PREVALENCE OF SOME FOOD POISONING PATHOGENS IN SQUABS AND WOODEN PIGEONS CARCASSES IN ASSIUT GOVERNORATE

(With 4 Tables)

By

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SUMMARY

Squabs and wooden pigeons carcasses were removed from commercial processing lines immediately after defeathering and evisceration. The carcasses as well as their organs; livers, hearts, gizzards and parts of intestines were examined for the presence of risk pathogens, Staphylococcus, Campylobacter jejuni, Salmonella and Clostridium perfringens. From the examined squab carcasses, S. typhimurium and C.
Porphyromonas could not be detected, while C. jejuni was positive in 8% and S. aureus in 16%. Wooden pigeon carcasses were contaminated with S. typhimurium (12%), S. aureus (16%) and C. jejuni (8%). For organs, livers were highly contaminated with Salmonella (8%), C. perfringens (48%) in case of wooden pigeons carcasses while in squab carcasses was 4% and 28%, respectively. The organs and the intestines were implicated as a major source for the presence of such pathogens.

Key words: Food poisoning pathogens, squabs, wooden pigeons.

INTRODUCTION

Pigeons carcasses during processing can be contaminated with a variety of pathogenic bacteria. The most important pathogens are salmonellae, campylobacter jejuni, Staph. aureus and Clostridium perfringens, which are undesirable and unavoidable. Salmonella typhimurium could be isolated from pigeons by many investigators (Parnas, 1980; Wathe and Wathe, 1980; Rosaf, 1981 and Polydorou, 1985). In domestic squabs examined by Khalifa and Abd Allah (1995), 2.5% of the examined samples were contaminated by Salmonella typhimurium. In feral pigeons, Salmonella typhimurium was isolated by Wilson (1960); Farrari et al. (1964), Wilson and MacDonald (1967), Goodchild and Tucker (1968), Shafahi et al. (1990) and Woerlen (1990).

Campylobacter jejuni was detected in different percentages pigeons in all over the world by many researchers. In wooden pigeons, Farrari et al. (1983); De Boer et al. (1983); Harog et al. (1983), Woerlen (1990) and Pitkala et al. (1992) could isolate the organism from the examined pigeons. The pathogen could be isolated from the intestinal content of wooden pigeons by Rosaf (1981), De Boer and Stigter (1984) and Woerlen (1990). However, C. jejuni was detected in 6% of squab gizzards and 10 squab livers (Khalifa, 1990). The organism failed to be detected in hearts and livers of feral pigeons (Woerlen, 1990) and in hearts and spleens of squabs (Khalifa, 1990).

Staphylococci are present on the skin of carcasses of pigeons, when they leave the slaughter plant. The commonest source of infection is the human food handler during further processing. Staphylococci can be found in the nose and on the hands of many humans and it is difficult to remove all of them from hand by ordinary washing (Breuner, 1977).
C. perfringens is ubiquitous and although large number occurs in
the intestinal tract of birds, this is not an organism that can removed
from there by any of the usual control measures (Brenner, 1977).
Little information on the prevalence of the risk pathogens in
pigeon carcasses in Egypt. The objectives of the research reported herein
were: 1. To study the predominance of salmonella, C. jejuni, S. aureus
and C. perfringens in pigeon carcasses. 2. The role of pigeon organs
(livers, gizzards and hearts) and intestines in spreading of these
pathogens.

MATERIALS and METHODS

Three-hundreds samples were collected from fifty random
domestic squabs and wooden pigeons carcasses. The samples were outer
surface swabs, inner surface swabs, livers, hearts, gizzards and parts of
intestines (25 each). Each sample was taken separately in a sterile plastic
bag. The cotton-tip swab was put in sterile peptone water. Next, the
samples were transferred to the laboratory within 30 min. where, they
were examined for the presence of the following pathogens: salmonella,
C. jejuni, S. aureus and C. perfringens.
1. Presence of Salmonella;
   This was determined by preenriching of the tested sample in buffered
peptone water (24 h, 37°C). Next, 0.1 ml was transferred to 10 ml of
tetrathionate broth (TT) and incubated for 24 h at 42°C. Loopfuls from
TT were streaked onto SS and XLD agar and incubated for 24 h at 37°C.
Isolates were biochemically and serologically confirmed according the
2. Presence of C. jejuni;
   This was done by inoculating the samples in nutrient broth
supplemented by growth and selective supplement (Proshon media,
Oxoid) without blood in a jar under microaerophilic atmosphere. After
incubation for 24h and 48h., campylobacter blood free selective medium
(Oxoid) was inoculated by streaking method. The plates were searched
for typical colonies (small gray drop like or gray slimy colonies) after 48
h of incubation. Typical colonies were confirmed using microscopic
analyses, catalase test, oxidase test and sensitivity to cephalothin and
nalidixic acid (Uyttendaele and Debevere 1996).
3. Isolation of Staph. aureus
   Enrichment of the samples were done in sodium chloride broth (10%)
at 37°C for 24 h. Three loopful after enrichment were streaked on Baird
Parker agar plates (Baird Parker, 1962) and then incubated at 37°C for 24h. The suspected colonies were confirmed by morphological characters, microscopic examination and coagulase test (Finegold and Martin, 1982).

4. Isolation of C. perfringens:

The samples were incubated in lactose sulphite broth (Beaven et al., 1986) for 48 h at 42°C. Loops from the incubation positive tubes (black precipitation and gas in Durham’s tube) were streaked onto SPS agar plates and were incubated anaerobically at 42°C for 48 h. The grown colonies confirmed by CAMP test.

RESULTS and DISCUSSION

From the results achieved in Table 2, it is evidently that Salmonella typhimurium could isolated from liver, gizzard and intestine of squabs with 4%, 4% and 8%, respectively. The organism failed to be detected in inner swabs and outer swabs of squabs (Table 1) and in hearts (Table 2). Comparing this data with that obtained in wooden pigeon carcasses, S. typhimurium was found in 12% and 12% in outer swabs and inner swabs respectively (Table 3) and in 4, 8 and 16% in liver, heart, and intestine, respectively (Table 4). In this respect, Parnes (1980) could isolated S. typhimurium from domestic and wild living pigeons. Nearly similar results were obtained by Khalafalla and Abd Allah (1995). They found that 2.5% of the examined squabs contained S. typhimurium. In a clinical specimens of pigeons examined by Criasovos et al. (1995), salmonella spp. could be isolated from 6 samples (1.5%).

The detection of salmonellae in pigeon carcasses may be attributed to hygiene measures adopted in the hatchery, breeding and rearing methods which help in the spread of infection. Once infected, carcass reaches the plant, it is difficult to have effective control measures. Salmonellae may be transmitted from infected to non-infected carcasses.

C. jejuni was more frequently isolated from intestine than liver, gizzard (Table 2) and inner swabs of squabs (Table 1). Each constituting incidence of 40%, 20%, 4% and 8% respectively. The fact that C. jejuni is often present at a high concentration in fecal materials which were released during defeathering and evisceration with subsequent contamination of the carcasses and edible organs (Kapperud and Rosef, 212.
1983, Stern et al., 1997). No isolates of C. jejuni could be found in hearts and the inner swabs of squabs. This finding was different from that recorded by Khalafalla, 1990, who found 10% of squab livers and 6% of squab gizzards contained C. jejuni. The organism failed to be detected in outer swabs of the squab carcasses (Table 1) and in the hearts (Table 2). This result comply with that recorded by Khalafalla (1990) who found that the squab hearts are free from C. jejuni. For wooden pigeons (Table 4), C. jejuni was present in higher incidence in the intestine (32%) and lower in the heart (4%). Casanova et al. (1995) reported 26.2% C. jejuni in cloacal specimens of pigeons in Barcelona.

Consumption of poultry meat has been identified as a risk factor for human campylobacteriosis in several developed countries (Schore et al., 1994). A part from consumption of raw or under cooked chicken meat (Deming et al., 1987), consumption of chicken liver (Hopkins et al., 1984) have been associated with campylobacter enteritis.

Staph. aureus was detected in outer swabs and inner swabs of squab carcasses in 16% and 16% respectively (Table 1) and in hearts, livers, gizzards and intestines of squab in 20, 28, 16 and 24%, respectively (Table 2). The higher incidence was in liver samples while the lower was in inner and outer swabs. On the other hand, the organism was present in 8% (hearts), 8% (livers), 12% (gizzards), 8% (intestines) as present in Table 4, while 12% (inner swabs) and 16% (outer swabs) of wood pigeon carcasses were positive (Table 3). Higher prevalence (16%) was in outer swabs and lower incidence was in liver, heart and intestine (each 8%).

Staphylococci account for the large majority of food poisoning outbreaks. Poultry meat is frequently and unavoidably contaminated during slaughter and processing as live birds carry staphylococci in brooded tissues, infected lesions, nasal sites, skin surfaces and arthritic joints (Brommer, 1977). The natural reservoirs of staphylococci are man and warm blooded animals. S. aureus is found in both healthy people and diseased people (Varnam and Evans, 1991). It may present at a number of sites of human bodies including the skin, nose, throat and hair and may be even present in stools (Holmberg and Blacke, 1984) and consequently they contaminate carcasses during processing.

From the findings presented in Table 2, it is evident that C. perfringens was present in the livers and intestine of the squabs in 28% and 12%, respectively. The pathogen were the etiology of necrotic enteritis and liver lesions in broiler chickens. The percentage of birds
condemned because of liver lesions was 2.9 (Schneitz et al. 1997). The organism failed to be detected in hearts, gizzards, inner and outer swabs of the examined squabs. In wooden pigeon carcases, C. perfringens was found in 8% of inner swabs (Table 3), and in livers, hearts, gizzards and intestines with levels of 12, 4.16 and 32%, respectively (Table 4). This result comply with that obtained by Fukata et al., 1986.

C. perfringens was isolated infrequently and in low numbers from the intestinal tract of broilers (Craven, 1997). The organism is involved in poultry disease and causes food poisoning in humans with poultry often the vehicle of infection (Labrie, 1991).

From this study, pigeon carcases were found to be contaminated with some of the pathogens. The majority of the microbial pathogens are part of gastrointestinal flora of pigeons. These organisms can also be associated with skin and feathers. Many of the bacterial pathogens can also be carried by plant employees (Goodfellow, 1992). The sources of cross contamination may be from, 1. One carcase touches another one. 2. One carcase touches a piece of evisceration equipment and another one touches the same location. 3. During the drawing process, the intestines are torn and the evisceration spoon become contaminated, thus contaminating other carcases. 4. Plant personnel who present visgtes for inspection can spread contamination from one carcase to another (Santikiewicz et al., 1999).

In conclusion, pigeons naturally can carry a variety of bacteria. The pathogens present either on or in live pigeon result in transfer of these pathogens to the retail product. The organs constitute a risk hazard for consumers. Wooden pigeon carcases and their organs carry more pathogens than in case of squabs and this may be due to the nature of the breeding, feeding and flying.

REFERENCES


Table 1: Prevalence of food poisoning pathogens in squab carcases.*

<table>
<thead>
<tr>
<th>Samples</th>
<th>Salmonella</th>
<th>S. aureus</th>
<th>C. perfringens</th>
<th>C. jejuni</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>No</td>
<td>%</td>
</tr>
<tr>
<td>On surface</td>
<td>0</td>
<td>0</td>
<td>4</td>
<td>16</td>
</tr>
<tr>
<td>In/on surface</td>
<td>0</td>
<td>0</td>
<td>4</td>
<td>16</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>0</td>
<td>8</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

*N=25 for each sample
Table 2: Prevalence of food poisoning pathogens in squab organs.*

<table>
<thead>
<tr>
<th>Samples</th>
<th>Salmonella</th>
<th>Sarcina</th>
<th>C. perfringens</th>
<th>C. jejuni</th>
</tr>
</thead>
<tbody>
<tr>
<td>Livers</td>
<td>1</td>
<td>4</td>
<td>7</td>
<td>28</td>
</tr>
<tr>
<td>Hearts</td>
<td>0</td>
<td>0</td>
<td>5</td>
<td>20</td>
</tr>
<tr>
<td>Gizzards</td>
<td>1</td>
<td>4</td>
<td>4</td>
<td>16</td>
</tr>
<tr>
<td>Intestines</td>
<td>2</td>
<td>8</td>
<td>6</td>
<td>24</td>
</tr>
<tr>
<td>Total</td>
<td>4</td>
<td>22</td>
<td>10</td>
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</tr>
</tbody>
</table>

*N=25 for each sample type

Table 3: Prevalence of food poisoning pathogens in wooden pigeon caecaes.*

<table>
<thead>
<tr>
<th>Sample</th>
<th>Salmonella</th>
<th>Sarcina</th>
<th>C. perfringens</th>
<th>C. jejuni</th>
</tr>
</thead>
<tbody>
<tr>
<td>Outer</td>
<td>3</td>
<td>12</td>
<td>4</td>
<td>16</td>
</tr>
<tr>
<td>Inner</td>
<td>3</td>
<td>12</td>
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<td>12</td>
</tr>
<tr>
<td>Total</td>
<td>6</td>
<td>24</td>
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<td></td>
</tr>
</tbody>
</table>

*N=25 for each sample.

Table 4: Prevalence of food poisoning pathogens in wooden pigeon organs.*

<table>
<thead>
<tr>
<th>Samples</th>
<th>Salmonella</th>
<th>Sarcina</th>
<th>C. perfringens</th>
<th>C. jejuni</th>
</tr>
</thead>
<tbody>
<tr>
<td>Livers</td>
<td>2</td>
<td>8</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>Hearts</td>
<td>1</td>
<td>4</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>Gizzards</td>
<td>0</td>
<td>0</td>
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<td>12</td>
</tr>
<tr>
<td>Intestines</td>
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<td>2</td>
<td>8</td>
</tr>
<tr>
<td>Total</td>
<td>7</td>
<td>9</td>
<td>22</td>
<td></td>
</tr>
</tbody>
</table>

*N=25 for each sample type