NON-O157 SHIGA TOXIN-PRODUCING ESCHERICHIA COLI (STEC)

THE ORGANISM/TOXIN

These organisms form a diverse group of *Escherichia coli* that are capable of producing shiga-toxin(s), as is *E. coli* O157:H7. However, they are of widely differing pathogenic potential, varying from those that can cause disease similar to that produced by *E. coli* O157:H7 to those that have never been associated with disease.

By definition all STEC must produce one of two toxins (denoted Stx1 and Stx2), but other factors are also involved in pathogenicity and it is the possession of these that seems to determine the virulence of any one serotype. Other factors known to be involved include the ability to adhere to intestinal cells, and the ability to produce a haemolysin.

An isolate possessing the ability to produce either Stx in the absence of other virulence determinants is unlikely to be a major pathogen.

Individual members are denoted by their O and H serotypes.

GROWTH AND ITS CONTROL

Growth:

The behaviour of these organisms is largely the same as for serotype O157:H7. Only basic information is given below as data on specific characteristics of individual serotypes are lacking. Refer to the E. coli O157:H7 datasheet for more detail.

Temperature: Optimum 37°C, minimum 7-8, maximum 46°C. Doubling time approx. 0.4h at 37°C.

pH: Optimum 6-7, range 4.4 to 9.0 or 10.0. The limit at the low pH end depends on the acidulant used. Mineral acids such as HCl are less inhibitory than organic acids (e.g. acetic, lactic) at the same pH. Growth was inhibited in the presence of 0.1% acetic acid (pH 5.1).

Atmosphere: Can grow in the presence or absence of oxygen. *E. coli* can grow at 8 and 9°C on beef under vacuum-packaged conditions, but not under 100% CO_2.

Water activity: Optimum growth is at a_w=0.995 minimum a_w= 0.950 (about 8% NaCl).

Survival:

Temperature: Survives well in chilled and frozen foods.

pH: Dies at pH values outside the range allowing growth. However, when exposed to low pH at low temperature, cells may survive for some time.

Inactivation (CCPs and Hurdles):

Temperature: Rapidly inactivated by heating at 71°C D time at 54.4°C = 40 min D time at 60 = 0.5–0.75 min D time at 64.3°C = 0.16 min Freeze thawing can lead to a reduction in numbers but the effect is strain dependent. D times may increase if the organism is heat shocked prior to heat treatment.

pH: Inactivated at pH values outside the range allowing growth at rates dependent on the conditions encountered. Inactivation is generally more rapid at warmer temperatures at low pH.

THE ILLNESS

For information on Haemorrhagic Colitis (HC), Haemolytic Uraemic Syndrome (HUS) and Thrombocytopaenic purpura (TTP) see the *E. coli* O157:H7 data sheet.

Information from the USA estimates that non-O157 STEC cause disease at half the rate as the O157:H7 serotype. A hospitalisation rate of 29.5% and case fatality rate of 0.8% have been estimated.

Non-O157 STEC serotypes: These are arranged below in three tiers of significance based on the history of the serotype in causing disease. (N.B. This list is likely to change over time as more STEC are recognised and the virulence of serotypes becomes better established).

Have caused HUS outbreaks or clusters: O26:H11, O111:H-, O113:H21.

Involved in HUS cases but not outbreaks: O2:H6, O5:H-, O6:H1,H4, O9:H-, O18:H7, O22:H8, O26:H-, O46:H31, O48:H21, O55:H6,H7,H10,H-, O75:H5, O86:H40, O91:H10,H21,H-, O98:H-, O103:H2,H7(?), O104:H-,H2, O105ac:H18, O111:H2,H8, O111ac:H-, O112ab:H2, O115:H10, O119:H6, O125ac:H-, O121:H19, 128ab:H2,H25, O118:H12,H16, O145:H25,H28,H-, O146:H8, O153:H25, O163:H19, O165:H-,H19,H25, O168:H-, OX3:H-.

Not implicated in cases of HUS to date: O1:H-,H1, O2:H5,H7,H29, O4:H10,H-, O5:H16, O6:H-, O18:H15,H-, O23:H7,H16, O25:H-, O26:H2,H32, O39:H4, O45:H-,H2, O50:H-,H7, O73:H19,H34, O78:H-, O82:H8, O84:H2,HNT, O91:H14,H40, O100:H32, O101:H-, O104:H21, O105:H18, O107:H27, O111:H5,H35,H49, O113:H7,H32, O114:H4,H48, O117:H4, O118:H-,H2,H12,H30, O119:H-, O121:H-, O125:H8,H-, O126:H-,H8, O128:H2,H8,H12,H-, O128ab:H-,H8, O146:H2,H21,H28,H31, O163:H-, O166:H12,

O172:H-, OX3:H2,H21.
Not implicated in human disease: O2:H-, O6:H34, O8:H19, O38:H21, O39:H49, O44:H-,H25,H28,H40, O46:H38, O63:H19, O69:H11, O76:H?, O84:H-, O88:H2,H25, O98:H25, O113:H-, O116:H21, O119:H25, O121:H7, O125:H19, O136:H12, O145:H8, O153:H31, O156:H-,H25, O162:H21, O165:H52, O166:H-,H15, O169:H19.

NZ Incidence: 1.8 cases/100,000 in 1999, 1.3 in 1998. N.B. This is for all shiga toxin-producing *E. coli* and in New Zealand serotype O157:H7 accounts for around 90% of the notified cases. There has been one death in New Zealand from serotype O113:H21.

Treatment: Dialysis, maintenance of fluid balance and treatment of hypertension in cases of HUS.

SOURCES

Human: Some serotypes appear to be restricted to man, e.g. O1, O55:H7 and H:10 and O148:H21.

Animal: Ruminant animals, notably bovines, seem to be a natural reservoir of many of the non-O157 STEC that cause disease in humans.

Food, environment, transmission routes: Little is known about the distribution of these organisms in food and the environment. However, it seems likely that the situation will be similar to that for serotype O157:H7. Non-O157 STEC are likely to be much more common than serotype O157:H7 in foods, but only a small proportion of the isolates appear to be pathogenic to humans.

Non-O157 STEC have been detected in beef, pork and lamb mince, and unpasteurised milk. It has been estimated that 85% of cases are foodborne.

OUTBREAKS AND INCIDENTS

Outbreaks: There have been no outbreaks of non-O157 STEC disease in New Zealand. The following are examples from overseas.

Fermented sausage: 158 cases, 23 HUS, 1 death. Serotype O111:H-. Control measure failure: incorrect fermentation.

Milk: 18 cases, 16 had HC. Serotype O104:H21. Control point failure: possible post-pasteurisation contamination.

Tap water: 5 cases, all HUS, 1 death. Serotype O26:H11 (possible involvement of O157 too).

Ice: 58 cases, 2 HUS. Serotype O111:H8.

Source not determined: 100 cases. Serotype O145:H-.

Epidemiological Studies: Cases caused by O118:(H16,H-) have been associated with rural environments and evidence for zoonotic transmission described. Similarly a cluster of 3 cases caused by serotype O103 was linked to exposure to a calf. Consumption of fish has been found to be a risk factor in sporadic cases (although difficult to rationalise from microbiological data), and contact with dogs and consumption of shellfish protective.

In parts of Europe, where non-O157 serotypes predominate in STEC infections, the pattern of transmission may be atypical, i.e. no association with beef products.

Person-to-person spread is as important with these serotypes as it is with *E. coli* O157:H7.

A study in Dunedin isolated O26:H11 and O128:H2 from the faeces of children.

ADEQUATE PROCESSING GUIDELINES

N.B. These guidelines have been derived from published information. Industry is advised to ensure that processing steps they are using are adequate to meet their particular food safety objectives.

Cook meats to:	**Internal temperature reached**	**Time**
Minced meats, hamburger patties (beef, veal, lamb, pork) + pork cuts	71°C	15 sec
Minced poultry	74°C	″
Meat cuts (beef, veal, lamb), fish, seafood	63°C	″
Poultry, breast	77°C	″
Poultry, whole	82°C	″
Hold foods at	≤ 5°C or ≥ 60°C	
Reheat cooked foods to	74°C	Instantaneous
Acidify foods to pH 3.6 or below		
Avoid cross contamination from raw to ready-to-eat foods		
Thoroughly wash all fruit and vegetables		

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