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Non-cancer mortality in poultry slaughtering/processing plant workers belonging to a union pension fund

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ABSTRACT

Background: The role of the biological environment in the occurrence of many chronic human diseases has been little studied. Humans are commonly exposed to transmissible agents that infect and cause a wide variety of subacute and chronic diseases in chickens and turkeys. The objective of this study is to investigate whether these agents cause similar diseases in humans, by studying workers in poultry slaughtering and processing plants who have one of the highest human exposures to these agents.
Methods: Mortality in poultry workers was compared with that in the United States general population through the estimation of standardized mortality ratios.
Results: Excess mortality from infectious and parasitic diseases was observed in the poultry workers. In addition, excess occurrences of deaths involving several sites of the cardiovascular, neurological, endocrine, gastrointestinal and reproductive systems, were observed, although the numbers involved were few in some instances.
Conclusion: The results indicate that poultry workers are at increased risk of dying from certain causes of death, including infections. This is consistent with other reports. Although it is possible that occupational exposure to transmissible agents present in poultry may be one of the causes of the excess occurrence of some of these diseases, other factors that were not considered because of the nature of the study design, could be equally important. Also, the small number of deaths involved in some instances calls for caution in interpreting the results. However, the study is important, as it has succeeded in newly identified areas that need further research, and which may have implications not only for workers, but also for the general population.

1. Introduction

The root cause of many chronic diseases in humans is still unknown. Chickens and turkeys destined for human consumption and their products are infected with a plethora of transmissible agents that cause a variety of diseases in the animals, including cancer, diseases of the nervous system, cardiovascular diseases, kidney diseases, etc. In addition, diseases that occur in humans such as leukemia, glioma, meningoencephalomyelitis, obesity, atrophy of the thymus, hypothyroidism, depression, paralysis, nephritis, aplastic anemia, gastrointestinal disease, etc. also occur in poultry (Diseases of Poultry, 2003; Ewert et al., 1990; Iwata et al., 2002; Payne, 1985; Whalen et al., 1988).

Thus, poultry birds are a potential source of infection for humans. Humans can be infected by direct contact with live or killed birds, their blood and secretions, consumption of raw or inadequately cooked poultry meat or other products such as eggs, and vaccination with vaccines grown in chicken embryo cells such as measles, mumps, etc. (Pham et al., 1999; Tsang et al., 1999). Serologic evidence indicates that humans are commonly infected with avian leukosis/sarcoma viruses, reticuloendotheliosis viruses and Marek's disease virus, that cause a wide variety of cancer, neurologic and other diseases in chickens and turkeys (Choudat et al., 1996; Johnson et al., 1995a,b).

The question therefore arises as to whether these agents also cause similar diseases in humans, especially those human diseases whose etiology is currently undetermined. We have been studying mortality in workers employed in poultry slaughtering/processing plants, who probably have one of the highest human exposures to these agents. We reason that if these agents cause disease in humans, it should be readily evident in this highly exposed group. We previously studied mortality in two separate cohorts of this type of workers, identified from union rosters in Maryland and Missouri (Johnson et al., 1986, 1997, 2009a,b, in press; Netto and Johnson, 2003). The new study of workers in poultry slaughtering and processing plants described later,

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reports the findings for non-malignant diseases, that were observed in the largest cohort studied to date. The results for cancer mortality for this new cohort have been separately published (Johnson et al., 2010).

2. Materials and methods

The study population consists of 20,132 workers who were working between 1972 and 1990 in 11 poultry slaughtering/processing plants located in five states in the United States (Arkansas, Louisiana, Maine, Missouri, Texas). The workers were identified from the roster of a Pension Fund administered by the United Food and Commercial Workers International Union. The cohort was completely defined, since with the aid of multiple types of records available at the Fund, we were able to check for completeness. They were followed up for mortality from January 1, 1972 to December 31, 2003, during which time 2454 of the workers died. Methods of follow-up included Social Security Administration, the National Death Index, Pension Benefit Information Inc., (a private company that identifies deaths), State Departments of Motor Vehicles, State Department of Vital Records, personal contact by telephone and mail, internet tracing methods, etc. One hundred and fifty-six subjects, (0.8% of the cohort) with missing date of birth, had their date of birth imputed based on the median year of birth of similar subjects with known date of birth who joined the union the same year as they did. This measure was deemed preferable, as the total person-years will be affected to a negligible degree, and thus adopted in favor excluding such subjects from the study, which might introduce bias.

Standardized mortality ratios (SMR) were estimated with the US general population as the comparison group, using the OCMAP Plus software from the University of Pittsburgh, USA. Altogether, a total of 130 separate causes of death from non-malignant diseases were investigated — see complete list in Johnson et al. (2009a). This is a marked improvement over occupational studies which typically in the past have used software that permitted the examination of usually less than 30 causes of non-malignant deaths, because of the practice of grouping causes together.

Information on race was available only for deceased individuals with a death certificate/known cause of death. Therefore for the SMR analyses, a race was randomly assigned to each of the 17,641 individuals in the study whose race was unknown, based on the racial distribution of deceased persons with known race. (A comparison of the current racial distribution of a random sample of more than half the membership of a similar union we had studied had revealed that it was not different from that in deceased workers).

The cohort was stratified by plant, and then stratified into four subgroups by race and sex (black males, black females, white males, and 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). Membership in the union or Fund was compulsory from the first day of employment. For deceased individuals, person-years were enumerated up to the date of death. For persons not known to have died, person-years were computed up to the date of termination of the study on December 31, 2003. Expected deaths were derived by multiplying the person-years in each cell by the corresponding gender-, calendar year-, age-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 chi-square distributions (Liddell, 1984). Because of the imputing of missing information on race, sensitivity analyses were conducted on the cohort as a whole, assuming the entire cohort was white, or black.

3. Results

Table 1 gives the distributions of subjects by location, and deaths by location and gender. Fifty-two percent of the cohort were females; 67% were born in 1950 or later. Of the total of 2454 deaths, 1661 (68%) were white. Table 2 gives the distribution of the cohort by age, person-years and years since first employment. The average duration of follow-up was 23.8 years.

The main findings of the SMR analyses are given in the Table 3. Statistically significantly elevated risks in the cohort as a whole, and affecting nearly all race/sex subgroups, were observed for deaths from, 1) ICD 001-009 (intestinal infectious diseases); 2) ICD 030-041 (other bacterial diseases); 3) ICD 240-246 (disorders of the thyroid gland); 4) ICD 295 (schizophrenic disorders); and 5) ICD 410-414 (ischemic heart diseases).

Statistically significantly elevated risks were recorded in women only, for deaths from mycoses; helminthiasis; benign neoplasms of the thyroid and other endocrine glands; diabetes; encephalitis; subarachnoid hemorrhage; and intracerebral hemorrhage. The SMR for pulmonary embolism and infarction, was significantly elevated in men only.

Significantly elevated risks confined to non-whites include, deaths from regional enteritis and ulcerative colitis; diseases of the prostate; and inflammatory disease of the ovary, fallopian tube, pelvic cellular tissue, and peritoneum.

Isolated elevated risks were observed for deaths from functional diseases of the heart in white males, and deaths from rheumatoid arthritis, etc.

A more detailed breakdown of individual conditions that constitute the group, for specific causes of death observed to be occurring in excess, is given in Table 4.

Significantly depressed risks were observed for deaths from cirrhosis of the liver, chronic liver disease, etc., accidental poisoning by solid, liquid, gas, etc., and from suicide, and self-inflicted injury.

The sensitivity analyses (not shown) indicate that each of the cause-specific SMRs for *the entire cohort* that is statistically significantly elevated in Table 3, still remains so when the entire cohort is assumed to be white. Also, all the other SMRs (whether statistically significant or not) either remained the same, or became increased by between 0% and 38%, except those for intracerebral hemorrhage and diseases of the ovary and fallopian tubes that increased by 50% and 89%, respectively. The SMR for suicide and self injury decreased from 0.7 to 0.6. When the entire cohort was assumed to be non-white, each of the cause-specific SMRs for the *entire cohort* decreased by 0% to 49%, with two exceptions – the SMR for disorders of the thyroid gland increased from 22.2 to 22.7, and the SMR for regional enteritis and ulcerative colitis increased from 1.9 to 2.0.

4. Discussion

In the most extreme case in the sensitivity analysis, the change in SMRs was less than 90%. Since the majority of the increased risks observed were at least 2-fold or much higher, imputing the race for non-deceased subjects in the analysis does not appear to have been associated with any serious bias. Furthermore, lost subjects were assumed to be alive, thus the reported cause-specific SMRs are conservative. Overall, this cohort did not demonstrate the “healthy worker effect”, possibly because of the influence of increased mortality from cardiovascular diseases, and all cancers (Johnson et al., 2010). This is not an infrequent finding in occupational studies in general, nor in studies in the meat and poultry industries (Guberan et al., 1993; Johnson et al., 2009a; McLean et al., 2004; Meijers et al., 1989).

Eighteen of the 130 causes of death examined were observed to be significantly in excess in this cohort of poultry workers. These include deaths from bacterial diseases, fungal diseases, helminth diseases and possibly viral diseases (encephalitis) and septicemia. Thus the workers appear to be dying at an increased rate from infections caused by virtually all the major groups of microorganisms. This finding is consistent with the fact that these workers have a high potential for exposure to poultry and their microbial agents: 1) they handle and are exposed to a large volume of animals daily; in a typical large poultry plant, more 75,000 chickens are killed and processed daily; 2) they have the most intimate contact with the blood, body fluids, and interior organs of the poultry birds; and 3) cuts from sharp knives and bone splinters, penetrating wounds, breaches of the skin as a result of dermatitis caused by irritant body fluids such as enzymes are frequent occurrences, and all provide ready access for micro-organisms that are present in the birds and their raw products, for entry into the body (Cai et al., 2005). Infection can also occur via the airborne route (Harris et al., 1962).

Table 1
Location and demographic make-up of poultry plants.

Plant	Located	State	No. of subjects	Deceased whites			Deceased nonwhites			All deaths
				Males	Females	Total	Males	Females	Total	
1 (12018)	Natchitoches	Louisiana	737	7	6	13 (15.5)	32	39	71 (84.5%)	84
2 (12030)	Alexandria	Louisiana	419	0	0	0 (0)	43	27	70 (100.0%)	70
3 (13019)	Carthage	Missouri	523	29	27	56 (88.9)	4	3	7 (11.1%)	63
4 (21055)	Arcadia	Louisiana	7	2	0	2 (100)	0	0	0 (0.0%)	2
5 (24010)	Lewiston	Maine	1296	98	53	151 (93.8)	9	1	10 (6.2%)	161
6 (24011)	Belfast	Maine	803	74	50	124 (96.1)	3	2	5 (3.9%)	129
7 (24012)	Belfast	Maine	1658	128	108	236 (97.5)	4	2	6 (2.5%)	242
8 (32710)	El Dorado	Arkansas	3319	47	27	74 (17.7)	170	174	344 (82.2%)	418
9 (32830)	Russellville	Arkansas	10227	512	441	953 (82.9)	112	85	197 (17.1%)	1150
10 (35045)	Lufkin	Texas	943	24	21	45 (41.3)	32	32	64 (58.7%)	109
11 (44220)	El Dorado	Arkansas	200	6	1	7 (26.9)	15	4	19 (73.0%)	26
Total			20,132	927	734	1661 (67.7)	424	369	793 (32.3%)	2454

The increased risk of infections is also consistent with our hypothesis that the excess occurrence of some of these diseases may represent occupational zoonoses. However, it is also possible that the close contact workers in the production line have with each other, could facilitate person-to-person transmission of infections at a high rate, and thus some of the diseases in excess may represent anthroponoses. High background rates in the underlying population from which the study population was derived such as populations of low socioeconomic status, or migrant populations, is also a possible explanation for the observed excess of infectious diseases. Thus the available data do not permit the determination of whether the excesses of infectious, parasitic and other diseases observed in the cohort were occupationally- or non-occupationally-induced. Similarly, deaths from individual organisms are too few to permit meaningful interpretation at this time.

It is not clear why deaths from leprosy a disease which is not a zoonosis should appear to be occurring in excess. Migrant workers in the study population from countries with high background rates of the disease compared to the very low rates recorded in indigenous US residents is one possible explanation.

The excess occurrence of deaths seen in the cohort also involved the endocrine, cardiovascular, reproductive, gastrointestinal, and the central nervous systems. Although it is possible that some of these diseases could have resulted from widespread dissemination of microorganisms during bacteremia, septicemia, or parasitemia, other candidates for explaining the excesses may include selection bias (poultry workers are from the lowest socioeconomic class, a group likely to have poor hygiene and higher prevalence of infection and many chronic diseases). Also, the poultry industry may disproportionately admit workers with existing chronic diseases into the workforce. However, in the case of disorders of the thyroid gland, the underlying condition responsible seems to be thyrotoxicosis, since 19 of the 21 deaths in this group were due to thyrotoxicosis, or post-surgical hypothyroidism which usually results after surgery for thyrotoxicosis. Thus this is unlikely to be a chance finding.

Increased risks in some cases involved all race/sex groups, while in others they were confined to a particular race or sex subgroups. This may reflect actual differences in the types of jobs conducted. These differences are well known to occur in the meat and poultry industries. For example, in one of our previous studies of cattle, pigs, and sheep abattoirs, slaughtering of animals was mainly done by blacks; prior to the 1970s meatcutters in supermarkets were almost exclusively white, and wrapping was predominantly a female job (Johnson, 1991, 1994). Also, we have conducted industrial hygiene assessments using antibodies against two of the viruses these workers are exposed to, and shown that antibody titers did vary depending on the task performed (Choi & Johnson, 2010; second manuscript currently under journal review). Similarly nested case–control studies of lung, pancreatic and liver cancers we have conducted within this cohort and others combined, indicated that risks varied with the type of job a worker performed, with slaughtering being one of the tasks associated with the highest risks (manuscripts currently under review). On the other hand, these different patterns of risk may reflect intrinsic susceptibility to particular diseases for some race/sex subgroups seen in the general population. It is well known for example that thyroid disease, is more common in females, and diseases of the prostate and diabetes tend to be more common in non-whites. The lack of sufficient subjects in a particular race or sex subgroup(s), or differences in age could also be the reason for the differences.

A comparison of the results with those of the only two other poultry cohorts in the literature studied for non-malignant disease occurrence, reveals that of nine distinct causes of death observed to be significantly occurring in excess in the Baltimore cohort (bacterial diseases, helminthiasis, diabetes, schizophrenic disorders, myasthenia gravis, ischemic heart disease, anterior horn disease, hypertension, and other diseases of kidney and ureter), only the last three were not

Table 2
The distributions of age, person-years and years since first employment of pension fund poultry workers.

Interval in years	Age distribution of poultry cohort			Total person-years by age		Years since first employment in poultry cohort		
	No. of subjects	Percentage	Cumulative percentage	No. of person-years	Percent of person-years	No. of subjects	Percentage	Cumulative percentage
0–9	0	0.00	0.00	0.00	0.00	526	2.61	2.61
10–19	5	0.02	0.02	4.17	0.00	3239	16.09	18.70
20–29	162	0.80	0.83	719.17	0.15	14,333	71.20	89.90
30–39	1277	6.34	7.17	22,583.87	4.72	2034	10.10	100
40–49	8710	43.26	50.44	20,4405.40	42.70	0	0	–
50–59	5925	29.43	79.87	14,9987.60	31.34	0	0	–
60–69	2452	12.18	92.05	59,776.74	12.49	0	0	–
70–79	1177	5.85	97.89	29,594.62	6.18	0	0	–
≥80	424	2.11	100	11,581.80	2.42	0	0	–
Total	20,132	100		478,653.37	100	20,132	100	

Table 3
Standardized Mortality Ratios for Non-cancers for the Period 1972 to 2003 – Poultry Workers (UFCW Pension Fund).

Cause of death (ICD 9th Revision)	Poultry Workers								
	Non-white Males N = 2,849	White Males N = 6,782	All Males N = 9,631	Non-white Females N = 3,550	White Females N = 6,951	All Females N = 10,501	All Non-whites N = 6,399	All Whites N = 13,733	All Groups N = 20,132
	Obs. SMR	Obs. SMR	Obs. SMR	Obs. SMR	Obs. SMR	Obs. SMR	Obs. SMR	Obs. SMR	Obs. SMR
Intestinal infectious diseases	1	2	3	0	1	1	1	3	4
(ICD 001-009)	8.0 (0.2 – 44.6)	8.6 (1.0 – 30.9)	8.4 (1.7 – 24.5)	-	2.9 (0.1 – 16.2)	**2.1 (0.1 – 11.6)**	**3.8 (0.1 – 21.3)**	5.2 (1.1 – 15.2)	**4.8 (1.3 – 12.2)**
Other bacterial diseases	3	9	12	7	13	20	10	22	32
(ICD 030-041)	0.6 (0.1 – 1.7)	1.8 (0.8 – 3.4)	1.2 (0.6 – 2.1)	**1.2 (0.5 – 2.4)**	2.2 (1.2 – 3.8)	**1.7 (1.0 – 2.6)**	**0.9 (0.4 – 1.7)**	2.0 (1.3 – 3.1)	**1.5 (1.0 – 2.1)**
Mycoses	0	0	0	4	1	5	4	1	5
(ICD 110-118)	-	-	-	**6.0 (1.6 – 15.5)**	1.6 (0.0 -9.0)	**3.9 (1.3 – 9.1)**	**2.6 (0.7 – 6.6)**	0.7 (0.0 – 3.6)	**1.6 (0.5 – 3.8)**
Helminthiasis	0	0	0	0	3	3	0	3	3
(ICD 120-129)	-	-	-	-	230 (47.5 – 673)	154 (31.7 – 450)	-	87.2 (18.0 – 255)	**54.8 (11.3 – 160)**
Benign neoplasm of thyroid/other unspec endocr	0	0	0	1	1	2	1	1	2
(ICD 226	-	-	-	**15.2 (0.4 – 84.8)**	15.7 (0.4 – 87.4)	**15.4 (1.9 – 55.8)**	**8.4 (0.2 – 46.8)**	8.7 (0.2 – 48.6)	**8.6 (1.0 – 30.9)**
Disorders of thyroid gland	5	4	9	3	9	12	8	13	21
(ICD 240-246)	43.8 (14.2–102)	33.4 (9.1–85.6)	38.5 (17.6–73.1)	**9.4 (1.9 – 27.4)**	23.0 (10.5–43.6)	**16.9 (8.7–29.5)**	**18.4 (8.0–36.3)**	25.4 (13.5 – 43.5)	**22.2 (13.8 – 34.0)**
Diabetes	11	15	26	13	26	39	24	41	65
(ICD 250)	1.0 (0.5 – 1.8)	1.1 (0.6 – 1.7)	1.0 (0.7 – 1.5)	**0.8 (0.4 – 1.3)**	1.6 (1.0 – 2.3)	**1.1 (0.8 – 1.6)**	**0.9 (0.5 – 1.3)**	1.3 (0.9 – 1.8)	**1.1 (0.8 – 1.4)**
Schizophrenic Disorders	3	0	3	2	2	4	5	2	7
(ICD 295)	43.0 (8.9–126)	-	18.1 (3.7 – 52.8)	**33.6 (4.1 – 121)**	20.9 (2.5 – 75.4)	**25.7 (7.0 – 65.9)**	**38.7 (12.6 –90.2)**	10.4 (1.3 – 37.6)	**21.8 (8.8 – 44.9)**
Encephalitis	0	0	0	0	2	2	0	2	2
(ICD 323)	-	-	-	-	17.8 (2.2 – 64.2)	8.9 (1.1 – 32.3)	-	8.0 (1.0 - 29.1)	4.2 (0.5 – 15.3)
Myasthenia Gravis	0	0	0	1	0	1	1	0	1
(ICD 358.0)	-	-	-	12.6 (0.3 – 70.1)	-	4.8 (0.1 – 26.8)	7.9 (0.2 – 44.0)	-	2.9 (0.1 – 16.0)
Ischemic Heart disease	79	182	261	56	119	175	135	301	436
(ICD 410-414)	1.4 (1.1 – 1.7)	1.4 (1.2 – 1.6)	1.4 (1.2 – 1.6)	1.1 (0.8 – 1.5)	1.4 (1.1 – 1.7)	1.3 (1.1 – 1.5)	1.3 (1.1 – 1.5)	1.4 (1.2 – 1.5)	1.3 (1.2 – 1.5)
Functional diseases of the heart	6	22	28	6	12	18	12	34	46
(ICD 426,427)	0.7 (0.3 – 1.5)	1.8 (1.1 – 2.7)	1.3 (0.9 – 1.9)	0.6 (0.2 – 1.4)	1.1 (0.5 – 1.8)	0.9 (0.5 – 1.4)	0.7 (0.3 – 1.2)	1.4 (1.0 – 2.0)	1.1 (0.8 – 1.5)
Subarachnoid hemorrhage	1	3	4	1	11	12	2	14	16
(ICD 430)	0.5 (0.0 – 3.0)	1.1 (0.2 – 3.1)	0.8 (0.2 – 2.2)	0.2 (0.0 – 1.3)	2.2 (1.1 – 3.9)	1.3 (0.7 – 2.3)	0.3 (0.0 – 1.2)	1.8 (1.0 - 3.0)	1.1 (0.7 – 1.9)
Intracerebral hemorrhage etc	2	9	11	1	16	17	3	25	28
(ICD 431,432)	0.3 (0.0–1.0)	1.5 (0.7 – 2.9)	0.8 (0.4 – 1.5)	0.1 (0.0 – 0.7)	2.3 (1.3 – 3.7)	1.2 (0.7 – 1.8)	0.2 (0.0 – 0.6)	1.9 (1.2 – 2.8)	1.0 (0.7 – 1.4)
Pulmonary embolism & infarction	4	6	10	4	3	7	8	9	17
(ICD 415)	1.8 (0.5 – 4.7)	2.4 (0.9 – 5.2)	2.1 (1.0 – 3.9)	1.3 (0.4 – 3.3)	0.9 (0.2 – 2.7)	1.1 (0.4 – 2.3)	1.5 (0.7 – 3.0)	1.6 (0.7 – 2.9)	1.5 (0.9 – 2.5)
Regional enteritis, ulcerative colitis, other	1	0	1	2	0	2	3	0	3
(ICD 555-556 , 558)	5.1 (0.1 – 28.6)	-	1.6 (0.0 – 8.7)	7.7 (0.9 – 27.9)	-	2.1 (0.3 – 7.7)	6.6 (1.4 – 19.3)	-	1.9 (0.4 – 5.6)
Cirrhosis/chron liver dis., liver abcess, etc.	7	8	15	5	11	16	12	19	31
(ICD 571-573)	0.5 (0.2 – 1.1)	0.4 (0.2 – 0.8)	0.5 (0.3 – 0.8)	0.5 (0.2 – 1.2)	0.9 (0.5 – 1.7)	0.7 (0.4 – 1.2)	0.5 (0.3 – 0.9)	0.6 (0.4 – 1.0)	0.6 (0.4 – 0.8)
Diseases of prostate	2	0	2	-	-	-	2	0	2
(ICD 600-602)	23.4 (2.8–84.5)	-	9.2 (1.1 – 33.2)				23.4 (2.8-84.5)	-	9.2 (1.1 – 33.2)
Disease of ovary, fallopian tubes	-	-	-	2	0	2	2	0	2
(ICD 614-620)				10.2 (1.2 – 36.7)	-	6.5 (0.8 – 23.5)	10.2 (1.2-36.7)	-	6.5 (0.8 – 23.5)
Rheumatoid arthritis , etc	2	0	2	0	1	1	2	1	3
(ICD 714,720)	25.0 (3.0 -90.2)	-	5.6 (0.7 – 20.2)	-	1.1 (0.0 – 6.2)	0.8 (0.0 – 4.7)	5.4 (0.7-19.5)	0.9 (0.0 – 4.8)	1.9 (0.4 – 5.7)
Accidental poisoning by sol/liq/gas/vap	7	7	14	0	2	2	7	9	16
(ICD E850-E869)	1.0 (0.4 – 2.0)	0.5 (0.2 – 1.1)	0.7 (0.4 – 1.2)	-	0.4 (0.1 – 1.6)	0.3 (0.0 – 0.9)	0.7 (0.3 – 1.4)	0.5 (0.2 – 1.0)	0.6 (0.3 – 0.9)
Suicide & self-inflicted injury	10	33	43	1	2	3	11	35	46
(ICD E950-959)	1.0 (0.5 – 1.8)	0.8 (0.6 – 1.1)	0.8 (0.6 – 1.1)	0.3 (0.0 – 1.8)	0.2 (0.0 – 0.6)	0.2 (0.0 – 0.6)	0.8 (0.4 – 1.5)	0.7 (0.5 – 0.9)	0.7 (0.5 – 0.9)
All causes of death	424	927	1351	369	734	1103	793	1661	2454
	0.9 (0.8 – 1.0)	1.3 (1.2 – 1.4)	1.2 (1.1 – 1.2)	**1.0 (0.9 – 1.1)**	1.3 (1.2 – 1.4)	**1.1 (1.1 – 1.2)**	**0.9 (0.9 – 1.0)**	1.3 (1.2 – 1.4)	**1.2 (1.1 – 1.2)**

Figures in parentheses are 95% confidence interval.

Table 4
Detailed listing of component diseases belonging to causes of death observed to be occurring in excess.

Cause of death (ICD) [Observed deaths]	Cause of death (ICD) [Observed deaths]	Cause of death (ICD) [Observed deaths]	Cause of death (ICD) [Observed deaths]	Cause of death (ICD) [Observed deaths]	Cause of death (ICD) [Observed deaths]	Cause of death (ICD) [Observed deaths]	Cause of death (ICD) [Observed deaths]
Intestinal infectious diseases (ICD 001-009) [N = 4]	Other bacterial diseases (ICD 030-041) [N = 32]	Mycosis (ICD 110-118) [N = 5]	Helminthiasis (ICD 120-129) [N = 3]	Benign neopl. thyroid and other unspecif. endoc (ICD 226-227) [N = 2]	Disorders of thyroid gland (ICD 240-246) [N = 21]	Regional enteritis, ulcerative colitis, other (ICD 555-556, 558) [N = 3]	Rheumatoid arthritis, etc. (ICD 714,720) [N = 3]
Cholera [N = 1]	Leprosy [N = 16]	Blastomycotic infection [N = 2]	Echinococcosis [N = 3]	Benign neoplasm of thyroid [N = 2]	Thyrotoxicosis with and without goiter [N = 11]	Other and unspec. noninfectious gastroenteritis and colitis [N = 2]	Rheumatoid arthritis and other inflammatory polyarthropathies [N = 3]
Other *Salmonella* Inf [N = 1]	Septicemia [N = 14]	Dermatophytosis [N = 1]	Other trematode infections	Benign neoplasm of other endocrine gls.	Acquired hypothyroidism [N = 8] *All 8 were post-surgical hypothyroid	Regional enteritis [N = 1]	Ankylosing spondylitis and other inflammatory spondylitis
Intestinal infections due to other orgs. [N = 1]	Meningococcal infection [N = 1]	Coccidioidomycosis [N = 1]	Schistosomiasis		Congenital hypothyroidism [N = 1]	Vascular insufficiency of intestine	
Ill-defined intestinal diseases [N = 1]	Bacterial infections in conditions elsewhere and unspecified site [N = 1]	Other mycoses [N = 1]	Other cestode infections		Other diseases of thyroid [N = 1] *Unspec. hypothy.	Ulcerative colitis	
Other food poisoning bacteria	Strep. sore throat and scarlet fever	Histoplasmosis	Trichinosis		Simple and unspecified goiter		
Amoebiasis	Erysipelas	Candidiasis	Filarial infection and dracontiasis		Nontoxic nodular goiter		
Other protozoal intestinal diseases	Diphtheria	Dermatomycosis, other and unspec.	Ancylostomiasis and necatoriasis		Thyroiditis		
Typhoid/paratyphoid	Tetanus	Opportunistic mycoses	Other intestinal helminthiasis				
Shigellosis	Whooping cough		Other and unspecified helminthiasis				
	Actinomycotic infections		Intestinal parasitism unspecified				
	Other bacterial diseases						
	Disease due to other mycobacteria						

in excess in the cohort reported here. Similarly, of the four causes of death that were significantly in excess in the Missouri cohort (bacterial diseases, diseases of the thyroid gland, helminthiasis, schizophrenic disorders), all four were also significantly in excess in this study. High risks of death from infectious and parasitic diseases and many organs and systems have also been reported in a cohort of workers employed in plants where cattle, pigs, and sheep were slaughtered and processed (Johnson et al., 2007). It is therefore concluded that there is good reason to suspect that the excess deaths from at least some of the diseases observed in this cohort may represent real risks associated with working in poultry slaughtering and processing plants.

The results for neurologic diseases warrant particular attention: the Baltimore study reported high risks of death in the entire cohort from both myasthenia gravis (SMR = 14.5, 95% CI 1.8–52.4) based on 2 deaths, and anterior horn disease (SMR = 3.9, 95% CI 1.1–9.9) based on 4 deaths (Johnson et al., 2009a). In the present study, there is an indication that myasthenia gravis may be occurring in excess also (SMR = 12.6 in nonwhite females, or SMR = 2.9 overall), although this was based on a single death. No death from anterior horn cell was recorded, but only 0.4 was expected (not shown). In the Missouri cohort no death from myasthenia gravis was recorded, and for anterior horn cell the SMR was 1.4 in females (Johnson et al., in press; Netto and Johnson, 2003). For schizophrenia, in the entire Baltimore poultry cohort, the SMR was 10.3, 95% CI, 0.3–57.5 (based on a single death); in the entire Missouri cohort, the SMR was 16.3, 95% CI 4.4–41.6 (N = 4); and in the current study, for the entire cohort the SMR is 21.8, 95% CI, 8.8–44.9 (N = 7). Although the number of deaths involved from these neurologic diseases in all three cohorts is relatively small, the very high relative risks observed for these causes in these cohorts and the consistency may indicate that this may be more than a chance occurrence.

5. Conclusion

We postulate that infection from a variety of microorganisms which these workers are exposed to at work, may be the underlying cause of the excess of at least some of these diseases. It is well known for example that poultry can be the source of human cases of salmonellosis, *E. coli* infection, coccidioidomycosis, Newcastle disease virus infection, psittacosis, etc. It is not possible from this type of retrospective study to rule out other possible explanations mentioned earlier. Thus it is possible that some of the associations observed are not related to the occupational experience of these workers. Furthermore small numbers of deaths were involved for some causes. Hence caution should be exercised in interpreting the results, which should be considered preliminary at this time, and worthy of further investigation. In spite of these limitations, the findings may be important not only for workers in this occupational group, but also for the general population which is also widely exposed to poultry zoonotic transmissible agents. Some of the diseases observed to be occurring in excess like schizophrenic disorders cause severe debilitating diseases and the cause is presently unknown. Hence any clues to their etiology warrant some consideration. Confirmatory studies of adequate statistical power are required, and nested case–control studies are needed to investigate these associations in greater detail. Similarly, small-scale clinical trials assessing the effect of adding antimicrobial or anti-parasitic therapy to the usual management of some of the neurologic and other diseases that are occurring in excess in this cohort, may provide an opportunity to test the hypothesis that these agents are involved in the occurrence of these diseases. Likewise, consideration should be given to initiating studies that will search for evidence of infection with poultry microbial agents in general population cases of diseases reported here to be in excess in poultry workers.

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