ESCHERICHIA COLI INFECTIONS IN BROILERS: A REVIEW. PART II: EPIDEMIOLOGY, PUBLIC HEALTH & ECONOMIC IMPACT.

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INTRODUCTION

The purpose of this paper is to provide a comprehensive source of information regarding the epidemiology, public health and economic impact of *E. coli* infections in broilers. Epidemiology may be defined as the study of patterns of disease that exist under field conditions [1]. Many *E.coli* serogroups [2,3] may be implicated in different forms of *E.coli* infections such as colibacillosis, colisepticemia, airsacculitis and cellulitis [2,3,4,5]. It is important to provide some information about whether some *E.coli* serogroups from poultry sources are implicated in human infections. A comprehensive review of *E.coli* infections can not rely solely on biologic (epidemiologic and public health) information [1]. Hence, the economic impact of *E. coli* infections in different parts of the world was included in the review.

مقدمة:

الغرض من كتابة هذه الورقة هو اضافة معلومات عن الأمراض التي تسببها الأشيرشية القولونية (*E.coli*) في الدجاج اللاحم خاصة فيما يتعلق بالوبائيات والصحة العامة والآثار الاقتصادية . يمكن تعريف كلمة الوبائيات بالدراسة التي تعنى بانتشار الأمراض والعوامل التي تؤثر على هذا الإنتشار تحت الظروف الطبيعية [1]. كثير من العترات المصلية الأشيريشية القولونيه [2,3] يمكن أن تعزل من اشكال مختلفة من الأمراض التي تسببها الباكتيريا مثل أمراض الكولي باسيلوسس (*colibacillosis*) والكولنيسيتسيميا (colisepticemia) والأيزساكيولايتس (airsacculitis) والسليولايتس (*cellulitis*) [2,3,4,5]. انه من المهم معرفة إذا كان

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هنالك عترات مصلية موجودة في الدواجن يمكن أن تنتقل للإنسان وتسبب له أمراض. هذه المراجعة للمعلومات الخاصة بباكتيريا الاشيريشية لم تقتصر على الجوانب الحيوية فقط وإنما إحتوت على مراجعة متكاملة وإشتملت على الآثار الإقتصادية للمرض في اجزاء مختلفة من العالم.

EPIDEMOILOGY:

The prevalence of different serotypes of *E.coli* varies from place to place. This might be due to differences associated with geographical region, environment and management systems. The accuracy and precision of prevalence estimates of *E.coi* serotypes are also affected by sampling methods, specimen tested and the characteristics of the diagnostic tests(s) (sensitivity and specificity) used [1]. The following briefly describes studies which have estimated the prevalence of *E.coli* serotypes among broiler chickens.

In Britain, *E.coli* serogroups O2 and O78 constitute 50% and 20% of the isolates from outbreaks of colisepticemia in chickens, while serogroups O11,O1 constitued 10% and 9% respectively[2]. This is in agreement with another study in which *E.coli* serogroups O2,O78,O73,O1 and O11 constituted 65%, 20%, 12.5%, 0.5% and 0.5% respectively> However, in chickens over 28 days of age, serogrup O2 constituted 96% of the *E.coli* isolates [3].

Escherichia coli serogroup O78 has been the most frequent serogroup isolated from cellulitis lesions in Canada. Other serogroups such as O2,O115,O21,O83,O161, O1 and O113 have been isloated less frequently [4,5].

In India, *E.coli* infection ranks second after coccidiosis as the cause of death in poultry [6]. Although isolation of serogroups O78,O24,O2,O86,O51, and O143 has been reported from cases of colisepticemia in chickens [7], serogroups O57, O24 and O2 are the predominant ones[8].

The three predominant serotypes involved in colisepticemia outbreaks in Saudi Arabia are O33:H4,O45:H10 and O78:H-. Serotypes such as O119:H27, O145:H25 and O132:H- have been involved to a lesser extent [9].

Serogroups O78 and O45 have been reported as the most common serogroups isolated from chickens in Australia [10].

In the United States, serogroups O78,O2, and O35 have been considered the predominant serogroups isolated from broiler chickens with colibacillosis [11,12].

Serogroups O78:K80,O2:K80,O127:K57 and O14O have been isolated from field cases of dermatitis in Germany [13].

In Israel, colibacillosis ranks second after chronic respiratory disease [14]. The most common serogroups were O78(39.8%), O2(20.4%), O1(12%) and O111(4.2%). This is in agreement with a previous study which reported that 61.9% of the strains isolated from diseased chickens and turkeys belonged to one or another of the three serological groups:O78:K80,O1:K1[15].

Several studies have indicated that there are complex factors which lead to the development of *E.coli* infection in chickens. However, mixed infection is thought to be characteried by severe lesions and higher mortality. Association between infection with pleuropneumonia-like organisms and infectious bronchitis virus or Newcastle disease virus[16,17], adenovirus or infectious bursal disease virus[18,19] or coronavirus [20] and *E.coli* infections in chickens has been documented. *Escherichia coli* infection may occur as a mixed infection with *Campylobacter jejunji* [21], *Eimeria tenella* and *Ascaridia galli* [22].

Few studies have investigated the effect of nutrition on the diseases caused by *E.coli*. Iron, ascorbic acid and corticosterone have been experimentally documented to be protective against infection with *E.coli*[23,24].

Stocking density has been suggested as an important risk factor for the development of dermatitis and cellulitis in broiler chickens [13,25,26,27]. Scratches are another risk factor which has been suggested of importance for the development of cellulitis[13,27,28]. Another factor which may contribute to the development of dermatitis and cellulitis in broiler chickens is poor litter conditions [25,26,29,30]. However, in a recent study on cellulitis, Goodhope et al.[31] argued that new litter was used and maintained in good

condition throughout the grow-out period in both affected and unaffected flocks.

A genetic effect on the development of dermatitis and cellulitis in broiler chickens has also been suggested [27]. This could be due to different levels of feathering in different breeds. It has been documented that the incidence of dermatitis [3] and *E.coli* infection [33] increased in genetic strain crosses with slow feathering. These findings have been confirmed in a more recent survey of broiler producers in which several mangers had mentioned that cellulitis is more common in slow feathering breeds [26]. Another study has reported breed difference in response to inoculation with infectious bronchitis virus and different strains of *E. coli* [34]. Also, breed has been reported to have an impact on the aggressiveness and nervousness of birds [35,36].

Based on the opinions of broiler producers in the United States, the most important factors associated with cellulitis included *E.coli*, chick quality, high early and total mortality, skin scratches, airsaccullitis, ventilation, sex, breed and age of breeder flock [26]. More recently, risk factors such as growing rate, food composition, air condition, litter condition, stocking density, feathering, and body structure have been reported [27,28,37].

PUBLIC HEALTH IMPACT:

The isolation of *E.coli* strains from broiler chickens and from people is well documented and a considerable overlap in the distribution of *E.coli* serogroups has been demonstrated [38,39,40,41,42,43]. In a study to investigate the distribution of *E.coli* strains, Linton et al. [39] compared antbiotic resistant O-serogroups of *E. coli* isolated from human feces with those isolate from rectal contents of broiler chickens, 38 different O-serogroups were found in chcikens only, 19 in people only and 46 in both chickens and people. However, results for the isolates from people were derived by different procedure and, therefore, a strict quantitative comparison was not possible. Moreover, in a study to determine whether *E.coli* isolated from infected humans and septicemic chickens were of common origin, Achtman et al. [40]

found that poultry and human isolates of *E.coli* O2:K1 are of the same clonal group and they could be distinguished only by their plasmid content. However, the difference in plasmid content suggests that these two groups are not overlapping.

It is important to stress the fact that most poultry, unlike other meat, is packed in polythene which avoids environmental and human contamination after factory processing. The organisms deteced on the carass are most probably of poultry or factory origin. In a survey of *E. coli* O157:H7 in 896 retail meat sample, Doyle [44] reported the isolation of this serotype from 3.7% of ground beef, 1.5% of pork, 2.0% of lamb, and 1.5% of poultry samples. The isolation of *E. coli* O157:H7 from poultry is supported by a previous experimental study in which *E. coli* O157:H7 was found capable of colonizing chicken ceca followed by prolonged fecal shedding [45]. Since no details of formal random sampling procedures were given, it is difficult to extrapolate these results to other meat samples. In a random sample of meats from processing plants in Ontario, Read et al.[46] isolated VT-producing *E.coli* from 24 of 225 samples of ground beef, from 9 of 235 samples of pork, and from none of 200 chicken samples. However, Irwin et al.[47] failed to identify any VT-producing strains of *E. coli* isolated from fecal samples of 500 broiler chickens from 50 farms in a slaughter house survey in Ontario.

There is some evidence that *E.coli* strains from poultry sources are associated with human infection. Poultry meat has been incriminated as a source of *E.coli* for patients in a hospital. Fecal *E.coli* serotypes of ward patients are generally similar to those present in food including poultry meat [38]. Supporting evidence that the food source of strains of *E.coli* appearing in patients feces was the finding that five patients had apparently acquired new serotypes after eating the contaminated food [38].

However, since the food was examined only as it was being served to the patients, there is a possibility that the poultry meat would have been contaminated in the kitchen. Among *E.coli* serotypes isolated from poultry, *E.coli* O157:H7 remains the most important because of its association with a severe disease in humans which may include

three different syndromes: hemorrhagic colitis [48,49], hemolytic uremic syndrome [50,51], and thrombotic thrombocytogenic purpura [52]. In one outbreak of *E.coli* O157:H7-associated hemorrhagic colitis in a nursing home in Ontario, Canada;ham, turkey and cheese sandwiches were epidemiologically implicated as the likely source of infection [53]. None of the above mentioned foods, however, was available for microbiologic analysis and, hence, the association lacks microbiologic confirmation. In another outbreak of haemorrhagic colitis and haemolytic uraemic syndrome associated with serotype O157:H7, turkey-roll sandwiches were significantly associated with the illness ($p<0.001$;RR=3.15)[54] Unfortunately, no epidemiologically implicated food remained for culture.

There is also evidence that avian *E.coli* may be transmitted to humans by direct contact. It has been observed that *E.coli* isolates from stools of poultry farm workers and from cloacal swabs from the birds show similar resistance patterns, indicating that the workers probably acquired the drug resistant organisms from the birds. However, the possibility of transfer of resistance was not discussed. This finding is supported in the same study by an experimental investigation in which the birds were orally inoculated with a laboratory strain of *E.coli* K12J5+Lac-, the organism was subsequently recovered from workers who handled the birds [43].

ECONOMIC IMPACT

Eschericha coli infections in broiler chickens are believed to be responsible for significant economic losses to the broiler industry. It would be diffficult to evaluate the monetary lssesdue to these infections because *E.coli* can be a primary or secondary cause of disease. However, it is estimated that 26% of all poultry condemnations in the United States were due to airsacculitis [55], and the annual losses due to colisepticemia were estimated at $100 million[56]. In Canada, respiratory colibacillosis causes significant loss in meat-type poultry. Losses in both broiler chickens and turkeys occur from three weeks of age to market, and as carcass condemnations [57]. However, the total cost may be much higher when reduced growth and low feed conversion rate are considered.

Cellulitis is emerging as an important cause of economic loss as a result of condemnation of severely affected carcasses or degradation, and from trimming of carcasses with mild lesions. In the United States, based on an average condemnation rate of 0.16% cellulitis is estimated to cost the broiler industry about $12 million annually. The full cost, however, is much greater due to downgrading and salvage of many affected carcasses, and is estimated to be between $18 and $20 million annually [26].

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