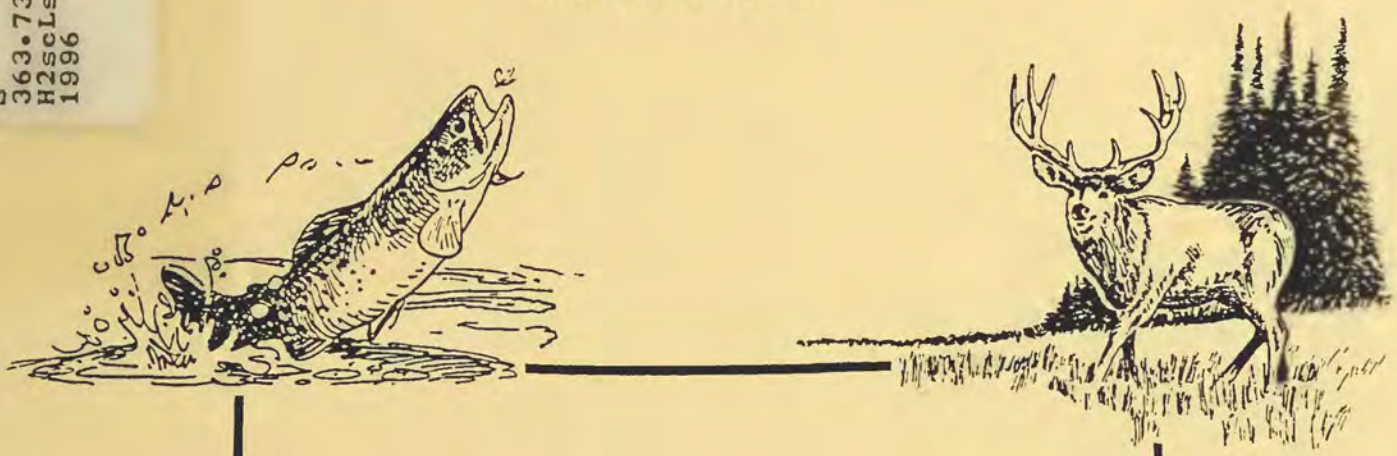
MONTANA STATE LIBRARY
3 0864 0009 9505 3

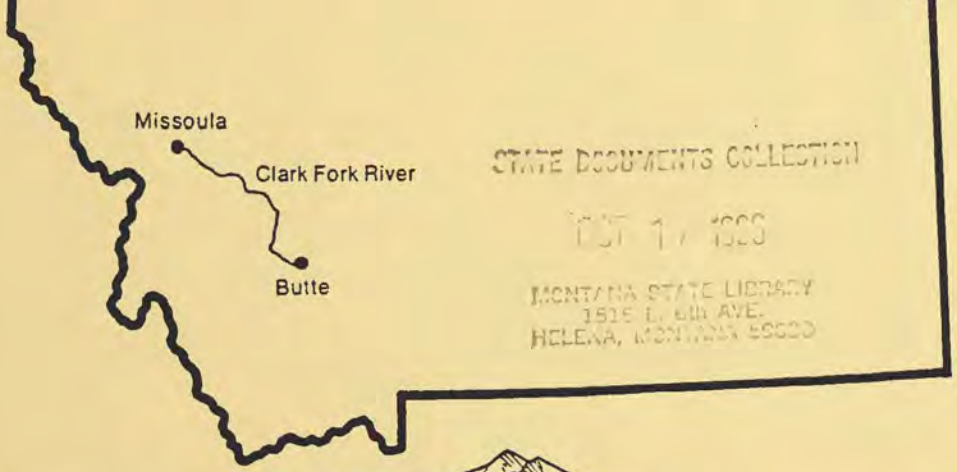
S
363.7394Consulting
H2scLsg Supplemental
1996 injury assessment
report
Hagler Bailly

STATE OF MONTANA
NATURAL RESOURCE DAMAGE PROGRAM

SUPPLEMENTAL INJURY ASSESSMENT REPORT:
CLARK FORK RIVER NPL SITES NRDA
LETHAL INJURIES TO SNOW GEESE
BERKELEY PIT
BUTTE, MT

JANUARY 1996





STATE DOCUMENTS COLLECTION
OCT 1 7 1996
MONTANA STATE LIBRARY
1515 E. 6th AVE.
HELENA, MONTANA 59620

Sue
MAY 2 6 1997
NOV 2 [illegible] 1999

SUPPLEMENTAL INJURY ASSESSMENT REPORT:

CLARK FORK RIVER NPL SITES NRDA

LETHAL INJURIES TO SNOW GEESE
BERKELEY PIT, BUTTE, MT

PREPARED FOR:

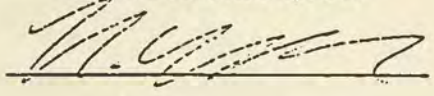
MONTANA NATURAL RESOURCE DAMAGE ASSESSMENT LITIGATION PROGRAM

PREPARED BY:

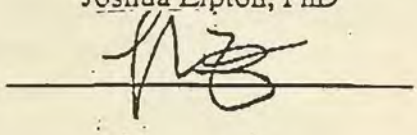
HAGLER BAILLY CONSULTING, INC.

Testifying Experts:

Hector Galbraith, PhD



Joshua Lipton, PhD



CONTENTS

Digitized by the Internet Archive
in 2011 with funding from
Montana State Library

http://www.archive.org/details/supplementalinju1996hagl

FIGURE

TABLES

ACRONYMS

ARCO	Atlantic Richfield Company
CBS	could not be sampled
CSU-VDL	Colorado State University Veterinary Diagnostic Laboratory
GI	gastrointestinal
MBMG	Montana Bureau of Mines and Geology
MDL-DLD	Montana Department of Livestock - Diagnostic Laboratory Division
NFWFL	National Fish and Wildlife Forensics Laboratory
NR	not reported
NRDA	Natural Resource Damage Assessment
NRDP	Natural Resource Damage Program
U.S. EPA	United States Environmental Protection Agency
U.S. NBS	United States National Biological Service
WSVL	Wyoming State Veterinary Laboratory

CHAPTER 1
INTRODUCTION

1.0 INTRODUCTION

This supplemental injury report evaluates injuries to wildlife resources that have resulted from releases of hazardous substances into the Berkeley Pit in Butte, MT.

In November, 1995, snow geese (Anser caerulescenes) carcasses were discovered floating in the Berkeley Pit. Over a period of several days, a reported 342 snow geese carcasses were removed from the pit waters (Billings and Cahalan, 1995; Billings, 1995a,b; The Montana Standard, 1995; Laceky, 1995). Several of the carcasses were retained for analysis to determine the cause of the deaths, and the remainder were removed and stored in plastic bags to await the results of laboratory analyses. Nineteen carcasses collected from the Berkeley Pit were submitted for analysis to determine the causes of death: Atlantic Richfield Company (ARCO) submitted six carcasses to the Colorado State University-Veterinary Diagnostic Laboratory (CSU-VDL); the U.S. EPA submitted four carcasses (plus two retrieved from the Mantel Ranch in Beaverhead County, MT) to the National Fish and Wildlife Forensics Laboratory (NFWFL) in Oregon; the Montana Natural Resource Damage Program (NRDP) submitted five carcasses to the Wyoming State Veterinary Laboratory (WSVL) at the University of Wyoming; and Montana Department of Fish, Wildlife, and Parks submitted four geese to the State of Montana Department of Livestock- Diagnostic Laboratory Division (MDL-DLD).

The Berkeley Pit is a former open-pit copper mine that since 1982 has been filling with water at an approximate rate of 5 million gallons per day (Figure 1). The Berkeley Pit is 1,780 feet deep, and the water surface measures approximately 675 acres (Maest et al., 1995). Inflows to the pit include water from flooded abandoned underground mine workings, groundwater, runoff, precipitation, and process waters from the former Weed Concentrator and the Montana Resources Concentrator (Maest et al., 1995). The pit water is acidic and contaminated with metals and metalloids; hazardous substances measured in pit water samples collected in June and November, 1995 include As, Ag, Cd, Cr, Cu, Ni, V, and Zn (MBMG, 1995).

All of the laboratories to which geese were submitted conducted necropsies on the carcasses and collected liver and kidney tissues, when possible, for residue analysis. Fungal, bacteriological, and histopathological examinations were also conducted. WSVL, MDL-DLD, and CSU-VDL have released the results of their examinations and analyses. NFWFL has released data pertinent to the geese that were collected at Mantel Ranch.

Figure 1. The Berkeley Pit, Butte, Montana.

The purpose of this report is to summarize the existing data on waterfowl mortalities in the Berkeley Pit, to present the results of the laboratory analysis reported to date by each of the laboratories that received goose carcasses, and to evaluate data relevant to the goose deaths in the context of the ongoing Clark Fork River Natural Resource Injury Assessment. This report is intended to supplement previous injury assessment reports presented by the State of Montana NRDP. The organization of this report is as follows: the remainder of Chapter 1 summarizes the principal conclusions of the report. Chapter 2 describes the methods used in developing the report. Chapter 3 presents information on injury determination and quantification, and presents an analysis of causality. Chapter 4 presents literature cited in the report. The pathology and toxicology reports from WSVL, MDL-DLD, and CSU-VDL are included as Appendices A, B, and C, respectively. The pathology reports from NFWFL for the two geese recovered from Mantel Ranch are included as Appendix D.

1.1 SUMMARY OF CONCLUSIONS

The post mortem examination data reported to date indicate:

- 100% of the geese retrieved from the Berkeley Pit that were examined showed necrosis or sloughing of the epithelium of the esophagus. All the geese also demonstrated necrosis or sloughing of the epithelium of one or more of the following: trachea, larynx, gizzard, and/or oral cavity. These symptoms are consistent with characteristics of acute Cu toxicosis reported in the literature, and with the effects of exposure to corrosive substances.

- 100% of the geese retrieved from the Berkeley Pit and examined at the WSVL exhibited severe acute stomatitis, esophagitis, and/or tracheitis. These symptoms are also consistent with characteristics of acute Cu toxicosis reported in the literature, and with the effects of exposure to corrosive substances.

- 100% of the intact geese retrieved from the Berkeley Pit and examined at WSVL or CSU-VDL exhibited kidney damage that is likely associated with attempts by the geese to eliminate excessive metal and hydrogen ions.

- 100% of the geese retrieved from the Berkeley Pit that were analyzed for metals residues exhibited elevated concentrations in the livers and kidneys. These data indicate that the birds were exposed to hazardous substances, that the hazardous substances are consistent with those measured in the Berkeley Pit, and that the metals were at concentrations observed to cause lethality in the literature.

- All but two of the geese collected from the pit were coated with an extraneous yellow pigment that deposited on feathers, eyes, and/or in digestive tracts of these birds. The yellow pigment is believed to be a coating of Fe and other metals salts.

- Other observations indicated that the geese collected from the pit were in otherwise good condition and appeared to be generally healthy, had empty stomachs, and had grossly normal major organs.

The necropsy data, metals residues, the common location and synchrony of the deaths, and water quality data collected from the Berkeley Pit consistently support the conclusion that acute metal toxicosis and sulfuric acid exposure caused the deaths of the 342 geese. The necropsy data (Appendices A, B, and C) are consistent with both acute metals toxicosis and with the well-documented corrosivity of sulfuric acid. The necropsy data are not consistent with hypotheses that bacterial or fungal disease, parasitism, exhaustion or starvation caused the deaths of the geese.

The data confirm that concentrations of hazardous substances in surface water resources of the Berkeley Pit are sufficient to cause injury to biological resources, and that groundwater sources to the Berkeley Pit serve as a pathway of injury to biological resources. Therefore, in addition to injury of biological resources, this wildlife kill confirms that water resources of the Berkeley Pit are injured [43 CFR § 11.62 (b) (v) and (iv) (c)], and that groundwater sources to the Berkeley Pit serve as pathways of injury to biological resources.

Moreover, "Migratory birds are protected by law: Congress has fixed a maximum fine for accidentally taking such a bird at $5,000 16 U.S.C. §707(a);18 U.S.C. §§3559,3571. That fine presumably reflects Congress's judgment of the social loss resulting from the death of such a bird, and in a representative democracy, Congress's judgment should be taken as at least a first approximation of the values of society." (Daum, 1992, pages 4-5).

CHAPTER 2
METHODS

In producing this report, data sources consulted included pathology reports from WSVL (Appendix A) at the University of Wyoming, MDL-DLD (Appendix B), CSU-VDL (Appendix C), and NFWFL (Appendix D), and toxicology reports from WSVL (Appendix A), the Michigan State University Animal Health Diagnostic Laboratory (for birds necropsied at MDL-DLD, Appendix B) and CSU-VDL (Appendix C). Recent Berkeley Pit water quality data was obtained from the Montana Bureau of Mines and Geology (MBMG, 1995). In addition, scientific literature regarding the significance of the presence of bacterial pathogens and non-pathogens in intestines and lungs, metal toxicosis in animals, health hazards of sulfuric acid, generation of acid mine drainage, and the ecology of snow geese and other migratory waterfowl were consulted. All references are cited in the text, and full citations are presented in Section 4.0.

CHAPTER 3
INJURY DETERMINATION AND QUANTIFICATION

3.1 INJURY DEFINITION

An injury to a biological resource has resulted from the release of a hazardous substance if the concentration of the hazardous substance is sufficient to cause the biological resource or its offspring to have undergone at least one of the following adverse changes in viability: death, disease, behavioral abnormalities, cancer, genetic mutations, and physiological malfunctions [43 CFR § 11.62 (f) (I)].

The relevant category of injury in this case is death. Biological responses for determining when death is a result of exposure to the release of hazardous substances include:

- Wildlife kill investigations. Wildlife kill investigations may be used when acute mortality has occurred to multiple wildlife species, or when detectable quantities of hazardous substances have adhered to, bound to, or otherwise covered surface tissues, or had been ingested or inhaled by dead or dying bird or mammal species [43 CFR § 11.62 (f) (4) (I) (C)].

3.2 WILDLIFE KILL DATA

The WSVL (Appendix A), MDL-DLD (Appendix B), CSU-VDL (Appendix C), and NFWFL (Appendix D) examined a total of 19 carcasses of snow geese collected from the Berkeley Pit, and the results of examinations of 15 of the geese have been released. Two of the fifteen had been scavenged before they were retrieved from the pit and were only partially intact. The NFWFL also examined the carcasses of two snow geese collected from the Mantle Ranch in Beaverhead County, MT, approximately 40 miles from the Berkeley Pit. All laboratories conducted necropsies on the carcases and collected liver and kidney tissues (when available) for contaminant analysis. Bacteriological, fungal, and histopathological examinations were also conducted. In addition, the NFWFL x-rayed the Mantel Ranch carcasses for evidence of trauma.

3.2.1 Necropsy Results

The birds examined were generally considered to be in good body condition. This determination was based on the body weights and the abundance and consistency of the subcutaneous and visceral fat. In addition, many of the organs (e.g., brain, heart, reproductive organs, muscle tissue, spleen, and thyroid gland) looked essentially normal in most of the birds examined.

Necropsy results have been reported for 13 intact snow geese carcasses collected from the Berkeley Pit. All of the carcasses exhibited necrosis and sloughing of the epithelium (linings) of the esophagus (Table 3-1). Ten of the thirteen exhibited necrosis and sloughing of the epithelium of the trachea. Seven of the same set of geese exhibited sloughing of the oral cavity (mouth), and six also demonstrated necrosis and sloughing of the lining of the larynx. All of the intact snow goose carcasses examined at the WSVL exhibited severe acute stomatitis, esophagitis, and tracheitis (inflammation of the mouth, esophagus, and trachea) (Table 3-1). In addition, two of the geese examined at CSU-VDL exhibited inflammation of the esophagus and/or gizzard (Table 3-1). None of the characteristics noted above were documented in either of the birds collected at Mantle Ranch (Table 3-1).

Gross observations from the pathology reports include notations of extraneous yellow pigment deposition (Table 3-2). Feathers of all but two of the carcasses retrieved from the Berkeley Pit were reportedly stained yellow, and yellow pigment deposition was noted in the gastrointestinal (GI) tracts of all but two of the intact carcasses. Yellow pigment deposition was also noted on the eyes of eight of the geese. Yellow pigment scraped from the trachea, larynx, crop, feathers, skin, esophagus, and eyes was analyzed by MDL-DLD and tested positive for Fe. The yellow pigment is likely a coating of Fe salts and salts of other metals dissolved in the pit waters (E.S.P. Williams, DMV, PhD,WSVL, pers. comm.). No yellow pigment deposition was noted on or in either of the birds collected from the Mantle Ranch.

No evidence of recent food ingestion or fatal trauma was reported for birds collected from the Berkeley Pit (Table 3-2). Gizzard contents of all intact carcasses collected from the Berkeley Pit were empty except for small amounts of grit. Gizzard contents were not reported for two of the geese submitted to CSU-VDL or for the geese collected at Mantle Ranch. Two of the birds collected at the pit had been scavenged and one bird had fractured ribs, but these injuries were not considered to be the cause of the deaths. The birds collected from Mantle Ranch both had injuries characteristic of a blunt trauma. The veterinary medical examiner's report suggests that the injuries were consistent with in flight collision with a radio tower or power line (Appendix D). None of the carcasses retrieved from the pit or the two from Mantel Ranch showed evidence of gunshot wounds.

Pulmonary edema (lung congestion) was reported for all but one of the birds collected from the Berkeley Pit (Table 3-2). Neither of the snow geese collected from Mantle Ranch showed

Table 3-1
Necropsy Results

Laboratory	Recovery Location	Goose Identification	Sloughing or Necrosis of Epithelium Reported					Inflammation Reported				
			Mouth	Larynx	Trachea	Esophagus	Gizzard	Mouth	Larynx	Trachea	Esophagus	Gizzard
WSVL (Appendix A)	Berkeley Pit	95A10,078-A[1]	CBS	CBS	CBS	CBS	CBS	CBS	CBS	CBS	CBS	CBS
		95A10,078-B	x	x	x	x	No	x	No	x	x	No
		95A10,078-C	x	No	x	x	No	x	No	x	x	No
		95A10,078-D	x	x	x	x	No	x	No	x	x	No
		95A10,078-E[2]	x	No	x	x	CBS	x	No	x	x	CBS
MDL-DLD (Appendix B)	Berkeley Pit	7-71	No	x	x	x	No	No	x	No	No	x
		7-72	No	x	x	x	No	No	No	No	No	No
		7-73	No	x	x	x	No	No	No	No	No	No
		7-74	No	x	x	x	x	No	No	No	No	No
CSU-VDL (Appendix C)	Berkeley Pit	956-12251 A	x	No	x	x	x	No	No	No	No	x
		956-12251 B	x	No	x	x	x	No	No	No	No	No
		956-13011 A	No	No	No	x	x	No	No	No	x	x
		956-13011 B	x	No	No	x	x	No	No	No	No	No
		956-13011 C	No	No	No	x	x	No	No	No	No	No
		956-13011 D	No	No	No	x	x	No	No	No	No	No
NFWL (Appendix D)	Mantle Ranch	ST 624701	No	No	No	No	No	No	No	No	No	No
		ST 02405	No	No	No	No	No	No	No	No	No	No

1. Carcass composed only of bones, feathers, and a portion of lung
2. Head of carcass only
CBS Could not be sampled
NR Not reported

Table 3-2
Gross and Microscopic Observations

Laboratory	Recovery Location	Goose Identification	Extraneous Yellow Pigment Deposition			Gizzard Content	Evidence of Trauma	Pulmonary Edema	Kidney Damage
			Feathers	Eyes	GI Tract				
WSVL (Appendix A)	Berkeley Pit	95A10,078-A	x [1]	CBS	CBS	CBS	Scavenged	x	CBS
		95A10,078-B	x	NR	x	Grit Only	No	x	x
		95A10,078-C	x	NR	x	Grit Only	Fractured Ribs	x	x
		95A10,078-D	x	NR	x	Grit Only	No	x	x
		95A10,078-E[2]	x	NR	x	CBS	Scavenged	CBS	CBS
MLD-DLD (Appendix B)	Berkeley Pit	7-71	x	x	x	Grit Only	No	x	NR
		7-72	x	x	x	Grit Only	No	x	NR
		7-73	x	x	x	Grit Only	No	x	NR
		7-74	x	x	x	Grit Only	No	x	NR
CSU-VDL (Appendix C)	Berkeley Pit	956-12251 A	x	x	x	Grit Only	NR	x	x
		956-12251 B	x	x	x	Grit Only	NR	x	x
		956-13011 A	x	x	x	Grit Only	NR	x	x
		956-13011 B	x	x	x	Grit Only	NR	x	x
		956-13011 C	NR	NR	NR	NR	NR	No	x
		956-13011 D	NR	NR	NR	NR	NR	x	x
NFWFL (Appendix D)	Mantle Ranch	ST 624701	No	NR	NR	NR	Fractured Keel Bone	No	No
		ST 02405	No	NR	NR	NR	Internal Hemorrhage	No	No

1. Carcass composed only of bones, feathers, and a portion of lung
2. Head of carcass only

CBS Could not be sampled
NR Not reported

any indications of lung congestion. Kidney damage, including gross observations of congestion and pale coloration and microscopic observations of dilated renal tubules that contain mineralized material, was documented in all of the intact carcasses examined by WSVL and CSU-VDL. The MDL-LDL did not include a report of kidney status.

3.2.2 Fungal, Bacteriological, and Parasitological Results

Table 3-3 presents the reported results of the fungal, bacteriological, and parasitological examinations of the snow goose carcasses. CSU-VDL and MDL-DLD attempted fungal cultures from carcasses with available lung tissue. The WSVL evaluated the potential of fungal infection by gross and microscopic examination of tissues. ARCO reported one confirmed case and one unconfirmed case of the fungal infection aspergillosis from the birds examined by CSU-VDL (ARCO, 1995). To be a confirmed case of a fungal infection such as aspergillosis, the fungus must be able to be cultured from the tissue of the carcass being examined, and the fungi need to be associated with typical fungal lesions in the tissues. No other laboratory reported fungal infections in any of the carcasses.

The WSVL prepared cultures prepared from heart and lung tissue of several of the birds to evaluate bacterial pathogenic activity. *Escherichia coli* and *Streptococcus* sp. were cultured from the lungs of two separate birds. No bacterial infection significant enough to result in death of the birds was detected by the veterinary pathologist or toxicologist. Parasitism was noted in one bird during the necropsy. A protozoal cyst in a muscle, a helminth in a vessel, and a sporozoan were identified in the single carcass.

The NFWFL did not attempt bacterial cultures during the gross examination, but looked for lesions suggestive of infectious disease. No bacterial infections or parasitism were noted for the Mantel Ranch geese.

The bacteriology division of the MDL-DLD prepared cultures from 3 of the 4 birds sampled. Cultures for general bacterial growth were prepared from the large intestine and sinuses, and attempts were also made to specifically isolate *Salmonella* sp. from the large intestine. *E. coli* was cultured from the intestines of two birds and *Hafnia alvei* was cultured from the intestines of a single bird. Cultures prepared from lung tissue were examined for bacterial and fungal growth, and Gram stains prepared from smears of lung tissue were examined for bacteria. No evidence of fungal or bacterial presence in lung tissue was observed. A fecal sample from one bird was examined for parasite ova using the floatation method; no ova were found. Nematode ova in the gizzard of one bird were, however, noted during the histopathological examination.

CSU-VDL detected *E. coli* in the lungs of one bird and in the lungs and liver of a second bird. A nematode was found in the ventriculus of one bird (Table 3-3).

Table 3-3
Fungal, Bacteriological, and Parasitological Results

Laboratory	Recovery Location	Goose Identification	Fungal Infection	Bacterial Infection	Parasitism
WSVL (Appendix A)	Berkeley Pit	95A10,078-A	No[1]	*Escherichia coli* (Lung)	No
		95A10,078-B	No	*Streptococcus* sp. (Lung)	Protozoal cyst (Muscle) Helminth (Vessel) Coccidia (Intestine)
		95A10,078-C	No	No	No
		95A10,078-D	No	No	No
		95A10,078-E[2]	CBS	CBS	CBS
NFWL (Appendix D)	Mantle Ranch	ST 624701	No	No	No
		ST 02405	No	No	No

Table 3-3 (cont.)
Fungal, Bacteriological, and Parasitological Results

Laboratory	Recovery Location	Goose Identification	Fungal Infection	Bacterial Infection	Parasitism
MDL-DLD (Appendix B)	Berkeley Pit	7-71	No	No	Nematode ova (Gizzard)
		7-72	No	*E. coli* (Intestine) *Hafnia alvei* (Intestine)	No
		7-73	NR	NR	NR
		7-74	No	*E. coli* (Intestine)	No
CSU-VDL (Appendix C)	Berkeley Pit	956-12251 A	*Aspergillus* sp. *Rhizopus* sp.	No	NR
		956-12251 B	*Aspergillus* sp.	No [3]	NR
		956-13011 A	No	*E. coli* (Lung)	Nematode (Ventriculus)
		956-13011 B	No	No	NR
		956-13011 C	No	No	NR
		956-13011 D	No	*E. coli* (Lung and Liver)	NR

1. Carcass composed only of bones, feathers, and a portion of lung
2. Head of carcass only
3. Unconfirmed

CBS Could not be sampled
NR Not reported

3.2.3 Metals Residues

Metal residue data from WSVL, MDL-DLD, and CSU-VDL have been released; the Mantel Ranch geese were not submitted for metals residue analysis. Kidney and liver tissues were analyzed for a variety metals and metalloids (i.e., Pb, Fe, Hg, As, Mb, Zn, Cu, Cd, Ba, Be, Co, Cr, Ni, Mn, P, B, Tl, Sb, Na, K, Ca, Mg, Se, V). Table 3-4 presents concentrations of a subset of the metals and metalloids that were analyzed in liver tissues and Table 3-5 presents concentrations in kidney tissues. Results from CSU-VDL are in mg/kg dry weight for Fe, As, Zn, Cu, Se, and Mn, and in mg/kg wet weight for Cd. Results from MDL-DLD (analyzed by the Michigan State University Animal Health Diagnostic Laboratory) and WSVL are in mg/kg wet weight. Lead, tin, and vanadium concentrations were below detection limits in all tissues sampled. Mercury concentrations ranged from below instrument detection limit to 0.21 mg/kg. Nickel concentrations were reported for only 3 birds and ranged from below instrument detection limit to 1.1 mg/kg.

Dr. Merl F. Raisbeck, Wyoming State Veterinary Toxicologist, indicated that the liver and kidney samples he analyzed are high in Mn, Zn, and Cu. In fact, he described the Cu concentrations to be "acute toxic" in some domestic birds. He reported that Fe and Mn concentrations in two of the birds would represent "intoxication" in domestic species. Liver concentrations of cadmium are also reported to be high (Appendix A).

Dr. Michael Slanker of the Michigan State University Animal Health Diagnostic Laboratory reported that the liver and kidney concentrations of Mg and kidney concentrations of Zn were elevated in the geese submitted to the State of Montana Department of Livestock - Diagnostic Laboratory, and that the liver and kidney concentrations of Mn were toxic (Appendix B).

Dr. Terry Spraker of CSU-VDL reported that the six geese examined had elevated concentrations of Cu, Mn, and Zn in the liver and kidneys, and that the observed kidney damage could have been caused when the geese attempted to eliminate, via excretion, the excess Cu, Mn, Zn and H+ ions (Appendix C).

3.2.4 Conclusions

Drs. E.S. Williams and M. Raisbeck of WSVL concluded that the cause of death of the birds they examined appeared to be due to contact with and ingestion of a highly corrosive and/or toxic substance. Contact with a corrosive substance would be consistent with the sloughing of the linings and inflammation of the mouth, larynx, trachea, esophagus, and gizzard (Appendix A). Dr. Raisbeck reported that the elevated metal concentrations may account for the kidney damage observed. Although the results of the necropsy, gross pathology, fungal, bacteriological, and parasitological examinations were similar to the birds examined at

Table 3-4
Concentrations of Metals and Metalloids in Liver Tissues.
Results from WSVL and Michigan State University in mg/kg wet weight; results from CSU-VDL in mg/kg dry weight for Fe, As, Zn, Cu, Se, and Mn, and mg/kg wet weight for Cd.

Laboratory	Recovery Location	Goose Identification	Fe	As	Zn	Cu	Cd	Se	Mn
WSVL (Appendix A)	Berkeley Pit	95A10,078-B	32	ND	151	115	1.9	NR	NR
		95A10,078-C	334	ND	126	54	1.5	NR	40.2
		95A10,078-D	1200	ND	216	174	2.4	NR	74.4
Michigan State University (Appendix B)	Berkeley Pit	7-72	1050	<0.50	281	64.7	1.81	<2.00	79
		7-74	845	<0.50	238	136	2.36	<2.00	36.1
CSU-VDL (Appendix C)	Berkeley Pit	956-12251 A	427	2.52	692	678	1.0	2.74	97.5
		956-12251 B	278	2.56	312	151	0.9	5.14	184
		956-13011 A	473	0.97	655	225	1.0	3.01	155
		956-13011 B	441	1.94	765	254	0.8	3.04	225
		956-13011 C	371	1.01	535	242	0.5	4.50	152
		956-13011 D	565	0.85	346	147	0.7	3.47	147

NR Not reported
ND Not detected

Table 3-5
Concentrations of Metals and Metalloids in Kidney Tissues.
Results from WSVL and Michigan State University in mg/kg wet weight; results from CSU-VDL in mg/kg dry weight for Fe, As, Zn, Cu, Se, and Mn, and in mg/kg wet weight for Cd.

Laboratory	Recovery Location	Goose Identification	Fe	As	Zn	Cu	Cd	Se	Mn
WSVL (Appendix A)	Berkeley Pit	95A10,078-B	126	ND	171	13.2	1.4	NR	54.3
		95A10,078-C	144	ND	119	29.1	2.6	NR	135
		95A10,078-D	176	ND	205	37.6	3.7	NR	29.8
Michigan State University (Appendix B)	Berkeley Pit	7-72	309	<0.50	215	44.8	1.87	<2.00	69.2
		7-74	831	<0.50	246	183	2.54	<2.00	32.3
CSU-VDL (Appendix C)	Berkeley Pit	956-12251 A	506	3.37	527	155	2.2	2.4	46
		956-12251 B	424	3.25	398	69.5	1.6	4.26	248
		956-13011 A	307	1.29	568	91.2	1.0	2.85	211
		956-13011 B	425	0.81	413	72.3	0.7	2.56	171
		956-13011 C	310	0.59	531	90.4	1.9	3.90	282
		956-13011 D	427	0.63	511	50.7	0.8	3.14	395

ND Not detected
NR Not reported

WSVL, Dr. Layton from MDL-DLD was unable to "absolutely" determine a cause of death for the birds examined in Montana. A lack of scientific knowledge of the effects of the water chemistry on geese was cited as the reason for the inability to determine the cause of death (Appendix B).

Dr. Terry Spraker of CSU-VDL noted that the ulceration and erosion of the upper digestive system from the esophagus to the ventriculus and elevated levels of Cu, Mn, and Zn in liver and kidneys of the geese could have occurred as a result of ingestion of the "acidic mineralized water in the lake" (Appendix C). Dr. Spraker observed that the "acidic nature of the water could have facilitated absorption of these heavy metals", that "The renal necrosis may have been associate with the kidneys trying to eliminate the excessive number of ions in the blood, especially Cu, Zn, and Mn." and that "The tubular necrosis may have also been associated with trying to eliminate excessive hydrogen ions secondary to the suspected acidosis."

3.3 BERKELEY PIT WATER QUALITY DATA

Sources of contamination of the surface water contained in the Berkeley Pit and groundwater released to the Berkeley Pit include underground mine workings, the walls of the Berkeley Pit, and acid mine drainage resulting from the oxidation and dissolution of pyrite and other metals and metalloid sulfides in the underground mine workings. Hazardous substances that have been identified in source materials, including the underground mine workings and pit walls and acid mine drainage, are Ag, As, Cd, Cu, Ni, Pb, Sb, V, Zn, sulfate, and sulfuric acid (Maest et al., 1995). Hazardous substances measured in pit water in samples collected by the MBMG in June and November 1995 include As, As, Cd, Cr, Cu, Ni, Pb, V, Zn, and sulfate (MBMG, 1995). At the pH of the pit water (2.56 in November, 1995), at least some portion of the sulfate is present as sulfuric acid.

Table 3-6 presents pH and concentrations of hazardous substances in surface water samples from the Berkeley Pit, collected on June 19 and November 15, 1995. It was during the November 15, 1995 water sampling event that MBMG personnel discovered the goose carcasses.

Table 3-6
Hazardous Substance Concentrations and pH Measured in Berkeley Pit Surface Water Samples. All metals and metalloid are dissolved concentrations in µg/L.
(Data Source: MBMG, 1995; preliminary results).

Analyte (metals/ metalloids in µg/L)	June 19, 1995 Sampling depth: 200 ft	Nov. 15, 1995 Sampling depth: 200 ft	Nov. 15, 1995 Sampling depth: surface	Nov. 15, 1995 Sampling depth: surface
pH	2.72	2.31	2.56	2.56
Ag	<5	<5	—	—
As	820	730	—	—
Cd	2,260	2163	2,320	2,340
Cr	95	57	—	—
Cu	198,000	204,000	188,000	189,000
Hg	<0.1	<0.1	—	—
Ni	1,060	1,107	1,224	1,246
Pb	145	111		
V	180	150	—	—
Zn	634,000	610,000	553,000	555,000

3.4 CAUSALITY EVALUATION

The postmortem examinations of the snow geese recovered from the Berkeley Pit included gross observations of body condition, metal toxicology, and histopathological, fungal, and bacteriological examinations. This section evaluates the likelihood that, based on the existing wildlife kill data, factors other than hazardous substances caused the deaths of the geese. Other factors considered include:

- Acute aspegillosis.
- Bacterial disease.
- Parasitism.
- Exhaustion or starvation.

3.4.1 Acute aspergillosis

Aspergillosis is a disease caused by a fungus (*Aspergillus*) that may be contracted from grain and can be fatal to geese. Of the 15 geese submitted for examination from Berkeley Pit, CSU-VDL reported that one of the six geese examined had a confirmed aspergillosis infection and one had an unconfirmed aspergillosis infection. The goose with the aspergillosis infection also exhibited necrosis of the trachea, esophagus, gizzard, and oral cavity, yellow discoloration of feathers, eyes, and GI tract, pulmonary edema, kidney damage, and elevated metals concentrations in liver and kidney tissues. The other laboratories (WSVL and MDL-DLD) reported no fungal infections in any of the carcasses. It is possible that among the 300+ geese that were not examined, there were individuals infected with the fungus. However, the U.S. Fish and Wildlife Service - Madison, WI has confirmed that there have been no snow geese kills along the Pacific flyway this year other than at the Berkeley Pit (L. Glasier, Madison Health Laboratory, U.S. FWS, Madison, WI, pers. comm.). Moreover, a 23 year record of snow goose deaths along the neighboring Central and Mississippi flyways (1969-1992) confirms that only 0.4% of the reported deaths of banded snow geese, or an average of 1.7 banded geese/year, have been attributable to disease (Cooke et al., 1995).

It is unlikely that the snow geese that died in the Berkeley Pit were the only snow geese on the Pacific flyway that ingested *Aspergillus*-tainted grain, and that all of the geese that ingested the *Aspergillus*-tainted grain succumbed to the fungus synchronously after landing on the Berkeley Pit. The existing data are not consistent with the hypothesis that a fungus killed the 342 geese.

3.4.2 Bacterial disease

The WSVL attempted to prepare cultures from heart and lung tissues specifically to evaluate the involvement of a bacterial pathogen. *E. coli* and *Streptococcus* sp. were detected in the lungs of two separate birds, but no bacterial pathogen infection that could have resulted in the death of the birds was detected. The bacteriology division of the MDL-DLD found *E. coli* in the large intestine of two birds and *Hafnia alvei* in the intestine of one bird, but found no bacterial pathogens that could have resulted in the deaths of the birds. CSU-VDL found *E. coli* in the lungs of one bird and in the lungs and liver of a second bird.

E. coli is normal intestinal flora of animals and is ubiquitous in the environment from fecal contamination, *Streptococci* are normal flora of the skin and mucous membranes of animals, and *Hafnia* spp. are considered to be non-pathogens (Scanlan, 1988). *E. coli* in the lungs is occasionally a secondary opportunistic pathogen in pneumonia (Scanlan, 1988), but in this case is probably a post-mortem contaminant. Streptococcal infections of the respiratory tract may be primary infections or secondary to other diseases (Scanlan, 1988). Given the absence of a consistent bacterial pathogen amongst the birds that were retrieved from the Berkeley Pit

and examined for bacterial infection, the lack of microscopic evidence of a bacterial disease, and the synchrony and localized nature of the goose deaths, it is unlikely that a bacterial disease caused the deaths of the 342 geese.

3.4.3 Parasitism

WSVL noted a helminth and a coccidia in one of four goose carcasses that could be examined, MDL-DLD found a single nematode ovum in the gizzard of one bird, and CSU-VDL found a nematode in the ventriculus of one goose.

Given the absence of a consistent parasite amongst the birds that were retrieved from the Berkeley Pit and examined for parasites, and the more common observation of absence of parasites completely, it is unlikely that parasitism caused the deaths of the 342 geese.

3.4.4 Exhaustion or starvation

WSVL, MDL-DLD and CSU-VDL noted that the stomachs and gizzards of most of the birds were empty (CSU-VDL did not report contents for two birds). However, this is normal for birds on migration as these were, and the same laboratories that reported that the geese collected from the pit were generally in good body condition. The geese exhibited normal body weights and abundance and consistency of subcutaneous and visceral fat. In addition, the brain, heart, reproductive organs, muscle tissue, spleen, and thyroid glands of the geese looked essentially normal in most of the birds examined. The geese showed no evidence of unusual stress that would normally be associated with exhaustion or starvation, i.e., emaciation, deleted fat stores, or low body weight. The data do not support the conclusion that the birds died of exhaustion or starvation.

The existing data do not suggest that the geese were killed by a fungal or bacterial disease or parasite, or that the geese died of starvation or exhaustion.

3.4.5 Evidence of Hazardous Substance Causality

The post mortem examination data reported to date by all of the laboratories are consistent in several regards: all of the geese retrieved from the Berkeley Pit that could be examined showed consistent necrosis or sloughing of the epithelium of the esophagus, in addition to necrosis or sloughing of the epithelium of the trachea, larynx, gizzard, and/or oral cavity; all of the geese retrieved from the Berkeley Pit and examined at the WSVL exhibited severe acute stomatitis, esophagitis, and/or tracheitis; all of the inact geese examined by WSVL or CSU-VDL exhibited kidney damage, and all of the geese retrieved from the Berkeley Pit that have

been subjected to toxicological analysis exhibited elevated metals concentrations in the livers and kidneys. Moreover, all but two of the geese collected in the pit were coated with the yellow pigment believed to be a crust of Fe and other metals salts from the pit water (E.S. Williams, pers. comm.; Appendix B) that deposited on feathers, eyes, and/or in digestive tracts of these birds. Several additional consistent observations were that the geese collected from the pit were in otherwise good condition and appeared to be generally healthy, that their stomachs were empty except for grit, and that their major organs were grossly normal.

Necropsy results and metals residues reported for the geese retrieved from the pit are consistent with both acute copper toxicosis reported by Henderson and Winterfield (1975), and with the well-documented corrosivity of sulfuric acid.

Metals Toxicosis

Henderson and Winterfield (1975) reported on the cause of death of 100 three-week old Canada geese that died following exposure to water treated with copper sulfate for algal control. The geese died within 9 hours of exposure to pond water containing approximately 240 mg/kg Cu. Necropsies of ten of the geese revealed extensive necrosis and sloughing of the mucosae of the proventriculus and gizzard, small hemorrhages in the liver, and a greenish discoloration of the lungs and contents of the digestive tract. All visceral organs were examined for histopathy, and "the most significant microscopic lesions were the necrotic changes of the proventriculus and gizzard, and a non-specific diffuse hydropic degeneration of the liver." They reported that the lesions of the proventriculus and gizzard were "those associated with copper toxicosis in other avian species." Toxicological analysis of the livers revealed liver copper concentrations of 56-97 mg/kg wet weight.

Henderson and Winterfield (1975) concluded that there appeared to be a mechanism for the rapid uptake of large amounts of Cu from the gastrointestinal tract since the deaths occurred shortly after ingestion and the liver contained Cu in concentrations substantially higher than normal. "Normal" liver Cu concentration for domestic chickens was reported as 3-26 mg/kg (Mebring et al, 1950, as cited in Henderson and Winterfield, 1975). Cu in the livers of the ten Canada geese examined by Henderson and Winterfield exceeded "normal" liver concentrations by two to thirty times. The Henderson and Winterfield data suggest that the acutely toxic Cu threshold in lesser snow goose liver is <56 mg/kg wet weight.

The residue analyses of the snow geese that died in the Berkeley Pit confirmed liver Cu concentrations ranging from 54 to 174 mg/kg wet weight. These concentrations greatly exceed "normal" Cu liver concentrations in domestic birds, and they exceed the high end of the range of Cu liver concentrations measured in the birds examined by Henderson and Winterfield (56-97 mg/kg wet weight). By comparison to the Henderson and Winterfield data, the Berkeley Pit geese certainly exceed the acutely toxic threshold for Cu liver concentration. Moreover, the necropsy data are consistent with the observations of several reseaserchers,

(Henderson and Winterfield, 1975; Poupoulis and Jensen, 1976; Jensen and Maurice, 1978), that acute copper toxicosis in birds is manifested primarily by erosion of the gizzard and proventriculus.

Concentrations of Cd, Cu, Mn, and Zn measured in snow geese from Washington and California are summarized in Table 3-7 (U.S. NBS, unpublished data). Both populations breed on Wrangel Island and then migrate to separate wintering territories in Washington and California (C. Hui, U.S. NBS, pers. comm.). The one banded goose that was recovered from the Berkeley Pit also bred at Wrangel Island, so it is likely that the data presented in Table 3-7 would have been representative of the expected range of trace element concentration in the livers of the Berkeley Pit geese had they reached their wintering destination and not been exposed to the pit waters. The Washington population is under investigation as a reference population for the California population, which is hypothesized to be declining as a result of exposure to trace metals in fertilizers and fungicides (C. Hui, U.S. NBS, pers. comm.).

Metals analysis of livers of 18 Washington geese and 16 California geese has indicated that the concentration of Cu is higher in the California geese, and concentrations of Fe, Mg, Mo and Zn are higher in the Washington geese (Hui, U.S. NBS, pers. comm.). The metals concentrations in the livers of the geese that died in the Berkeley Pit greatly exceed concentrations of Cd, Cu, Mn, and Zn measured in both of these subpopulations (Table 3-7). The average Cd concentration in the Berkeley Pit geese livers (1.35 mg/kg wet weight, n=11) exceeds the both average concentration of the Washington birds (0.74 mg/kg wet weight, n = 18) and the average concentration of the California geese (0.64 mg/kg wet weight, n=16).

The average Cu concetrations in the livers of the Berkeley Pit geese (282.8 mg/kg dry weight and 108.7 mg/kg wet weight) exceed the average Cu concentrations of the Washington and California geese (45.6 and 116.0 mg/kg dry weight and 14.7 and 40.0 mg/kg wet weight) by two to seven times. Likewise, the average Zn concentration in the livers of the Berkeley Pit geese (550.8 mg/kg dry weight and 202.4 mg/kg wet weight) exceed the average Zn concentrations of the Washington and California geese (108 and 172.6 mg/kg dry weight and 36.5 and 55.6 mg/kg wet weight) by three to five times.

Mn was also elevated in the Berkeley Pit geese: the average Mn concentration in Berkeley Pit geese livers (160.1 mg/kg dry weight and 57.4 mg/kg wet weight) exceeded the average Mn concentrations in the Washington and California geese (9.9 and 10.3 mg/kg dry weight and 3.23 and 3.56 mg/kg wet weight) by fifteen to over seventeen times.

Table 3-7
Comparison of Concentrations of Trace Metals in Livers of Lesser Snow Geese from Washington and California Subpopulations with Concentrations in Berkeley Pit Geese Livers. Source of Washington and California data: USNBS unpublished data.

Population	Statistic	Cd (mg/kg)		Cu (mg/kg)		Mn (mg/kg)		Zn (mg/kg)	
		(dry wt)	(wet wt)	(dry wt)	(wet wt)	(dry wt)	(wet wt)	(dry wt)	(wet wt)
Washington[1]	Average	2.30	0.74	45.57	14.66	9.90	3.23	172.59	55.63
California[2]		1.77	0.64	116.03	40.03	10.27	3.56	107.98	36.51
Berkeley Pit[3]		NA	**1.35**	**282.83**	**108.74**	**160.1**	**57.43**	**550.83**	**202.40**
Washington	Maximum	4.28	1.46	87.38	25.60	15.21	5.60	408.00	115.87
California		3.41	1.10	626.50	201.73	17.42	6.11	413.80	124.97
Berkeley Pit		NA	**2.4**	**678**	**174**	**225**	**79**	**765**	**281**
Washington	Minimum	0.52	0.17	19.52	5.89	6.44	1.78	77.53	27.60
California		0.22	0.09	24.51	7.40	6.52	2.26	55.56	20.06
Berkeley Pit		NA	**1.5**	**147**	**54**	**97.5**	**36.1**	**312**	**126**

1. n = 18
2. n = 16
3. For Cu, Mn, Zn dry weight, n = 6; for Cu, Zn wet weight, n = 5; for Mn wet weight, n = 4; for Cd wet weight, n = 11.

Comparison with previous accounts of acute Cu toxicosis confirms two conclusions:

- The necropsy data are consistent with previous observations of acute Cu toxicosis in birds: erosion of the gizzard and proventriculus (Henderson and Winterfield, 1975; Poupoulis and Jensen, 1976; Jensen and Maurice, 1978)

- The Cu concentrations in the livers of the Berkeley Pit geese exceed the acutely toxic threshold for snow geese.

Comparison with the U.S. NBS data (unpublished) confirms three additional conclusions:

- Concentrations of Cd, Cu, Mn, and Zn are greatly elevated in the livers of the Berkeley Pit geese. Concentrations of these elements in the Berkeley Pit geese livers exceeded baseline concentrations of trace metals in lesser snow geese by an order of magnitude.

- It is unlikely that the source of the elevated metals in the geese that died in the Berkeley Pit was metals-tainted grain or pesticides. Concentrations in the livers of Berkeley Pit geese greatly exceeded the "elevated" Cu concentrations of the declining California subpopulation, and also greatly exceeded the "elevated" concentrations of Cd, Mn, and Zn in the stable Washington subpopulation. Thus it is apparent from the comparison with the California and Washington subpopulations, that dietary exposure to metals elsewhere along the flyway or in wintering areas does not explain the magnitude of elevated concentrations nor the combination of metals that are present in elevated concentrations in the Berkeley Pit goose livers.

- The only plausible source of the metals in the livers of the Berkeley Pit geese at the extreme concentrations measured was the pit water. The Berkeley Pit water contained 2,320 µg/L Cd, 189,000 µg/L Cu, and 554,000 µg/L Zn in November, 1995. (Mn concentrations in pit water have not been reported.)

Sulfuric Acid Corrosivity

Sulfuric acid is a hazardous substance and a corrosive and poisonous liquid. It is present in the Berkeley Pit water as a product of the oxidation and dissolution of pyrite and other metal and metalloid sulfides in the underground mine workings and pit walls. The break down of pyrite may be described as (Stokes, 1901, as cited in Nordstrom, 1977):

$$FeS_{2(s)} + Fe_2(SO_4)_{3(aq)} \rightarrow 3FeSO_{4(aq)} + 2S^0_{(s)} \quad (3\text{-}1)$$

$$2S^0_{(s)} + 6Fe_2(SO_4)_{3(aq)} + 8H_20_{(aq)} \rightarrow 12FeSO_{4(aq)} + 8H_2SO_{4(aq)} \quad (3\text{-}2)$$

The leaching of other metal sulfides with sulfuric acid further releases sulfate and heavy metals and metalloids such as copper, cadmium, lead, arsenic, and zinc to the environment (Maest et al., 1994). In such an acidic environment, many of these metals, and in particular, copper and zinc, are severely astringent and would potentiate the corrosive effects of the acid (Williams and Raisbeck, 1995).

In humans, dermal contact with concentrated sulfuric acid may cause severe irritation or burns (Fisher Scientific, 1990; NIOSH, 1994). Contact with the eyes may cause acute corneal damage or blindness (Fisher Scientific, 1990). Sulfuric acid is a mucous membrane irritant, and ingestion can cause corrosion of membranes from the mouth to through the gastrointestinal tract (Fisher Scientific, 1990), and can be fatal (Essex Industrial Chemicals, 1987). Inhalation may cause acute damage to lung tissue, bronchitis, tracheitis, and pulmonary edema (Fisher Scientific, 1990; NIOSH, 1994). In humans, symptoms of exposure include irritation, burning, corneal damage, mucous membrane irritation, rapid breathing, nasal secretions, sneezing, and burning pain in the mouth, throat, esophagus, and abdomen (Fisher Scientific, 1990).

The effects of ingestion of sulfuric acid are consistent with gross pathology reports for the geese that died in the Berkeley Pit. The lesions of the upper digestive system and the minor lesions on the feet of some of the geese could have been caused by the acidity of the water (Dr. T. Spraker, Appendix C). In addition, the ingestion of the acidic water of the Berkeley Pit likely caused an acidotic state in the geese, and the observed renal tubular necrosis may have been caused by attempts to eleiminate excess hydrogen ions (Dr. T. Spraker, Appendix C). Two of the examining laboratories noted the likelihood that the geese suffered from severe acidosis (an accumulation of acids causing disturbance in the acid-base balance of the body)(Appendicies A and C). Moreover, in an acidic environment, copper and zinc are severely astringent and would potentiate the corrosive effects of the acid (Dr. M. Raisbeck, Appendix A).

3.4.6 Conclusions

The consistency and nature of the necropsy data, the consistency of elevated concentrations of hazardous substances, and H^+ in livers and kidneys of the geese, the common location and synchrony of the deaths, and the pit water quality data consistently support the conclusion that it was acute metal toxicosis, likely exacerbated by the sulfuric acid in the water, that caused the deaths of the 342 geese.

The data confirm that concentrations of hazardous substances in surface water resources of the Berkeley Pit are sufficient to cause injury to biological resources, and that groundwater sources to the Berkeley Pit serve as a pathway of injury to biological resources. Therefore, in addition to injury of biological resources, this wildlife kill confirms that surface water

resources of the Berkeley Pit are injured, and that groundwater sources to the Berkeley Pit serve as pathways of injury to both surface water and biological resources.

3.5 QUANTIFICATION SUMMARY

In November, 1995, 342 geese died of acute metal toxicosis and acidosis after ingesting hazardous substances in Berkeley Pit water. This is not an isolated occurrence, but only the most visible and recent instance of wildlife deaths associated with the Berkeley Pit. On November, 16, 1993, MBMG personnel who were sampling the pit waters documented in field notes and in photographs six birds floating on the pit water surface. It is possible that such wildlife kills have occurred in the past, but have gone undocumented. It is also likely that if brown or other highly pigmented bird species have in the past died in the Berkeley Pit, they were camouflaged by the color of the pit water.

As the pit has filled with water, it has likely become a more attractive stopover for migratory birds. It is reasonable to assume that in the future, such kills will continue to occur, since the Berkeley Pit is on the Pacific flyway. Snow geese populations wintering in the Pacific and southwestern states migrate along the Pacific flyway through western Montana to nest on Wrangel Island, northern Alaska, the Yukon, and the western Northwest Territories (Cooke et al, 1995; Dzubin, 1979). A banded goose recovered from the Berkeley Pit was from Wrangel Island, Ushakyskoye, Russia. Regular sightings of snow geese in the Butte, MT area during the winter period have been documented (Bergerson et al., 1992). Other waterfowl sighted in the Butte, MT area that may potentially use the Berkeley Pit as a migration stopover include loons, grebes, pelicans, cormorants, swans, geese, and ducks (Bergerson et al., 1992). The American white pelican, trumpeter swan, and common loon are all considered threatened, endangered, or sensitive species that have been reported in the Butte, MT area (Bergerson et al., 1992).

A number of attitudinal and economic studies clearly indicate that people care about and value preventing injuries to birds and other wildlife resulting from exposure to human pollution. For example, ARCO's economist (Desvousges et al., 1992, see also Boyle et al., 1994) found that households as far away as Georgia placed significant importance and values on preventing migratory waterfowl deaths resulting from landings in uncovered waste oil holding ponds in the Central flyway (Rocky Mountain states). Rowe et al. (1991; 1992) found that residents of Washington and British Columbia were willing to pay to prevent seabird mortality from exposure to oil spills in the Pacific Northwest, with the aggregate value across all household amounting to several hundred to several thousand dollars per bird. Similarly, in reviewing the costs of seabird rescue programs implemented to respond to oil spills in the Pacific Northwest, Rowe and Bird (1991) found values of several hundred to several thousand dollars per bird for four different spills, and up to tens of thousands of dollars per bird following the Exxon oil spill. Other studies have found much larger values associated with preserving populations of

unique species including eagles (e.g., Boyle and Bishop 1987, Stevens et al. 1991) and the whooping crane (Bowker and Stoll, 1988). We have found no studies that specifically address and value snow geese mortality resulting from exposure to hazardous substances.

Returning to the value of an individual bird, John Daum, Counsel representing Exxon, stated in the Exxon contingent valuation method report that "Migratory birds are protected by law: Congress has fixed a maximum fine for accidentally taking such a bird at $5,000 16 U.S.C. §707(a);18 U.S.C. §§3559,3571. That fine presumably reflects Congress's judgment of the social loss resulting from the death of such a bird, and in a representative democracy, Congress's judgment should be taken as at least a first approximation of the values of society." (Daum, 1992, pages 4-5). This fine is consistent with the valuation studies reported above for common and some threathened bird species, but is less than the values that have been explicitly estimated for unique species such at bald eagles and whooping cranes.

3.6 CONCLUSIONS

The post mortem examination data reported to date indicate:

- 100% of the geese retrieved from the Berkeley Pit that were examined showed necrosis or sloughing of the epithelium of the esophagus. All the geese also demonstrated necrosis or sloughing of the epithelium of one or more of the followingn: trachea, larynx, gizzard, and/or oral cavity. These symptoms are consistent with characteristics of acute Cu toxicosis reported in the literature, and with the effects of exposure to corrosive substances.

- 100% of the geese retrieved from the Berkeley Pit and examined at the WSVL exhibited severe acute stomatitis, esophagitis, and/or tracheitis. These symptoms are also consistent with characteristics of acute Cu toxicosis reported in the literature, and with the effects of exposure to corrosive substances.

- 100% of the intact geese retrieved from the Berkeley Pit and examined at WSVL or CSU-VDL exhibited kidney damage that is likely associated with attempts by the geese to eliminate excess metal and hydrogen ions.

- 100% of the geese retrieved from the Berkeley Pit that were analyzed for metals residues exhibited elevated concentrations in the livers and kidneys. These data indicate that the birds were exposed to hazardous substances, that the hazardous substances are consistent with those measured in the Berkeley Pit, and that the metals were at concentrations observed to cause lethality in the literature.

- All but two of the geese collected from the pit were coated with an extraneous yellow pigment that deposited on feathers, eyes, and/or in digestive tracts of these birds. The yellow pigment is believed to be a coating of Fe and other metals salts.

- Other observations indicated that the geese collected from the pit were in otherwise good condition and appeared to be generally healthy, had empty stomachs, and had grossly normal major organs.

The necropsy data, metals residues, the common location and synchrony of the deaths, and water quality data collected from the Berkeley Pit consistently support the conclusion that acute metal toxicosis and sulfuric acid exposure caused the deaths of the 342 geese. The necropsy data (Appendices A, B, and C) are consistent with both acute metals toxicosis and with the well-documented corrosivity of sulfuric acid. The necropsy data are not consistent with hypotheses that bacterial or fungal disease, parasitism, exhaustion or starvation caused the deaths of the geese.

The data confirm that concentrations of hazardous substances in surface water resources of the Berkeley Pit are sufficient to cause injury to biological resources, and that groundwater sources to the Berkeley Pit serve as a pathway of injury to biological resources. Therefore, in addition to injury of biological resources, this wildlife kill confirms that water resources of the Berkeley Pit are injured [43 CFR § 11.62 (b) (v) and (iv) (c)], and that groundwater sources to the Berkeley Pit serve as pathways of injury to biological resources.

Moreover, "Migratory birds are protected by law: Congress has fixed a maximum fine for accidentally taking such a bird at $5,000 16 U.S.C. §707(a);18 U.S.C. §§3559,3571. That fine presumably reflects Congress's judgment of the social loss resulting from the death of such a bird, and in a representative democracy, Congress's judgment should be taken as at least a first approximation of the values of society." (Daum, 1992, pages 4-5).

CHAPTER 4
REFERENCES CITED

ARCO. 1995. Status Report - Berkeley Pit Snow Geese. December 15.

Bergeron, D., C. Jones, D.L. Genter, D. Sullivan. 1992. P.D. Skaar's Montana Bird Distribution, Fourth Edition. Special Publication No. 2. Montana Natural Heritage Program, Helena. 116 pp.

Billings, E.P. 1995a. Fungus may have killed snow geese. The Montana Standard, November 17, 1995.

Billings, E.P. 1995b. 171 geese removed from pit. The Montana Standard, November 18, 1995.

Billings, E.P. and S. Cahalan. 1995. Geese perish in pit. The Montana Standard, November 16, 1995.

Bowker, J.M and J.R. Stoll. 1988. Use of Dichotomous Choice Nonmarket Methods to Value the Whooping Crane Resource. *American Journal of Agricultural Economics* **May**: 372-381.

Boyle, K.H. and R. C. Bishop. 1987. Valuing Wildlife in benefit-Cost Analyses: A Case Study Involving Endangered Species. *Water Resources Research* **23**(5): 943-950.

Boyle, K.J. W. H. Desvousges, F. R. Johnson, R. W. Dunford, and S. P. Hudson. 1995. "An Investigation of Part-Whole Biases in Contingent-Valuation Studies." *Journal of Environmental Economics and Management* **27**(1):64-83.

Cooke, F., R.F. Rockwell, and D.B. Lank. 1995. *The Snow Geese of La Pérouse Bay*. New York: Oxford University Press. 297 pp.

Daum, John F. 1992. Legal and Regulatory Aspects of Contingent Valuation. In *Contingent Valuation: A Critical Assessment* Cambridge Economics, Inc. Cambridge, Mass. April 2.

Desvousges, F. Reed Johnson, Richard W. Dunford, Kevin J. Boyle, Sara P. Hudson, K. Nicole Wilson. 1992. *Measuring Nonuse Damages Using Contingent Valuation: An Experimental Evaluation of Accuracy*. Research Triangle Institute monograph 92-1 prepared for and copyrighted by the Exxon Corporation. June.

Dzubin, A. 1979. Recent Increases of Blue Geese in Western North America. In R.L. Jarvis and J.C. Bartonek, eds. *Management and Biology of Pacific Flyway Geese - A Symposium.* Corvallis, OR: OSU Book Stores, Inc. pp. 141-175.

Essex Industrial Chemicals. 1987. Material Safety Data Sheet - Sulfuric Acid. June.

Fisher Scientific Chemical Division. 1990. Material Safety Data Sheet - Sulfuric Acid. May.

Henderson, B.M., and R.W. Winterfield. 1975. Acute Copper Toxicosis in the Canada Goose. *Avian Diseases.* **19**(2): 385-387.

Laceky, T. 1995. ARCO disputes report: Fungus didn't kill pit geese, lab tests say. Associate Press report in The Montana Standard, November 25, 1995.

Jensen, L.S. and D.V. Maurice, 1978. Effect of methionine on copper-induced growth depression and gizzard erosion. *Poul. Sci.* **57**:1530-1532.

Maest, A.S., J.J. Metesh, R.J. Brand, and G. Abdo. 1995. Butte Groundwater Injury Assessment Report - Clark Fork River Basin NPL Sites, Montana. Prepared by RCG/Hagler Bailly for State of Montana Natural Resource Damage Litigation Program. January.

MBMG. 1995. Fax from Montana Bureau of Mines and Geology to DEQ, U.S. EPA, ARCO, U.S. FWS, and NRDP presenting preliminary Berkeley Pit water quality results from 11/15/95 sampling event. November.

NIOSH. 1994. NIOSH Pocket Guide to Chemical Hazards. DHHS (NIOSH) Publication No. 94-116. Prepared by U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health. June.

Nordstrom, D.K. 1977. Hydrogeochemical and Microbiological Factors Affecting the Heavy Metal Chemistry of an Acid Mine Drainage System. A Dissertation Submitted to the Department of Applied Earth Sciences and the Committee on Graduate Studies of Stanford University in Partial Fulfillment of the Requirements for the Degree of Doctor of Philosophy. March.

Poupoulis, C. And L.S. Jensen. 1976. Effect of high dietary copper on gizzard integrity of the chick. *Poul. Sci.* **55**:113-121.

Rowe, R. D. W. D. Shaw, and W.D. Schulze. 1992. "Nestucca Oil Spill." In *Natural Resource Damages: Law and Economics.* K.M. Ward and J.W. Duffield (eds). John Wiley and Sons, Inc. New York City, NY. pp. 527-554.

Rowe, R. D. and L. Bird, 1991. *Revealed Values in the Pacific Northwest for Preventing Seabird Mortality Due to Oiling.* Prepared for Environment Canada and British Columbia Ministry of Environment by RCG/Hagler Bailly, Inc. November, 1991.

Stevens, T.H., J. Echeverria, R.J. Glass, T. Hagar, and T.A. More. 1991. Measuring the Existence Value of Wildlife: What Do CVM Estimates Really Show? *Land Economics* 67(4):390-400.

Scanlan, C.M. 1988. *Introduction to Veterinary Bacteriology.* Ames, IA: Iowa State University Press. 457 pp.

The Montana Standard. 1995. Dead geese toll reaches 340. November 21, 1995.

APPENDIX A

UNIVERSITY OF WYOMING

Wyoming State Veterinary Laboratory
Department of Veterinary Sciences
College of Agriculture

1174 Snowy Range Road
Laramie, WY 82070
Phone: (307) 742-6638
Fax: (307) 721-2051

December 6, 1995

Ms. Candace West
Montana Department of Justice
1310 East Lockey
Old Livestock Building
Helena, Montana 59620

Dear Ms. West,

Enclosed are the results of our examination of three intact and two partial snow goose carcasses (received 20 November, 1995; EPA form 8-17195, Project number 177, project name NRDA, Wyoming State Veterinary Laboratory accession numbers 95A10078 A-E). In summary, the intact carcasses and one head showed similar changes of extensive damage to the oral cavity, esophagus, and trachea (see enclosed photographs). The tissues of the birds were exposed by direct contact with a golden-yellow liquid which was on the feathers, exposed skin surfaces, digestive and respiratory tracts. Additional gross and/or microscopic damage was detected in lung, kidney, skeletal muscle, and intestine of some birds. The cause of death of these birds was a combination of the effect of corrosive liquid on tissues, alteration of pH in the bodies of the birds, and the effect of elevated metals in the tissues (manganese, zinc, copper, iron, and cadmium). The copper levels in the tissues are consistent with acutely toxic levels in other species of birds. The levels of iron and manganese in a few of the snow geese are also in the toxic range for domestic birds.

The birds were judged to be in good body condition as determined by weight and the amount of subcutaneous and visceral fat. We performed bacterial cultures on some of the birds to rule out involvement of a bacterial pathogen and did not detect any significant bacterial infections that could have resulted in death of the birds. By gross and microscopic examination we were able to rule out aspergillosis (a fungal infection that occasionally can cause death of birds).

If you have any questions about the results or their interpretation, please feel free to give us a call.

Sincerely,

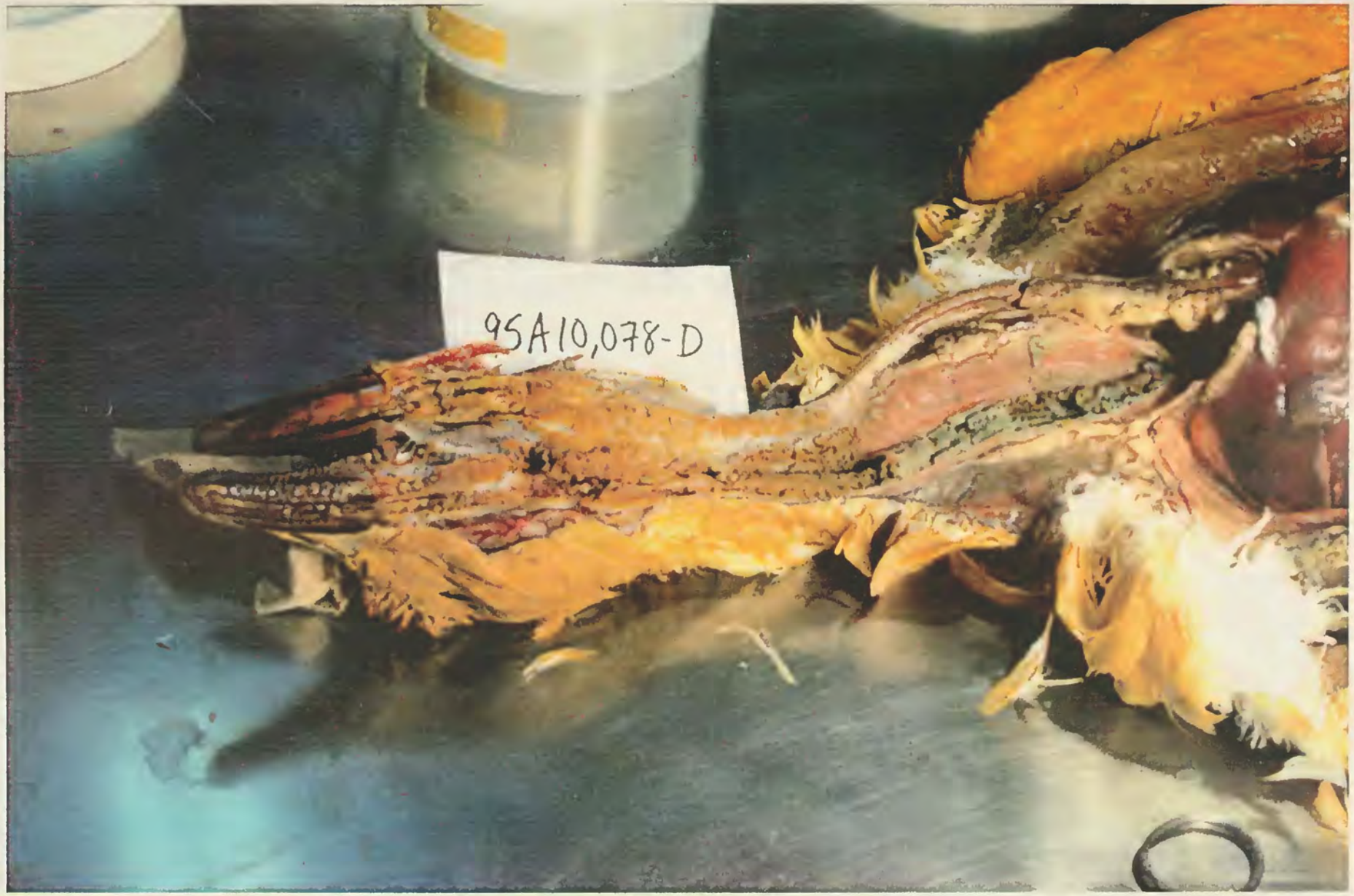
Elizabeth S. Williams, DVM, PhD, diplomate ACVP
Veterinary Pathologist

Meri Raisbeck, DVM, PhD, diplomate ABVT
Veterinary Toxicologist

DEC 07 1995

The University of Wyoming is an equal opportunity/affirmative action institution.

Excellence UW


95A10,078-D


95A10,078-B


95A19078-C

** WYOMING STATE VETERINARY LABORATORY **

UNIVERSITY OF WYOMING
1174 SNOWY RANGE ROAD, LARAMIE, WYOMING 82070
(307) 742-6638, WATS 1-800-442-8331

ACCESSION NUMBER: 95 A 10078

SAMPLE RECEIVED: 11/20/95
COUNTY: OS STATE: MT
OWNER:

VETERINARIAN:

WILLIAMS,BETH
1174 SNOWY RANGE ROAD
LARAMIE, WY 82070

MONTANA DEPT JUSTICE

SPECIES: GOOSE - NOS SEX: AGE: 0 0 ANIMAL ID:
SPECIMEN SUBMITTED: CARCASS

ANALYSIS COMPLETED	NUMBER DONE	FEE
NECROPSY	5	$ 0.00
HISTOPATHOLOGY	50	$ 0.00
BACTERIOLOGY	5	$ 0.00

RESULTS:

CARCASS
ASPIRATION
UNDETERMINED
SKIN
DERMATITIS
NECROTIZING INFLAMMATION
SKELETAL MUSCLE
MYOPATHY
DEGENERATION
CYST
MUSCLE
MYOSITIS, NOS(T-13001) (T-14...)
DEGENERATION
TRACHEA
TRACHEITIS (T-25000)
NECROTIZING INFLAMMATION
LUNG
PULMONARY EDEMA (T-28000)
HEMORRHAGE
ESCHERICHIA COLI
STREPTOCOCCUS, ALPHA HEMOLYTIC
NO MICROBIAL GROWTH
HEART
NO MICROBIAL GROWTH
ORAL MUCOUS MEMBRANE
STOMATITIS
ESOPHAGUS
ESOPHAGITIS
CECUM
TYPHLITIS
INFLAMMATION, ACUTE

KIDNEY
NEPHROSIS
INFLAMMATION, ACUTE

DIAGNOSIS:

WE WILL BE CLOSED FOR THE HOLIDAYS ON DECEMBER 25, 26 & JANUARY 1 & 2.

FINAL REPORT DATE: 11/30/95 SIGNED:_____, DIRECTOR.

WYOMING STATE VETERINARY LABORATORY

University of Wyoming
1174 Snowy Range Road
Laramie, WY 82070

PATHOLOGY REPORT

Acc #: 95 A 10,078 **November 27, 1995**

SPECIES: Snow Goose - A

VETERINARIAN: Williams

OWNER: Montana Department of Justice

GROSS FINDINGS:

This carcass is composed only of bones and feathers including wings and left leg. The head, neck, musculature of the body and internal organs are gone. The feathers are covered with yellow liquid. Only a portion of the left lung is present. The pH of the skin of the foot is 4.0 and the pH of the lung is 5.5-6.0.

MICROSCOPIC FINDINGS: HE stain

Sections of a small portion of lung are congested but otherwise appear microscopically normal.

FINAL DIAGNOSIS: Scavenged

COMMENTS: The cause of death is undetermined. The discoloration of the feathers indicates this bird was in contact with the same yellow fluid as the other birds in this group.

Reported by: E. L. Williams

WYOMING STATE VETERINARY LABORATORY

University of Wyoming
1174 Snowy Range Road
Laramie, WY 82070

PATHOLOGY REPORT

Acc #: 95 A 10,078 **November 27, 1995**

SPECIES: Snow goose - B

VETERINARIAN: Williams

OWNER: Montana Department of Justice

GROSS FINDINGS:

This adult male snow goose is in good body condition (2.7 kg). The feathers are discolored with yellow-brown liquid (pH 3.0). The pH of the oral cavity is 4.5. The skin ventral to the nostrils is dry and flakes easily from the underlying bill. A large plug of mucoid material is in the choana. The subcutaneous tissues surrounding the esophagus are edematous and hyperemic. The epithelium of the tongue is discolored yellow-brown and cracks when bent. The esophageal mucosa from the larynx to the middle of the neck is covered with mucoid necrotic grey material. The mucosa of the trachea is necrotic and covered by a thin fibrinonecrotic membrane. The lung is edematous, clear frothy fluid flows from the cut surfaces (pH 6.0), and most of the parenchyma is discolored grey. A small amount of grey fluid is in the abdominal, thoracic, and cervical air sacs. Vessels of the epicardium are congested and there is a slight increased amount of clear pericardial fluid (pH 6.5-7.0). The ventriculus contains gravel but no solid ingesta. There is little ingesta in the intestinal tract and the ceca are small. A single pale tan necrotic plug is present in the lumen and attached to the wall in one cecum. Green feces are in the cloaca. Other organs appear grossly normal.

MICROSCOPIC FINDINGS: HE stain, PAS, GMS stains

Skin of the face is characterized by superficial necrosis with golden discoloration of the external layer. Sections of the larynx and adjacent oral mucosa are characterized by severe necrosis of the epithelium with a sharp dividing line between the necrotic tissue and more normal epithelium. The epithelium of the esophagus is similar with marked excess mucus production and sloughing of the superficial layers of the epithelium. A layer of necrotic debris and epithelial cells is present. There is focal ballooning degeneration of the superficial layers of the epithelium. Vessels in the submucosa are congested and there is

moderate hemorrhage and edema. The esophageal lumen contains sloughed epithelial cells, unidentifiable yellow-gold material, and amphophilic debris. There is increased mucus production in the proventriculus with an accumulation of sloughed debris in the lumen. Cells in the lining of the ventriculus are swollen and vacuolated and there is focal hemorrhage. Yellow-gold material is present on the superficial layers. Sections of small intestine are moderately autolytic but the lumens contain sloughed debris and yellow-golden material. A few coccidial developmental forms are found in the intestine without inflammatory reaction. A section of cecum shows marked transmural necrosis with an accumulation of bacteria and unidentifiable debris in the lumen. There is a mixed inflammatory cell response and focal thrombosis of vessels in the wall. Yellow-golden material is in the lumen. The PAS and GMS stains contain a small amount of positive debris but no obvious fungi.

Sections of the trachea are characterized by extensive necrosis of the mucosa, hemorrhage, and vascular congestion. A few sections of skeletal muscle adjacent to the trachea show focal degeneration with a light infiltration of mononuclear inflammatory cells. Vessels in sections of an airsac are congested and eosinophilic debris is present on the epithelial surface. Vessels in the lung are extremely congested and one vessel contains cross sections of an unidentified parasite. Some airways contain eosinophilic debris. A few sections of fat are necrotic and highly eosinophilic.

The kidney contains a few dilated tubules which contain eosinophilic liquid and some mineralized material. A few tubules contain fragmented epithelial cells with pyknotic nuclei. A section of skeletal muscle contains a protozoal cyst.

Sections of thyroid gland, parathyroid gland, ganglion, brain, spleen, liver, and testicle are essentially normal.

FINAL DIAGNOSIS: Severe acute necrotizing stomatitis and esophagitis
Severe acute necrotizing tracheitis
Pulmonary edema
Acute necrotizing dermatitis
Acute mild tubular nephrosis
Aspiration
Mild degenerative myositis, trachea
Acute focal typhlitis
Parasitism: Protozoal cyst, skeletal muscle
Helminth, vessel
Coccidia, small intestine

COMMENTS: The cause of death appears to be due to contact with and aspiration and ingestion of toxic fluids. The lesions on the skin and in the oral cavity and trachea are consistent with contact with caustic materials. The low pH of the fluids could have contributed to severe physiologic imbalance (acid-base balance) and the high concentrations of heavy metals could have had damaged

kidney and other organs.

Reported by: Elizabeth S. Williams

WYOMING STATE VETERINARY LABORATORY

University of Wyoming
1174 Snowy Range Road
Laramie, WY 82070

PATHOLOGY REPORT

Acc #: 95 A 10,078-C **November 27, 1995**

SPECIES: Snow goose

VETERINARIAN: Williams

OWNER: Montana Department of Justice

GROSS FINDINGS:

This adult male snow goose is in good body condition (2.6 kg) based on the amount of subcutaneous and visceral fat. The feathers are discolored with yellow fluid. There is necrosis of the epithelium of the oral cavity which is brittle and yellow-brown in color. Necrotic debris and excessive mucus are on the mucosa of the esophagus. The ventriculus contains only gravel and the pH is 5.0-5.5. The tracheal mucosa is necrotic and the lumen contains a large clot of red-brown bloody material. The abdominal air sacs are discolored grey and the pH is 4.0-4.5. Seven ribs on the left side of the bird are fractured and there is hemorrhage in the associated lung, in the subcutis, and beneath the keel. The contents of the cloaca are mucoid. Other organs appear grossly normal.

MICROSCOPIC FINDINGS: HE stain

The lesions in this bird are similar to those observed in bird B. Sections of the tongue and oral cavity are characterized by superficial necrosis of the epithelium which is discolored golden. There is focal ballooning degeneration of the epithelium with foci of intercellular edema. Increased mucus exudation is present. Necrotic and unidentifiable debris is found in the lumen. Similar changes are found in the esophagus; in some areas the mucosa is hypereosinophilic. A superficial layer of debris and abundant mucus exudation are present in the lumen of the proventriculus. Splitting of the lining of the ventriculus occurs with lifting of the superficial portions. Necrosis occurs within a few glands. Sections of small intestine are moderately autolytic with focal necrosis of some villi. Debris is present in some intestinal crypts and yellow-golden material is found in the lumen. Some necrosis and eosinophilic debris occurs on the serosal surface.

The trachea is characterized by severe necrosis of the mucosa. Yellow foreign material admixed with eosinophilic granular material is on the necrotic membrane that remains. There are degenerative changes in the skeletal muscle adjacent to the trachea. Eosinophilic debris and yellow-gold material are found in airways of the lung, as well as marked vascular congestion and acute necrosis.

A few tubules in the kidney contain granular eosinophilic casts. Sections of pectoral muscle are degenerate with a mixed inflammatory cell infiltrate and focal mineralization. Sections of heart, spleen, liver, colon, testicle, and brain are essentially normal.

FINAL DIAGNOSIS: Severe acute necrotizing stomatitis and esophagitis
Severe acute necrotizing tracheitis
Pulmonary edema
Subacute degenerative myopathy, pectoral muscle
Trauma, fractured ribs
Acute pulmonary hemorrhage
Aspiration
Mild degenerative myositis, trachea
Mild acute tubular nephrosis

COMMENTS: The cause of death appears to be due to contact with and aspiration and ingestion of toxic fluids. The degenerative changes in the pectoral muscle and the trauma that resulted in fractured ribs and pulmonary hemorrhage are likely secondary to intoxication. The lesions on the skin and in the oral cavity and trachea are consistent with contact with caustic materials. The low pH of the fluids could have contributed to severe physiologic imbalance (acid-base balance) and the high concentrations of heavy metals could have had damaged kidney and other organs.

Reported by: E. Thomas Williams

WYOMING STATE VETERINARY LABORATORY

University of Wyoming
1174 Snowy Range Road
Laramie, WY 82070

PATHOLOGY REPORT

Acc #: 95 A 10,078 **November 27, 1995**

SPECIES: Snow goose - D

VETERINARIAN: Williams

OWNER: Montana Department of Justice

GROSS FINDINGS:

This adult female snow goose (2.4 kg)is in good body condition. Necrosis is present in the epithelium of the oral cavity including the tongue and larynx and the tissues are brittle and yellow-brown. The esophagus contains a large fibrinonecrotic plug which extends the entire length. The pH of the material in the esophagus is 4.0 - 4.5. Necrosis occurs in the tracheal mucosa and the lungs are very edematous; foamy clear fluid flows from cut surfaces. The abdominal and cervical air sacs are discolored grey (pH is 5.0). There is a slight increase in pericardial fluid which is pH 5.0. Petechial hemorrhages occur in the serosa of the intestines. There is essentially no ingesta in the digestive tract. Irregular pale foci are in the pectoral muscles. Other organs appear grossly normal.

MICROSCOPIC FINDINGS: HE stain

The lesions are similar to those in goose B and C. Sections of skin from the face and the foot have a layer of yellow foreign material on the surface with focal ulceration and little associated inflammatory cell reaction. A discrete line of necrosis separates viable from necrotic tissue in a few foci. The tongue, larynx, and esophagus are similar and all are covered with a yellow-gold discoloration of the superficial epithelium. Foci of necrosis, sloughing of superficial epithelium, and hemorrhage are present. A few foci of ulceration are infiltrated by a mixed inflammatory cell infiltrate. Increased amounts of mucus exudation are present. Necrosis of some cells within crypts, along with vacuolation of cells and a mixed inflammatory cell infiltration are present in the ventriculus. A few cross sections of small nematodes are in the muscularis of the ventriculus. The small intestine is autolytic but contains yellow debris in the lumen.

Sections of the trachea are characterized by necrosis and

hemorrhage of the mucosa. Yellow-gold foreign material and eosinophilic granular debris are on the surface of the trachea. Hemorrhage occurs in the muscularis and a mild mixture of mononuclear cells and macrophages are found beneath the mucosa. Vessels in the lung are extremely congested and eosinophilic debris is in airways and air sacs.

There is moderate degeneration, characterized by eosinophilia, swelling, and fragmentation, with little inflammatory cell infiltration in the pectoral muscle. Mineralized debris occurs in the lumens of some renal tubules and some tubular epithelium is vacuolated. A few granular eosinophilic casts are found in the tubular lumens.

Sections of heart, adrenal gland, proventriculus, spleen, liver, and brain are essentially normal.

FINAL DIAGNOSIS: Severe acute necrotizing stomatitis and esophagitis
Severe acute necrotizing tracheitis
Pulmonary edema
Acute necrotizing dermatitis
Subacute degenerative myopathy, pectoral muscle
Mild acute tubular nephrosis

COMMENTS: The cause of death appears to be due to contact with and aspiration and ingestion of toxic fluids. The degenerative changes in the pectoral muscle are likely secondary. The lesions on the skin and in the oral cavity and trachea are consistent with contact with caustic materials. The low pH of the fluids could have contributed to severe physiologic imbalance (acid-base balance) and the high concentrations of heavy metals could have had damaged kidney and other organs.

Reported by: Ephie D Williams

WYOMING STATE VETERINARY LABORATORY

University of Wyoming
1174 Snowy Range Road
Laramie, WY 82070

PATHOLOGY REPORT

Acc #: 95 A 10,078 **November 28, 1995**

SPECIES: Snow goose - E

VETERINARIAN: Williams

OWNER: Montana Department of Justice

GROSS FINDINGS:

Presented is only a head from a snow goose. The feathers are covered with yellow liquid. Necrosis of the skin ventral to the nares is present. Necrotic debris and abundant mucus is present in the oral cavity and larynx. The pH of the esophagus is 4.0 and necrotic debris and mucus are on the surface. A small amount of grain is admixed in this material. The mucosa of the trachea is necrotic and covered with a thin fibrinous membrane.

MICROSCOPIC FINDINGS: HE stain

Sections of the oral cavity and esophagus are characterized by superficial necrosis with a layer of yellow-gold material, debris, and mucus on the surface. Mucus glands are prominent and producing abundant mucus. There is severe necrosis of the tracheal mucosa with blood clots in the lumen. The periesophageal/peritracheal tissues show marked vascular congestion and edema. Focal superficial necrosis, with ballooning degeneration, and ulceration occur in sections of skin from the face.

FINAL DIAGNOSIS:
Severe acute necrotizing stomatitis and esophagitis
Severe acute necrotizing tracheitis
Focal necrotizing dermatitis

COMMENTS: The lesions in this bird are similar to those observed in the other birds. The cause of death is probably the same.

Reported by: Esther Williams

WYOMING STATE VETERINARY LABORATORY
1174 Snowy Range Road

UNIVERSITY OF WYOMING
LARAMIE, WY 82070

MICROBIOLOGY REPORT

Veterinarian Williams Owner Montana Accession# A-10078
Date 11-23-95 Species Goose ID/Number:

Specimen:	Results:*	Comments
Lung D+A	4	
Lung B	11	
Lung C	13	
Heart Swab B	13	

*See Number Code Below — ORGANISMS ISOLATED HAVE THE MORPHOLOGICAL, BIOCHEMICAL, AND/OR SEROLOGICAL CHARACTERISTICS OF:

1. ☐ Actinomyces	6. ☐ Pasteurella	11. ☒ Streptococcus -alpha
2. ☐ Campylobacter	7. ☐ Pseudemonas sp.	12. ☐ No significant isolates
3. ☐ Corynebacterium	8. ☐ Salmonella sp.	13. ☒ No bacterial isolates
4. ☒ Escherichia coli	9. ☐ Staphylococcus (coag+)	14. ☐ Mixed contaminants only
5. ☐ Klebsiella sp.	10. ☐ Staphylococcus (coag-)	15. ☐

SENSITIVITY RESULTS

	A	B		A	B
Amikacin			Novobiocin		
Ampicillin/Amoxicillin			Penicillin		
Augmentin/Clavomox			Polymyxin B		
Ceftiofur			Streptomycin		
Cephalothin			Sulfa + Trimethoprim		
Enrofloxacin			Sulfadiazine		
Erythromycin			Tetracycline		
Furacin/Nitrofurantoin			Tilmicosin		
Gentamycin			Triple Sulfa		
Lincomycin					
Methacillin					
Neomycin					

Comments

T-28000, T-28000, E15901
T-28000, E-25029
T-28000, E10003
T-32000, E10003

Report by Amy Boerger-Fields

WYOMING STATE VETERINARY LABORATORY

1174 Snowy Range Road
Laramie, Wyoming 82070
307-742-6638

TOXICOLOGY SECTION

NOV 26,1995

ACCESSION #: 10078-95

VETERINARIAN

Williams, Beth
1190 Jackson
Laramie, WY

OWNER

Montana Department of Justice

SAMPLE	*DATE REC'D*	*ANALYSIS*	*RESULTS*
Liver B	NOV 21,1995	ICP	
		Lead	None detected
		Iron	32 ppm
		Mercury	None detected
		Arsenic	None detected
		Molybdenum	0.8 ppm
		Zinc	151 ppm
		Copper	115 ppm
		Cadmium	1.9 ppm
		Barium	None detected
		Beryllium	None detected
		Cobalt	None detected
		Chromium	None detected
		Nickle	None detected
		Thallium	None detected
		Vanadium	None detected
Liver C	NOV 21,1995	ICP	
		Lead	None detected
		Mercury	None detected
		Arsenic	None detected
		Barium	None detected
		Beryllium	None detected
		Chromium	None detected
		Thallium	None detected
		Vanadium	None detected
		Manganese	40.2 ppm
		Iron	334 ppm
		Molybdenum	0.5 ppm
		Zinc	126 ppm
		Copper	54 ppm
		Cadmium	1.5 ppm
		Cobalt	0.3 ppm
		Nickle	0.3 ppm
Liver D	NOV 21,1995	ICP	
		Lead	None detected
		Mercury	None detected

		Arsenic	None detected
		Beryllium	None detected
		Chromium	None detected
		Vanadium	None detected
		Manganese	74.4 ppm
		Iron	1200 ppm
		Molybdenum	0.7 ppm
		Zinc	216 ppm
		Copper	174 ppm
		Cadmium	2.4 ppm
		Barium	0.2 ppm
		Cobalt	0.5 ppm
		Nickle	0.8 ppm
Kidney	NOV 21,1995	ICP	
		Lead	None detected
		Mercury	None detected
		Arsenic	None detected
		Beryllium	None detected
		Cobalt	None detected
		Chromium	None detected
		Nickle	None detected
		Thallium	None detected
		Vanadium	None detected
		Manganese	54.3 ppm
		Iron	126 ppm
		Molybdenum	0.8 ppm
		Zinc	171 ppm
		Copper	13.2 ppm
		Cadmium	1.4 ppm
		Barium	1.0 ppm
Kidney C	NOV 21,1995	ICP	
		Lead	None detected
		Mercury	None detected
		Arsenic	None detected
		Beryllium	None detected
		Cobalt	None detected
		Chromium	None detected
		Thallium	None detected
		Vanadium	None detected
		Manganese	135 ppm
		Iron	144 ppm
		Molybdenum	0.5 ppm
		Zinc	119 ppm
		Copper	29.1 ppm
		Cadmium	2.6 ppm
		Barium	6.5 ppm
		Nickle	1.1 ppm
Kidney D	NOV 21,1995	Lead	None detected
		Mercury	None detected
		Arsenic	None detected
		Beryllium	None detected
		Cobalt	None detected
		Chromium	None detected
		Nickle	None detected
		Thallium	None detected
		Vanadium	None detected
		Manganese	29.8 ppm

		Iron	176 ppm
		Molybdenum	0.6 ppm
		Zinc	205 ppm
		Copper	37.6 ppm
		Cadmium	3.7 ppm
		Barium	0.6 ppm
Brain B	NOV 21,1995	Cholinesterase	
Brain C	NOV 21,1995	Cholinesterase	
Brain D	NOV 21,1995	Cholinesterase	
Brain E	NOV 21,1995	Cholinesterase	
Esophagus B	NOV 21,1995		
Esophagus C	NOV 21,1995		
Esophagus D	NOV 21,1995		
Feathers B	NOV 21,1995		
Feathers C	NOV 21,1995		
Feathers D	NOV 21,1995		
Feathers E	NOV 21,1995		
Guts B	NOV 21,1995		
Guts C	NOV 21,1995		
Guts D	NOV 21,1995		
Lung C	NOV 21,1995		
Lung D	NOV 21,1995		
Esophageal content E	NOV 21,1995		

COMMENTS

As I indicated on Friday, all of the liver samples are high in Mn, Zn, Cu and Cd. Livers B and D are high in Fe. The kidneys are high in Mn, Zn, and Cu. The Cu concentrations are in the range described as "acute toxic" in domestic birds. Individual Fe and Mn results are also indicative of intoxication in domestic species. None of the results however, in itself, accounts for the necrotic lesions in the upper GI tract, although I believe that they may account for the renal damage you mentioned. According to the Bureau of Mines employee I talked to, the bulk of the acidity in the water results from pyritic (S) ores and is some variation of sulfuric acid. In such a matrix many of these elements, especially Cu and Zn, are severely astringent and would probably potentiate the corrosive effects of the acid.

Merl F. Raisbeck, DVM, PhD
Veterinary Toxicologist

Samples will be retained for 30 days from the date of this report. If you wish to have them returned, please contact us as soon as possible.

APPENDIX B

STATE OF MONTANA

DEPARTMENT OF LIVESTOCK-DIAGNOSTIC LABORATORY DIVISION

BOX 997 — BOZEMAN, MONTANA 59771 — PHONE (406) 994-4885

FINAL LABORATORY REPORT

SUBMITTEE	OWNER	DATE	CASE NO
Neil Andersen 1400 S. 19th Bozeman, MT 59715	MDFWP	12-14-95	7-71 7-72 7-73 7-74

7-71 GROSS: The carcass is of a male snow goose in poor to fair postmortem and thin nutritional state. There is moderate loss of pectoral muscle mass. The subcutaneous tissue has scant amount of fat and there are moderate amounts of visceral fat stores. The mucosa of the esophagus, trachea and larynx is covered by a yellow/brown moist membrane which easily scrapes away. The underlying surface is brown. The lungs are heavy, wet, and red. The liver is pale. The crop is empty except for a small amount of semifluid material and an invertebrate body (maggot or aquatic invertebrate). The proventriculus is empty and the ventriculus contains small amounts of grit. The small and large intestine contain minimal amounts of ingesta. Multifocally, there are (less than 10) discreet reddened slightly depressed regions on the feet and non-feathered portions of the leg. The feathers are bright yellow in extensive regions of the body and the corneas of the eyes are yellow and dry.

HISTOPATHOLOGY:

Multiple sections of lung, crop, esophagus and trachea and sections of small and large intestine with attached mesentery, skin, brain, skeletal muscle, heart, ventriculus, liver, larynx, eye and presumptive airsac are examined. Autolysis hampers accurate histologic assessment.

The connective tissue of the airsac is smudged and eosinophilic and is of low cellularity. Focally, the airsac surfaces are covered by pyknotic nuclei (many of which have morphology similar to the erythrocytic nuclei), a hypereosinophilic amorphous material, a basophilic granular material, and a dull brown/gold granular to amorphous material). Within the brown/gold material birefringent debris is present under polarized light.

The intestinal lumens contain sloughed epithelial cells, variable amounts of mucinous material, basophilic granular material and rarely plant fibers.

Focally, within the superficial tunica muscularis of the ventriculus, there are several nematode eggs surrounded by moderate numbers of leukocytes.

The tracheal mucosal surface is covered by a crystalline brown/gold material and a basophilic granular material. The epithelium is predominantly absent, however, few degenerate epithelial cells are still attached to the mucosal surface.

CONTINUED

Lab Fee: _____ Signature _____

STATE OF MONTANA

DEPARTMENT OF LIVESTOCK-DIAGNOSTIC LABORATORY DIVISION

BOX 997 — BOZEMAN, MONTANA 59771 — PHONE (406) 994-4885

FINAL LABORATORY REPORT

SUBMITTEE	Neil Anderson Page 2	OWNER	MDFWP	DATE	12-14-95	CASE NO	7-71 7-72 7-73 7-74

The mucosa of a large parabronchus within the lung is covered by a granular to homogenous basophilic material. The epithelial cells are intact, multifocally, beneath this debris. The submucosal blood vessels are prominent and filled with erythrocytes.

Particulate debris as described within the lumen of the parabronchus and trachea occurs on the surface of the lung.

Small amounts of debris as described in the tracheal lumen occurs on the epithelial surface of the esophagus and crop. The submucosal glands of these structures are ectatic and filled with a mucinous secretion.

Small amounts of debris as described within the trachea occur on the surface of the skin.

The structure presumed to be the larynx is denuded of epithelium and the epithelial surface is covered by mucinous debris containing pyknotic nuclei and heterophils. Deeper glandular lumens contain variable amounts of mucinous secretion, pyknotic nuclei and necrotic cells. The epithelial lining of these structures is absent.

The corneal and conjunctival surfaces are covered by basophilic homogenous to granular material and brown/gold granular crystalline material. The skeletal muscle myofibers between the conjunctival mucosa and the epidermis are basophilic and granular (mineralization).

MORPHOLOGIC DIAGNOSIS:
Extraneous pigment deposition, mucosa of trachea, parabronchi, esophagus, crop, and surfaces of airsac, lung, feathers/skin and eye
Laryngitis, heterophilic, moderate, acute, focal
Pulmonary fluid, severe
Endoparasitism, nematode ova, focal, with mild chronic ventriculitis, tunica muscularis of ventriculus
Autolysis, moderate

Lab Fee: CONTINUED Signature 7-71
7-72
7-73
7-74

STATE OF MONTANA

DEPARTMENT OF LIVESTOCK-DIAGNOSTIC LABORATORY DIVISION

BOX 997 — BOZEMAN, MONTANA 59771 — PHONE (406) 994-4885

FINAL LABORATORY REPORT

SUBMITTEE		OWNER		DATE		CASE NO	
	Neil Anderson Page 3		MDFWP		12-14-95		7-71 7-72 7-73 7-74

7-72 GROSS: The carcass is of a female snow goose in good nutritional and poor postmortem state. In multifocally extensive regions, the feathers are brown/yellow. Multifocally, discreet areas of the non-feathered portions of the leg and feet are dry and brown. The corneas are dry and yellow/brown. The laryngeal/tracheal/esophageal mucosa is covered by a dry yellow/brown granular membrane which easily scrapes away. The underlying tissue is grey/brown. The lungs are diffusely wet. The surfaces adjacent to the airsacs are green/brown and on cut surface the lung is red. The proventriculus and ventriculus have green surfaces. The ventriculus contains small amounts of grit. The crop and ventriculus are empty. The duodenum and cecum contain a red watery fluid. The colon has green watery contents.

HISTOPATHOLOGY:

Multiple sections of esophagus, trachea, lung, and crop and sections of proventriculus, brain, kidney, oviduct, uterus, skeletal muscle, heart, intestine, ovary, adrenal gland, liver, skin and pancreas are examined.

The proventriculus mucosal surfaces, multifocally, are covered by a basophilic granular material. Near the deeper recesses of this debris, cells have dark basophilic granular cytoplasm (presumptive mineralized epithelial cells). The mucosal stroma is collapsed in areas where few glandular mucosal structures are present.

The pulmonary vasculature is engorged with erythrocytes, multifocally. The epithelium of the bronchi is absent.

The esophageal and crop mucosa is covered by abundant amounts of mucinous material which in turn is covered by hypereosinophilic layers of smudged keratin. Small amounts of a brown/golden granular pigment also occurs on the surface.

The tracheal mucosa is covered by brown/gold granular crystalline material. The epithelium is absent.

In a section of skin, a focally extensive region of the epidermis is absent. The adjacent intact epidermis is basophilic and smudged. The deeper collagenous stroma is dull red and smudged.

Lab Fee: CONTINUED Signature

7-71
7-72
7-73
7-74

STATE OF MONTANA

DEPARTMENT OF LIVESTOCK-DIAGNOSTIC LABORATORY DIVISION

BOX 997 — BOZEMAN, MONTANA 59771 — PHONE (406) 994-4885

FINAL LABORATORY REPORT

SUBMITTEE	OWNER	DATE	CASE NO
Neil Anderson Page 4	MDFWP	12-14-95	7-71 7-72 7-73 7-74

The pancreas is autolyzed which precludes histologic assessment.

The duodenum contains variable amounts of basophilic granular material and fragmented villi.

MORPHOLOGIC DIAGNOSIS: Extraneous pigment deposition, mucosa of larynx, trachea, esophagus, crop, proventriculus, feathers/skin, and eye
Ulceration, skin
Pulmonary fluid, severe
Autolysis, moderate, body as a whole; severe pancreas

7-73 GROSS: The carcass is of a female snow goose in poor to fair postmortem and good nutritional state. In multifocally extensive regions the feathers are brown/yellow. The corneas are brown/yellow and dry. The larynx, esophagus, and tracheal mucosa are diffusely covered by a dry granular brown material that easily scrapes away from the underlying surface. The underlying surface is dull brown. Portions of the ventral lung have brown surfaces adjacent to the airsacs and is dark red on cross-section and are firm. The remainder of the lung is red and wet. The crop and proventriculus are empty. The ventriculus contains small amounts of grit. The small intestine, cecum and large intestine contain small amounts of grey/brown watery material. The fat stores are abundant.

HISTOPATHOLOGY:

Multiple sections of lung, trachea, esophagus and crop and sections of intestine, brain, kidney, liver, skeletal muscle, proventriculus, spleen, heart, and skin are examined.

The esophageal and crop mucosa are covered by a basophilic granular to linear material and a brown/golden granular to crystalline material. The underlying squamous epithelium is elevated from the underlying submucosal surface. The submucosal glands are filled with abundant amounts of mucin.

The proventriculus surface is covered by basophilic granular material and degenerate cells.

STATE OF MONTANA

DEPARTMENT OF LIVESTOCK-DIAGNOSTIC LABORATORY DIVISION

BOX 997 — BOZEMAN, MONTANA 59771 — PHONE (406) 994-4885

FINAL LABORATORY REPORT

SUBMITTEE		OWNER		DATE		CASE NO
Neil Andersen Page 5		MDFWP		12-14-95		7-71 7-72 7-73 7-74

The tracheal mucosa is absent and the surface is covered by a basophilic granular material and a brown/gold granular to crystalline material.

In a focally extensive region of the skin, the epidermis is absent and the underlying tissue is covered with a gold crystalline material. Marked mineralization of dermal collagen is present.

Gomori's iron stain of trachea, esophagus and lung revealed the mucosal pigment to stain positive for iron.

MORPHOLOGIC DIAGNOSIS: Extraneous pigment deposition with iron, mucosa of larynx, trachea, esophagus, crop, feathers/skin, and eye
Ulceration, skin
Pulmonary fluid, severe
Autolysis, moderate

7-74 GROSS: The carcass is of a male snow goose in poor postmortem and fair nutritional state. In multifocally extensive regions, the feathers are yellow/brown. The corneas are brown/yellow and dry. The esophagus, trachea and laryngeal mucosal surfaces are covered by a brown dull granular material which easily scrapes away. The underlying stroma is brown. The crop is empty. The proventriculus is empty. The ventriculus contains small amounts of grit. The lung are wet, heavy and red. The liver is pale. Multifocally, there are brown discreet regions on the non-feathered portions of the legs and feet.

HISTOPATHOLOGY:

Multiple sections of crop, esophagus, trachea and lung and sections of spleen, brain, liver, heart, proventriculus, skeletal muscle, intestine, eye, and ventriculus are examined.

The pulmonary vasculature is congested. The parabronchi epithelium is absent.

There is a focal region of degeneration in the cornified material of the mucosa of the ventriculus.

CONTINUED

Lab Fee:_____ Signature_____

7-71
7-72
7-73
7-74

STATE OF MONTANA

DEPARTMENT OF LIVESTOCK-DIAGNOSTIC LABORATORY DIVISION

BOX 997 — BOZEMAN, MONTANA 59771 — PHONE (406) 994-4885

FINAL LABORATORY REPORT

SUBMITTEE	OWNER	DATE	CASE NO
Neil Andersen Page 6	MDFWP	12-14-95	7-71 7-72 7-73 7-74

The tracheal epithelium is absent and the surface is covered by a gold/brown granular to crystalline material and basophilic granular material. The esophageal and tracheal mucosa are covered by small amounts of a basophilic material, mineralized laminations of keratin and mucin. The submucosal glands are filled with abundant amounts of mucin.

The conjunctival epithelium and corneal epithelium are covered by basophilic granular material. The deeper collagenous stroma of the cornea is hypereosinophilic.

There is marked autolysis of the intestinal mucosa.

Gomori's iron stain of trachea, lung, crop, and esophagus revealed the mucosal pigment to stain positive for iron.

MORPHOLOGIC DIAGNOSIS:
Extraneous pigment deposition with iron, mucosa of larynx, trachea, crop, feathers/skin, esophagus, and eye
Pulmonary fluid, severe
Degeneration, focal, moderate, cornified layers of mucosa, ventriculus
Autolysis, moderate

Bacteriology results of case 7-71, 7-72, and 7-74 are enclosed. The parasitology results of case 7-72 are enclosed with bacteriology report of 7-72.

A copy of the trace mineral analyses performed at Michigan State University Diagnostic Laboratory of liver and kidney from cases 7-72 and 7-74 are enclosed.

COMMENT: Neither an infections nor a significant inflammatory response was present in any of the four snow geese examined to account for their deaths. A consistent finding was the deposition of an extraneous material/pigment on the surfaces of the feathers and mucosal surfaces of the crop, esophagus, trachea, larynx and eyes of all the birds. The material was also present on the airsac surfaces and mucosal surface of the proventriculus in one bird. This material stained positive for iron with Gomori's iron stain. The mucosa itself was mostly intact in the crop and esophagus. Predominantly, the mucosa was absent in the airways. Lacking

Lab Fee: CONTINUED Signature 7-71 7-72 7-73 7-74

STATE OF MONTANA

DEPARTMENT OF LIVESTOCK-DIAGNOSTIC LABORATORY DIVISION

BOX 997 — BOZEMAN, MONTANA 59771 — PHONE (406) 994-4885

FINAL LABORATORY REPORT

SUBMITTEE		OWNER		DATE		CASE NO	
	Neil Andersen Page 7		MDFWP		12-14-95		7-71 7-72 7-73 7-74

in these organs (except in a focal region of the larynx of one bird) was an inflammatory cell response as well as rafts of necrotic cells. This indicates to me either, these birds died peracutely thus not enough time had elapsed to mount a minimal inflammatory cell response or the changes are a postmortem event secondary to autolysis and passive movement of the reservoir water into the luminal structures and lung.

Trace mineral analyses of the liver and kidney of the two birds tested revealed (as per interpreted by Dr. Michael Slanker, toxicologist, Michigan State University) elevated liver and kidney levels of magnesium (7-72, 7-74), kidney zinc (7-74) and toxic liver and kidney levels of manganese (7-72, 7-74). Unfortunately, there is a considerable lack of scientific studies in geese to derive well-defined tissue values (for high and toxic ranges) and with some elements, even their toxic effects. For instance, both birds have what is interpreted to be toxic levels of manganese, however, the only manifestation of manganese toxicosis that I have reference to is reported in turkeys and these birds only had growth retardation.

Hepatic and kidney zinc levels are elevated, however, interpretation is difficult. The reference Mineral Levels in Animal Health by R. Puls reports high levels in the liver to be 90 - 300 ppm wet weight, where as toxic levels are defined 200 - 1900 ppm. Kidney levels are: High -- 60 - 120 ppm; toxic -- 300 - 800 ppm. The values of these birds are in the region of overlap, thus making interpretation difficult. However, there are reports of subacute to chronic zinc toxicosis in ducks where the tissue levels were in the 200 range.

Kidney copper levels are very high, especially when I compare these values to sheep with copper toxicosis. However, toxic range for kidney copper is poorly defined in geese and lesions commonly seen in mammalian species were not present in these birds.

CONTINUED

Lab Fee:_____ Signature_____ 7-71
7-72
7-73
7-74

STATE OF MONTANA

DEPARTMENT OF LIVESTOCK-DIAGNOSTIC LABORATORY DIVISION

BOX 997 — BOZEMAN, MONTANA 59771 — PHONE (406) 994-4885

FINAL LABORATORY REPORT

SUBMITTEE	OWNER	DATE	CASE NO
Neil Andersen Page 8	MDFWP	12-14-95	7-71 7-72 7-73 7-74

The toxicologic tests performed are inconclusive, in part due to a lack of definitive scientific knowledge. The history is highly suggestive of a toxicity causing the death of this large number of birds found. It must be remembered that we only tested for what is present in the water based on water sampling. We don't know: the interaction of these elements in geese; the effects of the pH of the water; what forms of certain elements are present in the reservoir; the changing dynamics of the reservoir ecosystem; whether there are "hot spots" present; whether there are other toxins present that were not tested for; the movement of these animals and what they had come in contact with elsewhere; the effects of the high total dissolvable solids in the water which may have been present at the time of death; and I am sure there can be many others questions which could be posed. Based on the tests performed, my literature review and from conversations with other colleagues, I am unable to absolutely determine the cause of death of these birds.

Lab Fee: $220.00

Signature _____ 7-71

A. W. Layton, DVM, DACVP 7-72

bak 7-73

OWNER 7-74

FORM SV-51 20M (10-81)

STATE OF MONTANA - DEPARTMENT OF LIVESTOCK

DIAGNOSTIC LABORATORY DIVISION-BACTERIOLOGY

P.O. BOX 997 • BOZEMAN, MONTANA 59771 • PHONE (406) 994-4885

Diagnostic Lab No.	7-71	Tech	s	Species:	Avian	Age:	Sex:
				Breed:			
Date Out:	11/24/95	Date Received:	11/20/95	Name/No.	*		

Veterinarian:	Owner:
Neil Anderson FW & P 1400 South 19th Bozeman MT 59715	FW & P MSU Bozeman

Specific attempts to isolate Salmonella sp from the large intestine were negative.

Cultures prepared from the large intestine did not result in any bacterial growth.

Cultures prepared from the lung did not result in any bacterial growth.

Cultures prepared from the sinus swab did not result in any bacterial growth.

Cultures prepared from the lung did not result in any fungal growth.

No bacterial presence was noted on a gram stain prepared from direct smears of lung tissue. (1)

Pathologist: Dr. Layton

Charge 16.00 Diagnostic Lab No. 7-71

STATE OF MONTANA – DEPARTMENT OF LIVESTOCK

DIAGNOSTIC LABORATORY DIVISION-BACTERIOLOGY

P.O. BOX 997 • BOZEMAN, MONTANA 59771 • PHONE (406) 994-4885

Diagnostic Lab No.	7-72	Tech S	Species: Avian	Age:	Sex:
			Breed:		
Date Out: 11/24/95	Date Received: 11/20/95		Name/No. *		

Veterinarian:	Owner:
Neil Anderson FW & P 1400 South 19th Bozeman MT 59715	FW & P MSU Bozeman

Cultures prepared from the intestinal content resulted in:
Isolate : Escherichia coli
Isolate : Hafnia alvei

1+ to 4+ = rare colony to confluent growth
P = pure culture, M = mixed culture

Specific attempts to isolate Salmonella sp from the intestinal content were negative.

Cultures prepared from the lung did not result in any bacterial growth.

Examination of the fecal sample by the flotation method revealed no parasite ova. (1)

Pathologist: Dr. Layton

Charge 10.50 Diagnostic Lab No. 7-72

STATE OF MONTANA - DEPARTMENT OF LIVESTOCK

DIAGNOSTIC LABORATORY DIVISION-BACTERIOLOGY

P.O. BOX 997 • BOZEMAN, MONTANA 59771 • PHONE (406) 994-4885

Diagnostic Lab No.	7-74	Tech	S	Species:	Avian	Age:	Sex:
				Breed:			
Date Out: 11/24/95	Date Received: 11/20/95			Name/No.	*		

Veterinarian:

Neil Anderson FW & P
1400 South 19th
Bozeman MT 59715

Owner:

FW & P MSU

Bozeman

Cultures prepared from the intestinal content resulted in:
Isolate : Escherichia coli

1+ to 4+ = rare colony to confluent growth
P = pure culture, M = mixed culture

Specific attempts to isolate Salmonella sp from the intestinal content were negative.

Cultures prepared from the lung did not result in any bacterial growth.

Cultures prepared from the lung did not result in any fungal growth.

No bacterial presence was noted on a gram stain prepared from direct smears of lung tissue. (1)

Pathologist: Dr. Layton

Charge 13.50 Diagnostic Lab No. 7-74

REPORT OF LABORATORY EXAMINATION

ANIMAL HEALTH DIAGNOSTIC LABORATORY
P.O. Box 30076
Lansing, MI, 48909
Phone (517) 355-0281

TOXICOLOGY

Case Number: 1619906
Reported : 11/30/95
Received : 11/28/95
Case Origin: COURIERS (OTHER)

PRIVILEGED INFORMATION
NOT FOR PUBLICATION

Client Account: 15559

LAYTON, A. W.
MONTANA DEPT OF LIVESTOCK
DIAGNOSTIC LABORATORY DIVISION
BOX 997
BOZEMAN MT 59771

Phone: 406-994-4885

Owner:

MONTANA FISH WILDLIFE & PARKS

HISTORY : There has been 100% die-off of Snow geese. The birds were found in a mining pit reservoir. There has been minimal histologic lesions, none of which are suggestive of an infectious process. The crop, proventriciulous and ventriculus were empty except for a maggot found in 1 bird. The feathers were wet and discolored yellow/ brown. The esophagus and trachea mucosa were covered by a brown soft granular material. Complete history on file at the AHDL.

Name 1: 7-72 Age: ADULT
Breed: SNOW GOOSE Sex: UNDETERMINED

SPECIMEN: LIVER

Test: TISSUE MINERAL ANALYSIS (values in ppm)

Ba	0.061	Ca	321	Cu	64.7	Co	0.598
Fe	1050	Mg	360	Mn	79.0	Mo	0.500
P	3270	Zn	281	Sb	<1.00	As	<0.500
Cr	<0.200	Cd	1.81	Hg	<2.00	Pb	<0.500
Se	<2.00	Tl	<2.50	Na	667	K	2290
B	<1.00						

SPECIMEN: KIDNEY

Test: TISSUE MINERAL ANALYSIS (values in ppm)

Ba	0.330	Ca	321	Cu	44.8	Co	0.447
Fe	309	Mg	354	Mn	69.2	Mo	0.643
P	2680	Zn	215	Sb	<1.00	As	<0.500
Cr	<0.200	Cd	1.87	Hg	<2.00	Pb	<0.500
Se	<2.00	Tl	<2.50	Na	739	K	2500
B	<1.00						

MSU IS AN AFFIRMATIVE ACTION/EQUAL OPPORTUNITY INSTITUTION

Case Number: 1619906

Animal Comment:
The liver and kidney magnesium is greater than the expected 200 ppm or less. The liver and kidney manganese are in the toxic range (greater than 9 ppm).

Name 2: 7-74 Age: ADULT
Breed: SNOW GOOSE Sex: UNDETERMINED

SPECIMEN: LIVER

Test: TISSUE MINERAL ANALYSIS (values in ppm)

Ba	<0.050	Ca	179	Cu	136	Co	0.195
Fe	845	Mg	284	Mn	36.1	Mo	0.407
P	2760	Zn	238	Sb	<1.00	As	<0.500
Cr	<0.200	Cd	2.36	Hg	<2.00	Pb	<0.500
Se	<2.00	Tl	<2.50	Na	952	K	2860
B	<1.00						

SPECIMEN: KIDNEY

Test: TISSUE MINERAL ANALYSIS (values in ppm)

Ba	<0.050	Ca	171	Cu	183	Co	0.170
Fe	831	Mg	280	Mn	32.3	Mo	0.508
P	3070	Zn	246	Sb	<1.00	As	<0.500
Cr	<0.200	Cd	2.54	Hg	<2.00	Pb	<0.500
Se	<2.00	Tl	<2.50	Na	866	K	2600
B	<1.00						

Animal Comment:
Refer to animal #1 comments for magnesium and manganese. The kidney zinc concentrations are higher than the expected 120 ppm or less.

COMMENTS:
Other element concentrations are within expected ranges.

Michael R. Slanker 11/30/95
Clinical Toxicologist (517) 353-5275

APPENDIX C

Colorado State University

Diagnostic Laboratories
College of Veterinary Medicine
and Biomedical Sciences
Fort Collins, Colorado 80523-1644
(970) 491-1281
FAX: (970) 491-0320

January 9, 1996

Dr. Bill Stubblefield
ENSR
4413 West LaPorte Avenue
Fort Collins, CO 80521

Dear Bill:

Snow geese submitted by you from a die-off that occurred in Montana associated with a copper mine tailing pond have been necropsied. All six snow geese had similar lesions, however, the first bird necropsied did have a concurrent severe acute Aspergillosis infection of the lungs. Primary lesions found in these birds included varying degrees of ulceration and erosion of the upper digestive system from the esophagus to the ventriculus. This lesion may have been caused by ingestion of the acidic mineralized water in the lake. Birds also had elevated levels of copper, manganese, and zinc within the liver and kidneys. This too may have been acquired through ingestion of this water. The acidic nature of the water could have facilitated absorption of these heavy metals. If these birds did drink this acidic water, it could have caused an acidotic state within the bird. The

renal necrosis may have been associated with the kidneys trying to eliminate the excessive number of ions in the blood, especially copper, zinc, and manganese. The tubular necrosis may have also been associated with trying to eliminate excessive hydrogen ions secondary to the suspected acidosis.

One possible scenario to the cause of death in these birds would be that the birds were migrating south and had been flying for a day or two. They could have been mildly dehydrated. They landed on the lake and drank the acid mineralized water. The acidity of the water could have caused the lesions in the upper digestive system and the minor lesions on the feet. The birds could have absorbed an excessive amount of copper, manganese, zinc, and probably H+ ions. The body tried to compensate for the acidotic state and the excessive mineral levels by elimination of these elements via excretion from the kidneys. This then could have led to the renal tubular necrosis.

Terry R. Spraker, DVM/PhD

Colorado State University

Diagnostic Laboratories
College of Veterinary Medicine
and Biomedical Sciences
Fort Collins, Colorado 80523-1644
(970) 491-1281
FAX: (970) 491-0320

January 11, 1996

Dr. Bill Stubblefield
ENSR
4413 West LaPorte Avenue
Fort Collins, CO 80521

Dear Bill:

Snow geese submitted by you from a die-off that occurred in Montana associated with a copper mine tailing pond have been necropsied. All six snow geese had similar lesions, however, the first bird necropsied did have a concurrent severe acute Aspergillosis infection of the lungs. Primary lesions found in these birds included varying degrees of ulceration and erosion of the upper digestive system from the esophagus to the ventriculus. This lesion may have been caused by ingestion of the acidic mineralized water in the lake. Birds also had elevated levels of copper, manganese, and zinc within the liver and kidneys. This too may have been acquired through ingestion of this water. The acidic nature of the water could have facilitated absorption of these heavy metals. If these birds did drink this acidic water, it could have caused an acidotic state within the bird. The

renal necrosis may have been associated with the kidneys trying to eliminate the excessive number of ions in the blood, especially copper, zinc, and manganese. The tubular necrosis may have also been associated with trying to eliminate excessive hydrogen ions secondary to the suspected acidosis.

One possible scenario to the cause of death in these birds would be that the birds were migrating south and had been flying for a day or two. They could have been mildly dehydrated. They landed on the lake and drank the acid mineralized (possibly hypertonic) water. The acidity of the water could have caused the lesions in the upper digestive system and the minor lesions on the feet. The birds could have absorbed an excessive amount of copper, manganese, zinc, and probably H+ ions. The body tried to compensate for the acidotic state and the excessive mineral levels by elimination of these elements via excretion from the kidneys. This then could have led to the renal tubular necrosis.

Terry R. Spraker, DVM/PhD

Colorado State University

Diagnostic Laboratories
College of Veterinary Medicine
and Biomedical Sciences
Fort Collins, Colorado 80523-1644
(970) 491-1281
FAX: (970) 491-0320

January 23, 1996

Dr. Bill Stubblefield
ENSR
4413 West LaPorte Avenue
Fort Collins, CO 80521

Dear Bill,

This letter is to follow up my initial data transmittal to you on 11 January 1996. The raw data that was given to you was in the form of two reports (956-12251 and 956-13011). The remarks and letter that I wrote dated 11 January 1996 was my preliminary interpretation of the data that was available at that time. Subsequent to the initial report, additional data and information has been received that may alter my preliminary interpretation. Because the additional information may change my preliminary interpretation, I would be hesitant to form a final interpretation on the cause of death in these snow geese until we complete the Berkeley Pit (BMF-1) water ingestion trial with snow geese and additional analysis of the Berkeley Pit (BMF-1) water.

Terry R. Spraker, DVM/PhD

RCO

Environmental Affairs - Legal
555 Seventeenth Street
Denver, Colorado 80202
Telephone 303 293 4520
Facsimile 303 293 4295

Richard O. Curley, Jr.
Senior Attorney

January 26, 1996

VIA FEDERAL EXPRESS

Robert G. Collins, Esq.
Assistant Attorney General
Montana Department of Justice
Natural Resource Damage Litigation Program
Old Livestock Bldg.
1310 E. Lockey Ave.
P. O. Box 201425
Helena, MT 59620-1425

Re: Montana v. ARCO; Production of Recreation Computer Diskettes and Goose Documents

Dear Mr. Collins:

I enclose five (5) computer diskettes containing the non-fishing survey recreation data collected by Triangle Economic Research ("TER"). This letter is also to confirm that ARCO previously produced to the State all relevant documents created by TER relating to the data contained on these diskettes.

I also enclose a copy of documents generated by the Colorado Veterinary Diagnostic Laboratory of the College of Veterinary Medicine and Biomedical Sciences of Colorado State University relating to the geese found in the Berkeley Pit. If you have any questions about these materials, please contact me.

Very truly yours,

Richard O. Curley, Jr.

ROC/dl

c: w/o Enclosures
S. H. Foster, Esq./Holland & Hart
K. M. Ward, Esq./Harding & Ogborn

c:\docs\roc\outcorr\colins24.doc

JAN 29 1996

COLORADO VETERINARY DIAGNOSTIC LABORATORY
College of Veterinary Medicine and Biomedical Sciences
Colorado State University, Fort Collins, CO 80523
Phone 303-491-1281
Fax 303-491-0320

DL#: 956-12251
Date: 11-16-95

Vet/Clinic: NA
Owner: Bill Stubblefield
Animal ID: NA **Date Specimen Taken:** NA
Species: Avian **Breed:** Snow Geese **Age:** NA **Sex:** NA

History: These are two snow geese that were brought in by Bill Stubblefield for necropsy. These two birds are of approximately 250-300 snow geese that were found dead on a pond near a large copper mine.

Tissue Submitted: Two birds

GROSS NECROPSY: **Bird A:** Integument: The feathers of this bird have multiple areas of yellow brownish staining. This staining is covering the body, head and neck. The skin of the feet of this bird also have this brown yellowish discoloration. There are three small areas of blackish discoloration on the surface of the skin over the toes of this bird. There is an abundance of subcutaneous adipose tissue in this bird.

Cardiovascular system: The heart is normal in size, shape and color. There is an abundance of fat around the coronary groove.

Respiratory system: The trachea is normal. The lungs are severely edematous. There is a line of demarcation between the dorsal and ventral aspects of the lung. The dorsal aspects of the lung are relatively red whereas the ventral aspects of the lungs have a grey pale discoloration. There are also numerous white focal areas disseminated throughout the ventral posterior aspects of the lung. In this region the lung is extremely firm.

Digestive system: The mucosa of the mouth has a yellowish brownish discoloration. The epithelium of the esophagus has sloughed and in some areas the esophageal material appears to be slightly darkened. There are a few small ulcers or erosions at the proventricular/ventricular junction. The proventriculus and ventriculus are both empty and the ventriculus contains only a small amount of gravel. The small and large intestines appear to be relatively normal. However, very little food content is present in these organs. There is an abundance of adipose tissue within the abdominal cavity.

Lymphohematopoietic system: The spleen is small and normal in size, shape and color.

Urogenital system: This bird is a male and the testes are extremely small. The kidneys are slightly pale and have an abundance of fat covering the surface of them.

Musculoskeletal system: The muscle of this bird is normal.

Endocrine system: The thyroid glands are normal in size, shape and color.

Brain: The brain appears to be relatively autolyzed but otherwise no lesions are noted.

Special senses: The eyes appear to be dry. There is a small amount of yellowish discoloration on the surface of the cornea and conjunctiva of the eye.

Bird B: Integument: There are many focal areas especially on the ventral aspect of the bird, head and neck in which the feathers are discolored yellow brown. The feet are also discolored with this slightly yellowish brownish discoloration. There are several very small dark areas on the surface of the skin. In these areas, however the surface of the skin appears to be intact. There is an abundance of subcutaneous adipose tissue in this bird.

Heart: The heart is normal in size, shape and color.

Respiratory system: The trachea is relatively clean. Both lungs bilaterally are severely edematous. The ventral aspects of both lungs have a brownish gray discoloration. Air sacs are clean.

Digestive system: The mouth and tongue are covered with a slight grayish brownish discoloration. Some of the epithelium of the esophagus appears to be sloughed. There are a few ulcers at the proventricular/ventricular junction. The proventriculus and ventriculus are empty except for a small amount of grit and sand. The small and large intestines appear to be relatively normal. There is an abundance of adipose tissue throughout the mesenteries.

Lymphohematopoietic system: Spleen is of normal size and shape.

Urogenital system: The ovaries are small and appear to be immature. The kidneys are slightly pale and covered with adipose tissue.

Musculoskeletal system: The muscles are of normal mass and color.

Endocrine system: Thyroid glands are of normal size and shape.

Brain: The brain is normal grossly.

Special senses: The eyes appear to be slightly shrunken and have a small amount of this yellowish discoloration to the conjunctiva and cornea.

HISTOPATHOLOGY: Bird A: Lung: There are multifocal areas of consolidation throughout sections of lung. There are numerous thin septate branching hyphae present within these areas of consolidation and necrosis. There is a moderate degree of inflammation associated with these fungi. The inflammation is primarily mononuclear cells but not multinucleated giant cells. Special stains reveal fungal elements throughout these necrotic sections. The special stains do suggest that this is a fairly homogeneous population of fungus suggestive of aspergillosis. In areas of the lung that are adjacent to air sacs there is evidence of edema and a washed out appearance to the erythrocytes. This lesion is minimal in sections from this lung.

Heart: No significant lesions.

Proventriculus: The proventricular glands appear washed out and are not taking the hematoxylin stain. All of the cells appear to be eosinophilic. There is no evidence of inflammation in any of the proventricular glands. Some of the erythrocytes and some of the vessels within the proventriculus are also washed out and have this pale color. The nuclei have extremely minimal degree of staining. The junction between the proventriculus and esophagus which is covered by mucus secreting epithelial cells are relatively normal.

Liver: Hepatocytes are mildly diffusely swollen. The liver is moderately congested. There is a slightly increased number of lymphocytes and plasma cells within the adventitia of hepatic portal triad regions. In a few areas some of the smaller bile ducts are inspissated with bile.

Skeletal muscle: No significant lesions.

Esophagus: Two sections of esophagus are examined. The first being in the mid region and the second just above the proventriculus. The glands of the esophagus both within the mid region and near the proventriculus appear to be dilated. The esophagus in the mid region, however does show numerous cystic areas in the superficial layers of the squamous epithelial layer of the esophagus. These look like small blisters filled with a yellow non-staining material. There is some degree of sloughing of the esophageal epithelium however there is no reaction or inflam-

mation within the esophagus. The mucus glands within the esophagus appear to be markedly dilated in this region also.

Kidneys: There are a few tubules in which the tubular epithelium has undergone necrosis. The degree of tubular necrosis in this case is extremely mild. There is also an extremely minimal degree of basophilia within tubules that may be early areas of mineralization.

Small intestines: No significant lesions.

Ventriculus: The kaolin layer of this section of ventriculus appears to have undergone a coagulative-type of change and does appear altered. There are a few small areas of inflammation within the deeper layers of the kaolin layer. The kaolin layer does appear to be sloughing. The deeper glandular layer of the ventriculus is within normal limits.

Trachea: The tracheal epithelium is markedly disorganized and does show evidence of disruption and necrosis, and is covered with a yellow crystalline material.

Web of foot -- This section of web is characterized by having a relatively normal epidermal layer in thickness. The degree of keratin on the surface is within normal limits. There is an extensive amount of yellow-brown pigment on the surface of this section of skin which is probably some of the salts from the material from the pond from which these birds were found in. There is no evidence of inflammation within this section of tissue.

Soft palate -- The glandular layer within the soft palate is filled with mucus. The surface epithelium is somewhat separated but is mostly covered by this mucus layer. There is little to no inflammation within the soft palate. In other areas, the epithelial cells of the soft palate do show evidence of separation as if there is a mild to moderate degree of edema within the epithelial layer. In a few areas, there is an extremely small amount of inflammation within the superficial keratin layers of the soft palate.

Skin -- This section of skin is covered by an extremely thin layer of epidermis. The feather follicles are within normal limits.

Eyes -- Both eyes are examined. The corneas of both eyes have a dark or bluish discoloration to the outer corneal epithelium. One of the eyes also has an extensive degree of vacuolation and blister formation within the cornea. There is a smudged appearance to the cornea as if it has undergone a chemical

lesion. The epithelium of the conjunctiva is also disrupted and is covered with a brown granular material. The cells are sloughing and are deeply basophilic.

Bird B: Tissues from this bird have been frozen.

Lung: Alveolar capillaries are markedly congested in some areas and in other areas capillaries are empty of erythrocytes. There are numerous dust granulomas scattered throughout this section of lung. Fungal lesions are not found in the sections of lung from this particular bird.

Heart: No significant lesions.

Proventriculus: This section of tissue is taken at the junction of the proventriculus and the esophagus. In regard to the esophagus there does appear to be degeneration to the superficial squamous epithelial layer. Some of the glands of the esophagus appear to be slightly dilated. The superficial mucosal epithelial cells of the proventricular esophageal region have undergone necrosis and appear to be sloughing. There is a mild degree of inflammation within this region. This inflammation is primarily a few lymphocytes and a few heterophils. There is also a small amount of a yellow amorphous somewhat granular material found within the necrotic and dilated crypts in this region. There may also be early separation of the superficial layer of the squamous epithelial layer of the esophagus in this region.

Proventriculus: This section of proventriculus was taken at the proventricular/ventricular junction. The section of proventriculus including glands are similar to the first bird in that the cytoplasm appears to be washed out in this region, however the nuclei stain fairly well in these glandular structures. The surface epithelium of the section of ventriculus appears to have undergone acute degeneration and there is an extremely mild degree of inflammation in the deeper layers. However, this lesion is restricted primarily to the koalin layer and only extends to a minimal depth into the glandular layer of the ventriculus. There is also a small portion of the junctional area between the esophagus and the proventriculus which is covered by an abundance of a basophilic amorphous material on the surface suggesting necrotic or degenerated epithelial cells. There is only a mild degree of inflammation in this region.

Liver: No significant lesions.

Ovary: The ovary is small and immature.

Adrenal gland: No significant lesions.

Kidneys: A few of the tubules have undergone acute tubular necrosis. The tubules appear to be proximal and/or distal convoluted tubules. There is little to no inflammation around these necrotic tubules.

Esophagus: There is an extensive degree of separation within the outer layer of the esophagus. This appears to be due to edema within the stratum spongiosa layer. There may be early vesicle formation in this region, also.

Trachea -- Much of the tracheal epithelium has undergone a deep basophilic change. The epithelium is also markedly disorganized. There is little to no inflammation within the trachea, however, the epithelium does appear to have undergone an acute necrosis.

Proventriculus -- There appears to be a minimal degree of a basophilic discoloration or degeneration to a few areas within the kaolin layer of the ventriculus. The deeper glandular layer of the ventriculus and muscular layers are within normal limits.

Foot -- Two sections of foot are examined. The first section is from the distal part of the leg where a darkish area was observed grossly. This does appear to be an ulcerated area characterized by necrosis or burning of the epidermis, submucosa, and the deeper collagen layer of skin. This area is characterized by deep basophilia of the tissue with a slight loss of cellular detail. There is no inflammation in this region. The web of the toe is relatively free of any type of inflammatory reaction. Many of the vessels are filled with erythrocytes that are washed out and many of the cells appear to have lost some of their nuclear detail.

Skin -- This section of skin is from the body wall. This section does contain a large ulcerated area that is characterized by deep basophilic discoloration and loss of cellular detail. These areas are suggestive of burns.

Soft palate -- This section contains numerous large mucus crypts. Several of the crypts are infected and contain numerous inflammatory cells that are surrounded by an extensive number of lymphocytes and plasma cells. In some areas, the orifice of these tips appear to be slightly plugged with a yellow granular crystalloid-like material. This is the same material that has been seen in many parts of the body. This crystalloid material is probably condensation of some type of salt from the water. The epithelium in this area has undergone marked separation and early blister formation.

Ventriculus -- This second section of ventriculus is totally different from the first one described. This section has undergone extensive kaolin degeneration. The upper region of the glandular layer of the ventriculus is necrotic and has undergone a deep basophilic change and cells are markedly vacuolated. This is a relatively acute area of kaolin and glandular necrosis.

Small intestine -- This section has undergone advanced autolysis and is extremely difficult to interpret.

Eyes -- Both eyes are examined histologically. The corneas in these two eyes are relatively thin but show evidence of edema, especially in the posterior aspects of the cornea. The superficial corneal epithelium has undergone a deep dark basophilic change and there is extensive loss of cellular detail. The epithelium around the limbal junction is markedly vacuolated and has undergone this bluish discoloration. Many of the cells of the conjunctiva have lost their normal structure. The posterior aspect of the eye is within normal limits.

BACTERIOLOGY: Lung tissues from bird A revealed a heavy growth of two fungi, Aspergillus and Rhizopus. Pasteurella sp. were not isolated from the liver of either bird.

TOXICOLOGY: Attached.

DIAGNOSIS:

Bird A:

1. Pneumonia, multifocal, necrotizing, moderate lung associated with fungus typical of aspergillosis.
2. Nephrosis, necrotizing, tubular, mild, multifocal kidney.
3. Hepatocellular swelling, mild, diffuse, liver with bile inspissation, liver.
4. Blisters, mild, superficial, squamous epithelial layer, mid region, esophagus.
5. Washed out appearance/loss of basophilia, glandular epithelium, proventriculus.
6. Degeneration, diffuse, kaolin layer, ventriculus.
7. Acute necrosis, epithelial layer, trachea.
8. Excessive mucus production/degeneration, epithelial edema, soft palate.
9. Necrosis/cellular swelling, severe, diffuse, conjunctiva and cornea, eyes.

Bird B:

1. Necrosis, moderate, diffuse, superficial layers, esophagus, proventriculus and ventriculus.
2. Tubular necrosis, mild, diffuse, kidney.
3. Edema, diffuse, severe, lung.
4. Ulceration, skin, multifocal, skin of foot and skin of body.

5. Keratitis/conjunctivitis, acute, necrotizing, eyes, bilaterally.

Terry R. Spraker, DVM/PhD

Typed: 11/27/95 lm
11-18-95 ml
1-3-96 ea

COLORADO VETERINARY DIAGNOSTIC LABORATORIES
College of Veterinary Medicine and Biomedical Sciences
Colorado State University Fort Collins, CO 80523-1644
970-491-1281 FAX 970-491-0320

L A B O R A T O R Y R E S U L T S

OWNER: ENSR
VET: NONE
SPECIES: AVI
BREED: SNOW GEESE

DL Accession: 956-12251
Sample Received: 11/16/1995
Diagnostician: TRS

Specimen	Results	Comment Code
AEROBIC CULTURE		
LIV 0001	no significant growth	
LUNG 0001	no growth	
LIV 0002	no growth	
LUNG 0002	no growth	
FUNGAL CULTURE		
LUNG 0001	see comments	
LUNG 0002	see comments	
ARSENIC		
KID 0001	Dry Weight Analysis 3.37ppm	AS6
LIV 0001	Dry Weight Analysis 2.52ppm	AS6
KID 0002	Dry Weight Analysis 3.25ppm	AS6
LIV 0002	Dry Weight Analysis 2.56ppm	AS6
CADMIUM		
KID 0001	Wet Weight Analysis 2.2ppm	CD3
LIV 0001	Wet Weight Analysis 1.0ppm	CD2
KID 0002	Wet Weight Analysis 1.6ppm	CD3
LIV 0002	Wet Weight Analysis 0.9ppm	CD2
COPPER		
KID 0001	Dry Weight Analysis 155ppm	CuQ
LIV 0001	Dry Weight Analysis 678ppm	CuP
KID 0002	Dry Weight Analysis 69.5ppm	CuQ
LIV 0002	Dry Weight Analysis 151ppm	CuP
SOD NONSTAINED FEAT	Dry Weight Analysis 16.2ppm	
SOD STAINED FEATHER	Dry Weight Analysis 499ppm	
CYANIDE		
LUNG 0001	None Detected	
LUNG 0002	None Detected	
IRON		
KID 0001	Dry Weight Analysis 506ppm	IR9
LIV 0001	Dry Weight Analysis 427ppm	IR8
KID 0002	Dry Weight Analysis 424ppm	IR9
LIV 0002	Dry Weight Analysis 278ppm	IR8
SOD NONSTAINED FEAT	Dry Weight Analysis 117ppm	
SOD STAINED FEATHER	Dry Weight Analysis 10,410ppm	
LEAD		
KID 0001	Wet Weight Analysis 0.3ppm	LDE
LIV 0001	Wet Weight Analysis 0.4ppm	LDE
KID 0002	Wet Weight Analysis 0.4ppm	LDE
LIV 0002	Wet Weight Analysis 0.2ppm	LDE
MANGANESE		
KID 0001	Dry Weight Analysis 46.0ppm	MN4

LIV 0001	Dry Weight Analysis	97.5ppm	MN3
KID 0002	Dry Weight Analysis	248ppm	MN4
LIV 0002	Dry Weight Analysis	184ppm	MN3
SOD NONSTAINED FEAT	Dry Weight Analysis	5.22ppm	
SOD STAINED FEATHER	Dry Weight Analysis	307ppm	

MERCURY

KID 0001	Wet Weight Analysis	0.03ppm	ME6
LIV 0001	Wet Weight Analysis	0.02ppm	ME5
KID 0002	Wet Weight Analysis	0.03ppm	ME6
LIV 0002	Wet Weight Analysis	0.02ppm	ME6

SELENIUM

KID 0001	Dry Weight Analysis	2.40ppm	
LIV 0001	Dry Weight Analysis	2.74ppm	SER
KID 0002	Dry Weight Analysis	4.26ppm	
LIV 0002	Dry Weight Analysis	5.14ppm	SER

ZINC

KID 0001	Dry Weight Analysis	527ppm	ZIE
LIV 0001	Dry Weight Analysis	692ppm	ZID
KID 0002	Dry Weight Analysis	398ppm	ZIE
LIV 0002	Dry Weight Analysis	312ppm	ZID
SOD NONSTAINED FEAT	Dry Weight Analysis	46.2ppm	
SOD STAINED FEATHER	Dry Weight Analysis	705ppm	

FA TEST, CHLAMYDIA

LUNG 0001	Negative
SOD 0001	Negative
SPL 0001	Negative
LIV 0002	Negative
LUNG 0002	Negative
SPL 0002	Negative

REFERRAL LAB

MISC 0001	University of California*

COMMENT CODE	COMMENT NARRATIVE
AS6	ARSENIC Diagnostic level: Goose: Liver & Kidney (DW) - normal, < 2.0 ppm.
CD2	CADMIUM Diagnostic level: Goose: Liver (WW) - normal, < 0.6 ppm.
CD3	CADMIUM Diagnostic level: Goose: Kidney (WW) - normal, < 2.0 ppm.
CuP	COPPER Diagnostic level: Goose: Liver (DW) - normal, 24.0-120 ppm.
CuQ	COPPER Diagnostic level: Goose: Kidney (DW) - normal, 12.0-36.0 ppm.
IR8	IRON Diagnostic level: Goose: Liver (DW) - normal, 800-2,000 ppm.
IR9	IRON Diagnostic level: Goose: Kidney (DW) - normal, 600-1,400 ppm.
LDE	LEAD Diagnostic level: Goose: Liver & Kidney (WW) - normal, < 2.0 ppm.
ME5	MERCURY Diagnostic level: Canine: Liver & Kidney (WW) -

normal, < 0.1 ppm.

ME6 MERCURY Diagnostic level: Goose: Liver & Kidney (WW) - normal, < 0.1 ppm.

MN3 MANGANESE Diagnostic level: Goose: Liver (DW) - normal, 8-24 ppm.

MN4 MANGANESE Diagnostic level: Goose: Kidney (DW) - normal, 6-16 ppm.

SER SELENIUM Diagnostic level: Goose: Liver (DW) - 1.40-5.20 ppm.

ZID ZINC Diagnostic level: Goose: Liver (DW) - normal, 100-440 ppm.

ZIE ZINC Diagnostic level: Goose: Kidney (DW) - normal, 56.0-128 ppm.

SPECIAL NOTES

1 At least two fungal types isolated in heavy growth-Aspergillus sp and Rhizopus sp.

2 Please note that there is no published normal concentration of selenium in goose kidney available. Normal kidney selenium concentrations for poultry range from 2.00-4.80 ppm, dry weight basis.

3 *Referral results from the University of California: No organophosphate or organochlorine pesticides detected, see attatched sheets.

4 Please note that normal poultry feathers contain the following concentrations on a dry weight basis: Cu, 10-15 ppm; Fe, 80-180 ppm; Mn, 4-11 ppm; and Zn, 60-300 ppm.

5 No fungus isolated from tissues submitted on 11/18/95

FAX to :COLORADO STATE UNIVERSITY 970 491-0320

UNIVERSITY OF CALIFORNIA
VETERINARY DIAGNOSTIC
LABORATORY SYSTEM

P.O. Box 1770
Davis, CA 95617
(916) 752-8700

ACCESSION#:D9509308
District:
County: CO COLORADO
Case Coordinator: SNEWMAN

Submitter
COLORADO STATE UNIVERSITY
VETERINARY DIAGNOSTIC LAB
300 WEST DRAKE #E100
FORT COLLINS, CO 80523
(970) 491-1281

Owner:
NONE GIVEN
COMPLETE OWNER
INFORMATION NOT PROVIDED
SEE REFERENCE,

Agent or Collector:
Reference Number: 956-12251

Species: GAME BIRDS
Herd,Flock ID:
Date Taken: 11/16/95
Date Received: 12/06/95

4 Specimens submitted: 2 FAT, 2 BRAIN

FINAL REPORT Date Reported: 12/12/95

Electronically signed by:
Scott Newman, DVM

LABORATORY FINDINGS/DIAGNOSIS:
No organophosphate or organochlorine pesticides detected.

CLINICAL HISTORY:
No history given.

SPECIMEN SUMMARY:

Specimen Type	Breed	ID	Age	Sex	Qty
BRAIN	GOOSE	Multiple IDs			2
FAT	GOOSE	Multiple IDs			2

TOXICOLOGY:

The brain and fat samples contain none of the listed organophosphate or organochlorine pesticides in concentrations greater than the stated detection limits. MDL = method detection limit (lowest concentration detectable by our

ORGANOCHLORINE INSECTICIDES-EXTENDED SCREEN

BRAIN		Aldrin	BHC	Chlordane	p,p'DDD
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.25 ppm	.1 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		o,p'DDD	p,p'DDE	o,p'DDE	p,p'DDT
SPECIMEN.ID	MDL	.1 ppm	.1 ppm	.1 ppm	.1 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		o,p'DDT	Dicofol	Dieldrin	Endosulfan I
SPECIMEN.ID	MDL	.1 ppm	.1 ppm	.05 ppm	.05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		Endosulfan II	Endrin	Gamma Chlordane	HCB
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		Heptachlor	Heptachlor Epox	Lindane	Methoxychlor
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		Mirex	Toxaphene
SPECIMEN.ID	MDL	.05 ppm	2 ppm
956-12251A		Not Detected	Not Detected
956-12251B		Not Detected	Not Detected

FAT		Aldrin	BHC	Chlordane	p,p'DDD
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.25 ppm	.1 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

FAT		o,p'DDD	p,p'DDE	o,p'DDE	p,p'DDT
SPECIMEN.ID	MDL	.1 ppm	.1 ppm	.1 ppm	.1 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

FAT		o,p'DDT	Dicofol	Dieldrin	Endosulfan I
SPECIMEN.ID	MDL	.1 ppm	.1 ppm	.05 ppm	.05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

FAT		Endosulfan II	Endrin	Gamma Chlordane	HCB
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

FAT		Heptachlor	Heptachlor Epox	Lindane	Methoxychlor
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

FAT		Mirex	Toxaphene
SPECIMEN.ID	MDL	.05 ppm	2 ppm
956-12251A		Not Detected	Not Detected
956-12251B		Not Detected	Not Detected

ORGANOPHOSPHATE INSECTICIDES-EXTENDED SCREEN

BRAIN		ACEPHATE	AZINPHOS-METHYL	CARBOPHENOTHION	CHLORFENVINPHOS
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		CHLORPYRIFOS	COUMAPHOS	CROTOXYPHOS	CRUFORMATE
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		DDVP	DEF	DEMETON	DIAZINON
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		DICROTOPHOS	DIMETHOATE	DIOXATHION	DISULFOTON
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.2 ppm	.05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		EPN	ETHION	ETHOPROP	FENAMIPHOS
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		FENSULFOTHION	FENTHION	FONOPHOS	ISOFENPHOS
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		MALATHION	METHAMIDAPHOS	METHIDATHION	METHYL PARATHIO
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		MEVINPHOS	MONOCROTOPHOS	NALED	PARATHION
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		PHORATE	PHOSALONE	PHOSMET	PHOSPHAMIDON
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		PROPENOPHOS	PROPETAMPHOS	RONNEL	TERBUFOS
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		TETRACHLORVINPH	TRIAZOPHOS
SPECIMEN.ID	MDL	.05 ppm	.05 ppm
956-12251A		Not Detected	Not Detected
956-12251B		Not Detected	Not Detected

FAT		ACEPHATE	AZINPHOS-METHYL	CARBOPHENOTHION	CHLORFENVINPHOS
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

FAT		CHLORPYRIFOS	COUMAPHOS	CROTOXYPHOS	CRUFORMATE
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

FAT		DDVP	DEF	DEMETON	DIAZINON
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

FAT		DICROTOPHOS	DIMETHOATE	DIOXATHION	DISULFOTON
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.2 ppm	.05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

FAT		EPN	ETHION	ETHOPROP	FENAMIPHOS
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

FAT		FENSULFOTHION	FENTHION	FONOPHOS	ISOFENPHOS
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

FAT		MALATHION	METHAMIDAPHOS	METHIDATHION	METHYL PARATHIO
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

FAT		MEVINPHOS	MONOCROTOPHOS	NALED	PARATHION
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

FAT SPECIMEN.ID	MDL	PHORATE .05 ppm	PHOSALONE .05 ppm	PHOSMET .05 ppm	PHOSPHAMIDON .05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

FAT SPECIMEN.ID	MDL	PROFENOPHOS .05 ppm	PROPETAMPHOS .05 ppm	RONNEL .05 ppm	TERBUFOS .05 ppm
956-12251A		Not Detected	Not Detected	Not Detected	Not Detected
956-12251B		Not Detected	Not Detected	Not Detected	Not Detected

FAT SPECIMEN.ID	MDL	TETRACHLORVINPH .05 ppm	TRIAZOPHOS .05 ppm
956-12251A		Not Detected	Not Detected
956-12251B		Not Detected	Not Detected

COLORADO VETERINARY DIAGNOSTIC LABORATORY
College of Veterinary Medicine and Biomedical Sciences
Colorado State University, Fort Collins, CO 80523
Phone 303-491-1281
Fax 303-491-0320

DL#: 956-13011
Date: 11-29-95

Vet/Clinic: Bill Stubblefield/NA
Owner: NA
Animal ID: Geese **Date Specimen Taken:** NA
Species: Avian **Breed:** Geese **Age:** Yng **Sex:** M

History: These are four geese that were found on a copper mine in Montana.

NECROPSY: **Bird A:** Integument: Most of the feathers on the ventral aspect of the body, neck and head are covered with a yellowish discoloration. This yellowish discoloration is also prevalent on the feet and distal portion of the legs. There is an abundance of adipose tissue within the subcutaneous tissues.

Cardiovascular system: The heart is normal in size, shape and color.

Respiratory system: The trachea is normal. The lungs are extremely edematous and the ventral aspects of the lungs have a gray appearance.

Digestive system: The mucosal surface of the mouth has a light brownish, yellowish discoloration. The epithelium of the esophagus is sloughed in a few areas. There are a few erosions at the proventricular/ventricular junction. The kaolin layer of the ventriculus is yellow. The ventriculus and proventriculus are empty except for a small amount of grit. The rest of the digestive system appears to be relatively normal. There is an abundance of adipose tissue within the abdominal cavity.

Lymphohemopoietic system: The spleen is normal in size, shape and color.

Urogenital system: This is an immature male and the testes are normal and relatively small. The kidneys are slightly pale and are covered with an abundance of adipose tissue.

Musculoskeletal system: The muscle is of normal amount and color.

Endocrine system: Thyroid glands are normal in size, shape and color.

Nervous system: Brain is normal.

Special senses: The conjunctiva of the eyes have this yellowish discoloration that is probably the same material that is observed on the feathers.

Bird B: Integument: The feathers of the chest, abdomen, neck and head have a yellowish to light brownish discoloration. The conjunctiva of the eyes also contain this yellowish discoloration. The feet, especially the lower webs and metatarsal region, have a mild yellowish discoloration. There is an abundance of adipose tissue within the subcutaneous tissues of this bird.

Cardiovascular system: The heart is normal in size, shape and color.

Respiratory system: The trachea is within normal limits. The lungs are markedly edematous and the ventral half of the lungs have a grayish pale discoloration.

Digestive system: The mouth of this bird has a yellowish discoloration. The epithelium of the esophagus has sloughed. There are several erosions at the ventricular/proventricular junction. The ventricular kaolin layer is stained green. The proventriculus and ventriculus are empty except for grit. The remaining small and large intestines are relatively normal. There is an abundance of adipose tissue throughout the mesenteries. The spleen is normal in size, shape and color. The testes are small and considered normal. The kidneys are covered with a moderate amount of fat.

Musculoskeletal system: The muscle and bones are within normal limits.

Endocrine system: Thyroids are normal.

Nervous system: The brain is normal.

Bird C & D: The gross lesions in birds C & D are identical to the ones in A and B.

HISTOPATHOLOGY: Bird A/Slides 1-4/22-1, 22-2. Lungs -- The lungs are characterized by having a sharp line of demarcation between the dorsal aspect and the ventral aspect of the lungs. The dorsal aspects of the lungs appear to be relatively normal. The erythrocytes located within capillaries of the dorsal aspect of the lungs still contain a small to moderate amount of cytoplasm/hemoglobin, therefore, are stained relatively pink, whereas

the ventral aspects of the lungs are characterized by a total loss of eosinophilia of the erythrocytes. Peribronchi within this region are all open. Occasionally, there are small amounts of material that may have been aspirated into the lungs. In this region, there is a moderate degree of edema within some of the alveolar spaces. This edema was noted on the gross exam. In some of the peribronchi, there is unidentified foreign material. In some areas, the material has a gray-brownish color that is similar to some of the material that has been observed on the surface of the tissues of the birds.

Kidneys -- There is a moderate number of tubules that have undergone acute tubular necrosis within the kidney. There is little reaction associated with these areas of tubular necrosis. In other areas, the amount of tubular necrosis is more severe. The tubules that appear to be affected are the convoluted tubules. Occasionally, small areas of mineralization can be found.

Liver -- No significant lesions (NSL). The liver has undergone a moderate degree of autolysis and it has been frozen making it fairly difficult to interpret, however, there does appear to be inspissation of bile in some of the areas. This is characterized by small brownish granular-like material in between hepatocytes. It is difficult to tell if there is any necrosis within the liver due to the degree of autolysis.

Testes -- The testes are immature and within normal limits.

Intestines -- The intestines have undergone an extensive autolysis.

Heart -- There are several small foci of lymphocytes, plasma cells, and occasional multinucleated giant cell just under the epicardial surface. The myocardium is within normal limits. There is also a relatively large pyogranuloma within the myocardium. This pyogranuloma is filled with necrotic debris and is surrounded by multinucleated giant cells. The outer zone of this granuloma is composed primarily of lymphocytes, plasma cells, and macrophages. An acid-fast stain in this lesion in the heart did not reveal acid-fast organisms. A GMS stain also was negative for fungal agents. Whatever the cause of this pyogranuloma, the organism now appears to be absent.

Proventriculus -- The proventricular glands are all normal in shape and structure, however, all of the cells are deeply eosinophilic in color. There appears to be edema around the glands of the proventriculus. At least one of the glands is markedly dilated and contains parasites compatible in size and shape with tetrameres. At the proventricular junction, there is evidence of necrosis to the superficial mucosal layer. There is a mild accumulation of lymphocytes within this region, but these areas

are probably normal lymphoid aggregates. However, the lymphoid tissue in this region appears to have undergone necrosis.

Ventriculus -- The kaolin layer of the ventriculus has undergone extensive degeneration and separation and disorganization. The glandular layer of the ventriculus is relatively normal. There is one cross-section of a large nematode, probably capillary, at the kaolin glandular junction of this section of ventriculus.

Skin -- This section is within normal limits.

Bird B/Slides 5-8. Kidneys -- There are multifocal areas of tubular necrosis throughout the kidneys. In a few areas, mineralization of tubules is present. Occasionally, mild accumulations of inflammatory cells can be found within some of the interstitial tissues.

Liver -- The liver has undergone fairly extensive autolysis. No histological lesions are seen.

Spleen -- The lymphoid tissue is diffusely mildly depleted and the spleen has undergone autolysis.

Air sacs -- There is an extensive amount of a basophilic granular material on the surface of the air sacs. The identification of this substance is not determined but appears to be like mineralization.

Adrenal gland -- NSL.

Ovary -- The ovary appears to be immature and within normal limits.

Small intestines -- NSL, however, the intestines have undergone advanced autolysis.

Heart -- NSL.

Lungs -- The ventral half of the lungs are similarly affected as with other birds. In these areas, the erythrocytes all within capillaries are totally washed-out whereas the erythrocytes above this line that was observed on gross exam are within normal limits. The alveolar spaces are open and there is no evidence of inflammation within the ventral portion of these lungs. This line of demarcation was evident on the gross exam also. There is evidence of separation of the tissue of the lungs, especially around vessels, suggesting pulmonary edema. There was an extensive amount of pulmonary edema observed on the gross exam.

Soft palate -- There is an extensive amount of sloughed keratin on the surface of the soft palate. This is associated with sloughing of the superficial layer of epithelium of the soft palate. The thickness of the deeper layers of soft palate are within normal limits. The mucus glands of the soft palate are filled with mucus and appear to be relatively normal.

Proventriculus -- This section has undergone advanced autolysis and is extremely difficult to interpret.

Ventriculus -- The kaolin layer of the ventriculus is extremely pale and appears to have undergone degeneration. There are several areas in which there is moderate hemorrhage and accumulation of cellular debris within the deeper layers of the kaolin area at the kaolin glandular junction. These lesions appear to be relatively acute. There is another area in which there is a moderate accumulation of cellular debris in the deeper layers of the kaolin in this section of ventriculus. There is one dark basophilic line between the affected area of the kaolin and the more deeper unaffected area. The cause of this degree of basophilia or perhaps mineralization was not determined.

Small intestines -- Several sections are examined and all are within normal limits, however, these sections have undergone advanced autolysis.

Bird C/Slides 9-13. Kidneys -- There is a moderate number of tubules that have undergone severe tubular necrosis. There is little to no inflammation associated with these tubules. Convoluted tubules and some areas collecting duct tubules appear to be involved. The overall severity of this tubular necrosis is considered severe. Little to no inflammation is present within the interstitial tissues.

Spleen -- The lymphoid tissue of the spleen is severely depleted.

Small intestines -- These sections have undergone autolysis. No significant lesions are noted.

Liver -- Liver has undergone moderate autolysis. No significant lesions are observed in this section. Some of the cells of the liver have an unusual granular texture to the cytoplasm.

Skeletal muscle -- NSL.

Web of foot -- There is an accumulation of a non-staining debris on the surface of the epithelium of the foot. There is also one

small area of necrosis within the epithelium. This small plaque is a plaque of necrotic epithelial cells. There is no inflammation under this area of necrosis.

Aorta -- NSL.

Ovary -- The ovaries contain numerous small ova and are considered to be normal.

Adrenal glands -- NSL.

Proventriculus -- Some of the glands of the proventriculus are dilated and the proventricular esophageal junction is within normal limits. The esophagus has undergone autolysis but is otherwise within normal limits.

Heart -- NSL.

Lung -- The lesions in the lung in this bird are identical to birds A and B.

Bird D/Slides 14-17. Lungs -- There is an extensive amount of a basophilic material within some of the bronchi. This may be associated with aspiration of mineral material from the water. Several sections of lung also have this washed-out appearance. There is an extensive amount of edema throughout the sections of lung.

Liver -- There does appear to be an excessive amount of nuclear debris in sinusoids, some of which are probably not erythrocytic nuclei. There also appears to be an excessive amount of pigment within biliary canaliculi in a few areas in this section. There are also several areas in which there appears to be early hepatocellular necrosis. In these areas, the nuclei are pyknotic and the cytoplasm is more eosinophilic than what would be expected.

Kidneys -- A moderate to severe number of tubules have undergone acute tubular necrosis. The tubules involved appears to be primarily the convoluted tubules but occasionally collecting ducts can also be found that appear to be necrotic. Occasionally, small areas have a basophilic substance within the cytoplasm that appears to be early mineralization.

Skeletal muscle -- There appears to be a moderate amount of edema between the skeletal muscle bundles. There also appears to be an increased degree of separation between the muscle cells as if the muscle has undergone fairly extensive edema. On gross exam, some of the muscle did appear to be soft and this may be the reason for the slight soft texture to the muscle.

Heart -- NSL.

Ovaries -- The ovary is within normal limits. The ovary does contain a relatively normal number of ova.

Proventriculus -- This structure has undergone advanced autolysis but is within normal limits.

Esophagus -- There is an extensive degree of sloughing of the superficial epithelium of the esophagus. The deeper layers of epithelium are relatively normal and the mucus glands of the esophagus are dilated but relatively normal.

Ventriculus -- Two sections of ventriculus are examined. Both show early signs of degeneration of the kaolin layer but the glandular layers are within normal limits.

Skin -- The epidermis is of normal thickness. There is a moderate accumulation of a brown granular pigment, probably some type of salt on the surface of the skin, however, there is no evidence of inflammation in these regions. Feather follicles are within normal limits.

Small intestines -- Several sections are examined and all have undergone moderate to severe autolysis but no specific lesions are discernible.

Ventriculus -- Another section of ventriculus is examined and lesions are similar to what is described earlier.

TOXICOLOGY: Attached.

DIAGNOSIS:

Bird A.
1/Tubular necrosis, severe, diffuse, kidneys.
2/Pulmonary edema, severe, diffuse, lungs.
3/Epicarditis, chronic active, with large pyogranuloma.
4/Degeneration, severe, multifocal, kaolin layer, ventriculus.
5/Diffuse eosinophilia, severe, proventriculus.

Bird B.
1/Severe tubular necrosis, diffuse, kidney.
2/Pulmonary edema, severe, ventral aspects of lungs.
3/Degeneration, severe, kaolin layer, ventriculus.

Bird C.
1/Severe tubular necrosis, diffuse, kidneys.

Bird D.
1/Tubular necrosis, diffuse, severe, kidneys.
2/Pulmonary edema, diffuse, severe, lungs.
3/Edema, diffuse, moderate, skeletal muscle.
4/Degeneration, moderate, kaolin layer of ventriculus.

Terry R. Spraker, DVM/PhD

Typed: 12/11/95 lm
12-18-95 ml
1-3-96 ea

January 9, 1996

Dr. Bill Stubblefield
ENSR
4413 West LaPorte Avenue
Fort Collins, CO 80521

DL# 956-13011

COLORADO VETERINARY DIAGNOSTIC LABORATORIES
College of Veterinary Medicine and Biomedical Sciences
Colorado State University Fort Collins, CO 80523-1644
970-491-1281 FAX 970-491-0320

L A B O R A T O R Y R E S U L T S

OWNER: Spraker
VET: NONE
SPECIES: AVI
BREED: GEESE

DL Accession: 956-13011
Sample Received: 11/29/1995
Diagnostician: TRS

pecimen	Results		Comment Code
AEROBIC CULTURE			
LIV 0001	no growth		
LUNG 0001	Escherichia coli		04
LIV 0002	no growth		20
LUNG 0002	no growth		
LIV 0003	no growth		20
LUNG 0003	no growth		
LIV 0004	Escherichia coli		04
LUNG 0004	Escherichia coli		04
FUNGAL CULTURE			
LUNG 0001	see comments		
LUNG 0002	see commnents		
LUNG 0004	see comments		
ARSENIC			
KID 0001	Dry Weight Analysis	1.29ppm	AS6
LIV 0001	Dry Weight Analysis	0.97ppm	AS6
KID 0002	Dry Weight Analysis	0.81ppm	AS6
LIV 0002	Dry Weight Analysis	1.94ppm	AS6
KID 0003	Dry Weight Analysis	0.59ppm	AS6
LIV 0003	Dry Weight Analysis	1.01ppm	AS6
KID 0004	Dry Weight Analysis	0.63ppm	AS6
LIV 0004	Dry Weight Analysis	0.85ppm	AS6
CADMIUM			
KID 0001	Wet Weight Analysis	1.0ppm	CD3
LIV 0001	Wet Weight Analysis	1.0ppm	CD2
KID 0002	Wet Weight Analysis	0.7ppm	CD3
LIV 0002	Wet Weight Analysis	0.8ppm	CD2
KID 0003	Wet Weight Analysis	1.9ppm	CD3
LIV 0003	Wet Weight Analysis	0.5ppm	CD2
KID 0004	Wet Weight Analysis	0.8ppm	CD3
LIV 0004	Wet Weight Analysis	0.7ppm	CD2
COPPER			
KID 0001	Dry Weight Analysis	91.2ppm	CuQ
LIV 0001	Dry Weight Analysis	225ppm	CuP
KID 0002	Dry Weight Analysis	72.3ppm	CuQ
LIV 0002	Dry Weight Analysis	254ppm	CuP
KID 0003	Dry Weight Analysis	90.4ppm	CuQ
LIV 0003	Dry Weight Analysis	242ppm	CuP
KID 0004	Dry Weight Analysis	50.7ppm	CuQ
LIV 0004	Dry Weight Analysis	147ppm	CuP
CYANIDE			
LUNG 0001	None Detected		

ENS000048578

LUNG 0002	None Detected		
LUNG 0003	None Detected		
LUNG 0004	None Detected		
IRON			
KID 0001	Dry Weight Analysis	307ppm	IR9
LIV 0001	Dry Weight Analysis	473ppm	IR8
KID 0002	Dry Weight Analysis	425ppm	IR9
LIV 0002	Dry Weight Analysis	441ppm	IR8
KID 0003	Dry Weight Analysis	310ppm	IR9
LIV 0003	Dry Weight Analysis	371ppm	IR8
KID 0004	Dry Weight Analysis	427ppm	IR9
LIV 0004	Dry Weight Analysis	565ppm	IR8
LEAD			
KID 0001	Wet Weight Analysis	0.4ppm	LDE
LIV 0001	Wet Weight Analysis	0.3ppm	LDE
KID 0002	Wet Weight Analysis	0.3ppm	LDE
LIV 0002	Wet Weight Analysis	0.4ppm	LDE
KID 0003	Wet Weight Analysis	0.8ppm	LDE
LIV 0003	Wet Weight Analysis	0.2ppm	LDE
KID 0004	Wet Weight Analysis	0.7ppm	LDE
LIV 0004	Wet Weight Analysis	0.7ppm	LDE
MANGANESE			
KID 0001	Dry Weight Analysis	211ppm	MN4
LIV 0001	Dry Weight Analysis	155ppm	MN3
KID 0002	Dry Weight Analysis	171ppm	MN4
LIV 0002	Dry Weight Analysis	225ppm	MN3
KID 0003	Dry Weight Analysis	282ppm	MN4
LIV 0003	Dry Weight Analysis	152ppm	MN3
KID 0004	Dry Weight Analysis	395ppm	MN4
LIV 0004	Dry Weight Analysis	147ppm	MN3
MERCURY			
KID 0001	Wet Weight Analysis	0.02ppm	ME6
LIV 0001	Wet Weight Analysis	0.03ppm	ME6
KID 0002	Wet Weight Analysis	0.21ppm	ME6
LIV 0002	Wet Weight Analysis	0.03ppm	ME6
KID 0003	Wet Weight Analysis	0.03ppm	ME6
LIV 0003	Wet Weight Analysis	0.06ppm	ME6
KID 0004	Wet Weight Analysis	0.02ppm	ME6
LIV 0004	Wet Weight Analysis	0.03ppm	ME6
SELENIUM			
KID 0001	Dry Weight Analysis	2.85ppm	
LIV 0001	Dry Weight Analysis	3.01ppm	SER
KID 0002	Dry Weight Analysis	2.50ppm	
LIV 0002	Dry Weight Analysis	3.04ppm	SER
KID 0003	Dry Weight Analysis	3.90ppm	
LIV 0003	Dry Weight Analysis	4.50ppm	SER
KID 0004	Dry Weight Analysis	3.14ppm	
LIV 0004	Dry Weight Analysis	3.47ppm	SER
ZINC			
KID 0001	Dry Weight Analysis	568ppm	ZIE
LIV 0001	Dry Weight Analysis	555ppm	ZID
KID 0002	Dry Weight Analysis	413ppm	ZIE
LIV 0002	Dry Weight Analysis	765ppm	ZID
KID 0003	Dry Weight Analysis	531ppm	ZIE
LIV 0003	Dry Weight Analysis	535ppm	ZID
KID 0004	Dry Weight Analysis	511ppm	ZIE
LIV 0004	Dry Weight Analysis	346ppm	ZID
REFERRAL LAB			
MISC 0001	University of California*		

COMMENT CODE	COMMENT NARRATIVE
04	Light bacterial growth
20	Fungal results pending
AS6	ARSENIC Diagnostic level: Goose: Liver & Kidney (DW) - normal, < 2.0 ppm.
CD2	CADMIUM Diagnostic level: Goose: Liver (WW) - normal, < 0.6 ppm.
CD3	CADMIUM Diagnostic level: Goose: Kidney (WW) - normal, < 2.0 ppm.
CuP	COPPER Diagnostic level: Goose: Liver (DW) - normal, 24.0-120 ppm.
CuQ	COPPER Diagnostic level: Goose: Kidney (DW) - normal, 12.0-36.0 ppm.
IR8	IRON Diagnostic level: Goose: Liver (DW) - normal, 800-2,000 ppm.
IR9	IRON Diagnostic level: Goose: Kidney (DW) - normal, 600-1,400 ppm.
LDE	LEAD Diagnostic level: Goose: Liver & Kidney (WW) - normal, < 2.0 ppm.
ME6	MERCURY Diagnostic level: Goose: Liver & Kidney (WW) - normal, < 0.1 ppm.
MN3	MANGANESE Diagnostic level: Goose: Liver (DW) - normal, 8-24 ppm.
MN4	MANGANESE Diagnostic level: Goose: Kidney (DW) - normal, 6-16 ppm.
SER	SELENIUM Diagnostic level: Goose: Liver (DW) - 1.40-5.20 ppm.
ZID	ZINC Diagnostic level: Goose: Liver (DW) - normal, 100-440 ppm.
ZIE	ZINC Diagnostic level: Goose: Kidney (DW) - normal, 56.0-128 ppm.

SPECIAL NOTES

1 Please note that there is no published normal concentration of selenium in goose kidney available. Normal kidney selenium concentrations for poultry range from 2.00-4.80 ppm, dry weight basis.

2 *Referral results from the University of California: The 4 brain samples have a normal cholinesterase activity for most species of waterfowl. The brain & fat samples contain

ENS000048580

none of the listed organophosphate or organochlorine

4 pesticides in concentrations greater than the stated detection limits.

FAX to :COLORADO STATE UNIVERSITY ,970 491-0320

UNIVERSITY OF CALIFORNIA
VETERINARY DIAGNOSTIC
LABORATORY SYSTEM

P.O. Box 1770
Davis, CA 95617
(916) 752-8700

ACCESSION#:D9509311
District:
County: CO COLORADO
Case Coordinator: NEWMAN

Submitter
COLORADO STATE UNIVERSITY
VETERINARY DIAGNOSTIC LAB
300 WEST DRAKE #E100
FORT COLLINS, CO 80523
(970) 491-1281

Owner:
OWNER INFORMATION
NOT GIVEN
,

Agent or Collector:
Reference Number: 956-13011/DPO #309329

Species: GAME BIRDS
Herd/Flock ID:
Date Taken: 11/29/95
Date Received: 12/06/95

9 Specimens submitted: 4 FAT, 4 BRAIN

FINAL REPORT Date Reported: 12/12/95

Electronically signed by:
Scott Newman, DVM

LABORATORY FINDINGS/DIAGNOSIS:
Normal brain cholinesterase activity.
No organophosphate or organochlorine pesticides detected.

CLINICAL HISTORY:
Refer to DPO #309329 for payment.
No history given.

SPECIMEN SUMMARY:

Specimen Type	Breed	ID	Age	Sex	Qty
BRAIN	GOOSE	Multiple IDs			4
FAT	GOOSE	Multiple IDs			4

TOXICOLOGY:

The 4 brain samples have a normal cholinesterase activity for most species of waterfowl (normal = 14.6 -/- 5.7 uM/g,min). The brain and fat samples contain none of the listed organophosphate or organochlorine pesticides in concentrations greater than the stated detection limits. MDL = method detection limit (lowest concentration detectable by our test method).

FAT SPECIMEN.ID	MDL	CHLORPYRIFOS .05 ppm	COUMAPHOS .05 ppm	CROTOXYPHOS .05 ppm	CRUFORMATE .05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011D		Not Detected	Not Detected	Not Detected	Not Detected

FAT SPECIMEN.ID	MDL	DDVP .05 ppm	DEF .05 ppm	DEMETON .05 ppm	DIAZINON .05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011D		Not Detected	Not Detected	Not Detected	Not Detected

FAT SPECIMEN.ID	MDL	DICROTOPHOS .05 ppm	DIMETHOATE .05 ppm	DIOXATHION .2 ppm	DISULFOTON .05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011D		Not Detected	Not Detected	Not Detected	Not Detected

FAT SPECIMEN.ID	MDL	EPN .05 ppm	ETHION .05 ppm	ETHOPROP .05 ppm	FENAMIPHOS .05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011D		Not Detected	Not Detected	Not Detected	Not Detected

FAT SPECIMEN.ID	MDL	FENSULFOTHION .05 ppm	FENTHION .05 ppm	FONOPHOS .05 ppm	ISOFENPHOS .05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011D		Not Detected	Not Detected	Not Detected	Not Detected

FAT SPECIMEN.ID	MDL	MALATHION .05 ppm	METHAMIDAPHOS .05 ppm	METHIDATHION .05 ppm	METHYL PARATHIO .05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011D		Not Detected	Not Detected	Not Detected	Not Detected

FAT SPECIMEN.ID	MDL	MEVINPHOS .05 ppm	MONOCROTOPHOS .05 ppm	NALED .05 ppm	PARATHION .05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011D		Not Detected	Not Detected	Not Detected	Not Detected

CHOLINESTERASE

Specimen Information Id	Type	Result	MDL
956-13011A	BRAIN	12.2 UM/G/MIN	0.1 UM/G/MIN
956-13011B	BRAIN	13.3 UM/G/MIN	0.1 UM/G/MIN
956-13011C	BRAIN	13.4 UM/G/MIN	0.1 UM/G/MIN
956-13011-D	BRAIN	12.4 UM/G/MIN	0.1 UM/G/MIN

ORGANOCHLORINE INSECTICIDES-EXTENDED SCREEN

BRAIN		Aldrin	BHC	Chlordane	p,p'DDD
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.25 ppm	.1 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011-D		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		o,p'DDD	p,p'DDE	o,p'DDE	p,p'DDT
SPECIMEN.ID	MDL	.1 ppm	.1 ppm	.1 ppm	.1 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011-D		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		o,p'DDT	Dicofol	Dieldrin	Endosulfan I
SPECIMEN.ID	MDL	.1 ppm	.1 ppm	.05 ppm	.05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011-D		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		Endosulfan II	Endrin	Gamma Chlordane	HCB
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011-D		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		Heptachlor	Heptachlor Epox	Lindane	Methoxychlor
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011-D		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		Mirex	Toxaphene
SPECIMEN.ID	MDL	.05 ppm	2 ppm
956-13011A		Not Detected	Not Detected
956-13011B		Not Detected	Not Detected
956-13011C		Not Detected	Not Detected
956-13011-D		Not Detected	Not Detected

FAT SPECIMEN.ID	MDL	Aldrin	BHC	Chlordane	p,p'DDD
		.05 ppm	.05 ppm	.25 ppm	.1 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011D		Not Detected	Not Detected	Not Detected	Not Detected

FAT SPECIMEN.ID	MDL	o,p'DDD	p,p'DDE	o,p'DDE	p,p'DDT
		.1 ppm	.1 ppm	.1 ppm	.1 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011D		Not Detected	Not Detected	Not Detected	Not Detected

FAT SPECIMEN.ID	MDL	o,p'DDT	Dicofol	Dieldrin	Endosulfan I
		.1 ppm	.1 ppm	.05 ppm	.05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011D		Not Detected	Not Detected	Not Detected	Not Detected

FAT SPECIMEN.ID	MDL	Endosulfan II	Endrin	Gamma Chlordane	HCB
		.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011D		Not Detected	Not Detected	Not Detected	Not Detected

FAT SPECIMEN.ID	MDL	Heptachlor	Heptachlor Epox	Lindane	Methoxychlor
		.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011D		Not Detected	Not Detected	Not Detected	Not Detected

FAT SPECIMEN.ID	MDL	Mirex	Toxaphene
		.05 ppm	2 ppm
956-13011A		Not Detected	Not Detected
956-13011B		Not Detected	Not Detected
956-13011C		Not Detected	Not Detected
956-13011D		Not Detected	Not Detected

ORGANOPHOSPHATE INSECTICIDES-EXTENDED SCREEN

FAT SPECIMEN.ID	MDL	ACEPHATE	AZINPHOS-METHYL	CARBOPHENOTHION	CHLORFENVINPHOS
		.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011D		Not Detected	Not Detected	Not Detected	Not Detected

FAT		PHORATE	PHOSALONE	PHOSMET	PHOSPHAMIDON
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011D		Not Detected	Not Detected	Not Detected	Not Detected

FAT		PROFENOPHOS	PROPETAMPHOS	RONNEL	TERBUFOS
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011D		Not Detected	Not Detected	Not Detected	Not Detected

FAT		TETRACHLORVINPH	TRIAZOPHOS
SPECIMEN.ID	MDL	.05 ppm	.05 ppm
956-13011A		Not Detected	Not Detected
956-13011B		Not Detected	Not Detected
956-13011C		Not Detected	Not Detected
956-13011D		Not Detected	Not Detected

BRAIN		ACEPHATE	AZINPHOS-METHYL	CARBOPHENOTHION	CHLORFENVINPHOS
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011-D		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		CHLORPYRIFOS	COUMAPHOS	CROTOXYPHOS	CRUFORMATE
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011-D		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		DDVP	DEF	DEMETON	DIAZINON
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011-D		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		DICROTOPHOS	DIMETHOATE	DIOXATHION	DISULFOTON
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.2 ppm	.05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011-D		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		EPN	ETHION	ETHOPROP	FENAMIPHOS
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011-D		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		FENSULFOTHION	FENTHION	FONOPHOS	ISOFENPHOS
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011-D		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		MALATHION	METHAMIDAPHOS	METHIDATHION	METHYL PARATHIO
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011-D		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		MEVINPHOS	MONOCROTOPHOS	NALED	PARATHION
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011-D		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		PHORATE	PHOSALONE	PHOSMET	PHOSPHAMIDON
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011-D		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		PROFENOPHOS	PROPETAMPHOS	RONNEL	TERBUFOS
SPECIMEN.ID	MDL	.05 ppm	.05 ppm	.05 ppm	.05 ppm
956-13011A		Not Detected	Not Detected	Not Detected	Not Detected
956-13011B		Not Detected	Not Detected	Not Detected	Not Detected
956-13011C		Not Detected	Not Detected	Not Detected	Not Detected
956-13011-D		Not Detected	Not Detected	Not Detected	Not Detected

BRAIN		TETRACHLORVINPH	TRIAZOPHOS
SPECIMEN.ID	MDL	.05 ppm	.05 ppm
956-13011A		Not Detected	Not Detected
956-13011B		Not Detected	Not Detected
956-13011C		Not Detected	Not Detected
956-13011-D		Not Detected	Not Detected

APPENDIX D

NECROPSY EXAMINATION REPORT-CASE 95-0678 LAB- 5

Species:	Snow Goose	**Identification #:**	ST 624701
Sex/Age:	Female/Mature	**Specimen:**	Carcass
Weight:	2160 gm	**Date Received:**	November 27, 1995
Body Condition:	Good	**Date Examined:**	November 29, 1995
Post Mortem Condition:	Good		

HISTORY:

Carcass was recovered at the Mantle Ranch, Glen Mountain, Beaverhead County, Montana on November 16, 1995. It was first observed approximately November 11, 1995, by a ranch employee. The location is approximately 40 miles from the Berkley Pit, Butte, Montana.

EXTERNAL EXAMINATION:

The feathers are dry and have no discoloration. The skin of the feet is dry but does not have areas of sloughing epidermis. The feathers of the cloacal area show no evidence of diarrhea or other abnormal discharge. The wings are intact and there are no external lesions suggestive of trauma.

INTERNAL EXAMINATION:

The carcass was partially skinned and the breast removed to facilitate the examination of the internal organs. Subcutaneous and mesenteric fat deposits were normal in abundance and consistency. The muscle tissue was normal in color and consistency. The keel bone has been fractured and some hemorrhage is noted into the breast muscle tissue.

The mucosal membranes of the mouth, larynx, esophagus and trachea are normal. The proventriculus also is normal. Extensive hemorrhage from the base of the liver is noted into the body cavity. The liver is congested. The gall bladder is not distended. The kidneys are normal.

The lungs and air sacs are normal. The heart and the blood contained within the heart are normal. The brain is normal.

No lesions suggestive of infectious disease are visible on gross examination.

RADIOGRAPHIC EXAMINATION:

No significant radiographic findings on whole body x-rays.

SUBSAMPLES SAVED/SUBMITTED FOR ANALYSIS:

LAB-5A: Tissues for chemical analysis (kidney, liver)

DIAGNOSIS:

Internal hemorrhage from blunt trauma

COMMENT:

The source of the blunt trauma is unclear. Birds hitting radio towers or power lines would have such a lesion. The goose may have flown some distance before succumbing to the internal hemorrhage. The lesion is not a gunshot wound. No lesions suggestive of contact with corrosive mine waste water were observed.

VETERINARY MEDICAL EXAMINER'S REPORT 95-0678
Page 10 (Stroud) RKS

NECROPSY EXAMINATION REPORT-CASE 95-0678 LAB -6

Species:	Snow Goose	**Identification #:**	ST 02405 (Montana)
Sex/Age:	Female/Immature	**S pecimen:**	Carcass
Weight:	1755 gm	**Date Received:**	November 27, 1995
Body Condition:	Good	**Date Examined:**	November 29, 1995
Post Mortem Condition:	Good		

HISTORY:

Carcass was received from Jim Hagenbarth and was recovered at the Mantle Ranch, Glen Mountain, Beaverhead County, Montana on November 14, 1995. The location is approximately 40 miles from the Berkley Pit, Butte, Montana.

EXTERNAL EXAMINATION:

A open lesion in the skin of the neck 3.5 cm by 1 cm is present. The underlying muscle tissue has been chewed by a scavenger or predator. Blood staining of the feathers on the head, breast and right wing is evident. The feathers are dry and have no discoloration. The skin of the feet is dry but do not have areas of sloughing epidermis. The feathers of the cloacal area show no evidence of diarrhea or other abnormal discharge. The wings are intact.

INTERNAL EXAMINATION:

The carcass was partially skinned and the breast removed to facilitate the examination of the internal organs. Subcutaneous and mesenteric fat deposits were normal in abundance and consistency. The muscle tissue was normal in color and consistency. Extensive subcutaneous hemorrhage is noted in the tissues of the upper neck and throat area. A section of the trachea is absent from the neck tissues. An area of bruised subcutaneous tissue and muscle is noted on the left lower breast and abdomen.

The mucosal membranes of the mouth, larynx, esophagus and trachea are normal. The proventriculus also is normal. Hemorrhage from the base of the liver is noted into the body cavity and over the base of the heart. The liver is congested. The gall bladder is not distended. The kidneys are normal.

A pocket of clotted blood approximately 3 cm diameter is noted between the right rib cage and the right lung. The lungs and air sacs are normal. The heart and the blood contained within the heart are normal. The brain is normal.

No lesions suggestive of infectious disease are visible on gross examination.

VETERINARY MEDICAL EXAMINER'S REPORT 95-0678
Page 11 (Stroud) RKS

RADIOGRAPHIC EXAMINATION:

No significant radiographic findings on whole body x-rays.

SUBSAMPLES SAVED/SUBMITTED FOR ANALYSIS:

LAB-6A: Tissues for chemical analysis (kidney, liver)

DIAGNOSIS:

Internal hemorrhage from blunt trauma
Predator/scavenger damage

COMMENT:

The source of the blunt trauma is unclear. Birds hitting radio towers or power lines would have such a lesion. The goose may have flown some distance before succumbing to the internal hemorrhage. The lesion is not a gunshot wound. A predator or scavenger may have finally killed the injured bird by biting the neck or head. No lesions suggestive of contact with corrosive mine waste water were observed.

Richard K. Stroud

Richard K. Stroud DVM MS
Veterinary Medical Examiner