

Antibiotic resistance profiles of *Salmonella enterica* serovars isolated from chicken farms in Brazil.

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Abstract

The present study was conducted to characterize the resistance profile of the *Salmonella enterica* serovars Enteritidis, Kentucky, Senftenberg and Saint Paul isolated from chicken farms in Brazil. We selected 42 samples that have presented resistance to cefotaxime, tetracycline, enrofloxacin, gentamicin, chloramphenicol and sulfa/trimethoprim. PCR tests were conducted to detect genes associated with the resistance phenotype. Forty-four (44) primers were tested, although only 13 genes were detected. Fourteen samples had the product amplified to at least one gene. We concluded that the phenotype of resistance could have been caused by other genes that were not investigated in this study. Therefore, further analysis must be conducted in order to investigate the mechanisms of resistance in *S. enterica*.

Key words:

Salmonella spp., Antibiotic resistance, Poultry

Introduction

Salmonella is one of the main causes of food poisoning worldwide. The main source of infection in humans is contaminated poultry meat. However, the incautious use of antibiotics as growth promoters in farm animals can turn into a public health problem. Antimicrobial-resistant bacteria can be transferred through contaminated food jeopardize the treatment of Salmonellosis.

In order to understand better the mechanisms involved in antibiotic resistance, the aim of present study was to investigate the genes associated with the resistance phenotype found in 42 samples of *Salmonella enterica* isolated from chicken farms in Brazil. The samples selected presented resistance to cefotaxime, tetracycline, enrofloxacin, gentamicin, chloramphenicol and sulfa/trimethoprim. This project is the first step to further understanding of the genetic mechanisms underlying antibiotic resistance, thus new tools can be designed to combat this issue.

Results and Discussion

PCR analyses confirmed the presence of 13 genes in 14 samples (Chart 1). Two samples presented the genes *tea*, *aada* and *aac1*, which belong to different classes, while one sample presented the genes *stra* and *strb*, both associated to gentamicin resistance. Any of the samples tested have amplified products for genes associated with chloramphenicol resistance. Figure 1 is representative for the PCR results obtained in this study.

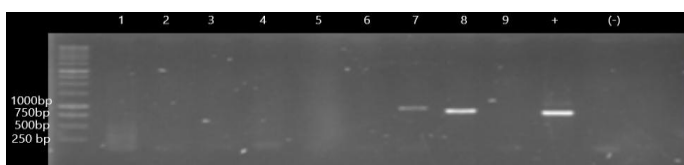


Figure 1. Representative image for the PCR products detected by agarose gel electrophoresis. Primer SHV, used for detection of β -lactamase genes. Amplicon size: 860bp. Lanes: 1 to 4: *S. Enteritidis*; 5 to 9: *S. Saint Paul*

Chart 1. Percentage of samples that presented a PCR amplification product for the genes tested.

Antibiotics	Samples per antibiotics/ Total of samples	Genes	% Per drug tested	% Total
Cefotaxime	11/42	<i>ctx</i>	9,09	0,42
		<i>shv</i>	27,27	1,26
Tetracycline	4/42	<i>tea</i>	50,00	0,84
		<i>qnrB</i>	8,30	0,42
Enrofloxacin	12/42	<i>qnrS</i>	24,90	1,26
		<i>parC</i>	8,30	0,42
		<i>parE</i>	8,30	0,42
		<i>dfx5</i>	100,00	0,42
Sulfa/trimethoprim	1/42	<i>strA</i>	11,10	0,42
		<i>strB</i>	11,10	0,42
		<i>aac4</i>	11,10	0,42
Gentamicin	9/42	<i>aadA</i>	22,20	0,84
		<i>aac1</i>	22,20	0,84
		<i>aac4</i>	11,10	0,42
		<i>aac4</i>	11,10	0,42

Conclusions

The resistance genes that were not found could be one never described or it is present in some mobile genetic elements other than plasmids. Genomic analysis should be done in order to characterize the other samples. A better understanding of these molecular mechanisms of resistance could help to control the selection of resistant strains.

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