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ORIGINAL ARTICLE

Mortality in the Baltimore union poultry cohort: non-malignant diseases

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Abstract
Background Workers in poultry plants have high exposure to a variety of transmissible agents present in poultry and their products. Subjects in the general population are also exposed. It is not known whether many of these agents cause disease in humans. If they do, we reason this would be readily evident in a highly exposed group such as poultry workers. We report here on mortality from non-malignant diseases in a cohort of poultry workers.
Methods Mortality was compared with that of the US general population, and with that of a comparison group from the same union. Risk was estimated by standardized mortality ratio, proportional mortality ratio, and directly standardized risk ratio.
Results Poultry workers as a group had an overall excess of deaths from diabetes, anterior horn disease, and hypertensive disease, and a deficit of deaths from intracerebral hemorrhage. Deaths from zoonotic bacterial diseases, helminthiasis, myasthenia gravis, schizophrenia, other diseases of the spinal cord, diseases of the esophagus and peritonitis were non-significantly elevated overall by all analyses, and significantly so in particular race/sex subgroups.
Conclusions Poultry workers may have excess occurrence of disease affecting several organs and systems, probably originating from widespread infection with a variety of microorganisms. The results for neurologic diseases could well represent important clues to the etiology of these diseases in humans. The small numbers of deaths involved in some cases limit interpretation.

Keywords Transmissible agents · Poultry slaughtering/processing · Viruses · Bacteria · Fumes · Neurologic diseases

Introduction

Over the years, we have been interested in whether the plethora of transmissible agents that are naturally present in animals used for food (cattle, pigs, sheep, and poultry), and their products may be the cause of chronic diseases in humans, including cancer, cardiovascular, and neurologic diseases. To investigate the role of poultry, we have used as our model subjects who work in poultry slaughtering and processing plants. The rationale is that this group of workers have the highest human exposure to these agents (Saif et al. 2003), and thus, if these agents cause disease in humans, it will be most readily manifested in this highly exposed group. Other potentially harmful occupational exposures in the industry include exposure to fumes emitted from the wrapping machine (Pauli et al. 1980; Vandervort and Brooks 1977; Johnson et al. 1999), smoke or aerosol emitted during smoking or cooking of poultry products

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(Nordholm et al. 1986; Vainiotalo and Matveinen 1993), and curing agents (Sen et al. 1974).

Only three cohort studies of these workers have been conducted to date (Fritschi et al. 2003; Johnson 1987a; Johnson 1987b; Johnson et al. 1997; Netto and Johnson 2003). One of them reported on only cardiovascular diseases and injury in workers employed in poultry abattoirs in Australia. The risk of cardiovascular diseases in poultry workers was similar to that for workers in red meat abattoirs. Unfortunately, an external non-meat group was not available for this comparison (Fritschi et al. 2003). We provided results for a cohort of poultry workers in a union in Missouri (Netto and Johnson 2003). A statistically increased risk was observed only for non-malignant respiratory disease in white males, for symptoms, senility and ill-defined conditions in white males and white females, and for all accidents in all females. We also previously studied on two separate occasions, 1954–1980 and 1954–1989 (Johnson 1987a; Johnson 1987b; Johnson et al. 1997), a cohort of 2,639 subjects who had *ever* worked in poultry slaughtering/processing plants, and who were members of a local meatcutters' union in Baltimore, Maryland. The individuals worked in six poultry plants located in Baltimore city and the eastern shore of Maryland. Only a few broad categories of cause of death were studied then, however, and only mortality from motor vehicle accidents was significantly elevated in the group (Johnson et al. 1997). We now report later, the results obtained after further extended follow-up from 1954 to the end of 2003 of a subset of this cohort ($N = 2{,}579$) who worked *exclusively* in poultry slaughtering and processing plants. We excluded 60 subjects from the original 2,639 because they had also worked in plants where cattle, pigs, and sheep were slaughtered. Also, this time, we were able to investigate 130 causes of death (see Table 1) when compared with less than 30 in previous follow-ups, and a total of 795 deaths have now occurred. The protocol for this study was approved by the UNT Health Sciences Center Institutional Review Board.

Materials and methods

The 2,580 subjects worked in any of six poultry slaughtering and processing plants during their entire membership in the Baltimore Meatcutters' Union, Local 27 (formerly Local 117). The union membership also included 6,052 subjects who had worked in companies outside the meat and poultry industries that were located in Baltimore and other parts of Maryland also, such as soft drinks manufacturing, oyster shucking, tanning, and soup manufacturing, which provides an internal comparison group.

Methods of follow-up include the National Death Index, Social Security Administration (SSA), Maryland State Department of Vital Records (MSDVR), Maryland State Department of Motor Vehicles, Health Care Financing Administration (HCFA), Veterans Administration, obituary notices, US Post Office, personal contact by telephone and mail, and internet tracing methods. The Pension Benefit Information Inc., a private company, was also used to identify deceased persons. This company matches subjects against US death records for all years from the 1800s to the present, also using information received from SSA, HCFA, and MSDVR, as well as the Civil Service Commission, Railroad Retirement Board, and the Department of Defense. Because of the extensive follow-up methods employed, subjects whose vital status was unknown were assumed to be alive at the end of study. Of the 795 deaths, we were able to obtain cause of death information for all but 34 (4.3%). All persons with a death certificate had complete date of birth information. However, date of birth information was missing for 197 poultry subjects (7.6%) not known to have died. Rather than excluding such persons from the analysis, these subjects were artificially assigned a date of birth based on the median year of birth of workers with known date of birth joining the union in a particular year—thus if a member without date of birth joined the union in 1975, he/she was assigned as his/her year of birth, the median year of birth for persons with known date of birth joining the union that particular year. This measure was deemed to be associated with negligible bias, since the total person-years will be affected to a negligible degree.

The main statistical analyses involved estimation of standardized mortality ratios (SMR), stratifying on age, calendar time, gender, race and plant, using the US general population as the comparison group, using the OCMAP Plus software from the University of Pittsburgh. Information on race was available only for deceased individuals with a death certificate/known cause of death. Therefore, to perform the SMR analyses, race was artificially assigned at random to each individual in the study without a death certificate/known cause of death, based on the known racial distribution of the union from a survey of its current membership carried out initially at the start of the study. The cohort was stratified by plant, and then stratified into four subgroups by race and sex (black males, black females, white males, white females), and each of these groups stratified according to age (5-year intervals) and calendar year at entry into the cohort (5-year intervals). Person-years were accumulated from January 1, 1954 for those who were already members of the union before that date. Those who became members later, person-years commenced on the date of membership. Person-years were enumerated up to the date of death, or date of termination of the study on December 31, 2003, whichever was earlier. Expected deaths were derived by multiplying the person-years in each cell by the corresponding gender-, calendar year-, age-

Table 1 List of causes of death investigated

Non-malignant causes of death	ICD 9th Rev
Certain infectious diseases	
1 Intestinal infectious diseases	001–009
2 Tuberculosis	010–018
3 Certain zoonotic bacterial diseases	020–027
4 Other bacterial diseases	030–041
5 Slow virus infections of CNS	046
6 Arthropod-borne viral fevers and viral hemorrhagic fevers	060–066
7 Mycoses	110–118
8 Helminthiases	120–129
9 Protozoal diseases	006,007,084–86,130,131
Diseases of the blood and blood-forming	
1 B12/folate deficiency	281.0–281.3, 281.9
2 Acquired hemolytic anemias	283
3 Aplastic anemia	284
4 Sideroblastic anemia	285.0
5 Purpura & hemorrhagic conditions	287
6 Diseases of white cells	288
Endocrine diseases	
1 Disorders of thyroid gland	240–246
2 Diabetes mellitus	250
3 Disorders of pancreas not diabetes	251
4 Diseases of parathyroid	252
5 Diseases of pituitary gland	253
6 Diseases of thymus gland	254
7 Diseases of adrenals	255
8 Ovarian dysfunction	256
9 Testicular dysfunction	257
10 Other diseases of endocrine glands	258, 259
Mental and behavioural disorders	
1 Senile & presenile psychotic conditions	290, 331
2 Alcoholic psychoses	291
3 Schizophrenic disorders	295
4 Affective psychoses/manic depressive reaction	296
5 Paranoid states	297
6 Neurotic disorders	300
7 Personality disorders	301
8 Alcoholic dependence/alcoholism	303
9 Drug dependence	304
10 Functional digestive disorders	564
Diseases of the nervous system	
1 Meningitis	320–322
2 Encephalitis	323
3 Intracranial and intraspinal abcess	324
4 Intracranial phlebitis & thrombophlebitis	325
5 Late effects of intracranial abcess/infec	326
6 Parkinson's disease	332
7 Multiple sclerosis	340
8 Hemiplegia & other paralysis	342, 344
9 Epilepsy	345

Table 1 continued

Non-malignant causes of death	ICD 9th Rev
10 Anterior horn disease	335
11 Other diseases of spinal cord	334, 336
12 Disorders of peripheral autonomic NS	337
13 Myasthenia gravis	358.0
14 Diseases of muscle, tendon, fascia	359
15 Diffuse diseases of connective tissue	446, 447, 710
16 Disorders of the eye & adnexae	360–379
17 Disorders of ear & mastoid process	380–389
Diseases of circulatory system	
1 Acute rheumatic fever	390–392
2 Chronic rheumatic heart disease	393–398
3 Hypertensive disease	401–405
4 Ischemic heart disease	410–414
5 Acute pericarditis	420
6 Acute & subacute endocarditis	421
7 Acute myocarditis	422
8 Functional diseases of heart	426, 427
Cerebrovascular diseases	
1 Subarachnoid hemorrhage	430
2 Intracerebral hemorrhage, etc.	431, 432
3 Occlusion/stenosis of pre-cerebral & cerebral arteries	433, 434
4 Transient cerebral ischemia	435
5 Pulmonary embolism & infarction	415
6 Aortic aneurysm	441
Diseases of respiratory system	
1 Pneumonia	480–486
2 Chronic bronchitis	491
3 Asthma	493
4 Pneumoconiosis	500–505
5 Resp conditions due to chemical- fumes/vapor fumes/vapor	506
6 Abcess of lung	513
7 Bronchiectasis	494
Diseases of digestive system	
1 Diseases of hard tissues of teeth; Gingival & periodontal diseases	521–523
2 Diseases of salivary glands	527
3 Stomatitis, etc.	528, 529
4 Diseases of esophagus	530
5 Stomach ulcer	531
6 Duodenal Ulcer	532
7 Gastrojejunal ulcer	534
8 Gastritis & duodenitis	535
9 Disorders of function of stomach	536
10 Acute appendicitis	540–543
11 Hernia of abdominal cavity	550–553
12 Regional enteritis; idiopat proctocolitis	555–556, 558
13 Peritonitis	567
14 Acute & subacute necrosis of liver	570
15 Cirrhosis and chronic liver disease liver abcess, and other disorders	571–573
16 Cholelithiasis	574

Table 1 continued

Non-malignant causes of death	ICD 9th Rev
17 Other disorder of gallbladder & biliary tract	575, 576
18 Diseases of pancreas	577
Diseases of genitourinary system	
1 Acute nephritis, nephrotic syndrome	580, 581
2 Chronic nephritis	582
3 Nephritis not specified as acute/chronic	583
4 Infections of kidney	590
5 Hydronephrosis	591
6 Calculus of kidney & ureter	592
7 Other diseases of kidney and ureter	593
8 Calculi of other parts of urinary system	594
9 Cystitis, other disord.bladder, urethritis	595–598
10 Diseases of prostate	600–602
11 Disorders of breast	610–611
12 Diseases of ovary, fallopian tube, etc.	614, 620
13 Diseases of uterus, cervix, vagina, vulva	615, 616
14 Endometriosis	617
15 Genital prolapse	618
16 Hemorrhage in pregnancy, plac. previa	640–641
17 Hypertension in pregnancy, childbirth, puerperium	642
18 Hydramnios	657
Diseases of the musculoskeletal system and connective tissue	
1 Rheumatoid arthritis, etc.	714, 720
2 Osteomyelitis, periostitis	730
Injury, poisoning and certain other consequences of external cause	
1 Transport accidents	E800–E829
2 Water transport accidents	E830–E838
3 Aircraft accidents	E840–E845
4 Accidental poisoning by solid & liquid substances, accidental poisoning by solid, liquid, gases & vapors	E850–E869
5 Accidental falls	E880–E888
6 Other accidents	E900–E928
7 Accidents caused by machinery	E919
8 Accidents caused by cutting and piercing instruments	E920
9 Suicide & self-inflicted injury	E950–E959
10 Fractures	800–829
11 Intracranial injury	850–854
12 Open wound	870–897
13 Injury to blood vessels	900–904
14 Superficial injury	910–919
15 Contusion with intact skin surface	920–924
16 Crushing injury	925–929
17 Foreign body entering through orifice	930–939
18 Burns	940–949
19 Injury to nerves & spinal cord	950–957

specific mortality rate for the United States general population. Observed and expected deaths for each cell were summed over all ages and calendar years, and over all strata, and the SMR estimated as the total observed number of deaths divided by the total expected. The 95% confidence intervals for the SMR were calculated according to a simple exact method that links both the Poisson and the chi-square distributions (Liddell 1984).

Table 2 The distribution of year of birth of poultry workers (1954–2003)

Interval	No. of subjects	Percentage	Cumulative percentage
1870–1900	14	0.5	0.5
1900–10	55	2.1	2.7
1910–20	155	6.0	8.7
1920–30	353	13.7	22.4
1930–40	641	24.9	47.2
1940–50	971	37.7	84.9
1950–60	390	15.1	100.0
Total	2,579		

We also estimated proportional mortality ratios (PMR) based on a similar method of stratification as was used to estimate the SMR, except that for each cell, the proportion of all deaths due to a given cause in the US population was multiplied by the total number of deaths in the corresponding cell of the study population to get the expected number of deaths. The ratio of corresponding observed to expected deaths is the PMR. The variance was calculated assuming a binomial distribution for the observations (Miettinen 1976). Estimation of the PMR was carried out because in the PMR analyses, date of birth and race information was available for all subjects.

Results

Table 1 gives a list of the 130 non-malignant causes of death investigated. These causes were based on only deaths coded as the underlying cause in the death certificate. Table 2 gives the distribution of the year of birth of the cohort, and Table 3 gives the number of subjects and person-years contributed by race and gender. Table 4 gives the results for causes of death for which a statistically significant SMR or PMR was observed in any race/sex subgroup or in the total cohort, when the observed number of deaths was two or more. However, the PMR results are not shown because they were virtually identical to those obtained with the SMR method. A total of 795 subjects (31% of the cohort) died during 1954–2003. High risks of death from zoonotic bacterial diseases and helminthiasis are seen in this poultry cohort suggesting that different types of microorganisms are causing disease in this population. A clear excess of diabetes in whites was observed (SMR = 3.6, 95% CI 2.0–5.8, not shown in Table 4), but not in blacks. Similarly, an excess of deaths was observed for disorders of the thyroid gland, senile and pre-senile psychotic conditions, anterior horn cell disease, myasthenia gravis, hypertensive disease, ischemic heart disease, diseases of the esophagus, peritonitis, and other diseases of the kidney and ureter, and transport accidents. On the other hand, a clear deficit of deaths was observed for intracerebral hemorrhage and other accidents.

We conducted SMR analyses also for the comparison group, but the results are not presented because the poultry group was significantly younger than the comparison group (47.2% of the poultry workers were born between 1870 and 1940 when compared with 67.8% of controls), and thus, the magnitude of the cause-specific SMRs in the poultry group cannot be directly compared with those in the comparison group. Hence, instead we conducted limited directly standardized risk ratio analyses in which death rates for specific diseases in the poultry group were compared with those in the comparison group, controlling for age, gender, race, and year of death, bearing in mind that this comparison may involve unstable rates because of the small numbers involved. As is seen in Table 4, in the direct standardization analysis, an elevated risk was no longer evident only for disorders of the thyroid gland, while the high risk of certain zoonotic bacterial diseases and helminthiasis became reduced but still persisted. However, these causes of death were based on only two deaths each. Thus, the results still hold to a large extent, irrespective of what method of analysis was used and irrespective of whether US rates are used for comparison or the internal control group was used.

Analysis by duration of employment or union membership was not carried out, because it was not possible to update employment information, since all of the plants had since closed down.

Table 3 Number of subjects, person-years at risk, number of deaths, by race and gender—1954–2003 (poultry workers)

	No. of subjects	Males			Females			Total	
		Person-years	No. of deaths	No. of subjects	Person-years	No. of deaths	No. of subjects	Person-years	No. of deaths
White	376	12,280.4	136	477	16,750.4	120	853	29,030.8	256
Non-white	994	31,951.8	339	732	25,330.1	200	1,726	57,281.9	539
Total	1,370	44,232.2	475	1,209	42,080.5	320	2,579	86,312.7	795

Since the race of subjects without death certificates was imputed, the numbers given for race and person-years in the table may not be exact. The race of deceased subjects is accurate

Table 4 Baltimore union poultry workers—standardized mortality ratios for the period 1954–2003

Cause of death	Non-white males No. obs. SMR (95% CI)	White males No. obs. SMR (95% CI)	Total males No. obs. SMR (95% CI)	Non-white females No. obs. SMR (95% CI)	White females No. obs. SMR (95% CI)	Total females No. obs. SMR (95% CI)	Total males & females No. obs. SMR (95% CI) [DSRR]@
Certain zoonotic bacterial diseases	0	1 295 (7.4–164)	1 52.9* (1.3–295)	1 106 (2.7–592)	0	1 78.8 (2.0–439)	2 63.3 (7.7–229) [3.4]
Helminthiasis	0	0	0	2 641 (77.6–2,316)	0	2 437 (53.0–1,580)	2 141 (17.1–511) [2.4]
Disorders of thyroid gland	1 12.0 (0.3–66.8)	0	1 9.7 (0.2–54.2)&	1 5.5 (0.1–30.8)&	0	1 4.1 (0.1–22.9)	2 5.8 (0.7–20.9)& [0.8]
Diabetes	6 0.7 (0.2–1.5)	5 2.6 (0.8–6.0)	11 1.0 (0.5–1.8)	10 1.1 (0.5–2.0)	11 4.3 (2.2–7.8)	21 1.8 (1.1–2.7)	32 1.4 (1.0–2.0) [1.3]
Senile & presenile psychotic conditions	0	0	0	3 3.3 (0.7–9.8)&	0	3 2.0 (0.4–5.8)	3 1.1 (0.2–3.3) [0.9]
Anterior horn disease	2 6.1 (0.7–22.0)&	1 4.5 (0.1–24.9)	3 5.4 (1.1–15.9)	0	1 3.7 (0.1–20.6)	1 2.1 (0.1–11.6)	4 3.9 (1.1–9.9) [∞]
Myasthenia Gravis	1 20.6 (0.5–115)&	1 43.2 (1.1–241)	2 27.9 (3.4–101)	0	0	0	2 14.5 (1.8–52.4) [8.0]
Hypertensive disease	8 0.8 (0.4–1.6)	6 5.7 (2.1–12.5)	14 1.3 (0.7 –2.2)	7 1.0 (0.4–2.0)	0	7 0.8 (0.3 –1.7)	21 1.1 (0.7–1.7) [1.7]
Ischemic heart disease	56 1.0 (0.7–1.3)	31 1.1 (0.8–1.6)	87 1.0 (0.8–1.3)	33 1.0 (0.7–1.4)	27 1.8 (1.2–2.6)	60 1.2 (0.9–1.6)	147 1.1 (0.9–1.3) [1.0]
Intracerebral hemorrhage etc.	0	0	0 *E* = 7.9 (0.0–0.5)	4 0.9 (0.3–2.4)	1 0.8 (0.0–4.6)	5 0.9 (0.3–2.1)	5 0.4 (0.1–0.9) [0.3]
Diseases of Esophagus	1 4.7 (0.1–26.0)	0	1 3.5 (0.1–19.3)	0	1 18.1 (0.5–101)&	1 6.7 (0.2–37.3)&	2 4.6 (0.6–16.5)& [2.7]
Peritonitis	0	0	0	1 4.8 (0.1–26.6)	1 14.2 (0.4–78.9)&	2 7.1 (0.9–25.8)&	2 3.2 (0.4–11.6) [2.3]
Other diseases of kidney & ureter	0	0	0	1 1.3 (0.0–7.5)	2 18.0 (2.2–65.0)	3 3.5 (0.7–10.3)&	3 1.5 (0.3–4.4) [0.6]
Transport accidents	16 1.2 (0.7–2.0)	11 2.8 (1.4–4.9)	27 1.6 (1.1–2.3)	3 1.1 (0.2–3.1)	2 1.1 (0.1–3.9)	5 1.1 (0.3–2.5)	32 1.5 (1.0–2.1)
Other accidents	2 0.3 (0.0–1.0)	1 0.6 (0.0–3.6)	3 0.4 (0.1–1.0)&	0	2 3.9 (0.5–14.2)	2 1.1 (0.1–4.0)	5 0.5 (0.2–1.1)
All causes of death	339 1.0 (0.9–1.1)	136 1.3 (1.1–1.6)	475 1.0 (1.0–1.1)	200 1.0 (0.9–1.2)	120 1.4 (1.1–1.6)	320 1.1 (1.0–1.3)	795 1.1 (1.0–1.2)

& = PMR was statistically significant

E = expected

@ DSRR = directly standardized risk ratios

Diabetes in whites ($N = 16$, SMR = 3.6**, 95% CI = 2.0–5.8)

Ischemic heart disease in whites ($N = 58$, SMR = 1.4, 95% CI = 1.1–1.8)

There were no deaths from slow virus infection of the CNS

Schizophrenia in (WF, $N = 1$, SMR = 71.4, 95% CI = 1.8–398)); (entire poultry cohort, $N = 1$, SMR = 10.3, 95% CI = 0.3–57.5)

Other diseases of the spinal cord in (NWF, $N = 1$, SMR = 25.2, 95% CI = 0.6–140); for entire poultry cohort ($N = 1$, SMR = 6.8, 95% CI = 0.2–37.8)

Discussion

This is the first time non-malignant mortality in workers in poultry slaughtering and processing plants has been studied in such detail (130 specific causes of death). This group of workers has one of the highest human exposures to a wide variety of transmissible agents present in poultry birds (viruses, bacteria, fungi, protozoa, helminths), and virtually all of them are exposed to a significant degree. In a typical large poultry plant, as many as 175,000 chickens are being killed daily. The workers are prone to cuts and other types of injury from sharp knives, bone splinters, etc., and to dermatitis from irritant enzymes and other body fluids from the birds, making it easy for microorganisms to enter the body via the skin. They also have intimate contact with blood and internal organs, and they are also exposed to microorganisms through the airborne route. In one study, a chicken carcass was artificially contaminated before plucking with readily identifiable marker bacteria; it was shown that the same organism could be recovered from the 265th carcass following the contaminated one through the mechanical feather-plucking machine (Mead 1976). Mckercher et al. (1978) studied infection in meat products from pigs infected with African swine fever virus and hog cholera virus. They showed that both viruses could be recovered in ham, even after it had been brined although absent when the brined ham was heated. They also recovered infectious viruses from pepperoni and salami sausages for up to 15 days after smoking or 22 days after slaughter. A serological study of antibodies to poultry oncogenic retroviruses in workers from a poultry plant in North Carolina indicated that virtually all the workers were infected and had demonstrable antibody levels to these viruses (Johnson et al. 1995a; Johnson et al. 1995b). No other human group has such a high potential for infection with poultry microorganisms on a daily basis in such large numbers as this group of workers. Thus, the wide variety of transmissible agents present in poultry is a prime suspect for explaining any excess risk of death from particular causes observed in the group. This is supported by the excess of zoonotic bacterial diseases and helminthiasis observed in the cohort.

Exposure to fumes during wrapping of meat and to aerosols and smoke during frying and smoking of poultry and to curing agents are the only other potentially harmful exposures that may occur in some poultry slaughtering and processing plants. However, we have no information on whether these exposures occurred in the plants studied. Moreover, typically only a handful of workers are usually engaged in cooking activities, hence it is unlikely that risks if any, associated with these activities can be detected in a cohort study of this type.

The excess of diabetes observed in the previous update (Johnson 1987a; Johnson 1987b; Johnson et al. 1997; Johnson et al. 2007) is also seen in the present follow-up. It could be related to non-occupational factors such as obesity, or it could reflect an increased susceptibility to infection, since it is well known that diabetes is a strong predictor of infection-related mortality (Bertoni et al. 2001), or it could be due to a zoonotic transmissible agent(s), since an infective origin of the disease has been postulated (Porte et al. 2003).

The significance of the increased risk of anterior horn cell disease, myasthenia gravis, and senile and pre-senile psychotic conditions (that includes Alzheimer's disease) is not known at the moment as the numbers of deaths involved are few, and the risks seem confined to particular subgroups. It is noted that a ten-fold increased risk of death from schizophrenia and a seven-fold increased risk of death for the category, other diseases of the spinal cord were observed in the entire poultry cohort (not shown in Table 4), but each of these conditions was based on only a single death, while none was reported in the much larger and older comparison group. The numbers of deaths involved for these neurologic diseases in the poultry cohort are sparse, but taken together, they suggest a consistent pattern. At the moment, all that can be said is that just as was previously hypothesized for cancers and other chronic diseases in the meat industry (Johnson 2005), a hypothesis that the present findings may perhaps be providing the first clues that cases of some of the neurologic diseases that occur in the general population may owe their origin to the presence of transmissible agents present in animals and animal products used for food, such as poultry, is plausible and would not be inconsistent with these results. The findings are also consistent with the recent report of an outbreak of progressive inflammatory neuropathy among swine slaughterhouse workers in a plant in Minnesota, illustrating that neurologic diseases are being caused by working in similar environments in the meat and poultry industries (Morbidity and Mortality Weekly Report (MMWR) 2008).

For hypertension, diseases of the esophagus, and peritonitis, the pattern of increased risks restricted to particular race/sex subgroups, and the small numbers of deaths involved also do not permit firm assessment of the significance of these findings at this time, as is the case with neurologic diseases. These could be chance findings, but they could be real, since the involvement of multiple sites in this study could be related to widespread infection, especially as similar findings have been reported in this same union for workers involved in the slaughtering and processing of cattle, pigs, and sheep (Johnson et al. 2007; Johnson and Zhou 2007). Also, in several instances (certain zoonotic bacterial diseases; for example, helminthiasis; myasthenia gravis,), the increased risks observed particularly in certain subgroups were extremely high suggesting a real association. Furthermore, the causes of death from neurologic diseases that were observed to be occurring in excess in the poultry

cohort were not observed to be in excess in the comparison group (SMR results not shown) that was much larger, older and had longer follow-up.

It is pertinent to note that the comparison group included a subset of workers engaged in oyster shucking, an occupational group with a possible pre-disposition to infection because of frequent cuts and injury to the skin associated with this job, and from being exposed to concentrated amounts of microorganisms present in oysters because of the filtering action of these shell fish. Thus, it may not be unexpected that when comparing the poultry group to the comparison group, the directly standardized risk ratio for certain zoonotic bacterial infection and helminthiasis were reduced though still elevated. We have no explanation for the deficit of deaths from intracerebral hemorrhage. Further follow-up might throw better light on these findings. It is interesting that it appears that non-malignant disease of the esophagus may be occurring in excess in the poultry cohort, since an excess of cancer of the esophagus was also observed in the cohort as well (Johnson et al. 2009), suggesting that the cancer may have been preceded by a pre-malignant lesion.

The quality of record keeping in the Baltimore Meatcutters Union from which this poultry cohort was originally derived was exceptional, to the extent that every subject who had ever applied to the union had a record, and this was independently checked by us by matching application slips with union dues records. Thus, selection bias is not an issue that could possibly explain the findings. Similarly, the assignment of race artificially to subjects for the SMR analysis does not appear to have created any significant bias in the results, since all diseases observed to be statistically significant in excess in the cohort in the SMR analysis were similarly observed to be statistically significant in excess in the PMR analysis in which complete information on race was available (except ischemic heart disease in white females, for which the PMR was 1.3 [95% CI 0.9–1.8]). In the analysis, persons whose vital status was unknown were assumed to be alive at the end of study; thus, the SMRs reported are an underestimation of the true risks.

In conclusion, the findings in this study are new. However, the retrospective cohort design that was used in the study cannot determine whether or not the excess of deaths observed were due to specific occupational exposures, nor was there sufficient statistical power, or opportunity to control for confounding factors. Furthermore, many comparisons were made, hence chance cannot be ruled out for some of the results. The logical next step is to extend follow-up and conduct nested case–control studies that will provide the opportunity to examine the role of candidate exposures in greater detail while controlling for occupational and non-occupational confounding factors. Because of these limitations and because it is the first time some of the findings are being reported, the results should be viewed as preliminary and hypothesis generating at the moment, and their full value may assume significance when they are confirmed in other much larger studies.

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References

Bertoni AG, Saydah S, Brancati FL (2001) Diabetes and the risk of infection-related mortality in the US. Diabetes Care 24:1044–1049

Fritschi L, Fenwick S, Bulsara M (2003) Mortality and cancer incidence in a cohort of meatworkers. Occup Environ Med 60:e4 (http://www.occenvmed.com/cgi/content/full/60/9/e4)

Johnson ES (1987a) Noncancer mortality in the meat industry: white males. J Occup Med 29(4):330–334

Johnson ES (1987b) Mortality from non-malignant diseases among women in the meat industry. Br J Ind Med 44:60–63

Johnson ES (2005) Meat workers—a suitable population for investigating possible role of some transmissible agents in chronic diseases in humans. Cellscience http://www.cellscience.com/journal/journalindex.asp July 27

Johnson ES, Zhou Y (2007) Non-cancer mortality in supermarket meat workers. JOEM 49(8):846–852

Johnson ES, Nicholson LG, Durack DT (1995a) Detection of antibodies to avian leukosis/sarcoma viruses (ALSV) and reticuloendotheliosis viruses (REV) in humans, by enzyme-linked immunosorbent assay (ELISA). Cancer Detect Prev 19:394–404

Johnson ES, Overby L, Philpot R (1995b) Detection of antibodies to avian leukosis/sarcoma viruses (ALSV) and reticuloendotheliosis viruses (REV) in humans, by Western blot assay. Cancer Detect Prev 19:472–486

Johnson ES, Shorter C, Rider B, Jiles R (1997) Mortality from cancer and other diseases in poultry slaughtering/processing plants. Int J Epidemiol 26(6):1142–1149

Johnson ES, Halabi S, Netto G, Lucier G, Bechtold W, Henderson R (1999) Detection of low level benzene exposure in supermarket wrappers by urinary muconic acid. Biomarkers 4:106–117

Johnson ES, Zhou Y, Sall M, El Faramawi M, Shah N, Christopher A, Lewis N (2007) Non-malignant mortality in meat workers—a model for studying the role of zoonotic transmissible agents in non-malignant chronic diseases in humans. OEM Online June 29, 10.1136/oem.2006.030825—Occup Environ Med 64:849–855

Johnson ES, Zhou Y, Yau CL, Prabhakar D, Ndetan H, Singh K, Preacely N (2009) Mortality from malignant diseases—update of the Baltimore union poultry cohort. Cancer Causes & Control. doi:10.1007/s10552-009-9452-6

Liddell FD (1984) Simple exact analysis of the standardised mortality ratio. J Epidemiol Community Health 38:85–88

McKercher PD, Hess WR, Hamdy F (1978) Residual viruses in pork products. Appl Environ Mecorbiol 35(1):142–145

Mead GC (1976) Microbiology of the poultry carcass and processing plant. R Soc Health J 96(4):164–167

Miettinen O (1976) Estimability and estimation in case-referent studies. Am J Epidemiol 103:226–235

Morbidity and Mortality Weekly Report (MMWR) (2008) Investigation of progressive inflammatory neuropathy among swine slaughterhouse workers—Minnesota, 2007–2008 57:1–3

Netto GF, Johnson ES (2003) Mortality in workers in poultry slaughtering/processing plants—the Missouri poultry cohort study. Occup Environ Med 60:784–788

Nordholm L, Espensen I, Jensen HS, Holst E (1986) Polycyclic aromatic hydrocarbons in smokehouses. Scand J Work Environ Health 12:614–618

Pauli G, Bessot JC, Kopferschmitt MC et al (1980) Meat wrapper's asthma: identification of the causal agent. Clin Allergy 10:263–269

Porte D Jr, Sherwin RS, Baron A (2003) Ellenberg & Rifkin's diabetes mellitus, 6th edn. McGraw-Hill Medical Publishing Division, New York

Saif YM, John Barnes H, Fadly AM, Swayne D, Glisson JR (eds) (2003) Diseases of poultry, 11th edn. Iowa State Press, Ames, IA

Sen NP, Donaldson B, Charbonneau C et al (1974) Effect of additives on the formation of nitrosamines in meat curing mixtures containing spices and nitrite. J Agric Food Chem 22(6):1125–1130

Vainiotalo S, Matveinen K (1993) Cooking fumes as a hygienic problem in the food and catering industries. Am Ind Hyg Assoc J 54:376–382

Vandervort R, Brooks SM (1977) Polyvinyl chloride film thermal decomposition products as an occupational illness. I. Environmental exposures and toxicology. J Occup Med 19:188–191