Environment International 37 (2011) 322–327

Contents lists available at ScienceDirect

Environment International

journal homepage: www.elsevier.com/locate/envint

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

Eric S. Johnson [a,*], Harrison Ndetan [b]

[a] University of North Texas Health Science Center, Department of Epidemiology, USA
[b] University of North Texas Health Science Center, Department of Biostatistics, USA

ARTICLE INFO

Article history:
Received 18 June 2010
Accepted 10 September 2010
Available online 12 November 2010

Keywords:
Infections
Infectious diseases
Chickens
Turkeys
Zoonosis

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,

* Corresponding author. Department of Epidemiology, School of Public Health, University of North Texas Health Science Center, 3500 Camp Bowie Blvd. Fort Worth, TX 76107, USA. Tel.: +1 817 735 0327; fax: +1 817 735 0443.
E-mail address: Eric.johnson@unthsc.edu (E.S. Johnson).

0160-4120/$ – see front matter © 2010 Elsevier Ltd. All rights reserved.
doi:10.1016/j.envint.2010.09.007

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).