INTRODUCTION

Shiga toxin-producing Escherichia coli (STEC) are a group of pathogenic Escherichia coli (E. coli) responsible for a spectrum of disease that includes hemorrhagic colitis and hemolytic uremic syndrome (HUS)¹

STEC can be broadly categorized into O157 and non-O157 groups based on their O surface antigen type.² Classical STEC detection algorithms were primarily targeted at biochemical differences specific to O157 STEC and missed a significant proportion of non-O157 STEC.³ Newer detection algorithms have been implemented aimed at improving the detection of non-O157 serogroups.⁴ In 2018, the Canadian Public Health Laboratory Network (CPHLN) published recommendations for detection of STEC including both O157 and non-O157 serogroups.⁴

The pathogenicity of STEC is conferred by the production of Shiga toxins. These toxins are the major virulence determinants of STEC and act by inhibition of protein synthesis.⁵ There are two types of Shiga toxins: Stx1 (comprised of subtypes 1a, 1c and 1d) and Stx2 (comprised of subtypes 2a-2g). Stx2 has been more strongly associated with severe disease and HUS.⁶

In this study, we aimed to analyze all non-O157 STEC isolates recovered from clinical stool samples in Alberta, Canada from 2018 to 2021. We retrospectively reviewed their demographic data, serotypes and Shiga toxin gene (stx) profile to describe the epidemiology of non-O157 STEC in Alberta.

METHODS

All stools submitted for bacterial pathogen detection in Alberta were tested for STEC in accordance with Canadian guidelines. Positive stool samples/enriched broth cultures were referred to Alberta Precision Laboratories- ProvLab for isolation. Mauve colonies on CHROMagar STEC plates underwent colony qPCR targeting the stx₁ and stx₂ genes. Serotyping was referred to the National Microbiology Laboratory. All non-O157 STEC from clinical samples were included in this retrospective analysis except for repeat isolations within one month.

RESULTS

A total of 729 isolates were identified from January 2018 to December 2021, representing 50 different serogroups. Serotyping data was not available for 7/729 isolates (0.9%)

Non-O157 Shiga Toxin Producing Escherichia coli (STEC) in Alberta, Canada from 2018-2021

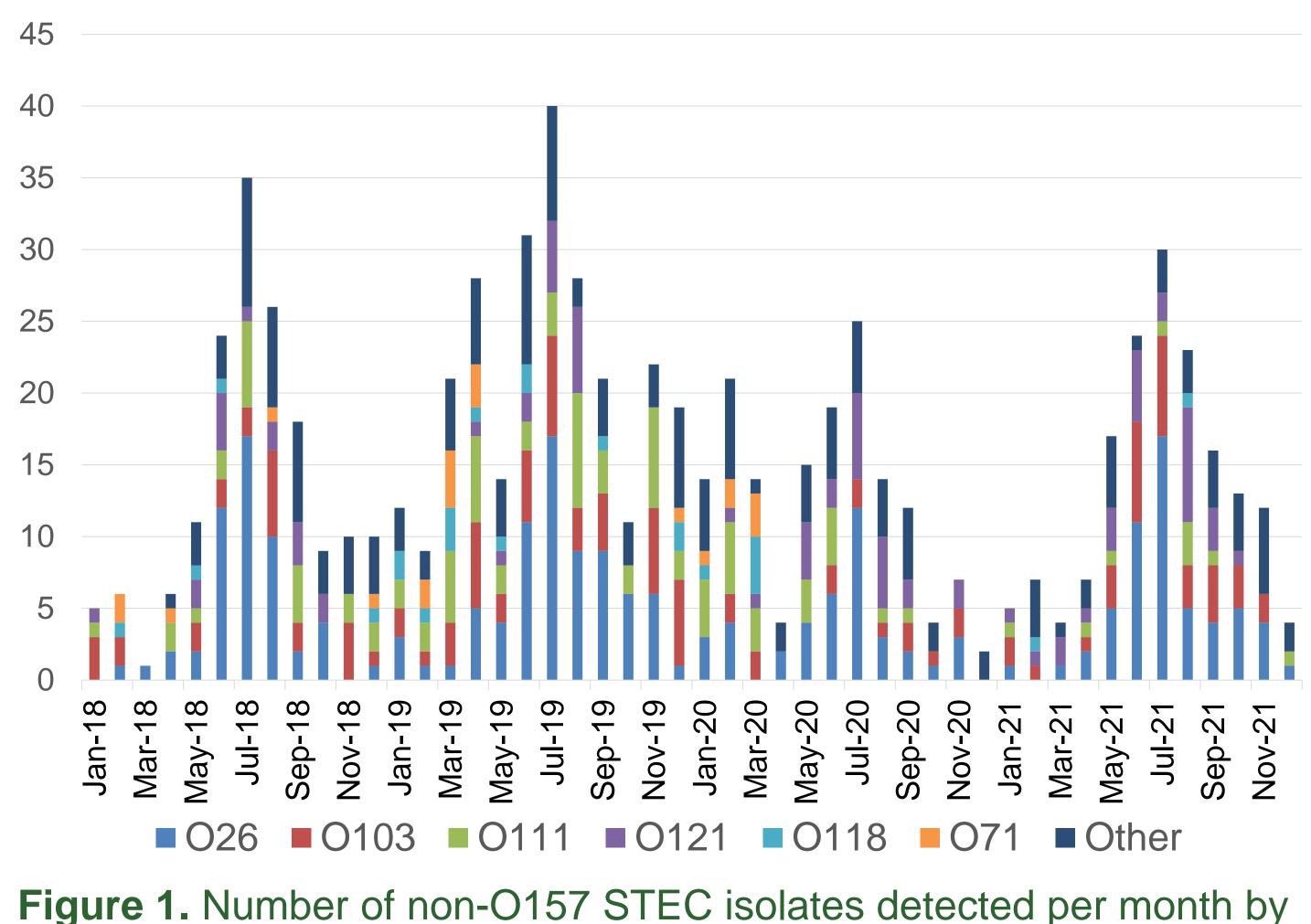
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RESULTS

Table 1. Number of serogroups and serotypes isolated during the study neriod

period			
Serogroup	Number of Isolates (%)	Serotype	Number of Isolates (% of serogroup
O26	221 (30.3)	H11	155 (70.1)
		H Non-motile	65 (29.4)
		H Undetermined	1 (0.5)
0103	116 (15.9)	H2	80 (69.0)
		H25	20 (17.2)
		H11	8 (6.9)
		H Non-motile	6 (5.2)
		H19	2 (1.7)
O111	93 (12.8)	H Non-motile	93 (100)
O121	80 (11.0)	H19	77 (96.3)
		H Non-motile	3 (3.7)
O118	24 (3.3)	H16	10 (41.6)
		H2	9 (37.5)
		H Undetermined	3 (12.5)
		H14	1 (4.2)
		H Non-motile	1 (4.2)
071	21 (2.9)	H11	16 (76.2)
		H8	3 (14.3)
		H Non-motile	2 (9.5)
Other	167 (22.9)	-	-
Unknown	7 (0.9)	-	-
Total	729 (100)	-	-

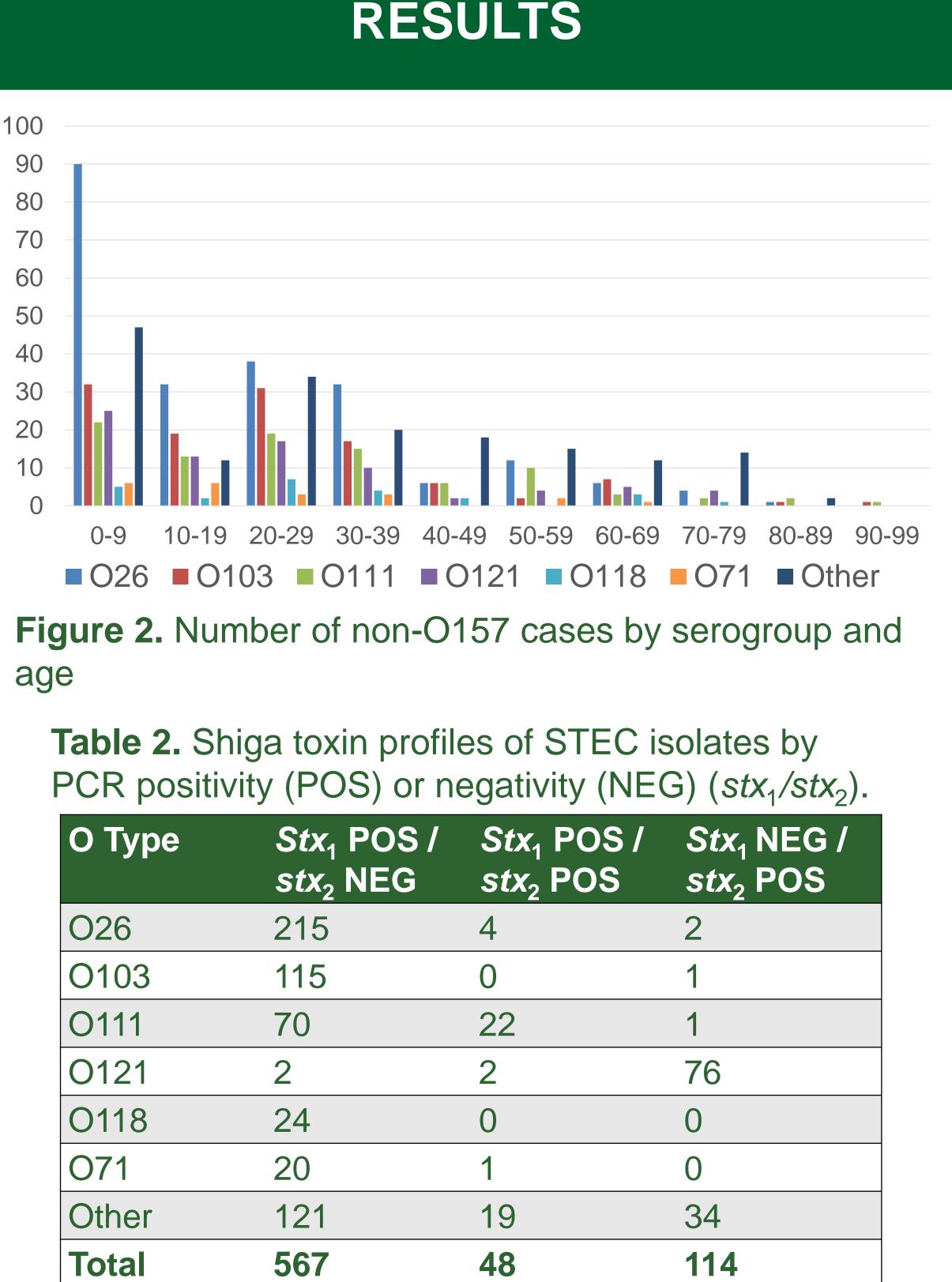


serogroup

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О Туре	Stx ₁ POS /		
	stx ₂ NEG		
O26	215		
O103	115		
O111	70		
O121	2		
O118	24		
071	20		
Other	121		
Total	567		

CONCLUSIONS

The epidemiology of non-O157 STEC in Alberta has not been previously described. Hundreds of cases are detected annually, particularly in the pediatric population. Non-O157 cases outnumber O157 cases; only 492 total O157 STEC cases were detected in the same region over the same time period. Non-O157 cases were concentrated in the summer months in keeping with prior reports.

The most prevalent serogroups isolated in Alberta were O26, O103, O111, O121, O118 and O71 which differs from British Columbia which has reported O121, O26, O103, O117 and O111 most commonly.⁷ This also differs from CDC FoodNet surveillance data which has reported O26, O103, O111, O121, O45 and O145 as their top 6.⁷ Although stx_1 positivity accounted for the majority of non-O157 isolates, 22% were positive for stx_2 which has been associated with more severe disease.⁶ These results underscore the importance implementing algorithms capable of detecting non-O157 STEC and highlight the need for further characterization of their virulence factors and clinical impact.