

## Mortality from malignant diseases—update of the Baltimore union poultry cohort

Eric S. Johnson · Yi Zhou · C. Lillian Yau ·  
Deepak Prabhakar · Harrison Ndetan ·  
Karan Singh · Nykiconia Preacely

Received: 19 March 2009 / Accepted: 6 October 2009 / Published online: 22 October 2009  
© Springer Science+Business Media B.V. 2009

**Abstract** We previously studied mortality up to 1989 in 2,639 members of a local union who had *ever* worked in poultry slaughtering and processing plants, because they were exposed to oncogenic viruses present in poultry. In this report, cancer mortality was updated to the year 2003 for 2,580 of the 2,639 subjects who worked *exclusively* in poultry plants. Mortality in poultry workers was compared with that in the US general population through the estimation of proportional mortality and standardized mortality ratios separately for each race/sex group and for the whole cohort. Compared to the US general population, an excess of cancers of the buccal and nasal cavities and pharynx (base of the tongue, palate and other unspecified mouth, tonsil and oropharynx, nasal cavity/middle ear/accessory sinus), esophagus, recto-sigmoid/rectum/anus, liver and intrabiliary system, myelofibrosis, lymphoid leukemia and multiple myeloma was observed in particular subgroups or in the entire poultry cohort. We hypothesize that oncogenic viruses present in poultry, and exposure to fumes, are candidates for an etiologic role to explain the excess occurrence of at least some of these cancers in the poultry workers. Larger studies

which can control for confounding factors are urgently needed to determine the significance of these findings.

**Keywords** Poultry slaughtering/processing · Cancer · Viruses · Fumes

### Introduction

Workers in poultry slaughtering and processing plants are exposed to oncogenic viruses that naturally infect and cause a variety of cancers in chickens and turkeys. These viruses include the avian leukosis/sarcoma viruses (ALSV), reticuloendotheliosis viruses (REV), and Marek's disease virus (MDV), some members of which are the most potent cancer-causing agent known in animals [1]. Other potentially carcinogenic occupational exposures include exposure to fumes emitted from the wrapping machine [2, 3], nitrosamines during the curing of poultry [4, 5], and to smoke or aerosol emitted during smoking or cooking of poultry products that contain polycyclic aromatic hydrocarbons and heterocyclic amines [5–7]. To date, however, only our two studies in Baltimore and Missouri in the USA, and a third from Australia, have reported on cancer mortality or incidence specifically in these workers [8–11]. The Australian study of union workers in poultry plants did not provide mortality data on any specific cancer type, but in cancer incidence analysis provided information on colon cancer and melanoma, neither of which was occurring in excess. Reports for other sites were not available because of small numbers [11]. The Missouri study also had low power, and although some cancers were observed to be slightly in excess, none was statistically significant [10]. We initially studied mortality in a cohort of 2,639 workers who had *ever* worked in poultry slaughtering and processing plants and who were members

---

E. S. Johnson (✉) · Y. Zhou · D. Prabhakar · N. Preacely  
Department of Epidemiology, School of Public Health,  
University of North Texas Health Science Center, 3500 Camp  
Bowie Blvd., Fort Worth, TX 76107, USA  
e-mail: ejohnson@hsc.unt.edu

Y. Zhou · C. Lillian Yau  
School of Public Health & Tropical Medicine, Tulane  
University, New Orleans, LA, USA

H. Ndetan · K. Singh  
Department of Biostatistics, School of Public Health, University  
of North Texas Health Science Center, 3500 Camp Bowie Blvd.,  
Fort Worth, TX 76107, USA

of a local union in Baltimore, Maryland, from 1954 to 1989 [8, 9]. Statistically significant increased standardized mortality ratios (SMR), relative risks, or odds ratio for all cancers, cancers of the liver, pancreas, rectum, lung, and hemopoietic and lymphatic systems were observed in previous studies of this cohort in the cohort as a whole or in at least one race/sex subgroup [8, 9, 12]. At the end of the last follow-up, 380 (14%) of the cohort had died [9]. We report here on an update of mortality in this cohort, but this time, restricting the analysis to 2,580 subjects (98%) who worked *exclusively* in poultry slaughtering and processing plants during their entire union membership. Also this time, a total of 51 different cancer types were investigated as compared with 25 and 33 in the previous follow-ups, and follow-up has been extended to the end of 2003, by which time 790 (31%) of the cohort were now deceased.

## Materials and methods

The source population was a total of 28 900 members of a local meatcutters' union in Baltimore, drawn from all sections of the meat and poultry industries. The study population was defined as all subjects in the union who had worked exclusively in six poultry slaughtering and processing plants during their union membership, between 1954 and 1979 ( $n = 2580$ ). Methods of follow-up and statistical analyses have been described in detail in previous publications [8, 9, 13]. In brief, 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, internet tracing methods, etc. 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 not identified as dead were assumed to be alive at the end of the study. Of the 790 deaths, we were able to obtain cause of death information for all but 34 (4.3%). All persons with a death certificate had complete information on date of birth. However, date of birth information was missing for 197 poultry subjects (7.5%), who are all not known to have died. Rather than excluding such persons from the analysis, their date of birth was imputed based on the median year of birth of workers with known date of birth joining the union during 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 who joined the union that particular year. This crude measure was deemed to be associated with negligible bias, since the total person-years will be affected to a very non-significant degree.

Statistical analyses involved estimation of SMR and proportional mortality ratios (PMR), stratifying on age, calendar time, gender, race, and plant, using the US general population as the comparison group. Information on race was available only for deceased individuals with a death certificate. Therefore to perform the SMR analyses, race was artificially assigned at random to each individual in the study without a death certificate, based on the known racial distribution of the union from a survey of its current membership carried out initially at the time the union was first studied in 1979. 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). Person-years were accumulated from 1 January, 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 31 December 2003 whichever was earlier. 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 [14].

A similar method of stratification was used to estimate the PMR, 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 observed to expected deaths is the PMR. The variance was calculated assuming a binomial distribution for the observations [15].

The protocol for this study was approved by the UNT Health Science Center Institutional Review Board.

## Results

The age distribution of the cohort is as follows: 8.6% were born before 1920, 38.5% were born between 1920 and 1939,

**Table 1** Number of subjects, person-years at risk, No. of deaths, by race and gender, 1954–2003 (poultry workers)

	Males			Females			Total		
	No. of deaths	No. of subjects	No. of person-years	No. of deaths	No. of subjects	No. of person-years	No. of deaths	No. of subjects	No. of person-years
White	252	372	12,131.7	132	478	16,764.3	120	850	28,896.0
Non-white	538	999	32,191.5	340	731	25,319.8	198	1,730	57,511.3
Total	790	1,371	44,323.2	472	1,209	42,084.1	318	2,580	86,407.3

The race of the number of subjects and person-years is artificially assigned

and 52.8% were born between 1940 and 1959. A total of 790 deaths were recorded for the 2,580 poultry workers, and the total number of person-years contributed was 86,407.3 (Table 1). Deaths from cancers overall were not significantly elevated in any race/sex group among the poultry workers (Table 2). In Table 2, the results are for causes of death for which more than one death occurred in the poultry cohort and for which a statistically significant SMR or PMR was observed in any race/sex subgroup or in the entire poultry cohort. Certain cancer types were observed to be in excess across race or sex subgroups—these are cancers of the base of tongue; palate and other unspecified mouth; nasal cavity/mid ear/accessory sinuses; liver/intrabiliary; malignant immunoproliferative disease/myeloma/plasma cell; and myelofibrosis. On the other hand, the observed excess of cancers of the tonsil and oropharynx, esophagus, rectum and recto-sigmoid junction, and lymphoid leukemia appears confined to a single race/sex group. The PMR results were similar to those obtained with the SMR analysis. Compared to the previous follow-up [9], the results were similar except that lymphoid leukemia and myelofibrosis are now newly observed to be occurring in excess.

## Discussion

It appears that cancers of the buccal, nasal, and pharyngeal cavities are occurring in excess generally in the poultry cohort and affecting multiple sites in this anatomical area and affecting both races or both sexes, for the most part. Small number of deaths and inability to control for possible confounding factors such as tobacco smoke and alcohol ingestion limit interpretation of the findings, but possible occupational factors that could be responsible for the excess include oncogenic retroviruses which induce a multiple of tumor types in chickens [1] and papilloma viruses. The prevalence of warts is known to be high in poultry workers and is known to be associated with the human papilloma virus type 7 which is uncommon in the general population [16]. Furthermore, it is now generally believed that at least certain types of oral cancer are caused by human papilloma viruses especially type 16 [17].

Similarly, the excess of cancer of the liver, myelofibrosis, and myeloma appears to affect more than one racial or gender group, suggesting a more general exposure such as exposure to oncogenic viruses as the prime suspect for a causal role. Again the inability to control for possible non-occupational confounding factors such as alcohol intake in the case of liver cancer, for example, limits the interpretation of the findings.

On the other hand, the excess of cancers of the esophagus, rectum and recto-sigmoid junction, and lymphoid leukemia appears confined to a single race/sex subgroup. This could be due to chance, or to the smaller number of deaths in females, or could be real, and reflect actual differences in exposures among the different race/sex subgroups. At the time the cohort was first assembled in the 1970s, certain tasks in the meat and poultry industries were highly related to race and gender. It is known, for example, in this cohort that killing and dressing activities were associated with black men, meatcutters in supermarkets were almost exclusively white men, while meat wrapping was predominantly carried out by women throughout the meat and poultry industries.

The results of this study should be considered as exploratory, as they begin to indicate which cancer types are likely to be caused by the poultry oncogenic viruses, if at all these viruses do cause cancer in humans. While this exposure is the most prevalent in the poultry industry, with virtually all the workers in this cohort exposed to them, it should be realized that there are other carcinogenic exposures which could be candidates to explain the excess occurrence of some of these cancers, especially those restricted to a single race/sex group.

We present in Table 2 the results for lung cancer because a statistically significant three- to fourfold increased risk for the cohort as a whole was observed in the initial follow-up of this poultry cohort up to 1980 in white females [8], which was hypothesized to be possibly associated with exposure to fumes emitted from the wrapping machine. This initial high risk has not been sustained in a subsequent follow-up up to 1989 for this group (SMR = 1.8) [9] and in this update (SMR = 1.6, 95% CI 0.8–2.8). This progressive decrease in risk could indicate that the initial findings were due to

**Table 2** Standardized mortality and proportional mortality ratios for cancers for the period 1954–2003

Cause of death		Poultry workers																		
		Non-white males		White males		All males		Non-white females		White females		All females		All non-whites		All whites		All groups		
		Obs.	SMR	PMR	Obs.	SMR	PMR	Obs.	SMR	PMR	Obs.	SMR	PMR	Obs.	SMR	PMR	Obs.	SMR	PMR	
Death from all malignant neoplasms	77	22	99	39	88	126	61	187												
		1.0 (0.8–1.2)	0.9 (0.6–1.4)	1.0(0.8–1.2)	1.0 (0.7–1.3)	1.3 (0.9–1.8)	1.1(0.9–1.3)	1.0 (0.8–1.2)	1.1 (0.9–1.4)	1.0 (0.9–1.2)	1.0 (0.8–1.2)	0.9* (0.7–1.0)	0.9 (0.8–1.0)	0.9 (0.8–1.0)	0.9 (0.8–1.0)	0.9 (0.8–1.0)	0.9 (0.8–1.0)	0.9 (0.8–1.0)	0.9 (0.8–1.0)	0.9 (0.8–1.0)
Base of tongue	2	1	3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		3.0(0.4–11.0)	7.2(0.2–40.2)	3.8 (0.8–11.0)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Palate/other unspec. mouth	1	0	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
		2.7 (0.1–14.9)	–	2.2 (0.1–12.3)	9.6 (0.2–53.3)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Tonsil; oropharynx	3	0	3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		5.3* (1.1–15.6)	–	4.6 (1.0–13.5)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Nasal cavity/mid ear, acc sinus	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		7.7 (0.2–43.0)	–	6.2 (0.2–34.4)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Esophagus	6	4	10	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		8.1* (1.6–42.0)	–	5.9* (1.1–33.2)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Recto-sigmoid, rectum, anus	4	0	4	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		3.2 (0.9–8.2)	–	2.3 (0.6–5.9)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Liver, intrabiliary	1	1	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
		3.4** (1.0–11.4)	–	2.2 (0.8–5.7)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Trachea, bronchus, lung	25	7	32	12	12	12	12	12	12	12	12	12	12	12	12	12	12	12	12	12
		1.0(0.6–1.4)	0.9 (0.3–1.8)	0.9 (0.6–1.3)	1.1(0.7–2.3)	1.6 (0.8–2.8)	1.1(0.9–2.1)	1.1 (0.7–1.5)	1.1 (0.7–1.9)	1.1 (0.7–1.5)	1.1 (0.7–1.5)	1.1 (0.7–1.5)	1.1 (0.7–1.5)	1.1 (0.7–1.5)	1.1 (0.7–1.5)	1.1 (0.7–1.5)	1.1 (0.7–1.5)	1.1 (0.7–1.5)	1.1 (0.7–1.5)	1.1 (0.7–1.5)

**Table 2** continued

Cause of death	Poultry workers																										
	Non-white males			White males			All males			Non-white females			White females			All females			All non-whites			All whites			All groups		
	Obs.	SMR	PMR	Obs.	SMR	PMR	Obs.	SMR	PMR	Obs.	SMR	PMR	Obs.	SMR	PMR	Obs.	SMR	PMR	Obs.	SMR	PMR	Obs.	SMR	PMR	Obs.	SMR	PMR
Malignant immunoglobulinoma, plasma cell	1	0.6 (0.0–3.3)	2	5.3 (0.6–19.1)	3	1.5 (0.3–4.2)	0	–	0	2.2 (0.1–12.5)	1	0.6 (0.0–3.2)	1	0.3 (0.0–1.9)	3	3.6 (0.8–10.6)	4	1.0 (0.3–2.7)	–	–	–	–	–	–	–	–	–
Lymphoid leukemia	0	0.6 (0.1–4.3)	0	3.9* (1.1–14.1)	0	1.4 (0.5–4.4)	2	–	2	1.7 (0.2–11.7)	0	0.5 (0.1–3.4)	2	0.3 (0.1–2.1)	0	2.7 (0.9–8.0)	2	1.0 (0.4–2.6)	–	–	–	–	–	–	–	–	–
Myelofibrosis	0	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
All deaths	340	1.0 (0.9–1.1)	132	1.3 (1.0–1.6)	472	1.0 (0.9–1.1)	198	1.0 (0.9–1.2)	120	1.4 (1.1–1.7)	318	1.1 (1.0–1.3)	538	1.0 (0.9–1.1)	252	1.3** (1.1–1.6)	790	1.1 (1.0–1.1)	–	–	–	–	–	–	–	–	–

Figures in parenthesis are confidence intervals

\* Statistically significant at the 95% confidence level; \*\* statistically significant at the 99% confidence level

chance. On the other hand, exposure to fumes containing benzene, polycyclic aromatic hydrocarbons, and phthalates from the wrapping machine, a possible occupational cause [3, 18–21], was significantly reduced after 1975 and could well explain these findings, as it did explain a similar decline in lung cancer risk over time in women in the meat department of supermarkets from the same union [21]. The study by Fritschi et al. also reported an elevated odds ratio of 1.5 for lung cancer for meat workers exposed to these fumes, although it was not statistically significant [11].

A cohort mortality study of this type does not control for non-occupational confounding factors and is not suitable for identifying the specific occupational exposures responsible. Apart from oncogenic viruses and fumes from the wrapping machine, exposure to carcinogens during frying/cooking or curing of poultry meat could also have occurred in this cohort although at a prevalence below 5% typically. This type of investigation is better handled using a nested case–control design, which we are conducting. Also, many comparisons were made and the numbers involved in many cases were small, thus interpretation should be tempered with caution.

Although every subject in the cohort has been followed up for at least 24 years, only 31% have died by the end of the study. Thus, it is anticipated that further follow-up to a point when the majority of the cohort will have died, will provide more definitive results. However, at this time, the study provides enough evidence to recommend that this occupational group warrants detailed investigation, and consideration should be given to the possible need to protect workers against candidate exposures that potentially may be responsible for the excess occurrences of some of these cancers. The group needs to be studied not only because of occupational reasons, but also because it is possible that the oncogenic viruses present in poultry and poultry products could be transmitted to subjects in the general population, from handling and ingestion of raw or inadequately cooked poultry products, from exposure to live birds, or from vaccination [22, 23]. Poultry slaughtering and processing plant workers have the highest known human exposure to these viruses (a typical worker will handle the internal organs of hundreds of birds a day and be exposed to their blood and secretions), and thus are a suitable group to investigate whether human exposure to poultry oncogenic viruses is associated with the development of cancer. It is worthwhile to note that exceedingly high risk of death from zoonotic bacterial diseases and helminthiasis (up to 100-fold) is being observed in this poultry cohort (manuscript in preparation), thus reinforcing the suggestion of the possible role of transmissible agents in the occurrence of the excess of some of these cancers.

Also, it is well established that some of these viruses are present in eggs destined for human consumption as reviewed by us [24] and as reported by us from a survey of eggs sold in supermarkets in the New Orleans metropolitan area [25, 26]. Furthermore as mentioned above, we and others have shown that members of the general population have antibodies in their blood against ALSV and REV [27–29]. Also, ALSV and REV are known to infect/transform human cells also in vitro [30–32] and to cause tumors in primates [24]. Finally, it was reported by the Centers for Diseases Control & Prevention that all stocks of measles, mumps, and yellow fever vaccines currently in use in the United States are contaminated with endogenous ALSV, and that in yellow fever vaccines from US and European manufacturers, the endogenous virus was of the infectious type [23, 33]. It is also important to note that exogenous ALSV-contaminated yellow fever vaccines caused malignant tumors when injected into chickens [34]. These observations have serious public health implications and reiterate the urgent need for studies to be conducted in subjects that have high exposure to the oncogenic viruses of poultry, such as workers in poultry slaughtering and processing plants.

**Acknowledgment** This update was funded by a grant (1 RO1 OH008071) from the National Institute for Occupational Safety & Health. Previous follow-ups were funded by the National Institute for Environmental Health Sciences and the National Cancer Institute (1 Ro1 CA 30410-3). Our appreciation and thanks go to the United Food & Commercial Workers International Union and the Baltimore Meatcutters' Union Local 27 (formerly Local 117) for their continuing support and collaboration over the years without which this research would not have been possible. There are no competing interests.

## References

1. Saif YM, John Barnes H, Fadly AM, Swayne D, Glisson JR (2003) Diseases of poultry, 11th edn. Iowa State Press, Ames, Iowa
2. Pauli G, Bessot JC, Kopferschmitt MC et al (1980) Meat wrapper's asthma: identification of the causal agent. *Clin Allergy* 10:263–269
3. 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
4. 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* 22:1125–1130
5. Jakszyn P, Agudo A, Ibanez R et al (2004) Development of a food database of nitrosamines, heterocyclic amines, and polycyclic aromatic hydrocarbons. *J Nutr* 134:2011–2014
6. Nordholm L, Espensen I, Jensen HS, Holst E (1986) Polycyclic aromatic hydrocarbons in smokehouses. *Scand J Work Environ Health* 12:614–618

7. 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
8. Johnson ES, Fischman HR, Matanoski GM, Diamond E (1986) Cancer occurrence in women in the meat industry. *Br J Ind Med* 43:597–604
9. Johnson ES, Shorter C, Rider B, Jiles R (1997) Mortality from cancer and other diseases in poultry slaughtering/processing plants. *Int J Epidemiol* 26:1142–1149
10. Netto GF, Johnson ES (2003) Mortality in workers in poultry slaughtering/processing plants—The Missouri Poultry Cohort Study. *Occup Environ Med* 60:784–788
11. 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>)
12. Metayer C, Johnson E, Rice JC (1998) Nested case–control study of tumors of the hemopoietic and lymphatic systems among workers in the meat industry. *Am J Epidemiol* 147:727–738
13. Johnson ES, Fischman HR, Matanoski GM, Diamond E (1986) Cancer mortality among white males in the meat industry. *J Occup Med* 28:23–32
14. Liddell FD (1984) Simple exact analysis of the standardised mortality ratio. *J Epidemiol Community Health* 38:85–88
15. Miettinen O (1976) Estimability and estimation in case-referent studies. *Am J Epidemiol* 103:226–235
16. Stehr-Green PA, Hewer P, Meekin GE, Judd LE (1993) The aetiology and risk factors for warts among poultry processing workers. *Int J Epidemiol* 22:294–298
17. Scully C (2002) Oral squamous cell carcinoma; from an hypothesis about a virus, to concern about possible sexual transmission. *Oral Oncol* 38:227–234
18. 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
19. Smith TJ, Cafarella JJ, Chelton C, Crowley S (1983) Evaluation of emissions from simulated commercial meatwrapping operations using PVC wrap. *Am Ind Hyg Assoc J* 44:176–183
20. Sokol WN, Aelony Y, Beall GN (1973) Meat-wrapper's asthma. A new syndrome? *JAMA* 226:639–641
21. Johnson ES (1994) Cancer mortality among workers in the meat department of supermarkets. *Occup Environ Med* 51:541–547
22. 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, 2005
23. Tsang SX, Switzer WM, Shanmugam V et al (1999) Evidence of avian leukosis virus subgroup E and endogenous avian virus in measles and mumps vaccines derived from chicken cells: investigation of transmission to vaccine recipients. *J Virol* 73:5843–5851
24. Johnson ES (1994) Poultry oncogenic retroviruses and humans. *Cancer Det Prev* 18:9–30
25. Pham TD, Lloyd Spencer J, Johnson ES (1999) Detection of avian leukosis virus in albumen of chicken eggs using reverse transcription polymerase chain reaction. *J Virol Methods* 78:1–11
26. Pham TD, Spencer JL, Traina-Dorge VL, Mullin DA, Garry RF, Johnson ES (1999) Detection of exogenous and endogenous avian leukosis virus in commercial chicken eggs using reverse transcription and polymerase chain reaction assay. *Avian Pathol* 28:385–392
27. Johnson ES, Nicholson LG, Durack DT (1995) 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
28. Johnson ES, Overby L, Philpot R (1995) 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
29. Choudat D, Dambrine G, Delemotte B, Coudert F (1996) Occupational exposure to poultry and prevalence of antibodies against Marek's disease virus and avian leukosis retroviruses. *Occup Environ Med* 53:403–410
30. Stenkvis B, Ponten J (1964) Morphological changes in bovine and human fibroblasts exposed to two strains of Rous sarcoma virus in vitro. *Acta Pathol Microbiol Scand* 62:315–330
31. Johnson ES, Griswold CM (1996) Oncogenic retroviruses of cattle, chickens and turkeys: potential infectivity and oncogenicity for humans. *Med Hyp* 46:354–356
32. Koo H-M, Brown AMC, Ron Y et al (1991) Spleen necrosis virus, an avian retrovirus, can infect primate cells. *J Virol* 65:4769–4776
33. Hussain AI, Johnson JA, da Silva-Freire M, Heneine W (2003) Identification and characterization of avian retroviruses in chicken embryo-derived yellow fever vaccines. Investigation of transmission to vaccine recipients. *J Virol* 77:1105–1111
34. Richman AV, Aulisio CG, Jahnes WG, Tauraso NM (1972) Avian leukosis antibody response in individuals given chicken embryo derived vaccines (36117). *Proc Soc Exp Biol Med* 139:235–237