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METHOD

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Evaluation of chromogenic selective agar (CHROMagar STEC) for the direct detection of Shiga toxin-producing *Escherichia coli* from faecal specimens

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Abstract

Shiga toxin-producing *Escherichia coli* (STEC) are zoonotic pathogens that cause symptoms of severe gastrointestinal disease, including haemolytic uraemic syndrome (HUS), in humans. Currently in England, STEC serotypes other than O157:H7 are not cultured at the local hospital laboratories. The aim of this study was to evaluate the utility of CHROMagar STEC for the direct detection of STEC from faecal specimens in a diagnostic setting, compared to the current reference laboratory method using PCR targeting the Shiga-toxin gene (*stx*) to test multiple colonies cultured on MacConkey agar. Of the 292 consecutive faecal specimens submitted to the Gastrointestinal Bacterial Reference Unit that tested positive for *stx* by PCR, STEC could not be cultured on MacConkey agar or CHROMagar STEC from 87/292 (29.8%). Of the 205 that were cultured, 106 (51.7%) were detected on both MacConkey agar and CHROMagar STEC and 99 (48.3%) were detected on MacConkey agar only. All 106 (100%) isolates that grew on CHROMagar STEC had the *ter* gene cassette, known to be associated with resistance to tellurite, compared to 13/99 (13.1%) that were not detected on CHROMagar STEC. CHROMagar STEC supported the growth of 36/40 (90%) isolates harbouring *stx2a* or *stx2d,* the subtypes most frequently associated with progression to HUS. Of the 92 isolates harbouring *eae,* an important STEC virulence marker, 77 (83.7%) grew on CHROMagar STEC. CHROMagar STEC is a useful selective media for the rapid, near-patient detection of STEC that have the potential to cause HUS.

INTRODUCTION

Shiga toxin-producing *Escherichia coli* (STEC) are zoonotic pathogens that cause symptoms of gastrointestinal (GI) disease in humans, including diarrhoea, bloody diarrhoea, abdominal pain and nausea. A sub-set of patients develop haemolytic ureamic syndrome (HUS), which is a severe condition associated with renal, neurological and/or cardiac complications, and can be fatal [1]. The STEC group is characterized by the presence of genes encoding Shiga toxin (Stx), *stx1, stx2* or both. There are ten Stx subtypes, 1a, 1c and 1d, and 2a-2g. STEC harbouring *stx2a* and *stx2d* alone or in combination with other *stx* subtype genes, are significantly associated with HUS [2–4]. STEC associated with more severe GI symptoms also have the intimin (*eae* for *E. coli* attaching and effacing) gene, located on a pathogenicity island called the locus of enterocyte effacement (LEE), and associated with the intimate attachment of the bacteria to the human gut mucosa [5].

In England, standard microbiology investigation protocols for the detection of STEC focus on the isolation of STEC serotype O157:H7 on selective media of non-sorbitol fermenting (NSF) colonies (http://www.hpa.org.uk/ProductsServices/MicrobiologyPathology/UKStandardsForMicrobiologyInvestigations/TermsOfUseForSMIs/AccessToUKSMIs/SMIBacteriology/smiB30InvestigationofFaecalSpecimensforEnteric/). Since 2013 an increasing number of local hospital laboratories in England have implemented commercial PCR assays targeting GI pathogens, including all STEC serotypes, as a first-line diagnostic detection method [6]. In England, faecal specimens from patients with symptoms of HUS and/or that are PCR positive for STEC but culture negative for STEC O157:H7, may be referred to the Gastrointestinal Bacterial Reference Unit (GBRU) at Public Health England for culture of non-O157 STEC. Current protocols for the detection of STEC other than serotype O157:H7 (non-O157 STEC) are technically demanding and labour intensive [6, 7].

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Abbreviations: eae, *E. coli* attaching and effacing; GBRU, Gastrointestinal Bacterial Reference Unit; HUS, haemolytic uraemic syndrome; LEE, Locus of Enterocyte Effacement; STEC, Shiga toxin-producing *Escherichia coli*; stx, Shiga toxin.

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In recent years, a range for different chromogenic media, containing a combination of inhibitors, metabolic substrates and dyes, have been developed with the aim to improve the culture of non-O157 STEC [8–11]. Inhibitors prevent the growth of Gram-positive bacteria, and STEC and other Enterobacteriaceae are differentiated by the colour of their colonies, resulting from the production of different metabolic by-products and their reaction with the dyes in the media. The implementation of selective agar for the detection of non-O157 STEC would facilitate rapid, near-patient testing at the local level, and improve the efficiency and cost effectiveness of the workflow at the reference laboratory. The aim of this study was to evaluate the utility of CHROMagar STEC for the direct detection of STEC from faecal specimens in a diagnostic setting, compared to the current reference laboratory method using PCR targeting the Shiga-toxin gene (*stx*) to test multiple colonies cultured on MacConkey agar.

METHODS

Faecal specimens submitted to local hospital diagnostic laboratories from hospitalized patients, and community cases reporting to their general practitioner with symptoms of GI disease were included in this study. All faecal specimens submitted to GBRU between April and June 2019 were inoculated onto MacConkey agar and into tryptone soya broth (TSB), and incubated at 37 °C, as per the standard protocol [7]. For the purposes of this evaluation, faecal specimens were also cultured onto CHROMagar STEC, and incubated at 37 °C. STEC appear as mauve-coloured colonies on CHROMagar STEC and can be differentiated from other Enterobacteriaceae and other Gram-negative bacteria that appear as blue and colourless colonies, respectively.

Following overnight incubation, DNA was extracted from TSB using Instagene (Bio-Rad Catalog No. 732–6030) and tested using the EU-RL VTEC real-time PCR primers and probes detecting *stx*1, *stx*2, *eae* (intimin) and O157*rfbE* (http://www.iss.it/binary/vtec/cont/VTEC_RealTime.pdf) on a Rotorgene Q (Qiagen, UK) as described previously [7]. For all faecal specimens that tested positive for *stx*, ten colonies were selected from the MacConkey agar and retested using the same PCR. Ten colonies were picked from the MacConkey agar because the agar is not non-selective for STEC, and PCR of ten colonies is required to maximize the chance of identifying the *stx*-positive colonies. In contrast, because CHROMagar STEC agar is selective for STEC, a single mauve colony was retested using the same PCR.

Those colonies harbouring the *stx* genes were sequenced, and serotype, *stx* profile, and the presence of *eae* and the *ter* gene cluster were determined from the WGS data, as described previously [12]. Briefly, genomic DNA was extracted, fragmented and tagged for multiplexing with Nextera XT DNA Sample Preparation Kits (Illumina) and sequenced using the Illumina HiSeq 2500. The reference databases, *SerotypeFinder* and *Virulencefinder*, containing the gene sequences encoding the O and H antigen groups, *eae* and the *ter* gene cluster were constructed as described by Joensen *et al.,* [13]. Using the *GeneFinder* tool (Doumith unpublished), fastq reads were mapped to the genes in the reference databases using Bowtie 2 [14]. Stx subtyping was performed as described by Ashton *et al.*, [15].

Comparisons were made with the presence and absence of *eae* in STEC and the presence of the *ter* gene cluster, and the ability to grow on CHROMagar STEC. A chi-squared test was used to assess statistical significance. A *P*-value of <=0.05 was deemed statistically significant. All statistical analyses were performed using stata 12.0 (Stata Corporation).

RESULTS

During the time frame of the study, 292 faecal specimens tested positive for *stx* by PCR. STEC could not be cultured on MacConkey or CHROMagar STEC from 87/292 (29.8%) consecutive faecal specimens submitted to GBRU. Of the 205 isolates that were cultured, 106 (51.7%) were recovered from both MacConkey agar using the 'ten-colony' method described above and CHROMagar STEC and 99 (48.3%) were detected on MacConkey agar only. Characteristics of the isolates of STEC cultured on both MacConkey agar and CHROMagar STEC, and those cultured on MacConkey agar alone, including serotype, *stx* profile and the presence or absence of *eae* and the *ter* gene cassette encoding resistance to tellurite, were compared.

Of the 106 isolates that were successfully cultured using both methods, 20 different serotypes were identified, and there were 36 different serotypes identified in the 99 isolates that were cultured using the current method (Tables 1 and 2). The most common non-O157 STEC serotypes that were cultured on CHROMagar STEC were O26:H11 (*n*=35/106, 33.0%) and O146:H21 (*n*=18/106, 17.0%). Of the STEC serotypes that could not be cultured on CHROMagar STEC, the most common were O91:H14 (*n*=21/99, 21.2%) and O128ab:H2 (*n*=11/99, 11.1%).

All 106 (100%) isolates that were isolated on CHROMagar STEC had the *ter* gene cassette, known to be associated with resistance to tellurite [16, 17]. Of those isolates that were not detected on CHROMagar STEC, 13/99 (13.1%) had the *ter* gene cassette. All 13 STEC isolates that had the *ter* gene cassette but were not detected from the faecal specimen when inoculated directly on to CHROMagar STEC, grew within 24 h on CHROMagar STEC when inoculated from the subculture that was detected using the MacConkey agar using the 'ten-colony' method (Table 2). This indicated that the failure to detect these isolates on CHROMagar STEC was caused by overgrowth of competing commensal bacteria, rather than inhibition on the selective agar.

The number of isolates testing positive for *eae* that were cultured using both methods (CHROMagar STEC and MacConkey ten-colony method) and the current MacConkey 'ten-colony' method only, was 77/106 (72.6%) and 15/99 (15.2%), respectively. Among 92 *eae*-positive STEC strains, 88 (95.7%) harboured the intact *ter* gene cluster and were tellurite resistant. In comparison, 41/114 (36.0%)

Table 1. stx subtypes, presence of eae and serotype of STEC that were cultured on CHROMagar

Stx subtype	No. (*n*=106)	Serotype
Stx1c	10	O146:H21 (8); O153-O178:H7 (2)
Stx1a+eae	31	O26:H11 (18); O103:H2 (3); O103:H8; O123:H2 (3); O71:H2 (2); O111:H8; O151:H2; O156:H25; O182:H25
Stx1c+stx2b	10	O146:H21 (9); O166:H28
Stx1a+stx2 a+eae	7	O26:H11 (5); O111:H8; O71:H8
Stx2b	11	O146:H21 (1); O146:H28 (8); O27:H30; O166:H28
Stx2a+eae	15	O26:H11 (10); O145:H28 (5)
Stx2c+eae	2	O26:H11 (2)
Stx2a+stx2 c+eae	1	O177:H25
Stxd +eae	1	O80:H2
Stx1a+stx2 c+eae+O157	2	O157:H7 (2)
Stx2c+eae+O157	10	O157:H7 (10)
Stx2a+eae+O157	1	O157:H7
Stx2a+stx2 c+eae+O157	5	O157:H7 (5)

Table 2. stx subtypes, presence of eae and serotype of STEC that could not be detected on CHROMagar

Stx subtype	No. (*n*=99)	Serotype
Stx1a	8	O91:H14 (3); O117:H7 (4); O152:H8
Stx1c	20	O78:H4 (4); O81:H21 (2); O126:H8 (2); O76:H19 (6); O128ab:H2; O146:H21; O43:H2; O38:H26 (2); O153-O178:H7
Stx1a+eae	8	**O84:H4 (2); O111:H8 (2); O103:H2 (1);** O103:H2 (2); **O108:H25**
Stx1a+stx2b	13	O91:H14 (13)
Stx1c+stx2b	19	O76:H19 (2); O128ab:H2 (6); O113:H4 (4); O112:H2; O38:H26 (2); O81:H21; O102:H6; O166:H28; O123:H10
Stx1a+stx2 c+eae	1	**O71:H8**
Stx1a+stx2 a+eae	4	**O26:H11 (2): O165:H25 (2**)
Stx2b	17	O91:H14 (5); O128ab:H2 (4); O?:H45 (3); O?:H8; O87:H16; O146:H21; O166:H28 (2)
Stx2c	1	O142:H16
Stx2a	2	O8:H19; O178:H19;
Stx2a+eae	1	**O145:H28**
Stx2d	2	O113:H4; O113:H21
Stx2d+eae	1	**O145:H25**
Stx2g	2	O2:H25; O?:H7

Serotypes highlighted in bold had the *ter* gene cluster; they were not detected from the faecal specimen when inoculated directly on to CHROMagar but grew within 24 h on CHROMagar when inoculated from the subculture that was detected using the MacConkey agar using the ten-colony method.

eae-negative isolates had the *ter* gene cluster. In this study, the presence of *eae* in STEC was significantly associated with the presence of the *ter* gene cluster (P=0.001) and the ability to grow on CHROMagar STEC (P=0.001). Therefore, CHROMagar STEC supported the growth of 77/92 (83.7%) isolates harbouring *eae*.

The most common *stx/eae* profiles exhibited by the isolates that were cultured on CHROMagar STEC were *stx1a+eae* (*n*=21/106, 19.8%), and *stx2a+eae* (*n*=15/106, 14.2%). Of the *stx* profiles exhibited by the isolates that could not be cultured on CHROMagar STEC, the most common were *stx1c* (*n*=20/99, 20.2%) *stx1c+stx2*b (*n*=19/99, 19.2%) and *stx2b* (*n*=17/99, 17.2%). Of the isolates that grew on CHROMagar STEC 30/106 (28.3%) had *stx2a* compared to 10/99 (10.1%) that failed to grow on CHROMagar STEC. Of the ten that failed to grow, six had the *ter* gene cassette, and grew on CHROMagar STEC following sub-culture. Therefore, CHROMagar STEC supported the growth of 36/40 (90%) isolates harbouring *stx2a* or *stx2d*.

DISCUSSION

Previous studies evaluating CHROMagar STEC have focused on the non-O157 serogroups most likely to cause severe symptoms, STEC O26, O103, O111, O121, O145 and O45, referred to in the literature as the 'top six'; while others have included a wider range of STEC serogroups [8–11, 17, 18]. With the exception of Wylie *et al.* [9], these studies have used sub-cultured archived collections of isolates of non-O157, rather than culturing directly from faecal specimens. In contrast, the purpose of the study described in this report was to evaluate the utility of CHROMagar STEC for the detection of non-O157 STEC serotypes in a real-time diagnostic setting.

No STEC was cultured using either method for 29.8% of the specimens tested. PCR is a more sensitive test than culture, and the most likely explanation for failure to culture is that

the bacterial load in the specimen was below the detection limit of either of the culture methods used [7]. In addition, competing commensal bacteria and various inhibiting factors, that are normally present in faecal specimens, could impact on the failure to recover STEC in these samples. Of the STEC cultured from consecutive faecal specimens that tested positive for *stx* submitted to GBRU during the study period, 51.7% were recovered using CHROMagar STEC. Using the same chromogenic agar, recovery rates for the detection of STEC O157 and the top six non-O157 STEC serogroups reported by Hirvonen *et al*. and Wylie *et al*. were 74.3 and 86.5%, respectively [8, 9]. Studies where a more diverse range of STEC serogroups were selected with the purposes of constructing a more challenging strain set report a lower recovery rate. In the study by Kalule *et al*. CHROMagar STEC agar supported the growth of less than half the strains tested (46.8%) [17].

All 106 isolates of STEC that grew on CHROMagar STEC had the *ter* gene cassette, which in STEC O157:H7 comprises eight genes *terZ, terA, terB, terC, terD, terE, terF* and *terW* [19, 20]. Correlation of growth of STEC on CHROMagar STEC with the presence of the *ter* gene cassette encoding resistance to tellurite is well documented in the literature [8–11, 17, 18]. Successful detection of STEC in a complex background requires an agar medium that suppresses the growth of background micro-organisms with minimal suppression of target STECs. Potassium tellurite has been added to agar media to facilitate the isolation of STEC O157:H7 since the mid-1990s, with resistance to tellurite being the primary principle of the selective medium [21]. The *ter* genes are commonly present in clinically relevant STEC isolates, including STEC O157:H7. However, some clinically relevant non-O157 STEC serogroups, such as O91 and O113, cannot grow on standard media containing 2.5 $\mu g\,ml^{-1}$ of potassium tellurite [16, 22].

Explanations as to why the STEC isolates harbouring the *ter* gene cluster were missed on primary culture included: (i) heavy growth of other tellurite-resistant bacteria masking the presence of the mauve STEC colonies and (ii) more than one strain of bacteria growing as mauve colonies on the agar plate and an *stx*-negative colony being selected for re-PCR. Furthermore, non-O157 STEC strains displayed mauve colouring with many shades on CHROMagar STEC agar, as described previously [23]. The mauve colour ranges from purple through to mauve to grey, and those colonies at the grey end of the spectrum may be more easily missed on the primary culture plate.

CHROMagar STEC was developed to facilitate the detection of the serotypes of STEC most commonly associated with severe GI disease, including HUS. Data from 2017, shows that the most common STEC serotypes causing severe GI disease in the UK are STEC O157:H7 and O26:H11 and both serotypes can be cultured on CHROMagar STEC [1, 6, 12]. Moreover, in this study, 75% of STEC harbouring *stx2a* or *stx2d*, the *stx* subtypes associated with more severe GI symptoms, were detected using the CHROMagar STEC, 90% including those that were resistant to tellurite but were missed on primary culture. The *eae* gene is a marker for virulence linked to human disease and may be beneficial to survival of the organism in the food chain [16]. In this study, the presence of *eae* in STEC was significantly associated with the presence of the *ter* cluster (P=0.001), and therefore the ability of STEC to grow on CHROMagar STEC [24]. The results from this study showed that the majority of STEC exhibiting the potential to cause severe gastrointestinal symptoms, specifically those harbouring *stx2a* or *stx2d* (36/40, 90%) and *eae* [77/92 (83.7%)], were detected using CHROMagar STEC.

Regardless of whether the diagnostic microbiology laboratory has implemented PCR for the detection of STEC (and other GI pathogens), the addition of CHROMagar STEC to the standard microbiological investigation protocols for faecal specimen will improve the detection of non-O157 STEC. Selected colonies identified on CHROMagar STEC can be sent to the reference laboratory for confirmation of STEC and typing. For laboratories that have implemented PCR, use of CHROMagar STEC will enable them to culture non-O157 STEC directly from the faecal specimens testing positive for *stx*. Non-O157 STEC is becoming an increasingly serious public health concern in the UK [6, 12, 25]. Robust and reliable detection of non-O157 STEC will enhance surveillance activities, facilitate outbreak detection and investigation, and improve the diagnosis of STEC-HUS.

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Conflicts of interest
The authors declare that there are no conflicts of interest.

Ethical statement
The authors declare that there is no requirement for ethical approval for this submission. This work was undertaken to inform the delivery of patient care and to prevent the spread of infection, defined as USUAL PRACTICE in public health and health protection.

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