

1 Haugum et al., Supplemental material, 20140507

2 Table S1. PCR primers used in the study.

Primer	Sequence (5'-3' direction)	Target gene	Amplicon size	Reference
<b>SLTI 1</b>	AAA TCG CCA TTC GTT GAC TAC TTC T	<i>stx1</i>	368 bp	Brian et al. 1992
<b>SLTI Rnew</b>	CCA TTC TGG CAA CTC GCG ATG CA			This study
<b>SLTI TaqMan</b>	FAM-AAC CTC ACT GAC GCA GTC TGT GGC AAG AGC-BHQ1			This study
<b>SLTII Fnew</b>	CAG TCG TCA CTC ACT GGT TTC ATC	<i>stx2</i>	283 bp	This study
<b>SLTII Rnew</b>	GGA TAT TCT CCC CAC TCT GAC AC			This study
<b>SLTII TaqMan</b>	HEX-CTG TCA CGG CAG AAG CCT TAC GCT TCA GGC-BHQ1			This study
<b>eae-Fny</b>	TTC ATT GAT CAG GAT TTT TCT GG	<i>eae</i>	105 bp	This study
<b>eae-Rny</b>	GCT CAT GCG GAA ATA GCC			This study
<b>eae-P</b>	FAM-ATA GTC TCG CCA GTA TTC GCC ACC AAT ACC			Nielsen et al. 2003
<b>efa1-F</b>	ATC AGA AGC CCG ACT ACG	<i>efa-1/lifA</i>	193 bp	This study
<b>efa1-R</b>	AAC ATT TGC CAG ACC AAG G			This study

3

- 4 Table S2. Number of virulence genes identified in various STEC serotypes by PCR analysis of 138 HUS-associated and non-HUS STEC strains
- 5 diagnosed at St. Olavs Hospital, Trondheim, Norway during the period 1996-2011.

Serogroup	<i>stx1</i>	<i>stx2</i>	<i>stx2</i> subtype	<i>eae</i>	<i>ehxA</i>	<i>nleB</i>	<i>nleE</i>	<i>ent</i>	<i>efa-1/lifA</i>	<i>nleA</i>	<i>nleF</i>	<i>nleH1-2</i>	<i>espK</i>	HUS-associated	non-HUS	Total
<b>O145</b>	18	8	2a	28	28	28	28	28	26	26	28	28	28	7	21	28
<b>O103</b>	18	0	0	20	21	21	21	21	21	4	21	21	21	2	19	21
<b>O157</b>	9	20	2a (n=2), 2a+2c (n=1), 2c (n=17)	20	20	20	20	20	15	20	19	20	20	0	20	20
<b>O26</b>	8	3	2a	11	11	11	11	11	11	11	11	11	11	1	10	11
<b>O121</b>	0	9	2a	9	9	9	9	9	9	9	9	9	8	5	4	9
<b>SF O157</b>	0	6	2a	9	9	9	9	9	9	9	9	9	9	9	0	9
<b>O111</b>	1	0	0	1	1	1	1	1	1	1	1	1	1	0	1	1

<b>Others</b>	24	25	2a (n=8), 2a+2c (n=3), 2b (n=10), 2c (n=1), 2d (n=2), 2g (n=1)	10	24	7	6	7	5	7	10	10	8	0	39	39
<b>Total</b>	78	71	-	108	123	106	105	106	97	87	108	109	106	24	114	138

6 Table S3. Comparison of potential virulence genes in 138 STEC strains diagnosed in patients where  
7 STEC was analyzed irrespective of symptoms (patients <2 years old) and on specific suspicion of STEC  
8 disease (patients ≥2 years old) at St. Olavs Hospital, Trondheim, Norway during the period 1996-  
9 2011.

Gene	Age <2 years n=50		Age ≥2 years n=88		P
	No. of strains	(%)	No. of strains	(%)	
<i>stx2</i>	20	(40)	51 <sup>1</sup>	(58)	0.05
<i>stx2a</i>	9	(18)	28	(31.8)	0.1
<i>stx1</i>	27	(54)	51	(58)	0.7
<i>eae</i>	44	(88)	64	(72.7)	0.05
<i>ehxA</i>	47	(94)	76	(86.4)	0.3
<i>nleB</i>	44	(88)	62	(70.5)	0.02
<i>nleE</i>	43	(86)	62	(70.5)	0.06
<i>ent</i>	44	(88)	62	(70.5)	0.02
<i>efa-1/lifA</i>	39	(78)	58	(65.9)	0.2
<i>nleA</i>	34	(68)	53	(60.2)	0.5
<i>nleF</i>	45	(90)	63	(71.6)	0.02
<i>nleH1-2</i>	45	(90)	64	(72.7)	0.02
<i>espK</i>	42	(84)	64	(72.7)	0.1

10 <sup>1</sup>This number does not include one SF O157:H- strain (St. Olav75, see Table 1) previously found to be  
11 positive for *stx2a* and one strain of unknown serotype (St. Olav154, see Table 1) previously found to  
12 be positive for *stx2b*.

13

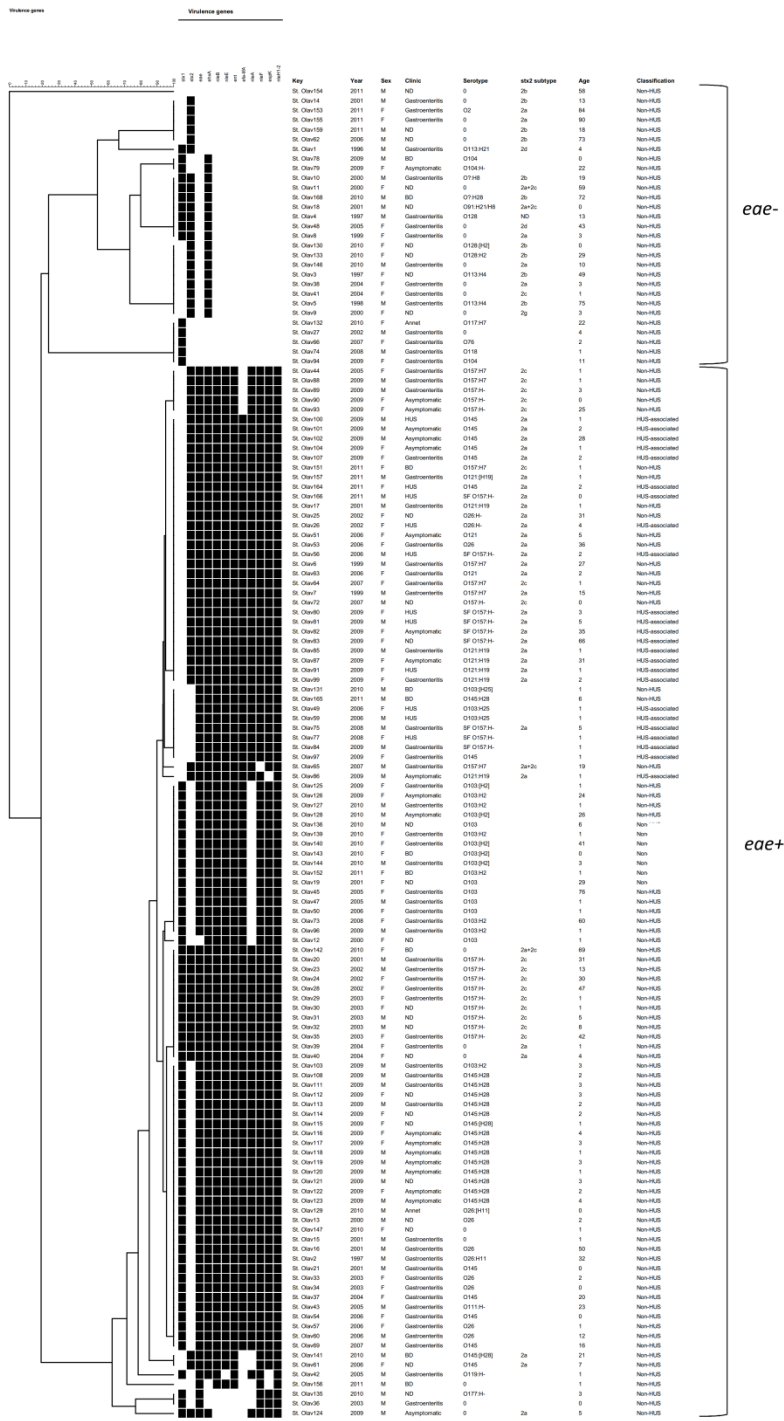


Figure S1

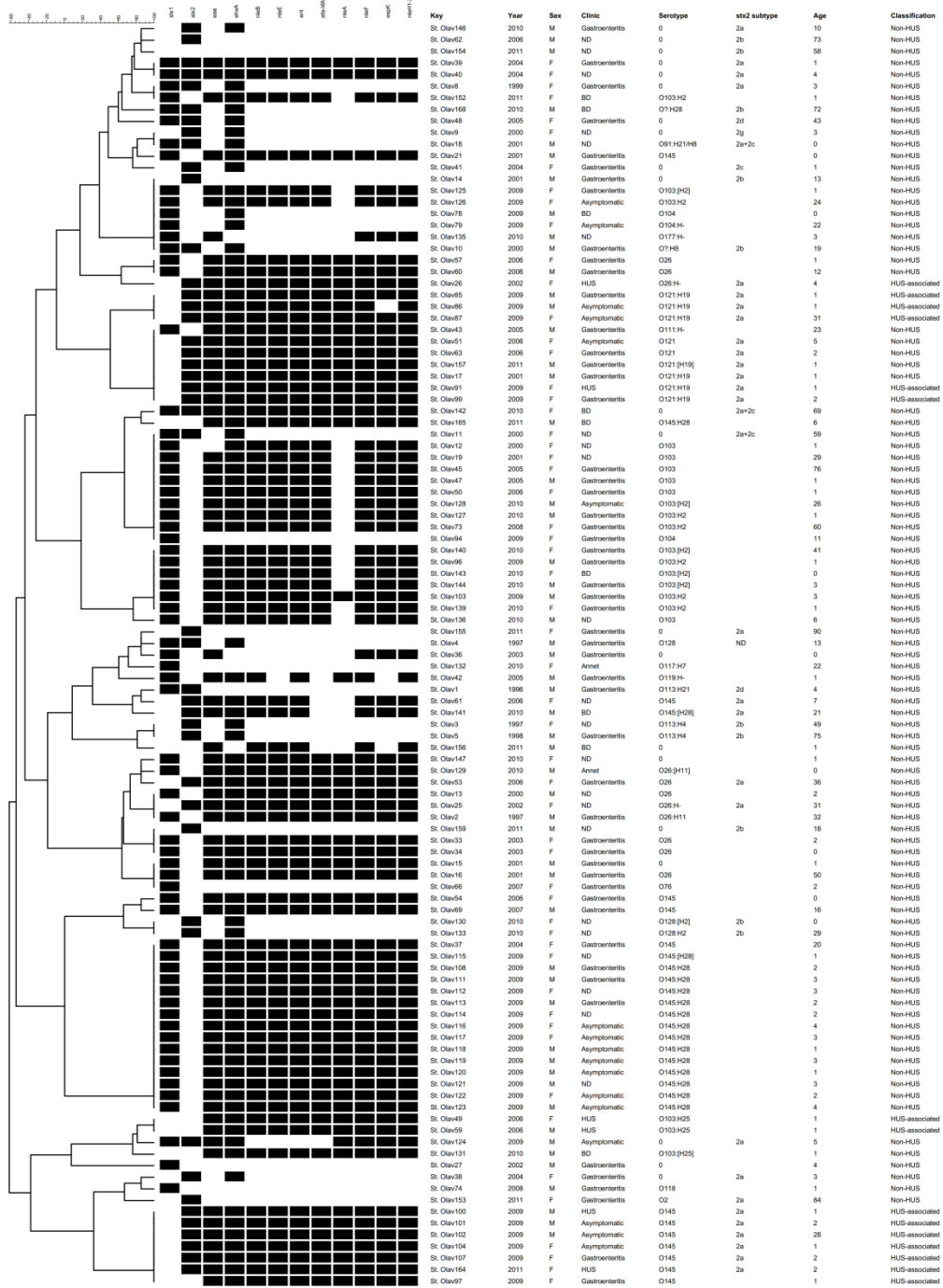


Figure S2

15

16

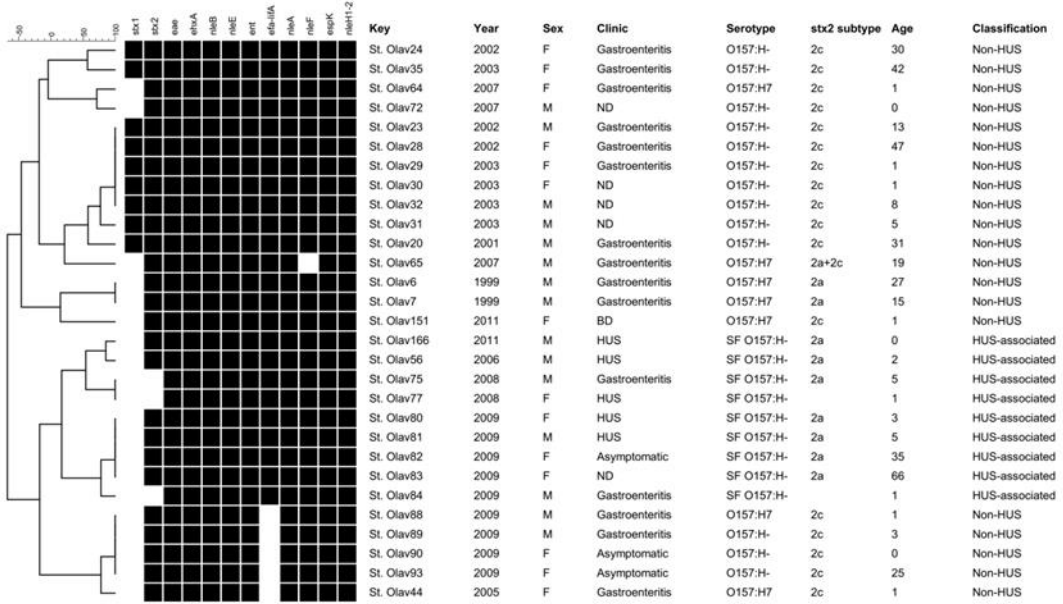


Figure S3

19 **Figure legends**

20 Figure 1. Cluster analysis of potential virulence genes in STEC. *eae* negative and *eae* positive STEC  
21 were mainly separated in two clusters. One exception was one *eae* negative strain that clustered  
22 among the *eae* positive strains due to the presence of some of the potential virulence genes. All HUS-  
23 associated strains clustered among *eae* positive strains and harbored all the potential virulence  
24 genes investigated in the study. For further details (serotype, *stx2* subtype etc.), please see Figure S1.

25

26 Figure S1. Cluster analysis of potential virulence genes in STEC. *eae* negative and *eae* positive STEC  
27 were mainly separated in two clusters. One exception was one *eae* negative strain that clustered  
28 among the *eae* positive strains due to the presence of some of the potential virulence genes. All HUS-  
29 associated strains clustered among *eae* positive strains and harbored all the potential virulence  
30 genes investigated in the study. Strains with serotype 0 did not belong to any of the serotypes tested  
31 for in the study.

32

33 Figure S2. MLVA dendrograms of the 109 non-O157 STEC strains, using seven repeat loci. The strains  
34 were distributed in 48 distinct MLVA genotypes. The virulence gene profile of each strain is  
35 displayed.

36

37 Figure S3. MLVA dendrograms of O157 and SF O157 with the virulence gene profile displayed. The  
38 strains were distributed in 17 distinct MLVA genotypes. The virulence gene profile of each strain is  
39 displayed.