actinomycetes, Mycobacterium, Salmonella spp, Streptococcus* and *Chlamydia psittaci*[12,15-18].

3.2 ALLERGENS AND PRO-INFLAMMATORY AGENTS

In general, occupation related respiratory symptoms result from airway inflammation caused by specific exposures of toxins, pro-inflammatory agents or allergens. Based on the underlying inflammatory mechanisms and subsequent symptoms a distinction between allergic and non-allergic respiratory diseases can be made. Non-allergic respiratory symptoms reflect a non-immune-specific airway inflammation, whereas allergic respiratory symptoms reflect an immune specific inflammation in which various antibodies can play a major role in the inflammatory response[14].

Allergenic agents

Allergy can be defined as acute or chronic symptoms and illness due to exposure to external agents to which the exposed subject is hypersensitive, because of a preceding specific immunologic sensitisation[14]. The agent A is called the *allergen*, and the specific immune response *allergic sensitisation.* All agents that can induce specific immune responses are also potential allergens. The term *allergen* can refer to a single molecule, a mixture of molecules, or a particle from which allergen molecules can be eluted. The latter may be dead material like animal skin scales or mite fecal particles, or viable or living (but non-pathogenic) propagules such as pollen grains, bacteria or mold spores[14]. *Occupational allergy* can be defined as allergic disease in which both sensitisation to the allergen *and* the induction of symptoms are caused by exposure to and allergen at the workplace. The cause-effect relation is usually rather obvious, since exposure to the "occupational allergen" practically only occurs in the work environment[14].

Pathogenic infectious agents are usually not called allergens. The only exception may be the respiratory pathology caused by spores of various molds, especially *Aspergillus fumigatus,* which can cause both a lung mycosis and simultaneously induce severe allergic reactions and hypersensitivity[14].

The most potent common allergens are mainly animal proteins, such as the house dust mite fecal proteins and the cat skin dander protein. According to various studies the urinary proteins may be strong allergens as well[19].

Pro-inflammatory agents

Although various substances in organic dust have been identified as possible causal agents in non-immune airway inflammation and related respiratory health effects, there are only few that are well characterised, of which almost all are of microbial origin. Most information is available on bacterial endotoxin and it is believed that bacterial endotoxin is one of the major causative agents for organic dust induced diseases. In addition, there are several other microbial components that are suspected to be involved in non-allergic respiratory symptoms and diseases, of which fungal β(1-3)-glucans and mycotoxins are probably the most important[14]. Microbial contaminated plant material as well as animal feces contribute largely in organic dust related exposure to these agents. Elevated exposures are therefore prevalent mainly in agricultural and related industries[14].

Endotoxins are integral components of the outer membrane of gram-negative bacteria and are composed of proteins, lipids, and lipopolysaccharides. The term "endotoxin" refers to the toxin as present on the bacterial cell wall, which is often liberated as a result of cell lyses[20,21]. Thomas' [22] suggestions that endotoxins are "read by our tissues as the very worst of bad news" and that in response to these molecules "we are likely to turn on every defence at our disposal," elaborate beautifully the toxic potential of these macromolecules[20].

Data on inhalation experiments on human volunteers are available. Pernis et.al.[23] themselves inhaled endotoxin aerosols derived from *E. Coli* in doses of 5, 10 and 20 μg. Acute effects were dry cough and shortness of breath accompanied with a decrease in FEV_1 (Spirometric lung function parameter: Forced Expiratory Volume in 1 Second). Cavanga et. al.[24] demonstrated a more than 10 % decrease in FEV_1 in

two of eight volunteers that were exposed to a dose of 80 μg *E. Coli* endotoxin. Except from these smaller studies where subjects where exposed to pure endotoxin, there are some larger studies in which healthy subjects were experimentally exposed to endotoxin containing cotton dust[25,26]. These studies showed similar health effects including fever, malaise, chest tightness, and breathing difficulties that were clearly associated with the level of endotoxin exposure but not with the level of dust exposure. Endotoxin related lung function changes and symptoms as reported in the above summarised experimental studies, have been confirmed in a large number of field studies conducted in various environments such as swine confinement workers, animal feed and grain workers, fibreglass workers, cotton mill workers, potato processing workers and poultry slaughter houses[27-33]. In most of these studies endotoxin exposure was much stronger associated with the studied health effects than dust exposure.

Due to financial and other limitations, this study will only investigate the prevalence of specific gram-negative bacteria and not specifically quantify the amount of endotoxins released. The study specifically investigates the prevalence of gram-negative bacteria in the poultry environment with an effect on human lung function, namely *Pseudomonas aeruginosa, Serratia marcescens, Salmonella spp and Escherichia coli (E. Coli).*

4. DECOMPOSITION GASES

Researchers have noted the presence of as many as 150 potentially toxic gases arising from the storage and handling of agricultural animal waste[34-36]. These gases include ammonia (NH_3), hydrogen sulfide (H_2S), methane (CH_4), carbon dioxide (CO_2), nitrogen compounds (NO_x) and trace gases associated with odour. The sources and properties of these gases are shown in Table 1.

Table 1. Sources and properties of gases emitted from poultry operations[12]

Gas	Source	Properties
Ammonia (NH_3)	• Manure decomposition • Composting • Commercial fertilizer handling, storage and manure application	• Sharp, pungent odour • Lighter than air
Hydrogen sulfide (H_2S)	• Bacterial decomposition in manure *without oxygen* (anaerobic)	• Heavier than air • Accumulates near the floor in enclosed buildings • Initially a rotten egg smell, but lethal concentrations paralyze the sense of smell
Methane (CH_4)	• Decomposition of manure *without oxygen* (anaerobic)	• No smell • Lighter than air
Carbon dioxide (CO_2)	• Anaerobic and aerobic decomposition or organic materials • Plant and animal respiration • Combustion of fossil fuels • Manure is not considered a major source of CO_2	• No smell • Heavier than air
Nitrogen oxides * (NO_x)	• NO_x is naturally generated by bacterial processes, decomposition and fires • Humans contribute primarily through burning fossil fuels	• NO and N_2O are colorless • NO_2 is reddish brown • NO_2 is the most common of NO_x • NO_2 is the main component of smog
Trace gases associated with odour	• Decomposition of manure	• Often have distinct smells

Nitrogen oxides (NO_x) include nitric oxide (NO), nitrogen oxide (NO_2) and nitrous oxide (N_2O)[12].

Many fatal or near-fatal incidents involving poultry manure handling activities have been documented[37,38]. Manure disturbing activity can result in the creation of oxygen-deficient, toxic and even explosive atmospheres[34,38,39]. There have been a number of reports of air contaminant measurements associated with disturbing

poultry manure[40,41]. One study found levels of ammonia routinely reaching or exceeding 100 ppm associated with tilling turkey confinements manure[40].

In the United States, worker exposure to potentially toxic gases have been frequently described in swine, poultry and cattle settings, with the relevant risk of exposure to each gas varying with the particular manure type and storage method[4].

In this project the study population will only be exposed to aerobic decomposition of (dry) poultry manure, therefore only the worker exposure to ammonia (NH_3) will be monitored.

4.1 AMMONIA (NH_3)

Ammonia is a colourless gas and has a characteristically pungent odour. In nature, most ammonia is produced by the decomposition of animal manures. The gas is classified as an irritant, and is lighter than air[42]. Ammonia has been identified in at least 135 of 1,631 National Priority List (NPL) hazardous waste sites in the United States[43]. The most important injurious effects of ammonia on humans are due to its irritative and corrosive properties. Exposures to ammonia as a gas cause chemical burns of the respiratory tract, skin, and eyes[43].

For the purposes of this study, the main focus is on the effects of ammonia on the *respiratory system.* Ammonia is an upper respiratory irritant in humans. Exposures to levels exceeding 50 parts per million (ppm) result in immediate irritation to the nose and throat; however, tolerance appears to develop with repeated exposure. Exposure to an air concentration of 250 ppm is bearable for most persons for 30-60 minutes. Acute exposures to higher levels (500 ppm) have been shown to increase respiratory minute volume. Accidental exposures to concentrated aerosols of ammonium salts or high concentrations of ammonia gas have resulted in nasopharyngeal and tracheal burns, airway obstruction and respiratory distress, and bronchiolar and alveolar edema[43]. Chronic occupational exposure to low levels of airborne ammonia (<25 ppm) had little effect on pulmonary function or odour sensitivity in workers at some factories, but studies of farm workers exposed to ammonia and other pollutants in livestock buildings, indicated an association between exposure to pollutants, including ammonia, and an increase in respiratory symptoms (such as bronchial reactivity/hyper responsiveness, inflammation, cough,

wheezing, or shortness of breath) and/or a decrease in lung function parameters. The contribution of ammonia to these respiratory symptoms is unclear[43].

Evidence also suggests that increasing levels of ammonia in poultry manure during decomposition will destroy *Salmonella* species and other pathogens that can potentially harm humans[12].

4.2 ODOUR

One of the primary complaints about poultry operations is odour, but odour is generally considered as a nuisance rather than a health risk. There is a difference between the psychological and physiological health effects related to odour exposure. *Psychological effects*, such as irritation, can result from exposure to odour and often occur at levels well below those that can harm human health. *Physiological effects* can occur through exposure to specific compounds that make up odour, for example, asphyxiation from exposure to elevated levels of hydrogen sulphide (H_2S) in a confined space[12].

It is difficult to evaluate odour and its health effects for the following reasons:

- Psychological and physical health effects are not necessarily independent.
- Odour from poultry manure is made up of about 160 compounds. Humans have many and varied responses to these compounds.
- The proportion and characteristics of odour contributed by each of the primary sources (barns, storages and land application) is not well understood.
- Odour intensity and odour offensiveness varies between individuals.
- Combining different odorants can have positive and negative effects on intensity and offensiveness. These effects are not easily predicted[12].

5 EXPOSURE LIMITS RELATED TO AIR QUALITY

Occupational health hazards for those working in concentrated animal feeding operations (CAFO's), have been long recognised. Research documents that current recommended or legal occupational exposure levels are not sufficient to protect workers[44]. The Occupational Safety and Health Administration (OSHA) has standards for non-specific dust (total dust and respirable dust) and for cotton dust, which is the only specific agricultural dust OSHA has a standard for[45]. The National Institute for Occupational Safety and Health (NIOSH) has recommended exposure limits for cotton dust, grain dust and wood dust[46], but these limits may not adequately protect workers exposed to organic dusts contaminated with microorganisms[45].

In the United States, there are four sources of recommendations in regards to occupational exposure limits. These include the American Conference of Governmental Industrial Hygienists (ACGIH), the American Industrial Hygiene Association (AIHA), the National Institute for Occupational Safety and Health (NIOSH) and the Occupational Safety and Health Administration (OSHA). The first two organizations (AIHA and ACGIH) are private professional organisations. The third, NIOSH, is a governmental educational and research organisation. OSHA is the only regulatory and enforcement agency of these four. The terminology for exposure limits is different for each of these organisations. AIHA, ACGIH, and NIOSH issue, respectively:

- Emergency Response Planning Guidelines/Workplace Environmental Exposure Level Guides (ERPGs/WEELs),
- Threshold Limit Values (TLVs) and
- Time Weighted Average Exposure Limits (TWAs).

OSHA issues Permissible Exposure Limits (PELs)[44].

The primary exposures of occupational concern in this project include ammonia (NH_3), total and respirable particulate matter (PM) and specific microorganisms (bioaerosols and endotoxin). However, none of the bodies mentioned above have specified limits for bioaerosols or endotoxin[44]. Table 2 lists the maximum indoor concentration levels for each of these bodies for the agents specified.

Table 2. Maximum concentration levels listed for occupational health[44]

	NH_3	Total Particulate Matter	Respirable dust	Bioaerosols	Endotoxin
AIHA	25 ppm	Not listed	Not listed	Not listed	Not listed
ACGIH	25 ppm	4 mg/m^3 (grain dust) 10 mg/m^3 (Nuisance dust)	3 mg/m^3 (grain dust)	Not listed	Not listed
NIOSH	25 ppm	4 mg/m^3 (grain dust)	Not listed	Not listed	Not listed
OSHA	50 ppm	10 mg/m^3 (grain dust) 15 mg/m^3 (Nuisance dust)	5 mg/m^3	Not listed	Not listed

ppm, parts per million.

The exposure criteria can be reported as:

- Time weighted average (TWA) exposure averaged over the full work shift (exposure levels in table 2);
- Short term exposure limit (STEL) recommendations for a 10- to 15-min exposure period; and
- Ceiling levels (CEIL) not to be exceeded at any time[4].

Donham[47] recommended maximum exposure values for the most common contaminants found in the swine confinement environment. These values are shown in Table 3. In most cases, the levels of gases and dust in these environments do not exceed the standards listed in Table 3 [48].

Table 3. Maximum indoor air contaminant (TWA) levels recommended for human health in swine buildings[47]

Air Contaminant	Recommended 8-hour TWA for human health
Total dust (mg/m^3)	2.40
Respirable dust (mg/m^3)	0.23
Endotoxin (g/m^3)	0.08
Ammonia (ppm)	7.00
Total microbes (cfu/m^3)	4.3×10^5

TWA, time weighted average; ppm, parts per million; cfu, colony forming units.

5.1 RELEVANCE OF LEGAL OR OTHER RECOMMENDED LIMITS TO OCCUPATIONAL AIR QUALITY

Regarding OSHA and NIOSH occupational health exposure regulations, the PEL's and TWA's listed for the hazardous substances found in CAFO's is not highly relevant. The reasons are as follows:

- The scientific literature documents that endotoxin is one of the most hazardous substances to CAFO workers[9]. However OSHA and NIOSH has no legal exposure limits for endotoxin[44].

- The legal exposure limits of OSHA and NIOSH for PM is based on a non-biologically active dust. However, the PM inside CAFO's is highly biologically active, (high concentrations of microbes, endotoxins, and glucans) and is hazardous at much lower levels than the published PEL's[49] and TWA's.

- The PEL's and TWA's are written assuming exposures to one toxic substance. CAFO's result in complex mixed exposures, which lowers the allowable exposure to each individual component of the mixture[49].

Therefore, the OSHA, NIOSH and other recommended limits are not highly relevant.

Although NIOSH, ACGIH and IAHA are more stringent than OSHA, research findings indicate that they are still much higher than they should be to offer adequate worker protection in mixed exposure situations like CAFO's[44].

In South Africa we also use the exposure limits of the Regulations for Hazardous Chemical Substances of 1995. Most of these exposure limits however, are adopted from international limits and guidelines. Because of the influence of geography, climate and degree of industrialisation on the agricultural air quality, the relevance of the foreign exposure limits is questionable.

6 MIXED EXPOSURES

Several published research manuscripts[50-52] document that the legal and recommended exposure limits for the toxic substances found in the agricultural environment are too high for CAFO's. In CAFO's there is a mixture of biologically active agents that can combine to produce respiratory and systemic effects at much lower levels[53]. The simultaneous exposure to these toxic substances may be additive or synergistic. When mixed exposures are present, and unless other data indicate differently, the effects of the toxic substances should be considered additive[44]. For example where C1, C2 and Cn are measured concentrations of hazardous substances, and T1, T2 and Tn are their respective threshold limit values (TLV's), the relationship is defined mathematically as follows[44].

$$C1/T1 + C2/T2 + Cn/Tn = < 1$$

There may be instances when the effects of two substances are greater than additive; this is defined as a synergistic interaction. If synergy is present, mixed exposures are even more hazardous than if the effects were merely additive. Cumro and Donham[53] defined such a relationship between NH_3 and PM in CAFO's. Data were analysed from an exposure-response study of 149 poultry CAFO workers. Analysis of this data-set revealed prominent dose-response relationships between increasing PM, NH_3 and endotoxin concentrations with corresponding cross-shift declines in worker lung function. As health effects to poultry workers from exposure to both dust and ammonia were less than half the published ACGIH TLV's, investigations were undertaken to study possible interactions between these substances. The results demonstrated that when workers are exposed to both PM and NH_3, the adverse effect on pulmonary function is up to 156% greater than the individual effects of these gases[53].

Assuming a typical swine CAFO winter concentration of 10 ppm of NH_3, and PM of 3.5 mg/m^3, and the TLV for grain dust of 4 mg/m^3; the correct relationship to determine if exposure limits are exceeded in this situation would be as follows:

$$\frac{[NH_3]}{\text{TLV of } NH_3} + \frac{[PM]}{\text{TLV of PM}} \times 1.56$$

An example of a typical swine building would be as follows:

$$\frac{10 \text{ ppm}}{25 \text{ ppm}} + \frac{3.5 \text{ mg/m}^3}{4 \text{ mg/m}^3} \times 1.56 = 2.0$$

In other words, a typical building might exceed the recommended limits by two times. Synergy of simultaneous dust and ammonia exposures in a working environment raises the question of redefining exposure limits for organic dust and ammonia when workers are exposed simultaneously to these substances[44].

7 SMOKING

Sterling and Weinkam[54] stated in 1978 that, in general, farm workers smoke less than individuals in most other occupations. This tendency is demonstrated by the results of general health surveys, cancer case-control studies and studies of respiratory disease among farm workers[54]. The most valid evidence of lower smoking rates among agricultural workers comes from studies on population-based comparison groups from the same area[4]. Reddy et al[55] conducted a study in 1996 to provide data on the South-African adult population's smoking status. They found that thirty-four percent of adult South Africans smoke. There was however no distinction made as to what percentage of the smoking population was in the agricultural industry. Farm workers in general have a relatively low prevalence of cigarette smoking in comparison to the general population, and given that, it is really striking that they have such major problems with airway disease. So even if it turns out not to be endotoxin or dust, there is something in the environment that is causing the farm workers to have problems with airway disease[8].

In this study, the subjects' smoking status and particular smoking habits were surveyed by means of an interviewer-administered questionnaire.

8 AIRWAY DISORDERS

Agriculture involves potential exposure to a wide range of respiratory toxins, many in concentrations higher than in other industries. Despite low rates of cigarette smoking, farmers have an increased prevalence of several acute and chronic respiratory diseases[4]. Studies indicate that the risk associated with developing respiratory disease appears to be more than threefold greater among those who are heavily exposed to inhalable dust generated in the agricultural environment[8]. The great majority of microorganisms present in the environment, especially in soil, manure or biosolids, have no negative effect on human health. However, following aerosolisation, some can be found in abnormally high concentrations in the air, and present a risk for individuals exposed to them[56].

There is increasing evidence that endotoxins are a significant contributor to respiratory disease. A dose-response to endotoxin and pulmonary function deterioration has been established in numerous studies[51-52,57-59]. Endotoxins are associated with the release of pro-inflammatory agents including tumour necrosis factor, interleukins, cytokines and inflammatory cells[60]. There are associated declines in pulmonary functions, primarily FEV_1 and symptoms including chest tightness, cough, dyspnea, and sputum production. Most CAFO research has focused upon swine confinement operations but recent studies have indicated similar dose-response findings in poultry operations[61].

Workers may not immediately notice any ill effects from airborne contaminants, but numerous cases of delayed responses have been documented. Symptoms such as chronic bronchitis, coughing, wheezing, and allergies can gradually develop over a long period of time[5]. The impact on the respiratory system may also vary considerably. Organic exposures may affect the airways and, depending on the antigenicity of the material and host susceptibility, may result in asthma, asthma-like syndrome, or chronic obstructive airway diseases[4].

Table 4. Principal bio-aerosols and their effects on human health[62]

PRINCIPAL BIOAEROSOLS	ORIGINS AND ASSOCIATIONS	REPORTED SYMPTOMS AND EFFECTS
BACTERIA: **Gram negative bacteria (*E. coli, Salmonella*); Thermophilic actinomycetes**	Abundant in environment and in humans. Outdoors, they originate in water, soil and plants, and they are associated with the presence of humans and animals.	Mucous membrane irritation, gastro-intestinal and respiratory problems (Gram negative bacteria and endotoxins), Hypersensitivity pneumonitis (thermophillic actinomycete).
MOULDS: ***Aspergillus fumigatus***	Ubiquitous in nature; proliferates well in humid conditions. *Aspergillus fumigatus* is thermo tolerant, sometimes pathogenic; it is found on manure, compost, wood and other organic material.	Allergic reactions, infections and irritation, organic dust toxic syndrome (ODTS). Concentrations of up to 10^5 cfu/m^3 of *Aspergillus fumigatus* would not be considered a risk for healthy individuals but one spore could be infectious for immunocompromised persons.
METABOLITES OR TOXINS **Endotoxins;** **Mycotoxins**	Ubiquitous, endotoxins are complexes that are integral parts of the outer membrane of Gram-negative bacteria. Their presence is often associated with organic dust. Mycotoxins (spores and propagules) are released by moulds.	The effects of endotoxins and their role as bioaerosol are not well known. Symptoms are cough, shortness of breath, fever, obstruction and inflammation of the lungs, and gastro-intestinal problems. The effects of mycotoxins are not well known. Symptoms are skin and mucous membrane irritations, dizziness, immunosupression, headache, nausea, and cognitive effects.

The principal respiratory disorders associated with agricultural exposures are asthma and other forms of airflow obstruction like chronic obstructive pulmonary disease (COPD); hypersensitivity pneumonitis (extrinsic allergic alveolitis); pulmonary fibrosis and infection (like tuberculosis). Organic dust toxic syndrome (ODTS), while not a localized disease, is a result of inhalation and therefore it is considered here. These disorders are outlined in Table 5 together with their most characteristic features.

Table 5. The principal occupational respiratory disorders of farm workers[63]

	ASTHMA	COPD	HYPERSENSITIVITY PNEUMONITIS	ODTS
Symptoms	Shortness of breath, wheezing, coughing.	Cough with phlegm, shortness of breath	Fever, shortness of breath, cough	Fever, malaise, cough, chest tightness
Lung function	Obstruction	Obstruction	Restriction	Normal or mild restriction
Bronchoalveolar lavage	Eosinophills	Neutrophils	Neutrophils, lymphocytes	Neutrophils
Trigger	Type I allergens, irritating agents	Organic dusts	Type III allergens	Endotoxins? Beta-1,3-glucans?
Pathogenesis	Inflammation, obstruction	Inflammation, particle deposition, obstruction	Inflammation of the interstitium	Inflammation of the airways and alveoli
Mechanism	IgE, mast cells, histamine, muscle contraction	Destruction of cilia, production of phlegm, emphysema	Macrophages, fibroblasts, fibrosis	Systemic cytokine response
Time course	Chronic	Chronic	Chronic	Acute

8.1 ASTHMA

Asthma is characterised by spastic contraction of the smooth muscle in the bronchioles, which causes extremely difficult breathing. The usual cause is contractile hypersensitivity of the bronchioles in response to foreign substances in the air. In about 70% of patients younger than 30 years of age, the asthma is caused by allergic hypersensitivity, espesially sensitivity to plant pollens. In older people, the cause is almost always hypersensitivity to non-allergenic types of irritants in the air, such as irritants in smog[64]. Asthma can also be defined as a disease characterised by variable airflow obstruction, airway hyper responsiveness, and airway inflammation[65]. When the asthmatic person breathes in the irritant that he or she is sensitive to, the irritant reacts with the mast cell-attached antibodies and causes the mast cells to release histamine, slow-reacting substance of anaphylaxis, eosinophilic chemotactic factor and bradykinin. The combined effects of these factors produce (1) localised edema in the walls of the small bronchioles, as well as secretion of thick mucus into the bronchiolar lumens, and (2) spasm of the bronchiolar smooth muscle. Therefore, the airway resistance increases greatly. The bronchiolar diameter becomes more reduced during expiration than during inspiration, therefore the asthmatic person can often inspire quite adequately, but has great difficulty expiring. Clinical measurements show (1) greatly reduced maximum expiratory rate and (2) reduced timed expiratory volume[64].

Objective signs of airflow obstruction are often associated with symptoms of chest tightness, wheezing, coughing and dyspnea[66].

Asthma-like syndrome

A distinction has been drawn between asthma and asthma-like syndrome, basing that distinction on an understanding of asthma as a relatively progressive condition frequently related to antigenic exposures. Asthma-like syndrome on the other hand, suggests mild, apparently reversible airway obstruction in the face of transient or mild increases in non-specific airway responsiveness resulting primarily from non-antigenic exposures[4]. It can be said that asthma-like syndrome is a non-allergic respiratory condition that is identical clinically to asthma but is not associated with persistent airway inflammation or airway hyper reactivity. The pulmonary deterioration can often be detected only by cross-shift testing[61]. The cross-shift decline in FEV_1 is generally less than 10 % but can be between 10% and 15%[4].

Occupational Asthma

Occupational asthma has been considered in the agricultural setting as encompassing both aggravation of previous asthma and the development of new asthma apparently triggered by elements in the workplace. In taking this approach, it is recognised that agricultural workers frequently grow up and live in the workplace[4]. A legal approach to the term "occupational asthma" will be avoided in this project, because this is an approach that is more geared to workers' compensation questions than to the understanding of the disease. Low concentrations of irritants may aggravate underlying asthma but do not usually cause asthma. Thus, chemicals common to the agricultural environment may contribute to the exacerbation of airflow obstruction in individuals with pre-existing asthma[4]. The prevalence of asthma specifically caused by exposure to agricultural dusts and fumes is not known. It is likely to depend on exposure and setting. Occupational asthma is estimated to account for between 5% and 15% of patients diagnosed with asthma[67]. Studies comparing asthma between the general population and agricultural population have shown a similar or even lower prevalence of asthma among the agricultural population[68], and additionally, they reflect a lower risk for the development of allergies among children living on farms. Living on farms may consequently exert a protective effect for the development of allergies[69-71]. Nevertheless, European poultry (odds ratio [OR] 1.9; 95% confidence interval [CI] 1.3 – 3.0) and flower farm workers ([OR] 2.2 95% [CI] 1.2-4.2) have been reported to be at higher risk for the development of asthma compared with other farm workers[72,73], and reversible airway constriction may be seen in up to 25% of confinement livestock or grain workers[74]. Data are sparse and inconclusive regarding the prevalence of asthma in agricultural workers; moreover, *no* data are available concerning the incidence of asthma among agricultural workers and their family members frequently exposed to *bioaerosols*[4].

The diagnosis of agricultural asthma is dependent on the demonstration of reversible airflow obstruction occurring in conjunction with inhalation of specific agents known to cause or exacerbate asthma. However, unlike other patients with occupational asthma, the agricultural worker commonly lives in the working environment and often works more than five days a week. Thus, the temporal relationship between exposures and symptoms may be difficult to establish[4].

8.2 CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

Chronic obstructive pulmonary disease (COPD) is a syndrome characterized and defined by a single physiological parameter: limitation of expiratory airflow[75]. In 1995 the American Thoracic Society (ATS) published a paper titled "Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease"[76]. During the same year, a European Respiratory Society (ERS) task force published its view on the assessment and management of patients with COPD[77]. In 2001, the Global Initiative for Chronic Obstructive Lung Disease (GOLD) was published. This was a collaborate effort between the US National Heart, Lung and Blood Institute (NHLBI) and the World Health Organization (WHO)[78]. An internationally accepted opinion, including the 1995 ATS statement, has defined COPD as a disease state characterised by chronic airflow limitation due to chronic bronchitis and emphysema. Chronic bronchitis has been defined in clinical terms: the presence of chronic productive cough for at least 3 consecutive months in 2 consecutive years. Emphysema has been defined by its pathologic description: an abnormal enlargement of the air spaces distal to the terminal bronchioles accompanied by destruction of their walls and without obvious fibrosis[79]. The definition of COPD has undergone major revision. COPD, like asthma, is now recognised as an inflammatory disease of the airways[80]. This statement is supported by extensive clinical and basic science research over the past 2 decades showing that asthma and COPD have different and distinct cellular and inflammatory mediator profiles[79]. The new ATS/ERS definition reflects these scientific advances: "COPD is a preventable and treatable disease characterised by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and is associated with an abnormal inflammatory response of the lungs to noxious particles or gasses, primarily caused by cigarette smoking. Although COPD affects the lungs, it also produces significant systemic consequences like muscular weakness, increased risk for atherosclerotic vascular disease, depression, osteoporosis, and abnormalities in fluids and electrolyte balance[79].

Several anatomical lesions contribute to airflow limitation. Inhibition of lung damage repair may lead to tissue destruction that characterizes emphysema, whereas abnormal repair can lead to the peribronchiolar fibrosis that causes airflow limitation in small airways[79]. Multiple pathogenetic mechanisms are likely to contribute to the development of COPD. The most important risk factor is cigarette smoking, which can affect the lungs by a variety of mechanisms[81]. Other exposures also contribute,

probably through similar pathways[75]. Agricultural workers are exposed to irritant gases and organic dust, which include grain dust, aeroallergens, endotoxins, insect antigens, beta –1,3-glucans, fungi and mycotoxins. Endotoxin and beta-1,3-glucans especially are thought to mediate macrophage activity and therefore, may induce neutrophilic inflammation of the respiratory tract[82]. Factors in addition to exposures, including both genetic[83] and acquired conditions[84], also play a role and likely account for much of the variable susceptibility of individuals to the effects of cigarette smoke and other exposures.

Von Essen[74] reported a high prevalence of chronic expectoration of phlegm (chronic bronchitis) among farm workers compared with controls in 1997. It has ranged between 3% and 30% in non-smoking farm workers. In the European Community Respiratory Health Survey[85], the prevalence of chronic bronchitis in winter in the general population aged 20-44 years, was shown to be 7.5% compared with 9.4% of animal farm workers within this age group in the study of European farmers ($p < 0.001$)[73]. The prevalence of COPD in animal and grain farm workers is related to exposure to large amounts of organic dust[86]. In pig farm workers, it has been shown that the prevalence of COPD is significantly related to time spent daily inside swine confinement buildings[73].

The relationship between COPD and systemic disorders

COPD is not only a disease of the lungs but is also a systemic inflammatory disorder. Muscular weakness, increased risk for atherosclerotic vascular disease, depression, osteoporosis, and abnormalities in fluids and electrolyte balance may all be consequences of COPD[79,87]. The most likely cause of death for patients with COPD, in fact, is cardiovascular disease[88]. This is true for both patients with mild disease and for those with more severe COPD. Interestingly, COPD, increases cardiovascular risk at all levels of disease[89]. The mechanisms that account for this increased risk are not well defined. However, they do not appear to be simply a measure of smoking, because the relationship is present even when epidemiological studies are adjusted for smoking behavior[87].

8.3 HYPERSENSITIVITY PNEUMONITIS

Hypersensitivity pneumonitis (HP), also called extrinsic allergic alveolitis, is an immune-system-mediated interstitial lung disease caused by the inhalation of several foreign environmental antigens, including avian proteins as well as endotoxin and mold spores[63,90]. HP does not fit clearly into any one of the four classic types of allergic reactions. The time lapse between exposure and symptoms, as well as possible involvement of antigen-antibody complexes, would suggest a type III reaction, while the late-phase cellular involvement with granuloma formation is more compatible with a type IV response[4].

HP in farmers or farm workers is also known as farmer's lung[63]. Causative agents in the farming environment are *Micropolyspora faeni, Thermophilic actinomycetes* and *Aspergillus* species growing in moldy hay, grain and other vegetable matter, as well as proteins derived from bird droppings or feather bloom. After sensitisation, low levels of exposure may trigger an attack of hypersensitivity pneumonitis. There are also some indications that cofactors are needed to induce the disorder in farm workers, for example bacterial endotoxin or fungal beta-1,3-glucans[4].

Patients with HP may present with an extensive variety of clinical and functional abnormalities. It remains unclear if the disease truly has different forms of presentation or is simply seen at different stages. The classically reported forms of HP are acute, sub acute and chronic. The *acute* form of HP is the most frequent presenting form and the easiest to characterise. The subject has typical acute symptoms including fever and chills, as well as pulmonary symptoms (chest tightness, dyspnea, cough). Symptoms appear within hours of exposure, generally becoming evident in the late afternoon. By morning, the fever has usually subsided, while dyspnea, although less severe than the evening before, may persist. *Sub acute* is used to describe a form of HP that is more insidious. In this form, dyspnea is manifested gradually over several weeks or months. No studies have explored variations in body temperature. The *chronic* form of HP is probably the long-term sequel of one or both of the other two forms. Chronic cough and sputum are common in this phase, and mild bronchospasm have also been reported[4]. Diagnostic criteria for HP have not been firmly established. One difficulty is that, although many symptoms, signs and clinical parameters are very sensitive, they are nonspecific[91]. Of the numerous criteria recommendations that have been published, the ones most frequently used are those of Richerson and associates[92] and Terho[93];

these criteria were limited to acute disease and need to be updated to include new information and account for the forms in the posed classification above[4]. It is evident though, that HP patients present with a restrictive pattern on pulmonary function studies[94].

The prevalence of HP in agricultural populations remains uncertain because of diagnostic limitations in epidemiological studies[63]. HP may occur in about 1% of farm workers[95]. Prevalence appears even lower if the diagnosis is verified by chest radiography. The incidence of clinically confirmed cases has been estimated at 0.2 - 0.5/1000 per year among those at risk[96]. The low attack rate of this disease despite widespread exposure to antigens in bioaerosols, argues for genetic predisposition in affected individuals[94]. The incidence and prevalence of HP also depend considerably on climatic conditions and farming practices, which in turn determine the likely degree of microbial contamination[4].

8.4 ORGANIC DUST TOXIC SYNDROME (ODTS)

ODTS is an acute inflammatory condition affecting airways and alveoli[4]. Pulmonary function tests are usually normal but may show mild restriction[63]. It is caused by the inhalation of large amounts of airborne organic dust[8] and it occurs even in the absence of exposure to antigens that cause hypersensitivity pneumonitis. This implies toxicity rather than hypersensitivity mechanisms[97]. ODTS was first reported in the mid-1970's and has been accepted as a distinct clinical entity since the mid-1980's[98,99]. ODTS is often seen following inhalation of mould dust containing large numbers of microorganisms[4], it typically follows exposures to mouldy grain, silage, hay and wood chips and is reported most commonly by farm workers and swine confinement workers[100].

The exact mechanisms of toxicity are not known, but endotoxin, fungal spores and mycotoxins are believed to play a role and the mechanism is non-immunogenic[101]. The amount and duration of exposure is important[100]. A prominent characteristic of ODTS is its appearance following the inhalation of dust in quantities that are clearly in excess of normal exposure[4]. In a study by Mamberg et al[102] in 1993, the average dose of mold spores associated with development of ODTS was 2×10^{10} spores. In another study, exposure to greater than 90-100mg/m^3 of respirable grain dust for 1 to 2 hours, provoked ODTS in two of six subjects[103]. Wintermeyer et al[104] exposed six volunteers to wood chip mulch dust for 60 to 120 minutes, the total dust

concentration ranged from 0.3 – 3.6 mg/m^3, three of the six volunteers developed symptoms of ODTS. In a negative study, 23 workers in the hanging department of poultry slaughterhouse plants were examined immediately before work on Monday morning and immediately after work on the same day. The mean level of total dust was 6.3 mg/m^3. None of the workers developed any symptoms of ODTS[105]. The level of endotoxin, rather than total dust, correlates with cross-shift changes in the forced expiratory volumes[106,107] and with signs of ODTS[108].

It has been estimated that 30% to 40% of workers exposed to organic dusts will develop ODTS[103,109]. The incidence of ODTS in European farmers has been estimated at between 20 and 190 per 10 000 farmers per year[4,97]. NIOSH issued a report in 1994 to increase the recognition of ODTS[100], it was noted that ODTS was a poorly recognised problem possibly affecting up to 40% of heavily exposed U.S. workers[111].

Schenker[4] suggested that, although the agents causing ODTS have not been fully identified, it would be useful to identify markers of exposure, which might predict symptoms better than dust levels do. A few such markers have been proposed, including endotoxin and total spore counts[4]. Another unresolved issue is whether the more relevant measure of exposure is the quantity of agent in the inhaled fraction or the quantity in the respirable fraction of dust[4]. The respirable fraction of swine farm dust is about 4% of the inhaled quantity[107] but could possibly induce a disproportionate share of the total response[4].

8.5 TUBERCULOSIS (TB) – A RESPIRATORY INFECTION

Tuberculosis (TB) is a potentially fatal contagious disease where the lungs are primarily involved, but the infection can spread to other parts of the body[112]. TB spreads by droplet infection. Aerosols containing TB bacteria may be exhaled or coughed out by infected humans or animals and then inhaled by susceptible humans[113]. Compared to some other infectious diseases, TB is not highly contagious. Only about one in three close contacts of a TB patient, and fewer than 15% of more remote contacts, are likely to become infected[112]. A number of factors that greatly increase the risk of progression of infection to disease, have been identified – age (infants and the elderly), silica exposure and silicosis and individuals infected with human immunodeficiency virus (HIV)[114,115]. Alcohol consumption and smoking are also important risk factors for TB[114].

In 1882, a time when TB caused every seventh death in Europe, the microbiologist Robert Koch discovered the tubercle bacilli[112]. The tubercle bacilli cause a peculiar tissue reaction in the lungs. This infection includes (1) invasion of the infected tissue by macrophages and (2) "walling off" of the lesion by fibrous tissue to form the so-called *tubercle* [64]. Tuberculosis in its later stages is characterised by many areas of fibrosis throughout the lungs, as well as reduced total amount of functional lung tissue. These effects cause:

- Increased "work" for the respiratory muscles to cause pulmonary ventilation and reduced vital capacity and breathing capacity.
- Reduced total respiratory membrane surface area and increased thickness of the respiratory membrane, causing progressively diminished pulmonary diffusing capacity; and
- Abnormal ventilation-perfusion ratio in the lungs, further reducing overall pulmonary diffusion of oxygen and carbon dioxide[64].

The presence of TB should be expected in any individual who presents with one or more of the following symptoms:

- Persistent cough of 3 weeks or more
- Sputum production, particularly if blood-stained
- Chest pain, shortness of breath
- Loss of appetite and loss of weight
- Night sweats and fever
- Malaise (general fatigue and weakness)[114].

Individuals with pulmonary TB do not run a high fever, but they often have a low-grade one. The TB Bacilli may travel from the lungs to lymph nodes in the sides and back[112].

Based on a Centers for Disease Control (CDC) survey addressing occupational and residential characteristics of tuberculosis cases from 1985 – 1989, it was estimated that farm workers accounted for 5% of all employed cases, leading to an estimate that the risk of tuberculosis in farm workers was sixfold that of the general population of employed adults[116]. A proportionate mortality study of 2206 pulmonary tuberculosis deaths occurring between 1979 and 1990 from the National

Occupational Mortality Surveillance database indicated that farm workers were at approximately twofold increased risk[117]. Greater risk for TB was seen among black than white male farm workers[4].

Three specific types of bacteria that are part of the Mycobacterium group cause TB: *Mycobacterium (M.) bovis, M. avium,* and *M. tuberculosis.* Bovine TB, caused by *M. bovis*, can be transmitted from livestock to humans. No other TB organism has as great a host range as bovine TB, which can infect all warm-blooded vertebrates. *M. avium* can affect all species of birds, as well as hogs and cattle. *M. tuberculosis* primarily affects humans and causes human TB, which is not a zoonotic disease[4,113,118]. Prior to the widespread introduction of milk pasteurisation, between 6 and 30% of human TB cases in developed countries were caused by *M. bovis* [119]. *M. avium* is the most common cause of TB in birds and is often found in soil and water. It can survive for a long period of time in the environment and can multiply in inanimate objects. Avian tuberculosis is transmitted by ingestion and inhalation of aerosolised infectious organisms from feces[120]. Although humans are more commonly infected with *M. tuberculosis* and *M. bovis*, human *M. avium* infections have considerably gained importance in the last two decades, mainly in HIV-infected individuals[120,121]. It is believed that immune-competent individuals are resistant to the strains of TB found in birds, but that immune-compromised individuals – such as those infected with HIV, those on chemotherapy, the elderly and children are at increased risk[120]. Martin and Schimmel[121] reports that poultry-to-man transmission of *M. avium* appears to be a very improbable event, and states that *M. avium* infection of farm poultry is hardly of any importance for poultry production as well as for human disease. In contrast, avian TB is a reportable zoonotic disease in Florida for both health and livestock officials, there the Department of Agriculture and Consumer Services must be notified of any birds infected with *M. avium* and the tuberculosis poultry flock must be depopulated[122]. In the 1890's, TB was recognised as an important health hazard in South Africa and in 1916 TB was classified as a compensable disease[114].

Before the development of effective antibiotics, many patients became chronically ill with increasingly severe lung symptoms. They lost a great deal of weight and developed a wasted appearance. This outcome is uncommon today – at least where modern treatment methods are available[112]. While most *Mycobacterium* infections are treatable with antibiotics, *M. avium* infection is the exception. *M. avium* is highly resistant to antibiotics. Surgical excision and lymph node removal are often

necessary to eliminate infection[122]. Although TB can be treated, cured and prevented if the people at risk take certain drugs, scientists have never come close to wiping it out. Few diseases have caused so much distressing illness for centuries and claimed so many lives[112].

9 STUDY DESIGN

According to Nelson et al[123], there are mainly two types of studies in industrial hygiene: experimental and observational. *Experimental studies* are studies with planned intervention on determinant factors suspected of altering the phenomenon under study, and *Observational studies* are studies with no deliberate human intervention. It is a study where conditions must be notified as found in a field study[123].

This was an observational, cross-sectional pilot study. A target population of 50 contract workers concerned with the removal and disposal of poultry manure were identified in the Potchefstroom district. (Note that, for this project, referral to "poultry farm worker" is a referral to poultry farm workers mainly concerned with the removal and disposal of the poultry manure, they have minimal to no involvement in the other phases of poultry farming.) Due to time and other limitations, all of these subjects could not be monitored. The Occupational Exposure Sampling Strategy Manual (OESSM) was used to determine the number of employees to sample in order to select the maximum risk employee(s). According to OESSM, for a target population of 50, a sample size of 18 will ensure with 90% confidence that at least one individual from the highest 10% exposure group is contained in the sample[124]. A random sample of 19 was drawn from the group of 50 for this project. Each person in the study population was examined only once. At the time of the examination both exposure monitoring and lung function tests were done, and the presence of potential confounding variables were determined. Personal monitoring was done by single exposure measurements taken for the full duration of the time-averaging period, i.e. 8 hours for an 8-hour workday. Area monitoring was done in poultry houses in three specific demarcated areas around Potchefstroom. Because of time and financial limitations, biological monitoring was done in two of the three areas. Ammonia monitoring was done in all three areas.

10 GENERAL AIM

The general aim of this study was to monitor the air quality of the work environment on poultry farms in the Potchefstroom district in the North-West province, South Africa.

11 OBJECTIVES

- To use personal sampling and gravimetrical methods to monitor the occupational exposure of the subjects to total- and respirable dust concentrations.

- To compare the occupational exposures of the subjects to the legal exposure limits of OSHA, NIOSH, Regulations for Hazardous chemical substances of 1995 and to the exposure limits recommended by Donham[47] for human health.

- To assess the lung functions of the subjects by means of Spirometry before and after the 8-hour working shift.

- To compare the baseline FEV_1, FVC and FEV_1/FVC% of each subject with his post shift FEV_1, FVC and FEV_1/FVC%, to determine if there is a cross-shift change in lung function.

- To use nomograms and predicted values to determine if the subjects have obstructive, restrictive or normal lung functions.

- To determine if there is a correlation between the concentration of organic dust to which the subjects were exposed and their lung functions.

12 HYPOTHESIS

The hypothesis for this project is that:

- The occupational exposures of the poultry farm workers will comply with the legal exposure limits.

- Poultry farm workers exposed to ammonia- and particulate matter concentrations above Donham's[47] recommended limits for human health will show acute symptoms of impaired respiratory function.

- The legal exposure limits for ammonia and particulate matter in South Africa do not offer adequate worker protection in mixed exposure situations like on poultry farms.

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CHAPTER 2

MANUSCRIPT: Exposure of poultry farm workers to ammonia, particulate matter and microorganisms in the Potchefstroom district, South Africa

This article is to be submitted to Occupational Health Southern Africa. Occupational Health Southern Africa is the official journal of the South African Society of Occupational Medicine (SASOM), the South African Society of Occuptional Health Nursing Practitioners (SASOHN), the Southern African Institute of Occupational Hygiene (SAIOH), and the Mining Medical and Other Health Care Professionals Association (MMOA).

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    Book references

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EXPOSURE OF POULTRY FARM WORKERS TO AMMONIA, PARTICULATE MATTER AND MICROORGANISMS IN THE POTCHEFSTROOM DISTRICT, SOUTH AFRICA

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ABSTRACT

Relatively few epidemiological data are available addressing pulmonary infections in the context of the agricultural work environment, especially for the South African population. The relevance of the current occupational exposure levels for agricultural workers is also questionable. The study aimed to: (1) determine if there is a correlation between the amount of occupational exposure to poultry farm dust and the lung function of the workers and to (2) determine if the legal exposure limits for particulate matter in South Africa offer adequate worker protection for poultry farm workers exposed to biologically active dust. Personal exposure to total and respirable dust were determined. Ammonia and biological monitoring was done, pre- and post shift lung function tests were performed and health and employment questionnaires were completed. The measured dust concentrations did not exceed the current legal limits and there were no statistically significant cross shift changes in the spirometric variables. The conclusion is that the occupational exposure on poultry farms in the Potchefsroom district does not have any adverse effects on the workers' lung function and the workers are adequately protected by the current legal limits in the short term.

INTRODUCTION

Agricultural production in South Africa has almost doubled in the past 30 years and it is clear that agriculture continues to play a fundamental role in the economy and daily existence of the populations in developing countries[1].

As early as 1555 and 1700, Magnus and Ramazzini warned about the dangers of inhaling grain dust[2,3]. Despite this early recognition the investigation of agricultural respiratory hazards has lagged behind the investigation of hazards in mining and other heavy industries[4]. Relatively few epidemiological data are available addressing pulmonary infections in the context of the agricultural work environment[5], especially for the South African population. Researchers state that it is organic dust that accounts for the most common exposure leading to agricultural respiratory disease[6]. Organic dust exposures may vary qualitatively as well as quantitatively from one occupation to another. Poultry house dust contains feed and fecal particles, feather barbules, skin debris, fungal fragments and spores, bacteria and bacterial fragments, viruses and particles of litter. Such dust was generally considered nuisance or inert, meaning it has little adverse effect on the lungs. New research shows that because poultry house dusts are largely organic and contain bacteria and other bioactive substances, it cannot be considered inert[7]. Pathogens from poultry manure can be transmitted to humans via air or fecel-oral transmission[8]. Some of the pathogens with an influence on human respiratory health are *Pseudomonas aeruginosa, Actinomycetes, Mycobacterium, Salmonella spp, Streptococcus* and *Chlamydia psittaci*[8-12]. It is believed that bacterial endotoxin is one of the major causative agents for organic dust induced diseases[13]. The term "endotoxin" refers to the toxin as present on the (gram-negative) bacterial cell wall, which is often liberated as a result of cell lyses[14,15]. A dose-response to endotoxin and pulmonary function deterioration has been established in numerous studies[16-20]. Endotoxins are associated with the release of pro-inflammatory agents and there are associated declines in pulmonary functions, primarily FEV_1 (forced expiratory volume in one second) and symptoms including chest tightness, cough, dyspnea, and sputum production[21].

Due to financial and other limitations, this study only investigated the prevalence of specific gram-negative bacteria in the poultry environment with an effect on human lung function, and did not specifically quantify the amount of endotoxins released. The study population was also exposed to the aerobic decomposition of (dry) poultry manure, and although the contribution of ammonia exposure to respiratory symptoms

is unclear[22], evidence suggests that increasing levels of ammonia in poultry manure during decomposition will destroy *Salmonella* species and other pathogens that can potentially harm humans[8].

Research documents that current recommended or legal occupational exposure levels are not sufficient to protect agricultural workers[23]. The Occupational Safety and Health Administration (OSHA) has standards for non-specific dust and cotton dust[24]. NIOSH has recommended exposure limits for cotton dust, grain dust and wood dust[25], but these limits may not adequately protect workers exposed to organic dust contaminated with microorganisms[24]. Donham[26] recommended maximum exposure values for human health for the most common contaminants found in the swine confinement environment. These values are much lower than the legal recommended levels and it includes values for endotoxin and microorganisms. In South Africa we are using the exposure limits of the Regulations for Hazardous Chemical Substances of 1995[27]. Most of these exposure limits however, are adopted from international limits and guidelines. Because of the influence of geography, climate and degree of industrialisation on the agricultural air quality, the relevance of the foreign exposure limits is questionable.

Despite low rates of cigarette smoking, farmers have an increased prevalence of several acute and chronic respiratory diseases[4]. Studies indicate that the risk associated with developing respiratory disease appears to be more than threefold greater among those who are heavily exposed to inhalable agricultural dust[6]. The principal respiratory disorders associated with agricultural exposures are asthma and other forms of airflow obstruction like chronic obstructive pulmonary disease (COPD); hypersensitivity pneumonitis (extrinsic allergic alveolitis); pulmonary fibrosis and infection (like tuberculosis). Organic dust toxic syndrome (ODTS), while not a localised disease, is a result of inhalation and therefore it is considered in this study[28].

The aims of this study were to: (1) determine if there is a correlation between the amount of occupational exposure to poultry farm dust and the lung function of the poultry farm workers and to (2) determine if the legal exposure limits for particulate matter in South Africa offer adequate worker protection for poultry farm workers exposed to biologically active dust.

METHODOLOGY

This was an observational, cross-sectional pilot study. A target population of fifty contract workers concerned with the removal and disposal of poultry manure were identified in the Potchefstroom district. According to the Occupational Exposure Sampling Strategy Manual (OESSM), for a target population of fifty, a sample size of eighteen will ensure with 90% confidence that at least one individual from the highest 10% exposure group is contained in the sample[29]. A random sample of nineteen was drawn from the group of fifty for this project. The Ethics Committee of the North-West University approved the study and informed consent was obtained from each subject after having the aims and objectives of the study explained, prior to testing. At the time of the examination both exposure monitoring and lung function tests were done, and the presence of potential confounding variables were determined.

Exposure to total and respirable dust were determined by means of personal sampling in the breathing zone. Monitoring was done by single exposure measurements taken for the full duration of the time-averaging period, i.e. 8 hours TWA for an 8-hour workday. In order to determine total dust concentration, 3-piece cassette holders were used and calibrated, battery-operated pumps (Gilian and SKC) provided a constant airflow of 2 L/min. For sampling of respirable dust, cyclone separator samplers were used and the calibrated pumps provided a constant airflow of 1.9 L/min. Fibreglass filters with a 37 mm diameter were used and the masses of all dust samples were determined gravimetrically.

Area monitoring for ammonia and bioaerosols were done in poultry houses in three specific demarcated areas around Potchefstroom. Because of time and financial limitations, biological monitoring was done in two of the three areas. The SKC Biosampler® was used for biological monitoring, air samples were drawn into swirling sterilised water and airflow was regulated at 12.5 L/min sonic flow. The Biosampler® was placed as close as possible to the workers at 0.5 m above floor level for a duration of 15 min. All samples were delivered to the microbiology laboratory of the North-West University within 24 hours. Qualitative analysis was performed to characterise airborne bacteria and fungi. The prevalence of the following gram-negative bacteria was investigated: *Pseudomonas aeruginosa, Serratia marcescens, Salmonella spp and Escherichia coli, Pasteurella spp, Actinomycetes, Saccharospora rectivirgula, Mycobacterium, Chlamydia psittaci, Staphylococcus* and *Strptococcus.* Ammonia Draeger tubes were used for ammonia monitoring in all

three areas. Personal monitoring for ammonia exposure was not done because ammonia is more of a concern for its corrosive and irritative properties than for its contribution to pulmonary illness, it is however an important measurement in this study due to the possible effect of ammonia on the microorganisms[22]. Weather conditions were also taken into account. The monitoring was done during summer. The atmospheric pressure was taken and a whirling hygrometer was used to determine the wet bulb and dry bulb temperature in order to calculate the relative humidity.

A portable device, the Spirobank_G, was used to conduct spirometric measurements before and after each work shift. All subjects were standing while performing the test and each subject was given a demonstration with clear instructions on how to perform the test prior to his first effort. The procedure was repeated to a maximum of eight times, until three reproducible results were achieved. The forced vital capacity (FVC), forced expiratory volume in one second (FEV_1) and the FEV_1/FVC ratio were analysed for baseline and post shift measurements. The spirobank_G calculates predicted values (FVC, FEV_1 and FEV_1/FVC) for each individual according to demographic data (i.e. height, weight, gender, ethnicity and age)[30,31].

Interviewer administered questionnaires were used to assess occupational and exposure histories and to detect symptoms of organic dust exposure. The questionnaire used is a slightly expanded and modified version of the American Thoracic Society's standardised questionnaire recommended for use in epidemiological surveys[32]. The interviewers were researchers with excellent knowledge and background of the study and administered the questionnaires in a choice of two languages (Afrikaans and English).

Statistical analysis

Throughout the statistical analysis it was assumed that the underlined distribution of the population is normal. Data were analysed by using the statistical software packages Microsoft Excel, Statistica© and SAS. A t-test for dependant data was performed in order to determine cross shift changes in spirometric measurements. ANCOVA's (covariance analysis) were performed to determine the correlation between dust concentrations and spirometric measurements while adjusting for smoking status. Frequency analysis was performed on the questionnaire data. P-values ≤ 0.05 were regarded as significant. Data were expressed as means, standard deviations and confidence intervals.

RESULTS

The characteristics of the subjects are shown in Table 1. They have a mean age of 37.00 ± 7.80 years, a mean body mass index (BMI) of 21.38 ± 2.42 kg/m^2 and a mean baseline FEV^1/FVC of 100.55 ± 15.69 %.

As shown in Table 2 and 3, the mean total dust concentration measured (3.96 ± 4.55 mg/m^3) falls below the legal limits of OSHA (15.00 mg/m^3), NIOSH (4.00 mg/m^3) and the Regulations for hazardous chemical substances of 1995 (10.00 mg/m^3). Fifty five percent of the measured total dust concentrations exceeded the 2.4 mg/m^3 that Donham[26] recommends for human health in the swine confinement environment. The mean respirable dust concentration (2.42 ± 2.13 mg/m^3) also falls well below the legal limits of OSHA (5.00 mg/m^3) and the Regulations for hazardous chemical substances of 1995 (5.00 mg/m^3), but all the measurements exceeded the maximum value that Donham[26] recommends for human health (0.23 mg/m^3). Figure 1 and 2 shows the measured dust concentrations in relation to the different recommended exposure values.

Table 4 reflects the weather conditions and the ammonia and biological monitoring results in the three demarcated areas around Potchefstroom. The following microorganisms were identified; *Actinomycetes, Staphylococcus, Streptococcus, Saccharospora rectivirgula* and *Mycobacterium*. Ammonia measurements in Area 1 exceeded the legal limits of both NIOSH (25.00 ppm) and the Regulations for hazardous chemical substances of 1995, as well as the recommended value of Donham[26] (see Table 5). Ammonia measurements in Area 2 did not exceed any legal limits but did exceed the recommended value of Donham[26]. Ammonia measurements in Area 3 did not exceed any of the legal or recommended limits. The outdoor temperatures were 23 °C wet bulb (Tw) and 29 °C dry bulb (Td) for area 1, 15 °C (Tw and Td) for area 2 and 16 °C (Tw) and 18 °C (Td) for area 3. The outdoor relative humidity was 65.93%, 100.00% and 88.60% in area 1, 2 and 3 respectively. The relative humidity inside the poultry houses was 65.97%, 77.65% and 82.82% in area 1, 2 and 3 respectively.

There is no statistical difference between the mean baseline FEV_1/FVC and the mean predicted FEV_1/FVC ($p = 0.88$), but when individually compared by using the guidelines of the American Thoracic Society (ATS) and the University of Iowa[33], there

are 3 individuals with mild to moderate obstruction, and 3 individuals with restrictive disorders. The results show no statistically significant cross shift changes in any of the measured variables, as reflected in Table 6. Table 7 clearly indicate that there is no significant correlation of the measured dust concentrations to any of the spirometric measurements.

The frequency analyses of the respiratory symptoms are shown in Table 8 and Figure 3. The symptoms are regarded as work-related if: (1) the age of the subject when the symptom first appeared is greater than the age the worker first started with the current employer, and one or both of (2) symptom is aggravated by environmental agents at work and (3) symptom improves on weekends and long holidays[34]. Among the 19 workers, 6 individuals suffered from symptoms characteristic for COPD (cough with phlegm, shortness of breath), keeping in mind that this was not clinically confirmed. Nine out of the nineteen subjects felt that their current job gives them health problems. Their main complaints were headaches, sore throats and coughing.

DISCUSSION

The present study was performed on poultry farm workers in the Potchefstroom district, South Africa, in order to determine if there is a correlation between the amount of occupational exposure to poultry farm dust and the lung function of the poultry farm workers.

Studies indicate that the risk associated with developing respiratory disease appears to be more than threefold greater among those who are heavily exposed to inhalable agricultural dust[6]. Since there is a combination of dust, ammonia and microorganisms, the occupational exposure is expected to have significant effects on pulmonary function.

If the baseline (measured before the work shift) FEV_1/FVC of each worker is individually compared to his predicted FEV_1/FVC by using the guidelines of ATS and the University of Iowa[33], there are three individuals with mild to moderate obstruction, and three individuals with restrictive disorders. However, the results show no statistical difference between the mean baseline and mean predicted FEV_1/FVC. The results also show no statistically significant *cross shift changes* in any of the measured spirometric variables (FEV_1, FVC and FEV_1/FVC). Thus, the occupational

exposures on the poultry farms at the time of the survey did not have any adverse effects on the workers' lung function. When reflecting on the *concentrations* of the different measured exposures, there is a fairly logical explanation for the unaffected lung volume measurements.

The measured dust concentrations were low. This is most probably due to the high humidity at the time of the survey and the excellent ventilation systems of the poultry houses. The sides of the poultry houses were completely open (only wire netting) and when the workers were busy removing the manure, there were at least four large extraction fans switched on. Although 55% of the measured total dust and all of the measured respirable dust concentrations exceeded the maximum values Donham[26] recommends for human health, the measured concentrations complied with the legal limits of OSHA[23], NIOSH[23] and the Regulations for hazardous chemical substances of 1995[27]. This can be an indication that the legal limits do, in fact, offer adequate protection for poultry farm workers in the Potchefstroom district, South Africa. It is also and indication that Donham's[26] recommended values for human health is not highly relevant or applicable in the Potchefstroom district.

There is also a strong possibility that the measured dust was not as biologically active as were expected. The measured ammonia concentrations were very high and evidence suggests that high levels of ammonia in poultry manure will destroy *Salmonella* species and other pathogens that can potentially harm humans[8]. The high relative humidity percentages at the time of the survey could also have affected the amount of *airborne* microorganisms. It could be that the dust particles (and therefore microorganisms) do not get airborne that easily, the particles are heavier and sag more rapidly. The following microorganisms were however identified with biological air monitoring; *Actinomycetes, Staphylococcus, Streptococcus, Saccharospora rectivirgula* and *Mycobacterium,* all of which have adverse effects on human lung function. The shortcoming regarding this aspect is that qualitative analysis was performed to characterise the airborne bacteria and fungi, but no quantitative tests were performed and therefore we do not know the *concentration* of microorganisms present. It is recommended that quantitative microbiological analyses be performed in future studies to determine how biologically active the measured dust really are.

Symptoms of organic dust exposure were detected with interviewer-administered questionnaires. Six of the nineteen individuals apparently suffered from symptoms

characteristic of COPD (cough with phlegm and shortness of breath), but COPD was not identified with the spirometric tests. Much of these symptoms were however identified as work-related via the questionnaire analyses (see Table 8 and Figure 3). It could mean that some of the individuals were in the beginning stages of developing COPD, but further studies is necessary to confirm this statement. With the baseline spirometric measurements there were three individuals identified with mild to moderate obstruction and three individuals with restrictive disorders, but there were no cross shift changes in their lung volumes and their dust exposure during the work shift had no direct contribution to their conditions. From the occupational and exposure history assessments it was found that only one of these six individuals was not previously exposed to respiratory hazards at work, but he also indicated that he has been a smoker for the last 22 years. The other five individuals had more service years in other occupations with pulmonary hazards than they do in their current job. Therefore it can be assumed that their lung conditions are a result of their previous occupational exposures. Nine out of the nineteen subjects felt that their current job gives them health problems. Their main complaints were headaches, sore throats and coughing.

The conclusion is that the occupational exposure to ammonia, particulate matter and microorganisms on poultry farms in the Potchefsroom district, South Africa, do not have any adverse effects on the workers' lung function and the workers are adequately protected in the short term, by the legal limits that are currently used in South Africa. This study does not safeguard against the possibility of the development of pulmonary disease over the long term. High exposure to these substances are most certainly expected to have adverse effects on human lung function, but due to the influence of geography and climate on the agricultural air quality, the concentrations of the hazardous substances on South African poultry farms may differ from the concentrations measured in other countries in the literature. The measured concentrations of these hazardous substances do not seem to pose any short-term threat to the poultry farm workers in the Potchefstroom district, South Africa.

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Table 1. Descriptive statistics

	Mean	SD	95% CI
Age (years)	37.00	± 7.80	[33.24; 40.76]
BMI (kg/m^2)	21.38	± 2.42	[20.22; 22.55]
Baseline FEV_1/FVC (%)	100.55	± 15.69	[92.99; 108.12]

Table 2. Particulate matter exposure – personal monitoring

	n	Mean	SD	95% CI
Total dust (mg/m^3)	11.00	3.96	± 4.55	[0.90; 7.02]
Respirable dust (mg/m^3)	8.00	2.42	± 2.13	[0.64; 4.32]

Table 3. Dust concentration levels (TWA) recommended for human health[23,26,27].

	OSHA (mg/m^3)	NIOSH (grain dust) (mg/m^3)	Reg. H. Chem. Sub. 1995 (mg/m^3)	Donham (mg/m^3)
Total dust	15.00	4.00	10.00	2.40
Respirable dust	5.00	Not listed	5.00	0.23

Table 4. Weather conditions, ammonia and microorganism exposure – area monitoring

Area	OUTDOOR			INDOOR					
	T_w (°C)	T_d (°C)	RH (%)	T_w (°C)	T_d (°C)	P_{atm} (kPa)	RH (%)	NH_3 (ppm)	Microorganisms
1	23.00	29.00	65.93	22.00	28.00	86.80	65.97	27.00	- *Actinomycetes* - *Staphylococcus* - *Mycobacterium*
2	15.00	15.00	100.00	17.00	21.00	85.90	77.65	23.00	- *Actinomycetes* - *Staphylococcus* - *Streptococcus* - *Saccharospora rectivirgula* - *Mycobacterium*
3	16.00	18.00	88.60	21.00	24.00	87.70	82.82	5.00	Not measured

Table 5. Ammonia levels (TWA) recommended for human health[23,26,27]

	OSHA (ppm)	NIOSH (ppm)	Reg. H. Chem. Sub. 1995 (ppm)	Donham (ppm)
Ammonia	50.00	25.00	25.00	7.00

Table 6. Cross shift changes in spirometric measurements

	Baseline (mean ± SD)	Post shift (mean ± SD)	Mean difference (baseline – post shift)	p
FEV_1 (L)	98.21 ± 25.75	103.35 ± 20.66	-5.14	0.35
FVC (L)	104.96 ± 34.89	108.63 ± 23.86	-3.67	0.72
FEV_1/FVC (%)	100.55 ± 16.69	98.01 ± 11.66	+2.54	0.56

Table 7. Association between dust exposure and lung function variables

	Baseline FEV_1	Baseline FVC	Baseline FEV_1/FVC	Post shift FEV_1	Post shift FVC	Post shift FEV_1/FVC
Total dust	p = 0.87	p = 0.88	p = 0.58	p = 0.91	p = 0.92	p = 0.70
Respirable dust	p = 0.45	p = 0.88	p = 0.40	p = 0.62	p = 0.95	p = 0.59

Table 8. Total and work-related respiratory symptoms

Symptoms	Number of individuals reporting symptoms	Number of individuals with work-related symptoms
Cough	12	11
Phlegm	7	5
Wheeze	7	4
Chest tightness	4	2
Shortness of breath	1	0

Figure 1.

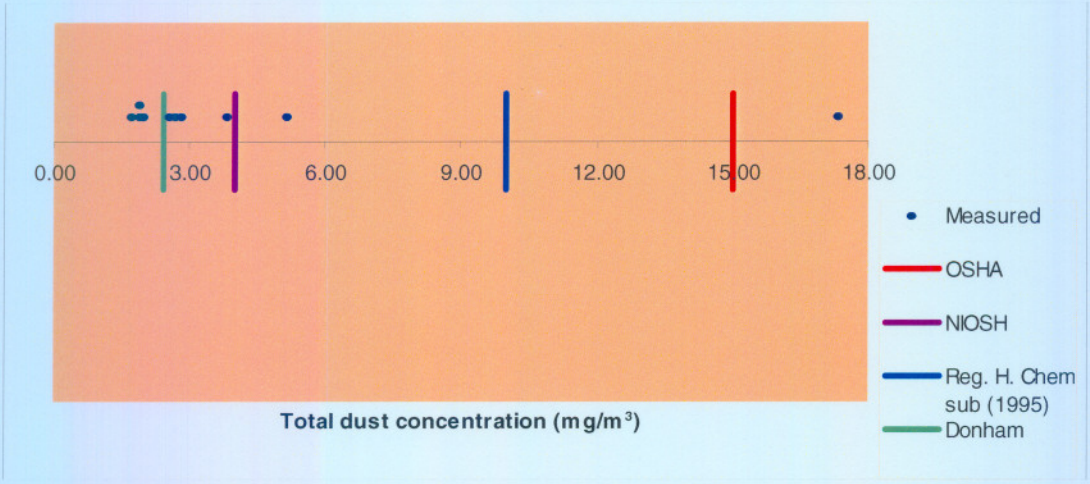


Figure 2.

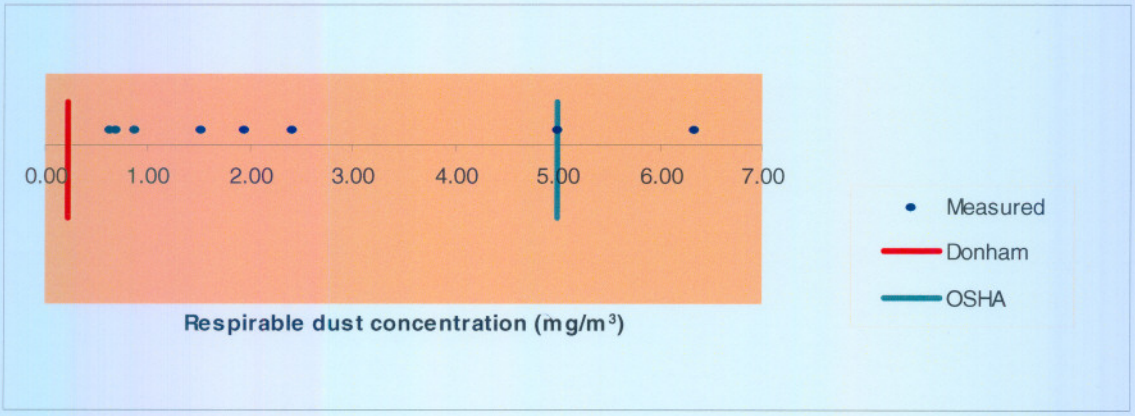
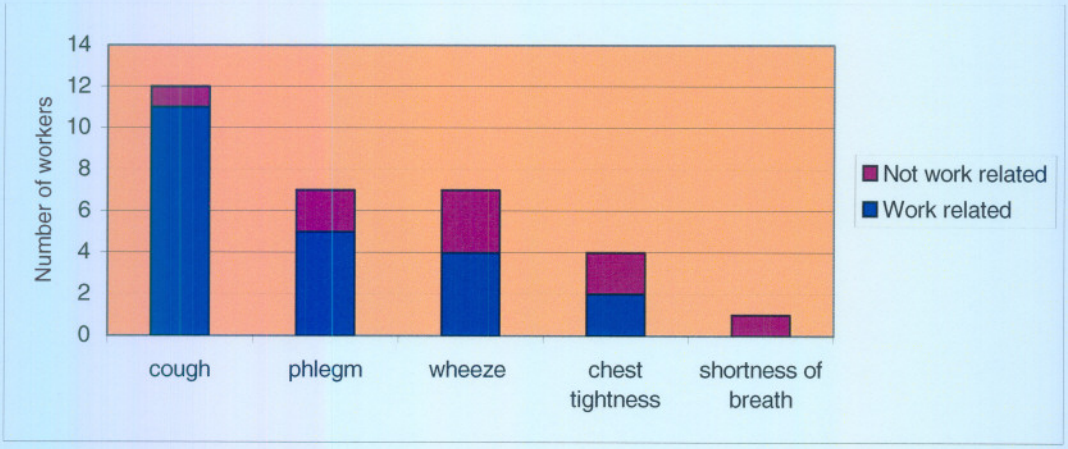


Figure 3.



LEGENDS OF TABLES

Table 1.

Descriptive statistics. n = 19. SD, standard deviation; CI, confidence interval; BMI, body mass index; FEV_1/FVC, forced expiratory volume in one second divided by the forced vital capacity.

Table 2.

Particulate matter exposure – personal monitoring. SD, standard deviation; CI, confidence interval.

Table 3.

Dust concentration levels (TWA) recommended for human health[23,26,27]. OSHA, Occupational Safety and Health Administration; NIOSH, National Institute for Occupational Safety and Health; Reg. H. Chem. Sub. 1995, Regulations for Hazardous Chemical Substances of 1995.

Table 4.

Weather conditions, ammonia and microorganism exposure – area monitoring. T_w, wet bulb temperature; T_d, dry bulb temperature; RH, relative humidity; P_{atm}, atmospheric pressure; NH_3, ammonia.

Table 5.

Ammonia levels (TWA) recommended for human health[23,26,27]. OSHA, Occupational Safety and Health Administration; NIOSH, National Institute for Occupational Safety and Health; Reg. H. Chem. Sub. 1995, Regulations for Hazardous Chemical Substances of 1995.

Table 6.

Cross shift changes in spirometric measurements. SD, standard deviation; FEV_1, forced expiratory volume in one second; FVC, forced vital capacity.

Table 7.

Association between dust exposure and lung function variables. FEV_1, forced expiratory volume in one second; FVC, forced vital capacity.

Table 8.

Total and work-related respiratory symptoms.

LEGENDS OF FIGURES

Figure 1.

Measured total dust compared to recommended and legal limits.

Figure 2.

Measured respirable dust compared to recommended and legal limits.

Figure 3.

Frequent respiratory symptoms

CHAPTER 3

GENERAL FINDINGS AND CONCLUSIONS

INTRODUCTION

In this chapter, a summary of the main findings from the article (Chapter 2) will be given. The results of the article will be interpreted and compared to the relevant literature. Conclusions will be drawn and recommendations will be made to researchers investigating respiratory hazards in the agricultural population of South Africa.

SUMMARY OF THE MAIN FINDINGS

The general aim of this study was to monitor the air quality of the work environment on poultry farms in the Potchefstroom district in the North-West province, South Africa. The specific aims were to: (1) determine if there is a correlation between the amount of occupational exposure to poultry farm dust and the lung function of the poultry farm workers, and to (2) determine if the legal exposure limits for particulate matter in South Africa offer adequate worker protection for poultry farm workers exposed to biologically active dust. It was hypothesised that: (1) the occupational exposures of the poultry farm workers will comply with the current legal exposure limits, (2) poultry farm workers exposed to ammonia- and particulate matter concentrations above Donham's[1] recommended limits for human health will show acute symptoms of impaired respiratory function and (3) the legal exposure limits for ammonia and particulate matter currently used in South Africa do not offer adequate worker protection during mixed exposure situations on poultry farms.

Results indicated that the mean total dust concentration measured (3.96 ± 4.55 mg/m^3) complied with the legal limits of OSHA[2] (15.00 mg/m^3), NIOSH[2] (4.00 mg/m^3) and the Regulations for hazardous chemical substances of 1995[3] (10.00 mg/m^3). The mean respirable dust concentration (2.42 ± 2.13 mg/m^3) also complied with the legal limits of OSHA[2] (5.00 mg/m^3) and the Regulations for hazardous chemical substances of 1995[3] (5.00 mg/m^3). The first hypothesis is thus accepted.

Fifty five percent of the measured total dust concentrations exceeded the 2.4 mg/m^3 that Donham[1] recommends for human health in the swine confinement environment and all of the respirable dust measurements exceeded Donham's[1] recommended value of 0.23 mg/m^3. Despite these dust concentrations, the spirometric values of the subjects were normal; there was no statistical difference between the mean baseline FEV_1/FVC and the mean predicted FEV_1/FVC ($p = 0.88$). Although, when

individually compared by using the guidelines of the American Thoracic Society (ATS) and the University of Iowa[4], there were three individuals with mild to moderate obstruction, and three individuals with restrictive disorders. However, results show no statistically significant cross shift changes in any of the measured variables and there is no significant correlation of the measured dust concentrations to any of the spirometric measurements. The dust exposures in excess of Donham's[1] recommended values did not have any adverse effects on the workers' lung function and therefore the second hypothesis is rejected.

Although 55% of the measured total dust and all of the measured respirable dust concentrations exceeded the maximum values Donham[1] recommends for human health, the measured concentrations did not exceed the legal limits of OSHA[2], NIOSH[2] and the Regulations for hazardous chemical substances of 1995[3]. This can be an indication that the current legal limits do, in fact, offer adequate protection in the short term, for poultry farm workers in the Potchefstroom district, South Africa. The third hypothesis is thus rejected but this does not safeguard against the possibility of the development of pulmonary disease over the long term.

COMPARISON OF FINDINGS WITH THE LITERATURE

When the results from this study are compared with results found in the literature, it is evident that certain findings contradicted those found in the literature, but also added to the available literature.

Contradictory findings in this study were that the measured dust concentrations in excess of Donham's[1] recommended values did not cause impaired lung function. Several published research manuscripts[5-7] document that the legal and recommended exposure limits for the toxic substances found in the agricultural environment are too high for CAFO's. In CAFO's there is a mixture of biologically active agents that can work synergistic to produce respiratory and systemic effects at much lower levels[8]. Cumro and Donham[8] defined such a synergistic interaction between NH_3 and PM. Data were analyzed from an exposure-response study of 149 poultry workers. Analysis of this data-set revealed prominent dose-response relationships between increasing PM, NH_3 and endotoxin concentrations with corresponding cross-shift declines in worker lung function. As these health effects occurred from exposure to both dust and ammonia concentrations less than half the

published ACGIH TLV's, investigations were undertaken to study possible interactions between these substances. The results demonstrated that when workers are exposed to both PM and NH_3, the adverse effect on pulmonary function is up to 156% greater than the individual effects of these gases. Similarly, the fact that there was no statistical difference between the mean baseline FEV_1/FVC and the mean predicted FEV_1/FVC ($p = 0.88$) was also a contradictory finding, since studies indicate that the risk associated with developing respiratory disease appears to be more than threefold greater among those who are heavily exposed to inhalable dust generated in the agricultural environment[9].

A result that added to the available literature was the low dust concentrations (3.96 ± 4.55 mg/m^3 total dust and 2.42 ± 2.13 mg/m^3 respirable dust) in the poultry houses of the Potchefstroom district, South Africa. Relatively few epidemiological data are available addressing pulmonary infections in the context of the agricultural work environment[10], especially for the South African population. High exposure to biologically active dust are most certainly expected to have adverse effects on human lung function, but due to the influence of geography and climate on the agricultural air quality, the concentrations of the hazardous substances on South African poultry farms may differ from the concentrations measured in other countries in the literature. The measured concentrations of these hazardous substances do not seem to pose any short-term threat to the poultry farm workers in the Potchefstroom district, South Africa.

CHANCE AND CONFOUNDING

It is important to reflect critically on some important factors that may have affected the results. There are some methodological issues that could have caused weaknesses in this study and therefore might have influenced the different outcomes.

The number of subjects included in this study could be questioned (n = 19). The reason for this was that the subjects for this pilot study were selected on a basis of availability. A target population of 50 contract workers concerned with the removal and disposal of poultry manure were identified in the Potchefstroom district. According to OESSM[11], for a target population of 50, a sample size of 18 will ensure with 90% confidence that at least one individual from the highest 10% exposure

group is contained in the sample. Despite the fact that the sample size complies with the guidelines of OESSM[11], the possibility of chance should be taken into account.

Confounders

Confounders such as smoking, previous occupational history and HIV-status could have influenced the results by causing over- or underestimation of the associations between lung function and biologically active dust. Smoking, identified as a confounder, was statistically adjusted for. Previous occupational history was taken into account in the interpretation of the results, but it was not statistically addressed. The HIV-status of the subjects was unknown in this study.

In the interpretation of the results of this dissertation, it was attempted to interpret statistical results from an occupational hygiene and physiological standpoint at all times, while keeping in mind that a statistical significance does not necessarily mean physiological significance and *vice versa*.

CONCLUSION

Occupational exposure to ammonia, particulate matter and microorganisms on poultry farms in the Potchefsroom district, South Africa, do not have any adverse effects on the workers' lung function and the workers are adequately protected in the short term, by the legal limits that are currently used in South Africa. This study does not safeguard against the possibility of the development of pulmonary disease over the long term. High exposure to these substances are most certainly expected to have adverse effects on human lung function, but due to the influence of geography and climate on the agricultural air quality, the concentrations of the hazardous substances on South African poultry farms may differ from the concentrations measured in other countries in the literature. The measured concentrations of these hazardous substances do not seem to pose any short-term threat to the poultry farm workers in the Potchefstroom district, South Africa.

RECOMMENDATIONS

The following recommendations are aimed at improving and retaining good respiratory health of poultry farm workers in the Potchefstroom district, South Africa:

- Educational information and/or material should be provided to all workers.
- Good engineering control should be applied, for example good house design. Increasing ventilation (natural and mechanical), and installing sensors to alert workers to elevated ammonia levels will help in achieving this goal.
- To reduce ammonia levels, the poultry houses should be kept as dry as possible by good insulation and ventilation to help reduce condensation.
- Dry floors along with short-term indoor manure storage will ensure low NH_3 production rates[10]. Installation of circulation fans can help to dry manure.
- An oil-misting dust control system can be used. Sprinkling canola oil will reduce dust and endotoxin levels as well as human respiratory effects[12].
- Good work organisation will help to reduce exposure to dust. Job rotation can reduce exposure time for an individual.
- Each worker should have the appropriate personal protective equipment (PPE) needed for the job to be performed.
- Only respiratory protection devices approved by NIOSH or the Mine Safety and Health Administration (MSHA) should be considered.
- Because of the wide range of potential exposures and because there are no applicable exposure limits for organic dust contaminated with microorganisms, NIOSH recommends that exposed workers wear the most practical respirator with the highest assigned protection factor (APF)[13].
- For additional information on respirator selection, consult the NIOSH Respirator Decision Logic[14].

From this study the following recommendations arise that are aimed at improving future research on occupational exposures on poultry farms:

- Quantitative microbiological analysis should be performed.

- When lung function is described as a study response, HIV-status should be included as an exclusion criterion where ethically possible.

- Ammonia is one of the most common gas hazards in agriculture and little is known about the effects of chronic ammonia exposure[15]. From this study, it seems as though most of the workers' complaints are due to ammonia exposure rather than microorganism exposure. Research should address *personal* ammonia exposure on poultry farms and the physiological effects of chronic ammonia exposure.

- It was noted that the poultry houses in the Potchefstroom district has excellent ventilation systems. This is partly due to the hot weather conditions in South Africa. Thus, by regulating temperature, dust concentrations are also controlled. Future research could be conducted on the different ventilation systems used in poultry houses and the effectiveness thereof, especially in different countries with different climates.

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APPENDIX A

INFORMED CONSENT

YUNIBESITI YA BOKONE-BOPHIRIMA
NORTH-WEST UNIVERSITY
NOORDWES-UNIVERSITEIT

INFORMED CONSENT

Exposure of poultry farm workers to ammonia, particulate matter and microorganisms in the Potchefstroom district, South Africa

Purpose

I understand the purpose of this project is to study the air quality of the work environment on poultry farms. The researchers will measure concentrations of dusts and bacterial products in the air during working days. The presence of current and past respiratory symptoms will be recorded among employees via standardized questionnaires. Lung function tests will be performed by using the spirometer.

This farm was randomly selected for this study, and all employees are being invited to participate.

Study procedures

The study involves three parts:

- Measuring exposures
- Performing lung function tests (spirometry)
- Recording symptoms (questionnaires)

Exposure measurement

Personal sampling
I understand that I will be asked to wear a small air-sampling device clipped to my collar, for the full work shift. It will be attached to an air pump worn on the belt. The device is collecting a sample of the air breathed onto a special filter, which will be analyzed later in a laboratory.

Environmental sampling
I understand that there will be a stationary air-sampling device in the working environment for a short period of time, during the work shift. The device is collecting a sample of the air in the workplace, which will later be analyzed in a laboratory.

Lung function tests – spirometry

I understand that I will be asked to perform a simple lung function test. This test involves breathing into a disinfected device, the spirometer. A researcher will fully explain and demonstrate the correct procedure and assist me in the performance of the test. The test will be repeated minimum three and maximum eight times, with at least 30 seconds rest between each attempt.

Questionnaires

I understand that an interviewer will ask a series of standard questions about symptoms I may have now, or have had in the past, and about my work history. Some additional information will also be collected to help interpret the results, e.g. age, smoking information and a brief family history.

YUNIBESITI YA BOKONE-BOPHIRIMA
NORTH-WEST UNIVERSITY
NOORDWES-UNIVERSITEIT

Confidentiality

I understand that participation in the study is completely voluntary and all results are confidential. No one other than the North-West University researchers will have access to personal information. Reports will include group information only; it will not be possible to identify individuals, or individual farms, from any report.

All computer files and documents will be coded with subject numbers, not names. The information will be stored securely and confidentially, and analyzed for the purpose of this study.

Contact

If I have any questions or want more information about the study, I may contact
Mr. MN van Aarde at (018) 299 2433 or Miss AC de Jager at (018) 299 2439.

Consent

I understand that my participation in this study is entirely voluntary and that I may refuse to participate in any or all parts of this study, or withdraw from the study at any time without jeopardy to my employment.

I have received a copy of this consent form for my own records.

I consent to participate in this study.

_____ _____
Name (print) Signature

_____
Date (dd/mm/yyyy)

_____ _____
Witness Signature

_____
Date (dd/mm/yyyy)

APPENDIX B

INVENTORY OF FARM CHARACTERISTICS

INVENTORY OF FARM CHARACTERISTICS

Farm_____ Location_____

Date _____ Visit _____

Class of animal:_____

Animal activity level: Low ☐ medium ☐ high☐

Choice of bedding material:_____

Type of floor: _____

Atmospheric pressure: _____

Temperature (indoor): _____

Temperature (outdoor): _____

Relative humidity (indoor): _____

Relative humidity (outdoor): _____

Ammonia concentration (indoor): _____

Ventilation system: _____

_____

Surface area of the poultry house: _____

Number of employees in poultry house: _____

Number of employees sampled: _____

APPENDIX C

GRAVIMETRIC DUST SAMPLING RECORD

GRAVIMETRIC DUST SAMPLING RECORD

Sample No: _____ Site _____ Date _____

Personal ☐ Static ☐

Person sampled _____

Measurement area _____

Sample type: Inhalable ☐ Respirable ☐

Job Description (type of activities) _____

Heterogeneous Group Size _____

Protection:

	Yes	No	Type
Respirators			
Protective Clothing			
Ventilation			

Sampling pump _____ Serial no _____

Average calibrated airflow rate **before** measurement _____ l/min

Average calibrated airflow rate **after** measurement _____ l/min

Average airflow rate _____ l/min

Deviation:

$$= \frac{\text{Airflow rate } \textbf{before} \text{ measurement} - \text{Airflow rate } \textbf{after} \text{ measurement}}{\text{Airflow rate } \textbf{before} \text{ measurement}} \times 100$$

= _____ %

Sampling times from _____ to _____

Total sampling times in minutes _____ min

Volume of air sampled = $\frac{\text{Average airflow rate (L/min) x Minutes Sampled}}{1000}$

= _____ m^3

Pump inspection Time 1 _____

Time 2 _____

Time 3 _____

Time 4 _____

Time 5 _____

Time 6 _____

Observations

_____

_____

_____

_____

_____

Control filter:

Average filter weight **before** measurement _____ mg

Average filter weight **after** measurement _____ mg

Filter weight before measurement – Filter weight after measurement = _____mg

Correction factor (C1) _____

Field Blank:

Average filter weight **before** measurement _____ mg

Average filter weight **after** measurement _____ mg

Filter weight before measurement – Filter weight after measurement = _____mg

Correction factor (C2) _____

Experimental filter:

Average filter weight **before** measurement _____ mg

Average filter weight **after** measurement _____ mg

Dust mass _____ mg _____ (correction factor) = _____mg

Dust Concentration: $\frac{\text{Dust mass (mg)}}{\text{Volume air } (m^3)}$

= _____ mg/m^3

Sampled by _____

GRAVIMETRIC DUST SAMPLING –CONTROL FILTERS

Date _____

Control filter 1: _____

Average filter weight **before** measurement _____mg

Average filter weight **after** measurement _____mg

Filter weight before measurement – Filter weight after measurement = _____mg

Control filter 2: _____

Average filter weight **before** measurement _____mg

Average filter weight **after** measurement _____mg

Filter weight before measurement – Filter weight after measurement = _____mg

Control filter 3: _____

Average filter weight **before** measurement _____mg

Average filter weight **after** measurement _____mg

Filter weight before measurement – Filter weight after measurement = _____mg

Control filter correction factor (C1) = _____

Field Blank:

Average filter weight **before** measurement _____ mg

Average filter weight **after** measurement _____ mg

Filter weight before measurement – Filter weight after measurement = _____mg

Field blank correction factor (C2) = _____

APPENDIX D

BIOSAMPLER FIELD SHEET

BIOSAMPLER FIELD SHEET

Sample No: _____ Site _____ Date _____

Personal ☐ Static ☐

Measurement point _____

Sampling pump _____ Serial no _____

Average calibrated airflow rate **before** measurement _____ l/min

Average calibrated airflow rate **after** measurement _____ l/min

Average airflow rate _____ l/min

Deviation:

$$= \frac{\text{Airflow rate } \mathbf{before} \text{ measurement} - \text{Airflow rate } \mathbf{after} \text{ measurement}}{\text{Airflow rate } \mathbf{before} \text{ measurement}} \times 100$$

= _____ %

Sampling times from _____ to _____

Total sampling times in minutes _____ min

$$\text{Volume of air sampled} = \frac{\text{Average airflow rate (L/min)} \times \text{Minutes Sampled}}{1000}$$

= _____ m^3

Observations

_____

_____

_____

_____

_____

Sampled by: _____

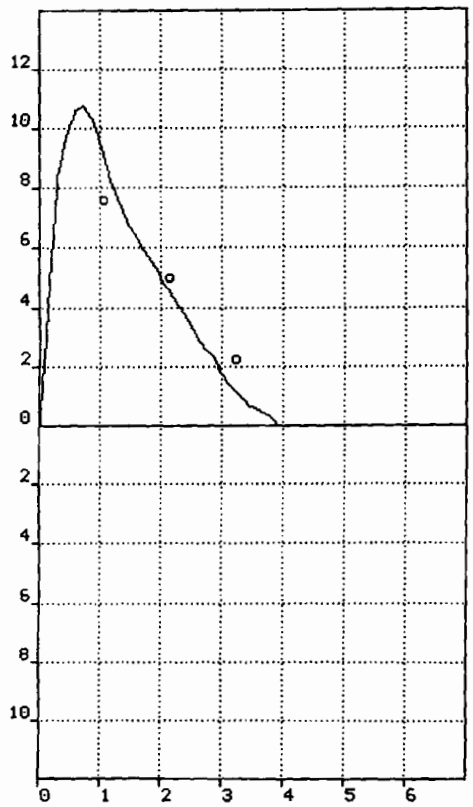
APPENDIX E

EXAMPLE OF SPIROMETRIC RESULTS

Name H12 - Baseline (34)

Date 07/14/05 Time 10:01
Age 24 Sex M
Height cm 161 Weight Kg 55

FLOW-VOLUME curve



Parameter		Predicted	PRE	%
*FVC	L	4.31	3.92	91
*FEV1	L	3.74	3.40	91
*PEF	L/s	9.00	11.06	123
FVC	L	4.31	3.92	91
FEV1	L	3.74	3.36	90
FEV1%	%	82.9	85.7	103
PEF	L/s	9.00	10.79	120
FEF25%	L/s	7.63	8.22	108
FEF50%	L/s	5.01	4.28	85
FEF75%	L/s	2.24	1.45	65
FEF2575	L/s	4.79	3.81	80
FET	s		6.00	
VEXT	mL		120	

*=Best Value
o=Predicted

INTERPRETATION: Normal Spirometry

REPRODUCIBILITY: FVC:Yes FEV1:Yes PEF:Yes

MIR spirobank/G ver.1.7

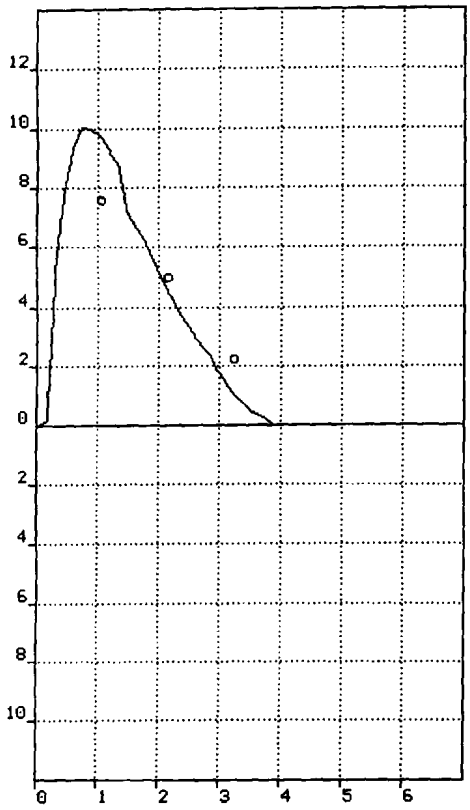
Signature .....

Name
Hla - Post Shift (46)

Date 07/14/05
Age 24
Height cm 161

Time 18:22
Sex M
Weight Kg 55

FLOW-VOLUME curve



Parameter		Predicted	PRE	%
*FVC	L	4.31	3.87	90
*FEV1	L	3.74	3.32	89
*PEF	L/s	9.00	10.30	114
FVC	L	4.31	3.87	90
FEV1	L	3.74	3.32	89
FEV1%	%	82.9	85.8	103
PEF	L/s	9.00	10.25	114
FEF25%	L/s	7.63	9.46	124
FEF50%	L/s	5.01	4.40	88
FEF75%	L/s	2.24	1.55	69
FEF2575	L/s	4.79	3.99	83
FET	s		16.48	
VEXT	mL		140	

*=Best Value
o=Predicted

INTERPRETATION: Normal Spirometry

REPRODUCIBILITY: FVC:Yes FEV1:Yes PEF:Yes

MIR spirobank/G ver.1.7

Signature .....

APPENDIX F

HEALTH AND EMPLOYMENT QUESTIONNAIRE

HEALTH AND EMPLOYMENT QUESTIONNAIRE

Thank you for your willingness to participate. Your cooperation is very important to the success of this study. The information obtained in the study is confidential and will not be released to another party without your permission.

Personal characteristics

STUDY _____

SITE _____

VISIT _____

SUBJECT _____

Date of interview _____ Time: _____

Last name: _____

First name: _____

Address: _____

_____

_____Postal Code [__ __ __-__ __ __]

Telephone: _____

Date of birth: __ __ /__ __ /__ __ __ __
dd mm yyyy

Sex: 1. Male_____ 2. Female_____

Race: 1. White _____ 2. Colored _____

3. Indian _____ 4. Black _____

5. Mixed (specify) _____

Standing height: _____cm

Weight: _____ kg

COLD

A. Do you currently have a cold?
1. Yes _____ 2. No _____

B. Have you had a cold in the last 6 weeks?
1. Yes _____ 2. No _____

COUGH

A. Do you usually have a cough? (Count cough with first smoke or first going out of doors. Exclude clearing throat.)

1. Yes_____ 2. No_____

If NO:

B. Do you usually cough at all on getting up or first thing in the morning?
1. Yes_____ 2. No_____

C. Do you usually cough at all during the rest of the day or night?
1. Yes_____ 2. No_____

If YES to any of above:

D. Do you usually cough like this most days, for 3 consecutive months or more during the year?
1. Yes_____ 2. No_____

E. For how many years have you had this cough? _____

F. Does the cough improve:
- On days off? 1. Yes_____ 2. No_____
- On long holidays 1. Yes_____ 2. No_____

G. Is there any thing or situation which makes your cough worse?
1. Yes_____ 2. No_____

If YES, specify _____

PHLEGM

A. Do you usually bring up phlegm from your chest? (Count phlegm with first smoke or first going out of doors. Count swallowed phlegm. Exclude phlegm from the nose.)

1. Yes_____ 2. No_____

If NO to A:

B. Do you usually bring up phlegm at all on getting up or first thing in the morning?

1. Yes_____ 2. No_____

C. Do you usually bring up phlegm at all during the rest of the day or night?

1. Yes_____ 2. No_____

IF YES TO ANY OF ABOVE:

D. Do you usually bring up phlegm like this most days for 3 consecutive months or more during the year?

1. Yes_____ 2. No_____

E. For how many years have you had trouble with phlegm? _____
(Number of years)

F. Does the phlegm improve:

- On days off? 1. Yes_____ 2. No_____
- On long holidays? 1. Yes_____ 2. No_____

G. Is there any thing or situation which makes the phlegm worse?

1. Yes_____ 0. No_____

Specify if YES: _____

CHEST COLDS AND ILLNESSES

A. If you get a cold does it usually go down to your chest? (I.e. more than half the time)

1. Yes_____ 2. No_____

B. During the past 3 years, have you had any chest illness that has kept you off work, indoors at home, or in bed?

1. Yes_____ 2. No_____

WHEEZING

Does your chest ever sound wheezy or whistling?
1. Yes_____ 2. No_____

A. When you have a cold?
1. Yes_____ 2. No_____

B. Occasionally apart from colds?
1. Yes_____ 2. No_____

C. Most days and nights?
1. Yes_____ 2. No_____

If YES to B or C:

D. Is the wheeze associated with chest tightness or difficulty breathing?

1. Yes_____ 2. No_____

E. For how many years has this been present? _____ (Number of years)

F. When does the wheeze occur MOST frequently?
1. At work _____
2. On return home _____
3. During sleep _____
4. No difference _____

G. Does the wheeze improve:

- on days off? 1. Yes____ 2. No____
- on long holidays? 1. Yes____ 2. No____

H. Is there any thing or situation which makes you wheeze?

1. Yes____ 2. No____

If YES, identify all that apply:

Exercise: 1.Yes___ 2. No___

Cold air: 1.Yes___ 2. No___

Tobacco smoke: 1.Yes___ 2. No___

Strong odor: 1.Yes___ 2. No___

Fumes or dust: 1.Yes___ 2. No___

Other: 1.Yes___ 2. No___

Specify: _____

I. Is the wheezing worse at any particular time of the year?

1. Yes___ 2. No___

If YES, when:

_____
(Season)

CHEST TIGHTNESS

A. Do you ever have episodes or attacks of chest tightness?

1. Yes____ 2. No____

B. If yes: is the chest tightness associated with difficulty in breathing?

1. Yes____ 2. No____

If YES to B:

C. For how many years has this been present? _____

D. Do you have chest tightness and difficulty breathing on most days?
1. Yes____ 2. No____

E. Does it improve:
On days off?
1. Yes____ 2. No____

F. On long holidays?
1. Yes____ 2. No____

G. Is there any thing or situation which makes your chest tightness worse?

1. Yes____ 2. No____

If YES, identify all that apply:

Exercise: 1.Yes___ 2. No___

Cold air: 1.Yes___ 2. No___

Tobacco smoke: 1.Yes___ 2. No___

Strong odor: 1.Yes___ 2. No___

Fumes or dust: 1.Yes___ 2. No___

Other, specify: _____

H. Is your chest tightness worse at any particular time of the year?
1. Yes____ 2. No____

If yes, when:

_____
(Season)

SHORTNESS OF BREATH

A. Are you troubled by shortness of breath when hurrying on the level or walking up a slight hill?

1. Yes____ 2. No____

If YES to A:

B. Do you have to walk slower than people of your own age, on the level, because of breathlessness?

1. Yes____ 2. No____

C. Do you have to stop for breath when walking at your own pace on the level?

1. Yes____ 2. No____

D. Do you ever have to stop for breath after walking about 100 yards (or a few minutes) on the level?

1. Yes____ 2. No____

E. For how many years have you had shortness of breath? _____
(number of years)

Which of the following statements best describes your breathing?

1) I rarely have trouble with my breathing. _____

2) I have regular trouble with my breathing but it always gets completely better. _____

3) My breathing is never quite right. _____

OTHER CONDITIONS

1. Have you ever had asthma?
1. Yes____ 2. No____

If YES to 1:

A. Do you still have it?
1. Yes____ 2. No____

B. Was it confirmed by a doctor?
1. Yes____ 2. No____

C. At what age did it start? _____
(Enter age)

D. If you no longer have it, at what age did it stop? _____
(Enter age)

2. Have you ever had hay fever?
1. Yes____ 2. No____

If YES to 2:

A. Do you still have it?
1. Yes____ 2. No____

B. Was it confirmed by a doctor?
1. Yes____ 2. No____

C. At what age did it start? _____
(Enter age)

D. If you no longer have it, at what age did it stop? _____
(Enter age)

PAST CHEST ILLNESSES

1. Did you have any lung trouble before the age of 16?
   1. Yes____ 2. No____

2. Have you ever had any chest injuries?
   1. Yes____ 2. No____

If yes, specify: _____

3. Have you ever had any chest operations?
   1. Yes____ 2. No____

If yes, specify: _____

4. Are you **currently** taking any medications for your breathing?
   1. Yes____ 2. No____

If yes, specify: _____

(CODE 1 if chest illness, injury, or operation interferes with current lung function)

TOBACCO SMOKING

SMOKING BY PEOPLE LIVING AROUND YOU:

When you lived at home:

1. Did your father smoke?
   1. Yes____ 2. No____

2. Did your mother smoke?

   1. Yes____ 2. No____

In your current household:

3. Do any members of your current household smoke (other than you)?

   1. Yes____ 2. No____

YOUR SMOKING:

1. Have you ever smoked **cigarettes**?
(No means less than 20 packs of cigarettes ever or less than one cigarette a day for one year.)

   1. Yes____ 2. No____

If YES:

A. Do you now smoke cigarettes (as of 1 month ago)?
1. Yes____ 2. No____

B. How old were you when you first started regular cigarette smoking?
_____years

C. If you have stopped smoking cigarettes completely, how old were you when you stopped?
_____years

D. How many cigarettes do you smoke per day now?
_____ cigarettes per day

E. On average, for the entire time that you smoked, how many cigarettes did you smoke per day?
_____ Cigarettes per day

PIPE SMOKING

1. Have you ever smoked a **pipe**?
1. Yes_____ 2. No_____

If YES:

A. Do you now smoke a pipe (as of 1 month ago)?
1. Yes_____ 2. No_____

B. How old were you when you first started smoking a pipe regularly?
_____ years

C. If you have stopped smoking a pipe completely, how old were you when you stopped?
_____ years

D. How frequently do you smoke pipe tobacco?
_____ per day.

ZOL SMOKING

1. Have you ever smoked **zol's** regularly(Yes means more than 1 zol a week for a year.)

1. Yes_____ 2. No_____

If YES:

A. Do you now smoke zol's (as of 1 month ago)?
1. Yes_____ 2. No_____

B. How old were you when you first started smoking zol regularly?
_____ Years

C. If you have stopped smoking zol completely, how old were you when you stopped?
_____ Years

D. How many zol do you smoke per day now?
_____ zol per day

E. What materials do you use to make a zol?
_____

FIRE MAKING METHODS

A. Do you make your fires inside or outside of the living house?
_____

B. What materials do you use to make the fire?
_____
_____

C. For how many hours a day are you exposed to the fire and or smoke thereof?
_____ Hours per day.

D. How many days in a week do you make a fire? _____ days per week.

E. Comments:
_____
_____
_____

FAMILY HISTORY

As some illnesses are associated with childhood and family history we would like to ask you about your family members, and about your childhood.

1. Was your biological father ever told by a doctor that he had any lung problems?
   Yes _____ No _____ Don't know _____

2. Was your biological mother ever told by a doctor that she had any lung problems?
   Yes _____ No _____ Don't know _____

3. Your country (province) of birth:
   _____

4. In what country did you spend the majority of the first 10 years of your life?
   _____

CURRENT JOB

The following questions are about your current job:

1. Industry type: _____
2. Job title: _____
3. What is your present work status?

1. _____ regular full time, working
2. _____ regular full time, but not working due to illness or disability
3. _____ regular full time, but not working due to vacation or other leave
4. _____auxiliary, working
5. _____auxiliary, but not working due to illness or disability
6. _____auxiliary, but not working due to vacation or other leave
7. _____other, specify: _____

4. If not working due to sickness, disability, or leave:
   What date did you last work? [__ __ /__ __/__ __ __ __]
   Dd mm yyyy
5. Total number of years in this job? _____
6. Total number of years with this employer? _____

PREVIOUS JOBS WITH THIS EMPLOYER

1. List previous jobs with this employer; start with the MOST RECENT and WORK BACKWARDS:

Nr.	From (year)	To (year)	Job title	Location	Comments, details
1					
2					
3					
4					
5					

PREVIOUS JOBS WITH OTHER EMPLOYERS:

1. Please list your jobs from the first job you had on leaving school that LASTED LONGER THAN SIX MONTHS. (Before you started working at your current employer).
   Start with your FIRST JOB since leaving school and WORK FORWARD.

	From(year)	To (year)	
1	_____	_____	Job title: _____ Type of company or industry: _____ _____ Comments: _____
2	_____	_____	Job title: _____ Type of company or industry: _____ _____ Comments: _____
3	_____	_____	Job title: _____ Type of company or industry: _____ _____ Comments: _____
4	_____	_____	Job title: _____ Type of company or industry: _____ _____ Comments: _____
5	_____	_____	Job title: _____ Type of company or industry: _____ _____ Comments: _____
6	_____	_____	Job title: _____ Type of company or industry: _____ _____ Comments: _____
7	_____	_____	Job title: _____ Type of company or industry: _____ _____ Comments: _____
8	_____	_____	Job title: _____ Type of company or industry: _____ _____ Comments: _____

2. In any job, have you ever been exposed to a high concentration of gas or fumes that made you sick or that sent you to first aid or to the doctor?
   1. Yes _____ 2. No _____

   Specify: _____

3. Have you ever had any health problems apart from accidents or injuries, that you feel were caused by your job with your **current** employer?
   1. Yes_____ 2. No_____

   Specify:_____

4. Have you ever had any health problems that you feel were caused by a job with **another** employer?
   1. Yes____ 2. No____

   Specify:_____

5. Have you ever left a job for health reasons only?
   1. Yes____ 2. No____

   If YES, what was the job and what was the nature of the problem?

   _____

   _____

There is no oil if olives are not squeezed...

No wine if grapes are not pressed...

No perfume if flowers are not crushed...

All the pressure I endured during the completion of this dissertation was just God bringing out the best in me!!