





Recommendations for the Public Health Management of Gastrointestinal Infections 2019

Principles and practice

A joint guidance from Public Health England and the Chartered Institute of Environmental Health

About Public Health England

Public Health England exists to protect and improve the nation's health and wellbeing, and reduce health inequalities. We do this through world-leading science, research, knowledge and intelligence, advocacy, partnerships and the delivery of specialist public health services. We are an executive agency of the Department of Health and Social Care, and a distinct delivery organisation with operational autonomy. We provide government, local government, the NHS, Parliament, industry and the public with evidence-based professional, scientific and delivery expertise and support.

Public Health England Wellington House 133-155 Waterloo Road London SE1 8UG Tel: 020 7654 8000 www.gov.uk/phe Twitter: @PHE_uk Facebook: www.facebook.com/PublicHealthEngland

Prepared by: Toyin Ejidokun, Jeremy Hawker, Lorraine Lighton, Karthik Paranthaman, Rhianwen Stiff and Gemma Ward. We are grateful for the contributions made by additional members of the guidelines working group, namely Bob Adak, Neil Anstey, Matthieu Pegorie and Ian Gray.

We are also grateful for the input provided by colleagues across Public Health England, the devolved administration, the Chartered Institute for Environmental Health and the Food Standards Agency. The document is being published as Interim recommendations.

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Executive summary

This guidance has been developed to provide a quick reference evidence-based guide to enable professionals in Public Health and Environmental Health departments undertake risk assessments which inform effective public health actions to minimise the risk of transmission of gastrointestinal infections in the general population and in community settings.

It replaces the former Public Health Laboratory Service Advisory Committee on Gastrointestinal Infections guidance titled "Preventing person-to-person spread following gastrointestinal infections: guidelines for public health physicians and environmental health officers" published in 2004.

The main changes from the previous guidance are:

- revision of the definition of the risk groups for transmission of gastrointestinal infections
- organism specific information including the control of the human source, case definitions, causative organism, update of the epidemiology and further relevant guidance and reference materials
- update of the information about specific organisms in line with the updated national guidance documents
- links to the list of notifiable diseases and the Health Protection (Notification) Regulations 2010
- information leaflet for minimising the spread of gastrointestinal infections
- stool sample collection information leaflet

Definitions

Carrier: A person without symptoms who excretes an infectious pathogen in their faeces or urine. These people are also known as 'excreters'.

Case: A person with gastrointestinal symptoms due to an infectious pathogen or a microbiological intoxication which may or may not be laboratory confirmed.

Chronic carrier: A person who continues to excrete a pathogen for over a year.

Clinical surveillance: Observation of, or by, a patient for the development of symptoms.

Contact: A person who is likely to have been exposed to a case of an infectious illness.

Convalescent carrier: A person who has recovered from their infectious illness but who continues to excrete the pathogen for up to a year.

Food poisoning: An illness caused by the consumption of food or water contaminated with bacteria and/or their toxins, or with parasites, viruses, or chemicals.

Gastrointestinal infection: any infection, from whatever source, of the gastrointestinal (digestive) tract.

Microbiological clearance: the reduction in the number of pathogenic organisms in a case's specimen below that detectable by conventional culture methods of laboratory testing or a negative PCR test.

Microbiological screening: The submission of stool samples by contacts to determine the presence or absence of pathogenic organisms either culture methods or PCR.

Outbreak: Two or more cases associated in time and place.

Sporadic case: A single case which does not appear to have any links to another known case or carrier.

Standard exclusion period: A minimum of 48 hours symptom free/no loose stools

Introduction

In 2004, a working group from the former Public Health Laboratory Service (PHLS)¹ published guidance on the prevention of person-to-person spread of gastrointestinal infections. This guidance has been well used by public health and environmental health professionals to provide evidence-based advice and take effective public health actions to minimise the risk of transmission.

Over the last 10 years, there have been substantial developments in our understanding of organism specific risks, transmission pathways and effectiveness of interventions to control spread. Furthermore, the past decade has seen important changes in health protection legislation in the United Kingdom, with health now being a function of the devolved administrations.

The aim of this guidance document, as with the previous version, is to provide clear and concise advice to professionals working in Public Health and Environmental Health Departments regarding the prevention of person-to-person transmission of gastrointestinal infections.

The focus of this document is on the prevention of transmission from sporadic cases in the general population and community setting. Where available, links are provided to more detailed disease-specific guidance documents where additional information may be sourced. This guidance is intended to provide evidence-based information for professionals to assess risks and implement appropriate public health interventions. It should not deter the reader from seeking expert assistance if required. Additional advice may be sourced, as appropriate, from local microbiologists and health protection teams/centres, epidemiologists or reference laboratories. Links to the relevant legislation to support the notification of infectious diseases or organisms are provided in Appendix I of this guidance.

Involving the Food Standards Agency

The Food Standards Agency (FSA) should be informed by Environmental Health (in Northern Ireland) or Public Health (in England and Wales) of any serious localised or non-localised incidents where food may be involved at the earliest opportunity using the on-line report form on the Agency's website available here:

http://incidents.foodapps.co.uk/login.aspx. General information about incidents and contact details for the FSA's Incidents team can be found here:

https://www.food.gov.uk/business-guidance/food-incidents

The FSA's Incidents team provides a 24/7 incident response capability in relation to all food and feed incidents ensuring that measures are taken to remove unsafe products from the market. They are also the National Contact Point for the European Commission's Rapid Alert System for Food and Feed (RASFF) through which information and alerts relating to food safety is exchanged.

General advice

Symptoms of gastroenteritis such as diarrhoea and vomiting may be due to a variety of causes including infections, toxins and non-communicable diseases. However, as a general principle, all cases of gastroenteritis should be regarded as potentially infectious unless there is good evidence to suggest otherwise. Transmission of gastrointestinal infection from person-to-person may occur through one or more of a variety of different pathways, including faecal-oral, foodborne, environmental and airborne routes. The usual mode(s) of transmission vary depending on the organism or agent concerned, and practitioners should be aware of the need to tailor advice based on the pathogen involved (if known) and the case's individual circumstances. For example, exclusion from work may be indicated for some infections where the case is employed as a food-handler, or advice on safe sexual practices may be indicated for organisms such as Shigella where transmission amongst men who have sex with men has resulted in outbreaks of illness. A liquid or semi-formed stool is more likely than a formed stool to contaminate hands and the environment and consequently poses a greater risk of spreading faecal pathogens. Formed stools voided by asymptomatically infected people, or people who have recovered from illness, may contain pathogens, but are less likely to transmit infection if good personal hygiene practices are adopted. Vomit, like liquid stool, may be highly infectious.

Cases and all household contacts should always be provided with advice aimed at minimising the potential for spread of infection including personal hygiene, safe preparation of food and the enteric precautions outlined in **Appendix II – Minimising the spread of gastrointestinal illness.**

The importance of good personal and domestic cleanliness cannot be over emphasized in preventing transmission. All persons involved in caring for a case (professional carers, family members, teachers) should also follow enteric precautions.

Individuals who have been requested to submit a stool sample for examination may be provided with the leaflet in Appendix III – Stool sample collection instructions.

The specific pathogens for which a sample is tested may vary in different laboratories. Some pathogens included in this guidance document do not form part of routine testing protocols in all laboratories and it may be necessary to make direct contact with the relevant testing laboratory to obtain further information regarding testing. If food is suspected as the source of an infection, it is important to liaise with the Public Health England Food, Water and Environmental (FWE) microbiology laboratory and the Food Standards Agency (FSA) promptly.

Exclusions from work, school and other institutional settings

All persons with gastroenteritis should be considered as potentially infectious to others and excluded from work, school or other institutional and social settings until a minimum of 48 hours symptoms free/no loose stools. The recommended exclusion duration and criteria for specific causes of gastroenteritis are provided within the relevant disease/organism specific section in the following pages. Some cases, or their contacts, may pose an increased risk of spreading the infection to other people (see Table 1) and additional measures may be required prior to re-commencing their usual activities, such as demonstration of microbiological clearance of the organism. Risk of transmission and illness will vary depending on host, agent, and environmental factors. Where required, a risk assessment should be conducted for each scenario and this should consider the factors that increase or decrease the likelihood of spread, before agreeing on interventions. In practice, each case, carrier or contact may require assessment on an individual basis in order that factors such as type of employment, provision of sanitation facilities at work, school or other institution and standards of personal hygiene can be considered. Discussion and agreement between the local Health Protection, Environmental Health and Public Health Microbiology teams is strongly recommended if considering an alternative to the exclusion advice provided in this guidance document and signposted disease specific guidance document. For example, a healthcare professional is very unlikely to spread Shigella in a healthcare setting where they wash hands so often and use appropriate personal protective equipment; however, a child wearing nappies may be of higher risk of spreading the infection.

The Food Standards Agency document *Food Handlers: Fitness to Work* provides regulatory and best practice advice for food businesses and food business employees with regard to illness, exclusion from work and returning to work: This is available at:

www.food.gov.uk/sites/default/files/media/document/fitnesstoworkguide.pdf

Local Authorities may exercise legal powers for health protection purposes, including exclusion, under Health Protection legislation in each devolved UK administration. For example, in England, the Health Protection Regulations 2010 is the most recent legislation and is supported by a companion toolkit that provides letters and notices to aid Environmental Health teams where circumstances require exclusion to be formalised.

Risk Group	Description	Additional Comments
Group A	Any person who is unable to perform adequate personal hygiene due to lack of capacity or ability to comply OR has lack of access to hygiene facilities.	Risk assessment regarding access to hygiene facilities should consider the availability of toilets /handwashing/hand drying facilities in a work/educational setting.
Group B	All children aged 5 years old or under (up to the sixth birthday) who attend school, pre-school, nursery or other similar child care or minding groups.	For children aged 5 years and under who do not attend school, risk assessment for clearance purposes should explore potential for transmission within other settings e.g. household or attendance at parties.
Group C	People whose work involves preparing or serving unwrapped ready to eat food (including drink).	Consider informal food handlers e.g. someone who helps to prepare food for charity and community events.
Group D	Clinical, social care or nursery staff who work with young children, the elderly, or any other particularly vulnerable people, and whose activities increase the risk of transferring infection via the faecal -oral route.	Risk assessment should consider activities such as helping with feeding or handling objects that could be transferred to the mouth.

People not in these defined risk groups present a minimal risk of spreading gastrointestinal illness and may return to any form of work/school/child care facility a minimum of 48 hours after their stools have returned to normal consistency and symptoms have stopped.

For all organisms, including those where there is no recommended action for isolated single cases, Public Health follow-up may be required in cluster/outbreak situations where local outbreak procedures should be followed.

Amoebiasis/Amoebic dysentery/Entamoeba histolytica

Cases should be advised to	follow usual enteric precautions. Specific advice on exclusion from
nursery, school or work set	
Public health follow-up	
required	Specific guidance exists.
i oquii ou	See 'Further relevant guidance and key references' below.
Cases	Clinical treatment advised for all confirmed cases
Cases	Enteric precautions and hygiene advice
Ocartosta	Obtain travel history
Contacts	Advise testing of symptomatic and asymptomatic household, co-
	traveller and sexual contacts
Exclusions	A minimum of 48 hours symptom free/ no loose stools
	No exclusion for asymptomatic cases
Microbiological	Repeat stool sample 1 week after treatment completion to confirm
clearance	treatment success (not for exclusion purposes)
Case definitions:	
A person with E. histolvtica	infection determined by demonstration of E. histolytica using PCR
on a stool specimen	
OR	
	se AND demonstration of trophozoites on stool microscopy OR
· ·	
· · · · · · · · · · · · · · · · · · ·	es of <i>E. histolytica</i> in intestinal/rectal biopsy by histopathology
OR	
	nosis of amoebic liver abscess and positive serology for antibodies
to E. histolytica	
Couloctive agents	
Causative agent:	
Cause	Entamoeba histolytica
	<i>Entamoeba histolytica</i> Humans are the only known reservoir
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Cause	
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Public Health England. Interim Public Health Operational Guidelines for Amoebiasis (Entamoeba Histolytica)

Available at www.gov.uk/government/publications/amoebiasis-public-health-operationalguidelines

US Food and Drug Administration (2012) Bad Bug Book - Foodborne Pathogenic Microorganisms and Natural Toxins Handbook. Second Edition Available at:

www.fda.gov/Food/FoodbornellInessContaminants/CausesOfIllnessBadBugBook

Bacillus species food poisoning/ Bacillus species

Control of human source: Cases should be advised to follow usual enteric precautions. Specific advice on exclusion from		
nursery, school or work settings is given below.		
Public health follow-up		
required	Not usually indicated unless case is identified as part of a	
	cluster/outbreak. In those circumstances, local outbreak	
	procedures should be followed.	
Cases	Enteric precautions	
•	Collect information on food consumption in 24-hour period prior	
	to symptom onset	
Contacts	No action required	
Exclusions	Cases: a minimum of 48 hours symptom free/ no loose stools	
Microbiological	None required	
clearance		
Case definitions:		
	east one clinical criterion OR at least one laboratory criterion.	
	sact one cannot ontenon on at least one laboratory entenon.	
Clinical: • sudder	n onset of nausea AND vomiting	
	inal cramps AND diarrhoea	
OR		
-	n of ≥10 ⁵ B. cereus organisms per gram or direct detection of B.	
	enterotoxin from epidemiologically implicated food in the setting of	
a	onteretexin nem epidenneregieany impliedted reed in the cetting of	
person or persons with diarrhoea or vomiting		
	n of the organism from the stools of 2 or more ill persons but not	
	e stools of controls, in an outbreak situation	
Causative agent:		
Cause	Bacillus species, mainly Bacillus cereus, which produce toxins	
	(enterotoxins)	
	Gastrointestinal infections also caused by <i>Bacillus subtilis</i> and	
	Bacillus licheniformis	
Reservoir		
Reservoir	Ubiquitous in the environment, including soil	
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	Bacillus cereus - mainly rice dishes (e.g. outbreaks of fried rice in
	Chinese restaurants), also pasta, meat or vegetable dishes and
	dairy products.
	Bacillus subtilis and licheniformis – mainly meat or vegetable with
	pastry products, cooked meat and poultry products, also bakery
	products and ethnic meats
	Possible transmission has been linked to organ preservation fluid
	and contaminated parenteral nutrition
	Person-to-person spread is not documented
Incubation period	Bacillus cereus
	Emetic syndrome - average 2-3 hours (range 1-6) hours
	Diarrhoeal syndrome - 8-12 hours (range 6-24 hours)
	Bacillus subtilis – 10 minutes–4 hours (average 2.5 hours)
	Bacillus licheniformis – 2–14 hours (average 8 hours)
Common clinical	Bacillus cereus - 2 clinical syndromes may occur caused by
features	different toxins:
	Emetic syndrome (heat-stable toxin) – nausea and vomiting,
	abdominal pain with or without diarrhoea. Generally, a mild illness
	lasting <12 hours
	<u>Diarrhoeal syndrome</u> (heat-labile toxin) – diarrhoea (which may
	be profuse and watery) and abdominal pain with or without
	nausea and vomiting lasting around 24 hours
	Bacillus subtilis – nausea, vomiting and diarrhoea
	Bacillus licheniformis – diarrhoea and abdominal pain
Period of infectiousness	Not applicable as no risk of person-to-person spread
Other relevant	Severe non-foodborne infection can occur in cases that are
information	immunocompromised, have intravascular catheters or are
	intravenous drug users. A high infectious dose is required. As
	this is via inoculation of bacillus into the bloodstream or growth
	within a wound, it is not foodborne and there is no person-to-
	person spread and no exclusion is required.
Further relevant guidance	and key references:

Public Health England: www.gov.uk/government/collections/bacillus-species-food-poisoning Hawker J, Begg N, Blair I, Reintjes R, Weinberg J & Ekdahl K. (2012) Communicable Disease Control and Health Protection Handbook – Third Edition. Wiley-Blackwell. US Food and Drug Administration (2012) Bad Bug Book - Foodborne Pathogenic Microorganisms and Natural Toxins Handbook. Second Edition Available at:

www.fda.gov/Food/FoodbornellInessContaminants/CausesOfIIInessBadBugBook

Botulism/ Clostridium botulinum toxin

Control of human source:		
Cases should be advised to follow usual enteric precautions. Specific advice on exclusion is given below.		
Public health follow-up	p YES	
required	A single case of botulism should be considered a potential public health emergency. Prompt actions should be	
	undertaken to identify the source. Specific guidance exists. See 'Further relevant guidance and key references' below.	
Cases	Antitoxin treatment based on clinical diagnosis as appropriate Obtain urgent risk factor history from case/parent/household contact May need to obtain food samples – liaison between Environmental Health and Health Protection Teams recommended	
Contacts	Clinical surveillance: seek medical help if unwell Obtain risk factor history – they may have been exposed to the same source	
Exclusions	None usually required. However, consider exclusion of cases of infant botulism from childminder/crèche settings because large numbers of organisms are excreted in faeces and there may be a risk of exposure to other infants	
Microbiological	None required	
clearance		
Case definitions:		

Foodborne botulism: Clinical syndrome and history compatible with foodborne botulism. Confirmation of a clinical diagnosis is by detection of botulinum toxin in serum or faecal specimens or detection and isolation of C. botulinum from faeces. Confirmation will also be obtained from isolation and toxin detection in food samples.

Infant botulism: Clinical syndrome and history compatible with infant botulism. Confirmation of a clinical diagnosis is by detection of C. botulinum in faeces by PCR and subsequent isolation of *C. botulinum* from infant faeces or rectal wash out or detection of botulinum toxin in these specimens as well as in serum.

Wound botulism: Clinical syndrome and history compatible with wound botulism. There is an association with substance misuse, especially injecting heroin. Confirmation of the clinical diagnosis is by the demonstration of botulinum toxin in serum or wound specimens, or by PCR detection and subsequent isolation of C. botulinum from specimens. In the UK, wound botulism is exclusive to drug injectors.

Causative agent:	
Cause	<i>Clostridum botulinum</i> neurotoxin. Cases also associated with neurotoxin produced by <i>Clostridium</i> butyricum and <i>Clostridium baratii.</i>
Reservoir	Widespread in the environment $-C$. botulinum heat resistant spores exist in soil, dust, untreated water and the gastrointestinal tracts of animals and fish. Under the appropriate anaerobic conditions, the spores germinate and produce toxin.

Epidemiology	Rare: 100-200 cases reported in the EU annually.
Lpidemology	Three naturally occurring forms of botulism: food-borne, wound
	and infant (or intestinal) botulism. Inhalation botulism is extremely
	rare.
Transmission	Foodborne: ingestion of food contaminated by toxin. A variety of
	meat, fish and vegetables have been implicated. Associated with
	under processed food, and home preservation.
	Wound: inoculation of spores that germinate in the tissue
	producing toxin and capable of causing systemic symptoms.
	Infant: ingestion of <i>C. botulinum</i> spores in food (e.g. in honey) or
	from the environment which germinate and produce toxin in the
	infant intestine. Persons with open lesions on their hands should
	wear gloves when handling soiled diapers from these patients.
	Cases of infant botulism caused by <i>C. butyricum</i> have been
	associated with pet terrapins in the UK and Ireland. Person-to-person spread does not occur in food or wound
	botulism. There is a risk of cross-infection to other infants with
	infant botulism due to excretion of organisms in faeces which may
	be prolonged. Stools should be discarded as hazardous material.
	Cross-infection control measures include scrupulous hand
	washing when handling infants and during nappy changing and
	avoiding close contact with other infants, including not sharing
	toys, bedding and cots.
Incubation period	Foodborne: 2 hours to 8 days (usually 12-72 hours). More severe
	disease may be associated with a shorter incubation period.
	Wound botulism: 4-21 days
Common clinical	Inhalation: few hours to 4 days
features	Characteristic symmetric descending flaccid paralysis of motor and autonomic nerves: slurred speech, double vision, difficulty in
reatures	swallowing, ptosis, respiratory muscle paralysis. In food botulism,
	diarrhoea and vomiting may precede neurological symptoms by a
	few hours. In infants, constipation is a frequent, often over-looked
	symptom.
Period of infectiousness	C. botulinum may be detected in the stool and although person-
	to-person spread does not occur for food or wound botulism, in
	infant botulism, cross-infection control measures should be
011	followed.
Other relevant information	The hospital microbiologist and Consultant in Communicable Disease Control/Health Protection should be
mormation	
	contacted urgently . If food is suspected as a source, the Food Standards Agency Incident Branch should be informed.
	Foodborne botulism is a public health emergency and may require
	a food product recall.
	Urgent arrangements should be made to contact the Botulism
	service at PHE Colindale for clinical risk assessment and testing
	of clinical specimens and suspect food by the Gastrointestinal
	Bacteria Reference Laboratory at Colindale.
	Botulism is a clinical diagnosis which laboratory tests can confirm
	but not refute. Antitoxin must be administered as soon as

	possible after symptom onset to prevent toxin binding at the site of action. Antitoxin should be given based on a clinical diagnosis and should not be delayed for awaiting laboratory testing results. Advice on clinical management of suspected cases of botulism is available from Dr Gauri Godbole on 07826 859642 in liaison with the regional Public Health laboratories and information on obtaining antitoxin for all forms of botulism is available via the Colindale duty doctor during working hours and out of hours.	
	NI arrangements : Supplies are strictly arranged by contacting a Consultant Microbiologist at the Regional Virus Laboratory Tel: 028 9063 2662 (Mon-Fri 9am-6pm), or outside office hours, the Microbiologist on call, via Royal Victoria Hospital Belfast switchboard Tel: 028 9024 0503.	
	The Consultant Microbiologist or Microbiologist on call will then contact Belfast City Hospital pharmacy (via BCH switchboard Tel: 028 9032 9241) to authorise supply to the requesting clinician or hospital.	
Further relevant guidance	and key references:	
Public Health England: www.gov.uk/government/collections/botulism-diagnosis-data-and- analysis		
Public Health England (July 2012) <i>Botulism: clinical and public health management</i> Available at: www.gov.uk/government/publications/botulism-clinical-and-public-health- management		
Hawker J, Begg N, Blair I, Reintjes R, Weinberg J & Ekdahl K. (2012) Communicable Disease Control and Health Protection Handbook – Third Edition. Wiley-Blackwell. Food Standards Agency Incident teams available at:		
https://www.food.gov.uk/e	enforcement/enforcework/report. US Food and Drug	

Administration (2012) Bad Bug Book - Foodborne Pathogenic Microorganisms and Natural Toxins Handbook. Second Edition

Available at:

www.fda.gov/Food/FoodbornellInessContaminants/CausesOfIlInessBadBugBook

Campylobacteriosis/ Campylobacter species

Control of human source:	
	follow usual enteric precautions. Specific advice on exclusion from
nursery, school or work set	•
Public health follow-up	
required	England and Northern Ireland: Not usually indicated unless case
required	is identified as part of an outbreak. In those circumstances, local
	outbreak procedures should be followed.
	Wales: Follow up of individual cases as per local protocol. If
	identified as part of a cluster or outbreak, local outbreak
	procedures should be followed.
Casaa	
Cases	Clinical treatment if thought appropriate by clinician
	Enteric precautions
Contacts	Not applicable
Exclusions	A minimum of 48 hours symptom free/no loose stools
Microbiological	None required
clearance	
Case definitions:	
	f gastroenteritis and identification of Campylobacter spp. from an
	en, most often from a stool specimen
Causative agent:	
Cause	Campylobacter jejuni accounts for most cases, followed by
	Campylobacter coli.
	C. fetus and C. lari are uncommon causes but may cause severe
	illness in immunosuppressed individuals
Reservoir	Gastrointestinal tract of birds (especially poultry) and mammals
	(e.g. cattle, sheep, domestic pets); C. coli is particularly
	associated with pigs.
	Campylobacter spp. cannot multiply outside the host but may
	exist in environmental sources such as soil, manure and water
	sources
Epidemiology	Campylobacter species are the commonest bacterial cause of
	infectious gastrointestinal disease in developed countries and one
	of the most common causes of traveller's diarrhoea in the UK
	The infection follows a seasonal pattern in temperate regions with
	a peak in the late spring/summer months.
Transmission	Primarily ingestion of contaminated food or drink (e.g. inadequate
	cooking of raw meats and offal, cross-contamination between raw
	and cooked foods, raw drinking milk), or water
	The organism is unable to multiply outside a host, but food-borne
	outbreaks do occur.
	Transmission may also be via direct contact with infected animals
	e.g. domestic pets or farm animals
	Person-to-person spread may occur, but the risk is low (mainly
	via young children who are not toilet trained)
Incubation period	Usually 2-5 days (range of 1-10 days)
Common clinical	Most cases have symptoms of diarrhoea, abdominal pain (which
features	may be prominent) and fever

	Some may experience bloody stools and vomiting and feeling
	generally unwell
	Infection may be asymptomatic (25-50%)
	Most cases are self-limiting within 2-3 days (80-90% resolve within 1 week)
	Complications are rare but potentially serious, including Guillain-
	Barre syndrome, reactive arthritis and haemolytic uraemic
	syndrome
Period of infectiousness	Cases are considered infectious whilst symptomatic
Other relevant	Infections are highest in children aged <5 years
information	Groups at highest risk are those with the increased exposure to a
	contaminated source including occupational contact with farm
	animals or raw poultry or meat, overseas travellers, men who
	have sex with men and family contacts of a case.
	The infectious dose is considered to be low.
Further relevant quidance	and key references:

Further relevant guidance and key references: Public Health England: www.gov.uk/government/collections/campylobacter-guidance-data-

and-analysis Public Health Wales: www.wales.nhs.uk/sitesplus/888/page/43695

Hawker J, Begg N, Blair I, Reintjes R, Weinberg J & Ekdahl K. (2012) *Communicable Disease Control and Health Protection Handbook – Third Edition*. Wiley-Blackwell. Centers for Disease Control and Prevention:

www.cdc.gov/nczved/divisions/dfbmd/diseases/campylobacter

Food Standards Agency:

www.food.gov.uk/science/microbiology/campylobacterevidenceprogramme

US Food and Drug Administration (2012) Bad Bug Book - Foodborne Pathogenic Microorganisms and Natural Toxins Handbook. Second Edition Available at:

www.fda.gov/Food/FoodbornellInessContaminants/CausesOfIIInessBadBugBook

Cholera/ Vibrio cholerae O1 and O139

Control of human source	
	follow usual enteric precautions. Specific advice on exclusion from
nursery, school or work settings is given below	
Public health follow-up	YES
required	
Cases	Clinical management as appropriate
Cuecco	Enteric precautions
	Obtain travel history
Contacts	No action required for asymptomatic close contacts
Contacts	Screen symptomatic co-travellers and household contacts
	Provide 'inform and advise' information to co-travellers
Exclusions	
	A minimum of 48 hours symptom free/no loose stools
Microbiological clearance	Not routinely required for cases in risk groups where risk
clearance	assessment of personal hygiene and facilities is satisfactory. A
	single microbiological clearance specimen may be required
	where sanitary facilities and personal hygiene are considered
Coop definitions:	inadequate.
Case definitions:	infection and inclution of tovigania Vibric chalarce O1 or O100 from
	infection and isolation of toxigenic <i>Vibrio cholerae</i> O1 or O139 from
	cal evidence of recent infection
Causative agent:	
Cause	Toxigenic Vibrio cholerae serogroups O1 (biotypes 'classical' and
	'El Tor') and O139
	Non-O1 and non-O139 may cause milder gastroenteritis but not
	cholera
Reservoir	Humans and the environment
Epidemiology	Cases in the UK occur in travellers returning from endemic areas
	(Africa, Asia, Central and South America, Caribbean)
	An average of 16 cases of cholera caused by Vibrio cholera O1
	and O139 have been reported in England and Wales between
	2004 and 2012. No confirmed cases have been reported from
	Northern Ireland since 2004.
Transmission	Transmission is via the faecal-oral route primarily via drinking
	water contaminated by faeces
	Consumption of contaminated food, especially shellfish, is also a
	route of transmission.
	A large infectious dose is required so secondary transmission is
	not likely in countries with good sanitation systems (e.g. UK).
Incubation period	Usually 24-72 hours (range 2 hours - 5 days) but is dependent on
-	the dose ingested
Common clinical	-,
features	quickly change to large volumes of pale fluid stools ('rice-water'
	stools).
	Cases usually recover spontaneously once dehydration is
	corrected.
	Severe disease is related to the infectious dose and health status
	of the case and occurs due to significant fluid loss and

	dehydration which can be fatal. Babies, children, the elderly and those with poor general health are most at risk of dehydration and severe disease.
Period of infectiousness	Cases are considered infectious whilst diarrhoea is present and up to 7 days after. Since secondary transmission is unlikely in the UK due to good sanitation, exclusion for 48 hours after first normal stool is usually applied. Occasionally, some cases might become 'carriers' for a few months
Other relevant information	Large epidemics are common following the breakdown of public health measures such as areas experiencing war, famine and natural disasters. A vaccination against cholera is available but is not considered to be highly effective and is therefore not generally recommended.
Further relevant guidance	and key references:
Disease Control and Health US Food and Drug Adminis Microorganisms and Natura Available at:	v.gov.uk/cholera Reintjes R, Weinberg J & Ekdahl K. (2012) <i>Communicable</i> <i>Protection Handbook – Third Edition</i> . Wiley-Blackwell. tration (2012) Bad Bug Book - Foodborne Pathogenic I Toxins Handbook. Second Edition mellInessContaminants/CausesOfIIInessBadBugBook

Clostridium difficile infection/ Clostridium difficile toxin

Control of human source:		
Community cases should be advised to follow usual enteric precautions. Specific advice on		
exclusion from nursery, school or work settings is given below. Cases in a hospital, care home		
or other institutional setting should be isolated and enteric precautions followed until a minimum		
of 48 hours symptom free/no loose stools, with management led by hospital/other appropriat		
infection prevention control		
Public health follow-up		
required	Not usually indicated unless case is identified as part of a	
	cluster/outbreak. In those circumstances, local outbreak	
	procedures should be followed.	
	Northern Ireland: Follow up of individual cases as per local	
	protocol. In short – collection of risk factor information; provision	
	of IPC advice to care/residential home setting where this applies	
	See 'Further relevant guidance and key references' below.	
Cases	Clinical management as appropriate	
	Enteric precautions	
	Isolation in healthcare and social care settings until considered	
	non-infectious	
Contacts	Clinical surveillance	
Contacts	Screen symptomatic contacts	
Exclusions	A minimum of 48 hours symptom free/no loose stools	
Microbiological	None	
clearance	None	
clearance		
Coop definitions		
Case definitions:	which is Cleatridium difficile CDH EIA (or NAAT) positive, and taxin	
Diarrhoeal stool specimen	which is <i>Clostridium difficile</i> GDH EIA (or NAAT) positive, and toxin	
Diarrhoeal stool specimen EIA positive (PPV = 91.4%)	which is <i>Clostridium difficile</i> GDH EIA (or NAAT) positive, and toxin), which makes it most likely that <i>C. difficile</i> is present	
Diarrhoeal stool specimen EIA positive (PPV = 91.4%) OR), which makes it most likely that <i>C. difficile</i> is present	
Diarrhoeal stool specimen EIA positive (PPV = 91.4%) OR Toxic megacolon or ileus v		
Diarrhoeal stool specimen EIA positive (PPV = 91.4%) OR Toxic megacolon or ileus v and toxin EIA positive), which makes it most likely that <i>C. difficile</i> is present	
Diarrhoeal stool specimen v EIA positive (PPV = 91.4%) OR Toxic megacolon or ileus v and toxin EIA positive OR), which makes it most likely that <i>C. difficile</i> is present vith specimen positive for <i>C. difficile</i> GDH EIA (or NAAT) positive,	
Diarrhoeal stool specimen EIA positive (PPV = 91.4%) OR Toxic megacolon or ileus v and toxin EIA positive OR Endoscopic/Computerised), which makes it most likely that <i>C. difficile</i> is present with specimen positive for <i>C. difficile</i> GDH EIA (or NAAT) positive, Tomographic evidence of pseudomembranous colitis with	
Diarrhoeal stool speciment EIA positive (PPV = 91.4%) OR Toxic megacolon or ileus w and toxin EIA positive OR Endoscopic/Computerised supportive clinical findings), which makes it most likely that <i>C. difficile</i> is present vith specimen positive for <i>C. difficile</i> GDH EIA (or NAAT) positive,	
Diarrhoeal stool specimen v EIA positive (PPV = 91.4%) OR Toxic megacolon or ileus v and toxin EIA positive OR Endoscopic/Computerised supportive clinical findings), which makes it most likely that <i>C. difficile</i> is present with specimen positive for <i>C. difficile</i> GDH EIA (or NAAT) positive, Tomographic evidence of pseudomembranous colitis with e.g. raised White Cell Count	
Diarrhoeal stool speciment EIA positive (PPV = 91.4%) OR Toxic megacolon or ileus v and toxin EIA positive OR Endoscopic/Computerised supportive clinical findings Causative agent: Cause), which makes it most likely that <i>C. difficile</i> is present with specimen positive for <i>C. difficile</i> GDH EIA (or NAAT) positive, Tomographic evidence of pseudomembranous colitis with e.g. raised White Cell Count <i>Clostridium difficile</i> toxins (A and B)	
Diarrhoeal stool specimen v EIA positive (PPV = 91.4%) OR Toxic megacolon or ileus v and toxin EIA positive OR Endoscopic/Computerised supportive clinical findings), which makes it most likely that <i>C. difficile</i> is present with specimen positive for <i>C. difficile</i> GDH EIA (or NAAT) positive, Tomographic evidence of pseudomembranous colitis with e.g. raised White Cell Count <i>Clostridium difficile</i> toxins (A and B) Human gastrointestinal tract	
Diarrhoeal stool speciment EIA positive (PPV = 91.4%) OR Toxic megacolon or ileus v and toxin EIA positive OR Endoscopic/Computerised supportive clinical findings Causative agent: Cause), which makes it most likely that <i>C. difficile</i> is present with specimen positive for <i>C. difficile</i> GDH EIA (or NAAT) positive, Tomographic evidence of pseudomembranous colitis with e.g. raised White Cell Count <i>Clostridium difficile</i> toxins (A and B) Human gastrointestinal tract Spores may be present on environmental surfaces contaminated	
Diarrhoeal stool speciment EIA positive (PPV = 91.4%) OR Toxic megacolon or ileus v and toxin EIA positive OR Endoscopic/Computerised supportive clinical findings Causative agent: Cause Reservoir), which makes it most likely that <i>C. difficile</i> is present with specimen positive for <i>C. difficile</i> GDH EIA (or NAAT) positive, Tomographic evidence of pseudomembranous colitis with e.g. raised White Cell Count <i>Clostridium difficile</i> toxins (A and B) Human gastrointestinal tract Spores may be present on environmental surfaces contaminated by symptomatic persons	
Diarrhoeal stool speciment EIA positive (PPV = 91.4%) OR Toxic megacolon or ileus v and toxin EIA positive OR Endoscopic/Computerised supportive clinical findings Causative agent: Cause), which makes it most likely that <i>C. difficile</i> is present with specimen positive for <i>C. difficile</i> GDH EIA (or NAAT) positive, Tomographic evidence of pseudomembranous colitis with e.g. raised White Cell Count <i>Clostridium difficile</i> toxins (A and B) Human gastrointestinal tract Spores may be present on environmental surfaces contaminated	
Diarrhoeal stool speciment EIA positive (PPV = 91.4%) OR Toxic megacolon or ileus v and toxin EIA positive OR Endoscopic/Computerised supportive clinical findings Causative agent: Cause Reservoir), which makes it most likely that <i>C. difficile</i> is present with specimen positive for <i>C. difficile</i> GDH EIA (or NAAT) positive, Tomographic evidence of pseudomembranous colitis with e.g. raised White Cell Count <i>Clostridium difficile</i> toxins (A and B) Human gastrointestinal tract Spores may be present on environmental surfaces contaminated by symptomatic persons	
Diarrhoeal stool speciment EIA positive (PPV = 91.4%) OR Toxic megacolon or ileus v and toxin EIA positive OR Endoscopic/Computerised supportive clinical findings Causative agent: Cause Reservoir	 which makes it most likely that <i>C. difficile</i> is present with specimen positive for <i>C. difficile</i> GDH EIA (or NAAT) positive, Tomographic evidence of pseudomembranous colitis with e.g. raised White Cell Count <i>Clostridium difficile</i> toxins (A and B) Human gastrointestinal tract Spores may be present on environmental surfaces contaminated by symptomatic persons <i>C. difficile</i> is found in faeces of approximately 3% of healthy 	
Diarrhoeal stool speciment EIA positive (PPV = 91.4%) OR Toxic megacolon or ileus v and toxin EIA positive OR Endoscopic/Computerised supportive clinical findings Causative agent: Cause Reservoir	 which makes it most likely that <i>C. difficile</i> is present with specimen positive for <i>C. difficile</i> GDH EIA (or NAAT) positive, Tomographic evidence of pseudomembranous colitis with e.g. raised White Cell Count <i>Clostridium difficile</i> toxins (A and B) Human gastrointestinal tract Spores may be present on environmental surfaces contaminated by symptomatic persons <i>C. difficile</i> is found in faeces of approximately 3% of healthy adults, 66% of healthy infants, 7% of asymptomatic care-home residents and 20% of elderly patients on long-term wards without 	
Diarrhoeal stool speciment EIA positive (PPV = 91.4%) OR Toxic megacolon or ileus v and toxin EIA positive OR Endoscopic/Computerised supportive clinical findings Causative agent: Cause Reservoir	 which makes it most likely that <i>C. difficile</i> is present with specimen positive for <i>C. difficile</i> GDH EIA (or NAAT) positive, Tomographic evidence of pseudomembranous colitis with e.g. raised White Cell Count <i>Clostridium difficile</i> toxins (A and B) Human gastrointestinal tract Spores may be present on environmental surfaces contaminated by symptomatic persons <i>C. difficile</i> is found in faeces of approximately 3% of healthy adults, 66% of healthy infants, 7% of asymptomatic care-home residents and 20% of elderly patients on long-term wards without causing symptoms of disease (i.e. asymptomatic carriers). 	
Diarrhoeal stool speciment EIA positive (PPV = 91.4%) OR Toxic megacolon or ileus v and toxin EIA positive OR Endoscopic/Computerised supportive clinical findings Causative agent: Cause Reservoir	 which makes it most likely that <i>C. difficile</i> is present with specimen positive for <i>C. difficile</i> GDH EIA (or NAAT) positive, Tomographic evidence of pseudomembranous colitis with e.g. raised White Cell Count <i>Clostridium difficile</i> toxins (A and B) Human gastrointestinal tract Spores may be present on environmental surfaces contaminated by symptomatic persons <i>C. difficile</i> is found in faeces of approximately 3% of healthy adults, 66% of healthy infants, 7% of asymptomatic care-home residents and 20% of elderly patients on long-term wards without causing symptoms of disease (i.e. asymptomatic carriers). Clinical infection occurs when the normal flora of the gut is 	
Diarrhoeal stool speciment EIA positive (PPV = 91.4%) OR Toxic megacolon or ileus v and toxin EIA positive OR Endoscopic/Computerised supportive clinical findings Causative agent: Cause Reservoir	 which makes it most likely that <i>C. difficile</i> is present with specimen positive for <i>C. difficile</i> GDH EIA (or NAAT) positive, Tomographic evidence of pseudomembranous colitis with e.g. raised White Cell Count <i>Clostridium difficile</i> toxins (A and B) Human gastrointestinal tract Spores may be present on environmental surfaces contaminated by symptomatic persons <i>C. difficile</i> is found in faeces of approximately 3% of healthy adults, 66% of healthy infants, 7% of asymptomatic care-home residents and 20% of elderly patients on long-term wards without causing symptoms of disease (i.e. asymptomatic carriers). Clinical infection occurs when the normal flora of the gut is disturbed, usually using antibiotics, enabling <i>C. difficile</i> to grow 	
Diarrhoeal stool speciment EIA positive (PPV = 91.4%) OR Toxic megacolon or ileus v and toxin EIA positive OR Endoscopic/Computerised supportive clinical findings Causative agent: Cause Reservoir	 a), which makes it most likely that <i>C. difficile</i> is present b), which makes it most likely that <i>C. difficile</i> is present b), which makes it most likely that <i>C. difficile</i> GDH EIA (or NAAT) positive, c) Tomographic evidence of pseudomembranous colitis with e.g. raised White Cell Count c) Clostridium difficile toxins (A and B) Human gastrointestinal tract Spores may be present on environmental surfaces contaminated by symptomatic persons c. difficile is found in faeces of approximately 3% of healthy adults, 66% of healthy infants, 7% of asymptomatic care-home residents and 20% of elderly patients on long-term wards without causing symptoms of disease (i.e. asymptomatic carriers). Clinical infection occurs when the normal flora of the gut is disturbed, usually using antibiotics, enabling <i>C. difficile</i> to grow and produce toxins. The main risk factors are antibiotic use and 	
Diarrhoeal stool speciment EIA positive (PPV = 91.4%) OR Toxic megacolon or ileus v and toxin EIA positive OR Endoscopic/Computerised supportive clinical findings Causative agent: Cause Reservoir	 which makes it most likely that <i>C. difficile</i> is present with specimen positive for <i>C. difficile</i> GDH EIA (or NAAT) positive, Tomographic evidence of pseudomembranous colitis with e.g. raised White Cell Count <i>Clostridium difficile</i> toxins (A and B) Human gastrointestinal tract Spores may be present on environmental surfaces contaminated by symptomatic persons <i>C. difficile</i> is found in faeces of approximately 3% of healthy adults, 66% of healthy infants, 7% of asymptomatic care-home residents and 20% of elderly patients on long-term wards without causing symptoms of disease (i.e. asymptomatic carriers). Clinical infection occurs when the normal flora of the gut is disturbed, usually using antibiotics, enabling <i>C. difficile</i> to grow 	

	It is the most important cause of healthcare-associated diarrhoea
	in developed countries.
Transmission	Person-to-person spread from symptomatic patients either directly or indirectly via contaminated hands of healthcare/other care workers
	Via contact with environmentally contaminated surfaces e.g.
	commodes
	Spread does not occur from asymptomatic carriers.
Incubation period	Difficult to establish incubation period
	Among patients commencing antibiotics, diarrhoea usually starts
	within 1-2 days of commencing antibiotics but can occur several
	weeks after antibiotic treatment
Common clinical	Watery diarrhoea (ranging from mild to severe) with or without
features	fever, nausea, loss of appetite and abdominal pain
	Complications include dehydration, pseudomembranous colitis,
	toxic megacolon, intestinal perforation and death in severe cases
Period of infectiousness	Most infectious when symptomatic
	Infectiousness reduces with treatment and decreasing severity of
	symptoms
	Stopping the implicated antibiotics (if possible) may be indicated
Other relevant	<i>C. difficile</i> spores are hardy and may remain on environmental
information	surfaces for many weeks. Thorough environmental cleaning with
	suitable agents e.g. chlorine containing products is required to
	reduce transmission.
Further relevant guidance	
i unner reievant guluance	

Public Health England: www.gov.uk/government/collections/clostridium-difficile-guidance-data-and-analysis

Public Health Wales: www.wales.nhs.uk/sites3/page.cfm?orgid=379&pid=13577

Department of Health:

https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/215135/dh_13 3016.pdf

Public Health England (June 2013) Updated guidance on the management and treatment of Clostridium difficile infection

Available at: www.gov.uk/government/publications/clostridium-difficile-infection-guidance-onmanagement-and-treatment

Hawker J, Begg N, Blair I, Reintjes R, Weinberg J & Ekdahl K. (2012) Communicable Disease Control and Health Protection Handbook – Third Edition. Wiley-Blackwell. Centers for Disease Control and Prevention:

www.cdc.gov/HAI/organisms/cdiff/Cdiff_infect.html

Clostridium perfringens food poisoning/ Clostridium perfringens enterotoxin

Control of human source:		
	follow usual enteric precautions. Specific advice on exclusion from	
	nursery, school or work settings is given below.	
Public health follow-up		
required	Not usually indicated unless case is identified as part of a	
	cluster/outbreak. In those circumstances, local outbreak	
	procedures should be followed.	
Cases	Clinical management as appropriate	
Cases	Enteric precautions	
Contacts	No action required	
Exclusions	A minimum of 48 hrs symptom free/no loose stools	
Microbiological	None required	
clearance	None required	
Case definitions:		
	ea and/or abdominal pain (rarely vomiting) and detection of	
	erotoxin from a stool specimen	
Causative agent:		
Cause	Clostridium perfringens enterotoxin	
Reservoir	Ubiquitous in soil and gastrointestinal tract of mammals and birds;	
Itesel voli	frequently present in raw meat.	
	Enterotoxin is produced only by some strains and thus C.	
	<i>perfringens</i> can live in the human intestine without producing	
	symptoms of disease. Only strains able to produce enterotoxin	
	cause gastrointestinal illness.	
Epidemiology	Reported cases in the UK are higher in autumn and winter	
Lpidemiology	months.	
	There are estimated to be >100,000 UK cases per year, but these	
	are greatly under-reported and under-detected.	
	<i>C. perfringens</i> food poisoning outbreaks are particularly	
	associated with institutional catering where food is inadequately	
	refrigerated before serving allowing the bacterium to grow.	
	Enterotoxigenic strains can also cause non-foodborne infections	
	or antibiotic associated diarrhoeal illness and outbreaks	
Transmission	Food poisoning occurs via ingestion of high numbers of C.	
	<i>perfringens</i> vegetative cells in contaminated foods, especially	
	meat and meat products. The organism can grow at temperatures	
	of 15-50°C and heat-resistant spores survive normal cooking	
	temperatures. Inadequate storage and insufficient reheating of	
	contaminated food allows growth of the organism to high	
	numbers. C. perfringens vegetative cells are ingested with the	
	food and then sporulate and release toxin in the small intestine	
Incubation period	Usually 8-18 hours (range 6-24 hours)	
Common clinical	Diarrhoea (watery and often violent) and abdominal pain	
features	Symptoms resolve within 24 hours for most cases	
Period of infectiousness	Not applicable as no risk of person-to-person spread	

Other relevant information	The elderly, very young and those with underlying medical conditions may experience more severe disease. Testing for this enterotoxin is not routinely undertaken, and a specific request will need to be made to the reference laboratory
	to have this performed Likewise, the ability of <i>C. perfringens</i> isolates to encode enterotoxin genes which can be determined by PCR and is performed by the PHE Reference Laboratory at Colindale
Further relevant guida	ance and key references:
5	www.gov.uk/clostridium-perfringens

Hawker J, Begg N, Blair I, Reintjes R, Weinberg J & Ekdahl K. (2012) *Communicable Disease Control and Health Protection Handbook – Third Edition*. Wiley-Blackwell.

Cryptosporidiosis/ Cryptosporidium species

Control of human source	
	follow usual enteric precautions. Specific advice on exclusion from
nursery, school or work set	•
Public health follow-up	
required	Specific guidance exists. See 'Further relevant guidance and key
loquilou	references' below.
Cases	No specific treatment is currently licenced within the UK.
	Clinicians should seek expert advice for profoundly
	immunosuppressed patients.
	Complete questionnaire.
Contacts	Clinical surveillance
	Screen symptomatic contacts
Exclusions	A minimum of 48 hrs symptom free/no loose stools
	Cases should not use swimming pools for 2 weeks after diarrhoea
	and vomiting symptoms have stopped
Microbiological	None required
clearance	
Case definitions:	
	of a gastrointestinal illness AND laboratory evidence of
	s or DNA in an appropriate sample, usually stool/faeces.
Causative agent:	
Cause	Cryptosporidium, a protozoan parasite. C.hominis and C. parvum
	cause most laboratory confirmed cases in the UK. Species are
	determined by reference genotyping.
Reservoir	Gastrointestinal tracts of humans and animals. Asymptomatic
	carriage has been documented in humans and animals.
Epidemiology	One of the most common protozoal causes of gastroenteritis in
	the UK.
Transmission	Approximately 40% of laboratory confirmed cases occur in
	children below 5 years of age. Most cases are acquired within the
	UK; approximately 20% report recent foreign travel.
	Ingestion of oocysts
	Faeco-oral spread:
	Direct or indirect contact with infected animals.
	Person to person spread, particularly in households, healthcare
	and nurseries.
	Water contaminated directly or indirectly with faeces.
	Outbreaks have been associated with public and private water
	supplies, swimming pools and, more rarely, contaminated food. Seasonal outbreaks are associated with farm visits to feed and
	handle lambs and calves.
Incubation period	Incubation period is dose dependent. Usual range 3 – 12 days
	(usual median 5-7 days)
Common clinical	Profuse watery diarrhoea accompanied by abdominal cramps.
features	(96% of patients who present for consultation), vomiting (65%),
IGALUIGO	mild fever (59%), and loss of appetite.

	Mean duration of symptoms reported as 12.7 days, but they may
	persist for up to a month.
	After apparent cessation, recurrence of symptoms is reported in
	around 1/3 of cases.
	Profoundly immunocompromised patients may experience
	chronic or intractable disease, and potentially life-threatening
	complications.
Period of infectiousness	Whilst symptomatic and for up to 2 weeks after symptoms have
	stopped.
Other relevant	Immunocompromised individuals (particularly people with
information	profound T cell immunodeficiencies) are at increased risk of
	experiencing severe/prolonged symptoms and of complications.
	Complications may be severe and life threatening, and may
	include pancreatitis, sclerosing cholangitis and biliary cirrhosis
	(rare) or pneumoretroperitoneum / pneumomediastinum (very
	rare).
	Clinicians treating immunocompromised cases should seek
	expert advice.
	Laboratories may not routinely test for <i>Cryptosporidium</i> species
	so prompt microbiological diagnosis should be discussed with
	routine diagnostic laboratories.
	Oocysts are highly resistant to disinfection with levels of
	chlorination usually used in drinking water treatment and
	swimming pools.
Further relevant guidance	
	wales.nhs.uk/sitesplus/888/page/44044
	Unit: www.wales.nhs.uk/sites3/page.cfm?orgid=457&pid=25284
	s://www.gov.uk/government/collections/cryptosporidiosis-
guidance-data-and-analysis	
o	Guidance for the investigation of Cryptosporidium linked to
swimming pools.	Culture for the investigation of Oryptospondium inned to

Available at: www.wales.nhs.uk/sites3/page.cfm?orgid=457&pid=49029

US Food and Drug Administration (2012) Bad Bug Book - Foodborne Pathogenic Microorganisms and Natural Toxins Handbook. Second Edition Available at:

www.fda.gov/Food/FoodbornellInessContaminants/CausesOfIIInessBadBugBook

Cyclosporiasis/ Cyclospora cayetanensis

Control of human source:	
	follow usual enteric precautions. Specific advice on exclusion from
nursery, school or work set	
Public health follow-up	
required	Not usually indicated unless case is identified as part of a
loquilou	cluster/outbreak: in these circumstances local outbreak
	procedures should be followed.
Cases	Clinical treatment with Trimethoprim-sulfamethoxazole
Cubbo	Obtain travel history and complete questionnaire
	Enteric precautions
Contacts	No action required
Exclusions	A minimum of 48 hours symptom free/no loose stools
Microbiological	None required
clearance	none required
Case definitions:	
	d the identification of Cyclospora cayetanensis oocysts in a stool
sample	a the lacitation of Cyclospora cayetariensis obcysts in a stool
	e required as cases may not shed sufficient oocysts in stools)
Causative agent:	
Cause	Cyclospora cayetanensis, a protozoan parasite
Reservoir	Humans
Epidemiology	Cases are usually associated with travel to Central or South
Lpidemology	America, the Caribbean islands, Indian subcontinents and South
	East Asia
	Infection occurs worldwide however the parasite is not endemic
	in the UK. Since 2015, large outbreaks have been reported in the
	UK from travellers returning from Mexico.
Transmission	Direct person-to-person spread is unlikely.
Tranomicolori	Cyclospora cayetanensis is transmitted by ingesting infective
	oocysts. Oocysts are excreted in faeces of human hosts in a non-
	infective form. They must then sporulate (mature) over 7-15 days
	in the environment to become infective.
	Ingestion of sporulated oocysts from sources such as drinking
	water, and fresh foods cause infection.
	Outbreaks linked to imported fresh berries, herbs and salad
	leaves have been documented in developed countries.
Incubation period	Usually 7 days (range 1-14 days)
Common clinical	Watery diarrhoea which may be prolonged
features	Other symptoms often include abdominal pain, fatigue, nausea,
	flatulence, weight loss and loss of appetite. Vomiting, headache
	and fever may also occur.
	Some cases may be asymptomatic.
Period of infectiousness	Direct person-to-person spread is unlikely
Other relevant	Immunocompromised cases may remain infected for several
information	months, but treatment will clear infection.

	In a cluster/outbreak situation, a travel history should be sought and if none, a detailed food history (including raw fruits, salads,
	herbs and imported foods) should be undertaken.
Further relevant guidance and key references:	

Further relevant guidance and key references:

Public Health England: www.gov.uk/guidance/cyclospora-clinical-and-travel-guidance

Questionnaire: http://phenet.phe.gov.uk/Resources/duty-doctors/Documents/20170710-Cyclospora- Questionnaire-V4.docx or Select Survey

Hawker J, Begg N, Blair I, Reintjes R, Weinberg J & Ekdahl K. (2012) *Communicable Disease Control and Health Protection Handbook – Third Edition*. Wiley-Blackwell.

Centers for Disease Control and Prevention: www.cdc.gov/parasites/cyclosporiasis US Food and Drug Administration (2012) Bad Bug Book - Foodborne Pathogenic Microorganisms and Natural Toxins Handbook. Second Edition Available at:

www.fda.gov/Food/FoodbornellInessContaminants/CausesOfIIInessBadBugBook

Enteric fever: typhoid and paratyphoid fevers/ Salmonella enterica subsp. Enteric/ serovar Typhi (commonly *S. typhi*) and Salmonella enterica subsp. enterica serovar Paratyphi – A, B and C (commonly *S.* Paratyphi A, B and C)

Control of human source:	
	follow usual enteric precautions. Specific advice on exclusion from
nursery, school or work sett	
Public health follow-up required	
Cases	Clinical management of cases as appropriate
	Enteric precautions Clinical sample as soon as possible from any possible case for diagnosis
Contacts	 If the case's infection is likely travel related: Co-travelling contacts who have consistently similar exposures to case and who are in risk-groups A-D require ONE faecal sample as soon as possible for screening, "Warn and inform" and hygiene information. All other contacts require "Warn and inform" & hygiene advice information, but no screening samples unless symptomatic.
	If the case's infection is <i>not</i> travel-related: Consider extensive investigation to identify source (even if case is not in risk group). Household and other close contacts should provide one faecal sample for screening and should receive "Warn and inform" and hygiene advice.
	<i>Any</i> contact who is/becomes symptomatic, or who has a positive screening sample should be managed as a case.
	A wider risk assessment of child-care/education/employment setting may be required if a case attended such a session whilst symptomatic.
Exclusions	Possible case: Whilst symptomatic and for a minimum of 48 hours after symptoms have stopped. Group C – anyone suspecting they are suffering from this illness or have previously had it, or who has a lot of contact with someone who has it, should be excluded from food handling and food handling areas until medical clearance
	Probable/confirmed case not in a risk group: Whilst symptomatic and for a minimum of 48 hours after symptoms have stopped.
	Probable/confirmed case in risk group A-D:

	Exclusion from risk activities or redeployment until microbiological clearance.
	Asymptomatic contacts: exclusion not required.
Microbiological	Probable/confirmed case not in risk group:
clearance	Microbiological clearance not required
	Recovered/asymptomatic possible case in risk groups A-D: I faecal sample obtained
	Probable/confirmed case in risk groups A-D: Faecal sampling should commence 1 week after completion of antibiotic treatment. THREE consecutive negative samples required, taken at least 48 hours apart.
Casa definitions	

Case definitions: Confirmed Case:

A person with *S*. Typhi or *S*. Paratyphi infection determined by the Public Health England Gastrointestinal Bacteria Reference Unit

OR

A person with documented confirmatory evidence from a recognised overseas reference laboratory

Probable Case:

Local laboratory presumptive identification of Salmonella Typhi or Paratyphi on faecal and/or blood culture or culture of another sterile site (e.g. urine), with or without clinical history compatible with enteric fever.

OR

A returning traveller giving a clinical history compatible with enteric fever and documentation of a positive blood/faecal culture (or positive PCR for S.Typhi / S.Paratyphi on blood) and/or treatment for enteric fever overseas.

Possible Case:

A person with a clinical history compatible with enteric fever and where the clinician suspects typhoid or paratyphoid as the most likely diagnosis

ÔR

A person with clinical history of fever and malaise and/or gastrointestinal symptoms with an epidemiological link to a source of enteric fever e.g. if they have 'Warn and inform' information OR

A returning traveller reporting a diagnosis abroad with positive serological testing or Salmonella PCR from faeces but no documented evidence of a positive blood or faecal culture positivity.

Causative agent:	
Cause	Salmonella enterica subsp. enterica serovar Typhi (commonly S.
	Typhi).
	Salmonella enterica subsp. enterica serovar Paratyphi – A, B, C
	(commonly
	S. Paratyphi A, B and C).

Reservoir	The main reservoir for both typhoid and paratyphoid is the human intestinal tract
Epidemiology	 Majority of cases (95%) reported in the UK are related to travel to endemic areas. In developed countries where standards of sanitation are high, the diseases are sporadic and are mainly associated with foreign travel. In the UK, approximately 55% of enteric fever cases are due to <i>S</i>.
Transmission	Typhi and 45% to S. Paratyphi (majority paratyphoid A).Primarily faecal-oral following ingestion of food or water contaminated by faeces (or, occasionally, urine) of acutely ill cases or chronic carriers.Direct faecal-oral transmission can also occur in poor hygiene conditions and, rarely, through sexual contact.The risk of contracting typhoid and paratyphoid fever is highest for travellers to areas of high endemicity. The estimated incidence of typhoid among travellers to developing countries is
Incubation period	 3–30 cases per 100,000 travellers. Incubation period depends on host factors and the size of the infectious dose. S.Typhi: usually 8-14 days; but can range from 3-60 days. S.Paratyphi: usually 1-10 days National guidance suggests onset of travel related infection can occur up to 28-60 days after end of travel.
Common clinical features	Typhoid fever: Insidious onset of a systemic illness: symptoms may include sustained fever, marked headache, malaise, anorexia, abdominal pain, diarrhoea. There is a wide variation in clinical severity. Complications may include intestinal haemorrhage or perforation (about 1-4% of cases), renal failure or osteomyelitis. Other rare complications include cholecystitis, meningitis and pneumonia. The case-fatality rate of 10–20% observed in the pre-antibiotic era can fall below 1% with prompt antibiotic therapy. 5–20% of patients may experience relapses. In the UK, faecal carriage and relapse rates are estimated at <3%. Paratyphoid fever:
	 Clinically similar but usually less severe than typhoid. Complications are less common. Relapses may occur in up to 9% of cases. S.Paratyphi C infections are rare. Enteric fever can be successfully treated with antibiotic therapy and general medical support. Treatment should be subject to clinical opinion and antibiotic sensitivity.

bacteria. Cases are not considered infectious prior to symptom onset. Further risk assessment may be required for convalescent and chronic carriers in risk groups to consider potential ongoing risk to public health, and appropriate interventions. S.Typhi: - Approximately10% of untreated patients will excrete bacteria for at least 3 months after the onset of acute symptoms. - Approximately 2-5% become chronic carriers, which may last many years. S.Paratyphi: - Most people will excrete bacteria for 5-6 weeks after onset of acute symptoms - A small minority continue excreting for months or even years. Other relevant information Serovar Paratyphi B var. Java is associated with gastrointestinal disease and is difficult to distinguish by conventional microbiological tests from invasive biotypes associated with paratyphoid fever. Further relevant guidance and key references: Public Health England: www.gov.uk/government/collections/typhoid-and-paratyphoid-guidance-data-and-analysis Public Health Wales: www.wales.nhs.uk/sitesplus/888/page/43751 US Food and Drug Administration (2012) Bad Bug Book - Foodborne Pathogenic Microorganisms and Natural Toxins Handbook. Second Edition	Period of infectiousness	Variable Reaple are infectious for the duration of exerction of	
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Escherichia coli infections/ Escherichia coli other than STEC

Control of human source	· · · · · · · · · · · · · · · · · · ·
	follow usual enteric precautions. Specific advice on exclusion from
nursery, school or work settings is given below	
Public health follow-up	Practice varies across the UK
required	England: Yes
	Northern Ireland: No routine follow-up
Cases	Obtain potential risk factor history
Contacts	Clinical surveillance – others may have been exposed to the same risk
Exclusions	Cases and symptomatic contacts in risk groups: a minimum of 48 hours symptom free/ no loose stools Group C - anyone who has household contact with someone with E. Coli 0157 should inform their business manager; exclusion should be considered for such food handlers if managers are
	concerned they have poor hygiene or if contact with the infected person is unavoidable.
Microbiological	Medical clearance should be sought for Group C. This will usually
clearance	require 2 consecutive, negative, faecal samples, the second
	sample being taken 48 hours after the symptoms have stopped
	naturally.
Case definitions:	
Enteropathogenic (EPEC) Diffuse-adherent (DAEC) o	itis may be classified as (STEC, EHEC), Enterotoxigenic (ETEC), Enteroinvasive (EIEC), Enteroaggregative (EAEC, EAggEC), r Cytolethal distending toxin producing (CDT producing).
Causative agent:	- Frankarishia aali
Cause	Escherichia coli
Reservoir	Gastrointestinal tract of humans and animals.
Epidemiology	May be associated with travel to developing countries. May cause cases of gastroenteritis and outbreaks in developed countries.
Transmission	Faecal-oral from person to person (EPEC), foodborne (ETEC, EPEC, EIEC) or waterborne (ETEC, EPEC, EIEC) spread.
Incubation period	Reported range from 1 hour to 7 days. Most cases within about 10-50 hours (ETEC, EIEC) or about 8-18 hours (EPEC, EAEC).
Common clinical features	Diarrhoea (all types), often watery. Abdominal pain common (ETEC, EPEC, EIEC). Nausea, vomiting and fever may occur (all) and/or blood and mucus (EIEC, EAEC).
Period of infectiousness	Whilst symptomatic and for 48 hours after diarrhoea has stopped.
Other relevant	Excretion often longer than 48 hours after remission, but
information	infectious risk low if normal stools.
Further relevant guidance and key references:	
Public Health England: www.gov.uk/government/collections/escherichia-coli-e-coli-guidance- data-and-analysis Public Health Wales: www.wales.nhs.uk/sitesplus/888/page/43885 Hawker J, Begg N, Blair I, Reintjes R, Weinberg J & Ekdahl K. (2012) <i>Communicable</i>	

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Giardiasis/ Giardia duodenalis

Control of human source:		
Cases should be advised to	Cases should be advised to follow usual enteric precautions. Specific advice on exclusion from	
nursery, school or work settings is given below		
Public health follow-up		
required	120	
Cases	Enteric precautions	
Cuscs	Antimicrobial treatment is required	
	Undertake enhanced surveillance as per local protocol.	
Contacts	Screen symptomatic contacts	
Contacts	Practice may vary across the UK (household contacts may be	
	screened in Wales)	
Exclusions	A minimum of 48 hours symptom free/no loose stools	
	Cases should not go swimming for 2 weeks after symptoms have	
	stopped.	
	Northern Ireland have not routinely provided this advice	
Microbiological	None required	
clearance		
Case definitions:		
	pensistent with Cierdia and Cierdia and a state of the time	
	consistent with Giardiasis and Giardia spp. cysts or trophozoites	
	n via routine diagnostic laboratory methods.	
Causative agent:		
Cause	Giardia spp. Giardia duodenalis (syn. Giardia lamblia; syn.	
	Giardia intestinalis)	
Reservoir	Gastrointestinal tracts of humans and animals.	
Epidemiology	Cases may be associated with recent foreign travel.	
	Family clusters are common.	
Transmission	Faecal-oral spread, by direct or indirect contact with the faeces of	
	infected people or animals:	
	- Person-to-person spread is common, particularly within	
	families/households.	
	- Waterborne, including swimming in contaminated recreational	
	water.	
	- Direct contact with infected animals	
	- Foodborne transmission	
	- Sexual transmission, particularly amongst MSM	
	- Direct contact with infected animals (rare)	
	Outbreaks have been associated with infected food handlers,	
	drinking water and swimming pools.	
Incubation period	Usually 5-16 days (median 7-10 days); extremes of 1-28 days	
	reported	
Common clinical	Diarrhoea, abdominal pain, malaise, flatulence and, less often,	
features	nausea.	
	Prolonged diarrhoea, malabsorption and weight loss may occur.	
Desired of lafe of	Asymptomatic infection also occurs particularly in children.	
Period of infectiousness	Whilst symptomatic and for up to 2 weeks after symptoms have	
	stopped.	

Other relevant information	 Risk of transmission to others decreases after symptoms have stopped, but cysts continue to be shed after symptoms have stopped. Since cysts are resistant to normal chlorine levels used in swimming pools, cases should not go swimming for 2 weeks after symptoms have stopped due to the potential to contaminate the pool environment and cause onward transmission. Cysts are moderately resistant to disinfection with levels of chlorination usually used in drinking water treatment and swimming pools.
Fullnel relevant guida	nce and key relefences.
Further relevant guidance and key references: Public Health England: www.gov.uk/giardia US Food and Drug Administration (2012) Bad Bug Book - Foodborne Pathogenic Microorganisms and Natural Toxins Handbook. Second Edition Available at:	

www.fda.gov/Food/FoodbornellInessContaminants/CausesOfIIInessBadBugBook

Hepatitis A/ Hepatitis A virus

Control of human	
Control of human source:	
Cases should be advised to follow usual enteric precautions. Specific advice on exclusion from nursery, school or work settings is given below	
Public health	YES
follow-up	Take action based on notification of a CONFIRMED or PROBABLE case.
required	Specific guidance exists. See links to further relevant guidance and key
0	references below.
Cases	Clinical management as appropriate
	Hygiene advice
	Complete national surveillance questionnaire to identify possible source of
	infection
	www.gov.uk/government/publications/hepatitis-a-case-questionnaire
	Undertake risk assessment particularly if case occurs in a non-household
Contonto	setting
Contacts	All contacts should be provided with hygiene advice.
	Active or passive immunisation may be indicated for contacts in specific
	age-groups, and settings and for those with specific pre-existing medical
	conditions. For details, consult Public health control and management of
	hepatitis A Brognant or brogstfooding contacts should be treated the same on other
	Pregnant or breastfeeding contacts should be treated the same as other contacts.
	Wider prophylaxis beyond household contacts is not usually indicated if a
	single case is identified in a hospital, secondary school or workplace.
Exclusions	Exclude case from work, school or nursery until 7 days after the onset of
LACIUSIONS	jaundice or in the absence of jaundice, from the onset of symptoms such
	as fatigue, nausea or fever
	Exclude close contacts who fulfil ALL of the following criteria: are food
	handlers, have not been immunised within 14 days of exposure, cannot
	restrict activities to those which do not involve preparing and handling
	unwrapped ready-to-eat foods until 30 days post-exposure and cannot
	achieve scrupulous hand hygiene
Microbiological	None required.
clearance	
Case definitions:	
Confirmed:	

Confirmed:

A person that meets the clinical case definition AND is confirmed through IgM and IgG antibodies to hepatitis A

OR

A person with hepatitis A RNA (HAV RNA) detected regardless of clinical features OR

An asymptomatic person with no recent history of immunisation with anti-HAV IgM from oral fluid or serum AND an epidemiological link to a confirmed hepatitis A case

Probable:

A person that meets the clinical case definition and has an epidemiological link to a confirmed hepatitis A case

OR

A person that meets the clinical case definition AND has IgM antibody to the hepatitis A virus (anti-HAV IgM)

Virologist to request quantitative IgG and IgM results and to consider the laboratory findings in the broader clinical and epidemiological context		
Causative age		
Cause	Hepatitis A virus	
Reservoir	Human gastrointestinal tract	
Epidemiolog y	Hepatitis A is no longer endemic in the UK and cases represent eithe importation following acquisition abroad or the importation of contaminated food. Frozen food or food components have been associated with outbreaks in mainland Europe, Ireland and the USA. Clusters, often in families or social groups, commonly occur around the primary case but onward transmission is otherwise uncommon	
Transmissio n	Faeco-oral route. Transmission can also occur during sexual contact, particularly amongst MSM and through injecting drug use. Transmission within households is very common. Children <6 years are particularly effective transmitters, especially in schools	
Incubation period	Average = 28 days (Range 15-50)	
Common clinical features	Extremely variable. Severity of illness increases with increasing age 80-95% of infections in children <5 years of age are asymptomatic, while in adults 70-95% of infections result in clinical illness Common symptoms include malaise, fever and jaundice	
	Fulminant hepatitis occurs rarely (approximately 1% of notified cases), bu rates are higher with increasing age and in those with underlying chronic live disease (e.g. chronic hepatitis B or C infection)	
Period of	Two weeks before the onset of symptoms to one week after the onset o	
infectiousnes s	jaundice. Where jaundice is not reported, a history of dark urine or pale stools should be enquired about. If there are no symptoms of jaundice, onset o other symptoms (such as fatigue, nausea, and fever) should be used. Shedding may continue for many weeks but does not appear to be associated with transmission of infection A chronic carrier state is not known to follow acute infection	
Other	Improved standards of living and hygiene in the UK have led to a dramatic	
relevant information	decline in incidence of hepatitis A infection, and it is no longer a commor childhood infection in the UK Prevalence of antibodies to HAV is declining with consequently high susceptibility amongst people born in the UK – a fact which may be overlooked when visiting family members in high endemicity countries Infection is followed by lifelong immunity against further clinical illness IgM reactivity in the absence of acute hepatitis A virus infection may be detected in older patients including those with pre-existing liver disease and should be interpreted with care.	

Public Health England: Public health control and management of hepatitis A (June 2017) www.gov.uk/government/uploads/system/uploads/attachment_data/file/363023/Guidance_for _the_Prevention_and_Control_of_Hepatitis_A_Infection.pdf

Questionnaire: www.gov.uk/government/publications/hepatitis-a-case-questionnaire

Public Health Wales: www.wales.nhs.uk/sitesplus/888/page/43692

US Food and Drug Administration (2012) *Bad Bug Book - Foodborne Pathogenic Microorganisms and Natural Toxins Handbook.* Second Edition Available at: www.fda.gov/Food/FoodbornelllnessContaminants/CausesOfIllnessBadBugBook

Hepatitis E/ Hepatitis E virus

Control of human source		
Cases should be advised to follow usual enteric precautions		
	YES	
Public health follow-up	Public Health action for CONFIRMED cases	
required		
	Specific guidance exists. See 'Further relevant guidance and key references' below.	
Casaa		
Cases	Clinical treatment as appropriate.	
	Complete surveillance questionnaire.	
	Obtain travel history	
	Immunocompromised individuals, pregnant women and those	
	with a history of liver disease, liver injury or heavy alcohol	
	consumption could be at risk of more serious or prolonged illness	
Contacts	No action required	
Exclusions	None required.	
Microbiological	None required	
clearance		
Case definitions:		
•	in a patient with acute hepatitis	
 HEV IgM AND IgG positi 		
 HEV RNA positive (with 	or without detectable HEV antibodies)	
	E infection in a person with acute hepatitis:	
 HEV RNA persisting for 	at least 3 months (with or without detectable HEV antibodies)	
Causative agent:		
Cause	Hepatitis E virus Genotypes 1-4	
	Hepatitis E virus Genotypes 1-4 Humans (G1/2) and animals including swine (G3/4)	
Cause		
Cause Reservoir	Humans (G1/2) and animals including swine (G3/4)	
Cause Reservoir	Humans (G1/2) and animals including swine (G3/4) Endemic/epidemic (G1/2) in countries with poor sanitation	
Cause Reservoir	Humans (G1/2) and animals including swine (G3/4) Endemic/epidemic (G1/2) in countries with poor sanitation (Africa, Asia and Central America)	
Cause Reservoir Epidemiology	Humans (G1/2) and animals including swine (G3/4) Endemic/epidemic (G1/2) in countries with poor sanitation (Africa, Asia and Central America) Zoonotic (G3/4) in industrialised countries including UK In developed countries, a zoonosis primarily through consumption	
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undertaken to exclude G1. If a G1 infection is identified in a pregnant woman she may require closer monitoring due to the potential serious outcome of G1 infection in pregnancy Immunocompromised individuals presenting with acute hepatitis E should be investigated for pre-existing persisting infection and the development of persistence

Further relevant guidance and key references:

Public Health England (January 2015) *Hepatitis E: public health operational guidelines* Available at: www.gov.uk/government/publications/hepatitis-e-health-protection-response-toreports-of-infection

Public Health Wales: www.wales.nhs.uk/sitesplus/888/page/55047

Food Standards Agency: http://www.food.gov.uk/science/microbiology/hepatitis-e

Histamine fish poisoning (previously known as scombrotoxic fish poisoning, scombroid, pseudo allergic fish poisoning, mahi mahi flush)/ histamine poisoning

Control of human source:	
	o follow usual enteric precautions.
Public health follow-up	NO– unless in an outbreak situation
required	Involvement of the Food Standards Agency may be
required	indicated.
Cases	Obtain full food history: identify potential individuals, restaurant,
Cases	
0	supplier and country of origin of food
Contacts	Clinical surveillance. Fellow consumers of the fish may also
	experience symptoms.
Exclusions	None required
Microbiological	None required
clearance	
Case definitions:	
A person with clinical symp	toms and a food history consistent with marine biotoxin ingestion
	potential food sources such as cheese)
	al presentation but toxins may be identified from the suspected
food source.	
Causative agent:	
Cause	Histamine.
Cause	
	Inadequate refrigeration allows multiplication of bacteria that
	contain the enzyme histidine decarboxylase (HDC). HDC
	converts histidine in fish tissues to histamine. Subsequent
	cooking / smoking does not diminish the levels of histamine.
	Histamine can also be present as a consequence of fermentation
	in the production of foods such as certain cheeses or sausages
Reservoir	Inadequately preserved and improperly refrigerated fish.
	Approximately 100 different species have been implicated:
	- scombroid dark-meat fish e.g. tuna, mackerel, skipjack, bonito,
	marlin (most commonly);
	- nonscombroid species e.g. mahi-mahi (dolphin fish), amber jack,
	sardine, yellowtail, herring, and bluefish;
	- whitefish (very rarely)
	- has also been associated with the consumption of cheese and
	other fermented foods
Enidomiolog	
Epidemiology	Accounts for approximately 5% of food-borne disease outbreaks
	reported to US Centers for Disease Control and Prevention
	(CDC).
	During 1998-2008, 262 confirmed and 71 suspected outbreaks
	were reported to CDC.
	Seasonal variation is observed with more cases occurring during
	summer months.
	Between 2001 and 2007, there were 2 reported incidents to the
	UK Food Standards Agency linked to histamine in cheese;
	between 2008 and 2015, there were twenty such reported
	incidents

Person-to-person spread does not occur.	
2 minutes – 2 hours after ingestion	
Flushing, sweating, rash, diarrhoea, vomiting, abdominal pain and	
headache. Occasionally, a metallic taste or burning/swelling of	
the mouth.	
Symptoms usually resolve within a few hours.	
Cases with a history of atopy or those taking certain medications	
(e.g. isoniazid or doxycycline which slow histamine metabolism	
by the liver) may have more severe symptoms and/or prolonged	
illness.	
Rare complications include bronchospasm, angioedema,	
hypotension, pulmonary oedema, and cardiogenic shock.	
Long term health consequences have not been reported.	
Person-to-person spread does not occur.	
Suspected fish/foods should be discarded to prevent further	
cases as cooking, canning, smoking or other processing does not	
diminish the levels of histamine.	
Temperature control is vital at all stages after catching, including	
display for sale.	
Those eating the same meal may experience variation in	
symptom severity due to:	
- individual differences in sensitivity to or metabolism of histamine	
- size of portion consumed	
 amount of histamine in consumed portion 	
 whether the portion was from the same fish 	
Diagnosis is based on clinical symptoms and history of	
fish/suspect food consumption. Laboratory tests for cases are not	
indicated, and levels of plasma or urinary histamine/histamine	
metabolites correlate poorly with clinical severity. Uneaten	
portions of suspect fish/suspect food may be tested for histamine	
levels by Public Analyst Laboratories.	
The Food Standards Agency should be informed if an outbreak or	
wider problem is suspected.	
and key references:	
icrobiological safety of food- Discussion paper:	
It/files/acm_1193_histamine%20in%20cheese%20(paper).pdf	
Medscape: http://emedicine.medscape.com/article/1009464-overview#a0101	
Centers for Disease Control and Prevention:	
www.cdc.gov/nczved/divisions/dfbmd/diseases/marine_toxins	
US Food and Drug Administration (2012) Bad Bug Book - Foodborne Pathogenic	
Microorganisms and Natural Toxins Handbook. Second Edition	
Available at:	
ellInessContaminants/CausesOfIIInessBadBugBook	
S	
ov.scot/downloads/Risk_Management.pdf	

Listeriosis/ Listeria monocytogenes

Control of human source	:	
	Cases should be advised to follow usual enteric precautions. Specific advice on exclusion from	
nursery, school or work settings is given below.		
Public health follow-up	Practice varies across the UK	
required	England: Yes	
	Northern Ireland: Does not have an enhanced surveillance	
	system	
Cases	Clinical management as appropriate	
	Obtain food history	
	Undertake enhanced surveillance as per local protocol	
	Discuss need for additional actions with the local Health Protection	
	Team	
Contacts	No action required	
Exclusions	None required	
Microbiological	None required	
clearance		
Case definitions:		
A person with symptoms	consistent with Listeriosis infection and Listeria monocytogenes	
detected in normally steri	le sites (e.g. blood or CSF) using routine diagnostic laboratory	
methods.		
All isolates of L. monocyte	ogenes should be sent to the Laboratory of PHE Gastrointestinal	
Bacteria Reference Unit, C	olindale for whole genome sequencing	
Causative agent:		
Cause	Listeria monocytogenes	
Cause Reservoir	Gastrointestinal tracts of humans, birds, cattle, sheep and other	
	Gastrointestinal tracts of humans, birds, cattle, sheep and other animals.	
	Gastrointestinal tracts of humans, birds, cattle, sheep and other animals. Widespread in the environment: soil, vegetation, water,	
	Gastrointestinal tracts of humans, birds, cattle, sheep and other animals. Widespread in the environment: soil, vegetation, water, silage/sewage, mammal/fish/bird faeces.	
	Gastrointestinal tracts of humans, birds, cattle, sheep and other animals. Widespread in the environment: soil, vegetation, water, silage/sewage, mammal/fish/bird faeces. Occurs in raw foods, food components and ready to eat foods:	
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Reservoir	Gastrointestinal tracts of humans, birds, cattle, sheep and other animals. Widespread in the environment: soil, vegetation, water, silage/sewage, mammal/fish/bird faeces. Occurs in raw foods, food components and ready to eat foods: most commonly in foods because of contamination from sites in food production environments	
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Reservoir	Gastrointestinal tracts of humans, birds, cattle, sheep and other animals. Widespread in the environment: soil, vegetation, water, silage/sewage, mammal/fish/bird faeces. Occurs in raw foods, food components and ready to eat foods: most commonly in foods because of contamination from sites in food production environments Listeriosis is a rare but severe systemic infection that includes bacteraemia, meningitis, encephalitis and in pregnant women can lead to miscarriage and stillbirth. It most often affects those who have a weakened immune system including pregnant women, their unborn and new born infants, the elderly and individuals who are immunocompromised by a pre-existing medical condition or	
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	(e.g. pâté and sliced meat), smoked fish, butter, olives and melon in the US.
	Mother-to-baby transmission is important:
	- in utero transmission,
	- vertical transmission during birth, or
	e
	- person-to-person spread soon after delivery
	Direct contact with infected animals can occasionally cause infection
	Pregnant women, individuals who are immunocompromised and
	those (< 1 month and >60 years of age) are more susceptible to infection.
	L. monocytogenes can be present in the faeces of approximately
	5% of the population but is likely to be transitory
Incubation period	For invasive disease, the incubation period ranges from 1-70 days
Common clinical	Initial symptoms of listeriosis include fever and flu-like symptoms,
features	
reatures	which may or may not be preceded by a febrile gastroenteritis.
	Pregnant women may be asymptomatic or have mild symptoms.
	A person of any age and immune-state may experience any of the
	following symptoms or remain asymptomatic. Below are the most
	common presentations for particular patient groups.
	Healthy adults and older children:
	- Asymptomatic infection
	- Acute gastroenteritis with fever
	- Non-specific symptoms such as fever, muscle aches, headache
	(often goes undiagnosed/unrecognised).
	Pregnant women
	- no/mild non-specific flu-like symptoms (as above)
	- Foetal loss, stillbirth, pre-term delivery with severe infection in the
	newborn (some with pre-term delivery) and neonatal meningitis.
	Immunosuppressed persons / older adults
	- Septicaemia, meningitis or meningo-encephalitis
	Immunocompetent persons can also present with severe disease
	such as septicaemia or meningitis
Period of	Not applicable except at and shortly after delivery due to contact
infectiousness	(hand or fomites) from an infected infant to an apparently healthy
Intectiousness	infant who develops meningitis
Other relevant	
information	L. monocytogenes can grow over a wide temperature range in contain foods from $<0^{\circ}$ C (refrigeretor temperature) to about 40° C
mormation	certain foods from $\leq 0^{\circ}$ C (refrigerator temperature) to about 40° C.
	Investigation of food and food preparation areas (including
	isolation of <i>L. monocytogenes</i>) is essential for control of foodborne
	illness.
Further relevant guidance	
Public Health England: w	ww.gov.uk/government/collections/listeria-guidance-data-and-
analysis	
Hawker J, Begg N, Blair I,	Reintjes R, Weinberg J & Ekdahl K. (2012) Communicable

Hawker J, Begg N, Blair I, Reintjes R, Weinberg J & Ekdahl K. (2012) *Communicable Disease Control and Health Protection Handbook – Third Edition*. Wiley-Blackwell.

Food Standards Agency: www.food.gov.uk/science/microbiology/listeria Centers for Disease Control and Prevention: www.cdc.gov/listeria US Food and Drug Administration (2012) Bad Bug Book - Foodborne Pathogenic Microorganisms and Natural Toxins Handbook. Second Edition Available at:

www.fda.gov/Food/FoodbornellInessContaminants/CausesOfIlInessBadBugBook

Marine algal shellfish poisoning syndromes and ciguatera poisoning/ Marine biotoxins

Control of human sources	
	person transmission. Cases should be advised to follow usual
enteric precautions.	
Public health follow-up	NO – unless in an outbreak situation
required	Not usually indicated unless case is identified as part of a
required	
	,
	procedures should be followed. Involvement of the Food
	Standards Agency may be indicated.
Cases	Obtain food history: identify potential individual, restaurant,
	supplier or growing area
Contacts	Clinical surveillance. Other consumers of the fish/shellfish may
	also experience symptoms.
Exclusions	None required
Microbiological	None required
clearance	
Case definitions:	
A person with clinical sympt	oms and a food history consistent with marine biotoxin intoxication.
	cal presentation, but toxins may be identified from the suspected
food	······································
Causative agent:	
Cause	Multiple naturally occurring biotoxins produced by marine
Ouuse	organisms, retained by certain filter feeding bivalves and fish.
	Some carnivorous gastropods, crustaceans and fish concentrate
	the toxin in the food chain, leading to toxic effects following
Decemuein	ingestion by humans Seafood
Reservoir	
Epidemiology	Seasonal variation is observed with more cases occurring during
	summer months when dinoflagellates growth is greatest.
	Likely to be an under-reported cause of food-poisoning due to
	mild cases being un-recognised un-diagnosed by healthcare
	professionals.
	The most common syndromes are diarrhetic shellfish poisoning,
	ciguatera poisoning, neurotoxic shellfish poisoning, paralytic
	shellfish poisoning and amnesic shellfish poisoning
Transmission	Consumption of seafood contaminated by toxin. Person-to-
	person spread does not occur. Toxins can survive most cooking
	and freezing processes applied to food
Incubation period	Few minutes to 24 hours after ingestion
Common clinical	Symptoms vary depending on the specific causative agent and
features	amount ingested.
	Ciguatera poisoning:
	Nausea, vomiting, diarrhoea, cramps, excessive sweating,
	headache and muscle aches. Neurological symptoms may also
	occur including altered sensation (burning or pins-and-needles),

weakness, itching, dizziness, reversal of temperature sensation	I
altered taste sensations, nightmares, or hallucinations. Onset: minutes to 6 hours after ingestion Duration: 1-4 weeks	
Rarely fatal	
Due to ciguatera toxins produced by dinoflagellates tha accumulate in tropical reef fish (barracuda, grouper, sea bass snapper, mullet and others). Cases have occurred in UK due to consumption of imported fish.	,
Paralytic shellfish poisoning:	
Numbness or tingling of face, arms, and legs, headache dizziness, nausea and incoordination. Muscle paralysis and respiratory failure can occur in severe cases and may be fatal Onset: 15 minutes to 10 hours after ingestion (usually within 2 hours)	I
Due to a different red-brown coloured dinoflagellate whose toxin concentrates within certain shellfish (mussels, cockles, clams scallops, oysters, crabs, and lobsters). Associated with red alga tides. Cases have occurred in UK due to consumption of UK grown and imported shell fish.	,
Diarrhetic Shellfish Poisoning: Diarrhoea, nausea and abdominal pain. Onset: 30 minutes to 12 hours, duration 3-4 days. Due to dinoflagellate whose toxin accumulates in certain shellfish (mussels, cockles, scallops oysters and crabs). Associated with red algal tides. Cases have occurred in UK due to consumption of UK grown and imported shellfish.) , ;
Neurotoxic shellfish poisoning: Numbness, tingling in the mouth, arms and legs, incoordination and gastrointestinal upset. Some patients report temperature reversal Onset: 1-3 hours Duration: 2-3 days Rarely fatal Due to a third type of dinoflagellate toxin found in oysters, clams	•
and mussels	
Amnesic shellfish poisoning: Diarrhoea and vomiting, and occasionally dizziness, headache disorientation, and permanent short-term memory loss. In severe poisoning, seizures, focal weakness or paralysis and death may occur	;
Onset: within 24hours of consumption May cause long-term problems with short-term memory.	

	Rare syndrome caused by a toxin made by the diatom <i>Nitzchia pungens</i> and concentrated in mussels and other shellfish
Period of infectiousness	Person-to-person spread does not occur Suspected shellfish/fish should be discarded to prevent further cases as cooking, canning, smoking or other processing does not diminish the levels of toxic chemicals
Other relevant information	Diagnosis is based on clinical symptoms and relevant history of fish/shellfish consumption. Laboratory tests for cases are not indicated. Uneaten portions of suspect fish may be tested for specific toxin, but this does not aid treatment of the case.
Further relevant guidance a	nd key references:
Further relevant guidance and key references: Centers for Disease Control and Prevention: www.cdc.gov/nczved/divisions/dfbmd/diseases/marine_toxins US Food and Drug Administration (2012) <i>Bad Bug Book - Foodborne Pathogenic</i> <i>Microorganisms and Natural Toxins Handbook</i> . Second Edition Available at:	

www.fda.gov/Food/FoodbornellInessContaminants/CausesOfIIInessBadBugBook

Norovirus gastroenteritis/ Norovirus

Control of human source:	
Community cases should be advised to follow usual enteric precautions. Specific advice on	
exclusion from nursery, school or work settings is given below.	
Cases occurring within hospital, care homes or other institutional settings should follow usual	
enteric precautions and be managed under the appropriate local policy.	
Public health follow-up	
required	Not usually indicated unless case is identified as part of a
	cluster/outbreak. In those circumstances, local outbreak
	procedures should be followed.
	Specific guidance exists. See 'Further relevant guidance and key
	references' below.
Cases	Enteric precautions
Contacts	Clinical surveillance
	Group C – persons with household contact should inform the food
	business manager.
Exclusions	A minimum of 48 hours after symptoms have stopped/no loose
	stools
	Group C – best practice to exclude <i>suspected</i> infected persons
Microbiological	None required
clearance	
Case definitions:	
A symptomatic person and	aboratory identification of <i>Norovirus</i> from a clinical specimen, most
often a stool specimen.	
Causative agent:	
	Noroviruses (formally known as Norwalk like viruses and small
Causative agent:	<i>Noroviruses</i> (formally known as Norwalk like viruses and small round structured viruses)
Causative agent:	
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Common clinical	······································
features	vomiting and watery diarrhoea. These may be accompanied by
	fever, headache, abdominal pain and/or aching limbs.
Period of infectiousness	Whilst symptomatic and for 48-72 hours after diarrhoea has
	stopped.
Other relevant	
information	and ability to survive in the environment for several days all
	contribute to the high number of outbreaks caused by Norovirus.
	Immunity is short-lived; infection with one strain of Norovirus is
	not protective against other strains.
	Laboratories may not routinely test for Norovirus, hence prompt
	discussion with routine diagnostic laboratories may be indicated.
Further relevant guidance	
-	v.gov.uk/government/collections/norovirus-guidance-data-and-
analysis	
	wales.nhs.uk/sitesplus/888/page/43919
	ciation, Healthcare Infection Society, Infection Prevention Society,
	ncare Infections, NHS Confederation (March 2012) Guidelines for
	us outbreaks in acute and community health and social care
settings	
	overnment/publications/norovirus-managing-outbreaks-in-acute-
and-community-health-and-	U
0,	lorovirus Working Group (July 2007) <i>Guidance for the</i>
	nfection in Cruise Ships. Available at:
	Iblications/norovirus-managing-infection-in-cruise-ships
Food Standards Agency: www.food.gov.uk/science/microbiology/norovirus and Food	
Standards Agency: www.food.gov.uk/science/microbiology/norovirus and	
www.food.gov.uk/sites/default/files/multimedia/pdfs/publication/fitnesstoworkguide09v3.pdf	
.	tration (2012) Bad Bug Book - Foodborne Pathogenic
•	al Toxins Handbook. Second Edition
Available at:	malling and Constants (Constants Of Illing and Doug Doug Doug
www.rda.gov/Food/Foodboi	nellInessContaminants/CausesOfIIInessBadBugBook

Rotavirus infection/ Rotavirus

Control of human source:	
	follow usual enteric precautions. Specific advice on exclusion from
nursery, school or work settings is given below.	
Public health follow-up	NO
required	
Cases	Clinical management as appropriate
	Enteric precautions
Contacts	Clinical surveillance and screening of symptomatic contacts
Exclusions	A minimum of 48 hours symptom free/no loose stools
Microbiological	None required
clearance	
Case definitions:	
Laboratory detection of Rot	avirus in a symptomatic person
Causative agent:	
Cause	Rotavirus
	3 serogroups (A, B and C) with A being the most common
Reservoir	Humans
	Animal reservoirs exist but animal-to-human transmission does
	not occur
Epidemiology	Main cause of viral gastroenteritis in children in developed and
	developing countries
	Most cases occur in children aged 6 months -2 years
	Most cases in the UK occur in winter and spring, with a peak in
	March
	Outbreaks in settings such as nurseries are common, though with the implementation of a vaccination programme in the UK from
	2014, the incidence has decreased, and the epidemiological
	picture may change.
Transmission	Person-to-person spread via faecal-oral route is most common
Tunishinission	Transmission may also occur via contact with contaminated
	environmental surfaces. The virus is resistant to many
	disinfectants (inactivated by chlorine)
Incubation period	1 - 4 days
Common clinical	Watery diarrhoea and vomiting with/without fever, abdominal pain
features	and dehydration
	Vomiting usually resolves within 1-3 days and diarrhoea within 5-
	7 days but it can take up to 2 weeks
Period of infectiousness	Infectious from 2 days before symptom onset to 10 days after
	symptoms resolve. May be longer in immunocompromised
	individuals
Other relevant	Oral Rotavirus vaccine at 2 and 3 months is now part of the routine
information	childhood vaccination schedule in the UK. Those involved in
	nappy changing of recently vaccinated babies should observe
	good personal hygiene as traces of the vaccine virus may be
	excreted in faeces and may enable onward transmission,
	particularly to persons with weakened immune systems

If a child is tested for Rotavirus near the date of immunisation,
the vaccine virus may be detected in stool and where the child
presents with symptomatic gastroenteritis, testing for other
infectious aetiologies should be considered. Up-to-date
information on Rotavirus surveillance in England and Wales can
be found on the PHE website:
www.gov.uk/government/statistics/norovirus-national-update.

Further relevant guidance and key references:

Public Health England: www.gov.uk/government/collections/rotavirus-guidance-data-andanalysis

Hawker J, Begg N, Blair I, Reintjes R, Weinberg J & Ekdahl K. (2012) *Communicable Disease Control and Health Protection Handbook – Third Edition*. Wiley-Blackwell. NHS Choices: www.nhs.uk/Conditions/Rotavirus-gastroenteritis/Pages/Causes.aspx and:

www.nhs.uk/Conditions/vaccinations/Pages/rotavirus-vaccine-questions-answers.aspx#which US Food and Drug Administration (2012) Bad Bug Book - Foodborne Pathogenic Microorganisms and Natural Toxins Handbook. Second Edition Available at:

www.fda.gov/Food/FoodbornellInessContaminants/CausesOfIIInessBadBugBook

Salmonellosis excluding enteric fever/ Salmonella species excluding S. Typhi and S. Paratyphi

Control of human source:	
Cases should be advised to follow usual enteric precautions. Specific advice on exclusion from	
nursery, school or work set	
Public health follow-up required	Practice varies in the UK Northern Ireland: Each case followed up and a case questionnaire completed. Risk factor data reviewed, surveillance data reviewed etc. for all cases to identify emerging cluster
Cases	Enteric precautions Complete enhanced surveillance questionnaire.
Contacts	Clinical surveillance Screen symptomatic contacts
Exclusions	A minimum of 48 hours symptom free/no loose stools
Microbiological	None required
clearance	
Case definitions:	
	a Salmonella spp. Infection determined by the local microbiology
laboratory.	
Causative agent:	Colmonollo on n
Cause	Salmonella spp. Excluding S. Typhi. and S. Paratyphi A, B and C.
Reservoir	Gastrointestinal tract of wild and domestic animals, birds (especially poultry), reptiles, amphibians (for example, terrapins), and occasionally humans.
Epidemiology	There are >2500 serotypes of <i>Salmonella</i> . <i>Salmonella</i> Enterica serovar, <i>S.</i> Enteritidis and <i>S.</i> Typhimurium are the most commonly identified in the UK, Europe and USA. Cases often appear sporadic, but outbreaks occur in the general population and <i>institutions</i> .
Transmission	Predominantly through consuming foodstuffs (most often red and white meats, raw eggs, milk, and dairy products) following contamination of cooked food by raw food or failing to reach adequate cooking temperatures. Person to person spread, usually during the acute diarrhoeal phase of the illness and contact with infected animals can also cause infection. Waterborne outbreaks have also been reported.
Incubation period	Most commonly 12-48 hours but range of 4-120 hours has been
	reported Ingested dose will influence incubation period, symptoms and disease severity.
Common clinical	Symptoms include watery and sometimes bloody diarrhoea,
features	abdominal pain, headache, nausea, vomiting and fever.
	Duration of 4-7 days.
	Usually resolve without treatment.

	1	
	Septicaemia may occur and requires prompt hospitalisation and antibiotic therapy. The elderly, infants, and those with impaired	
	immune systems are more likely to have severe illness and	
	develop complications.	
Period of infectiousness	Cases are considered infectious whilst symptomatic.	
	However, organisms are excreted by convalescent carriers,	
	asymptomatic carriers and (rarely) chronic carriers.	
	Cases with diarrhoea, infants and faecally incontinent adults pose	
	a greater risk of transmission than do asymptomatic people.	
	Children aged <5 years may shed organisms for up to a year	
	(median 10 weeks). Over the age of 5 years, the maximum	
Other relevant	duration of shedding appears to be up to 12 weeks (median 4). Rates have fallen in the UK since the mid-1990s due to factors	
information	including greater public awareness about food safety, and the	
mormation	compulsory vaccination of the UK egg-laying flock against	
	Salmonella E <i>nteriditis</i> .	
	Secondary cases are common in outbreaks.	
	Food handlers who practice good hygiene are very rarely	
	responsible for initiating outbreaks.	
	Many reptiles, including those kept as pets, carry salmonella in	
	their guts without exhibiting symptoms but may transmit infection	
	to humans. Specific advice on reducing the risk of transmission is available from PHE (see below).	
Further relevant guidance a		
)	w.gov.uk/government/collections/salmonella-guidance-data-and-	
analysis		
	Public Health England – reducing the risks of salmonella infection from reptiles:	
0 0 1	ublications/salmonella-reducing-infection-from-reptiles	
	wales.nhs.uk/sitesplus/888/page/43751	
0	US Food and Drug Administration (2012) Bad Bug Book - Foodborne Pathogenic	
Available at:	al Toxins Handbook. Second Edition	
	rneIllnessContaminants/CausesOfIIInessBadBugBook	
www.iuu.gov/i 000/i 00000		

Sapovirus gastroenteritis/ Sapovirus

Control of human source	
	e advised to follow usual enteric precautions. Specific advice on
-	nool or work settings is given below.
	rring within hospital, care homes or other institutional settings
	ols to those developed for norovirus.
Public health follow-up	NO – unless in an outbreak situation
required	
Cases	Enteric precautions
Contacts	Clinical surveillance
Exclusions	Cases: A minimum of 48 hours symptom free/no loose stools
	Symptomatic contacts in risk groups: A minimum of 48 hours
	symptom free/no loose stools
Microbiological	None required
clearance	
Case definitions:	
	Sapovirus from a stool specimen from a person with diarrhoea.
	by electron microscopy or molecular methods
Causative agent:	
Cause	Sapovirus (formerly known as a human classic Calicivirus)
Reservoir	Humans
Epidemiology	Responsible for about 9% of cases of gastroenteritis in the
Epideimelogy	community and a similar proportion of those presenting to
	primary care
	Infection mainly in under-5's, although adult outbreaks do occur
	Outbreaks are most often in child care facilities, often with high
	attack rates. May also occur in hospitals, nursing homes, cruise
Transmission	ships and colleges
Transmission	Mostly person-to-person via the faeco-oral route
	Environmental contamination may occur, and waterborne or
	foodborne transmission may be possible
Incubation period	1-3 days (median 1.7 days)
Common clinical	Diarrhoea, often with abdominal pain/cramps and vomiting.
features	Vomiting usually a less prominent feature than in Norovirus
	infections
	Low grade fever, myalgia or headache may also occur
	Symptoms are usually mild and self-limiting
	Asymptomatic infection may occur
Period of infectiousness	Whilst symptomatic and for 48 hours after diarrhoea has
	stopped
Other relevant	Faecal excretion of the organism lasts for up to 2 weeks and
information	faeces have been shown to contain high levels of virus
Further relevant guidance	
	w.gov.uk/government/collections/gastrointestinal-infections-
guidance-data-and-analysis	
	Reintjes R, Weinberg J & Ekdahl K. (2012) <i>Communicable</i>
Disease Control and Health Protection Handbook – Third Edition. Wiley-Blackwell.	

Shiga toxin producing *Escherichia coli* (STEC) gastroenteritis (STEC O157 and non-O157), Haemolytic uraemic syndrome (HUS)

Control of human source:	
	follow usual enteric precautions. Specific advice on exclusion from
nursery, school or work sett	
Public health follow-up	Practice may vary across the UK
required	YES
	Public Health action for confirmed and probable cases
	Specific guidance exists. See 'Further relevant guidance and key
	references' below.
Cases	Undertake national enhanced surveillance questionnaire as
	guided by results of microbiological testing and risk assessment
	as per national guidance
	Clinical management as appropriate
	Enteric precautions
Contacts	Practice varies across the UK
	England: Screen symptomatic contacts and
	recovered/asymptomatic contacts in risk group B of cases of
	STEC O157. Management of contacts of non-O157 STEC should
	be guided by results of microbiological testing and risk
	assessment as per national guidance
	Wales: Screen all household contacts
Exclusions	NI: Screen contacts in high risk groups
Exclusions	England: STEC 0157
	Cases not in a risk group:
	Symptomatic cases:
	Until 48 hours symptom free
	Recovered/asymptomatic cases:
	No exclusion required
	Cases in risk groups A - D:
	Symptomatic cases:
	Exclude until microbiological clearance obtained
	Recovered/asymptomatic cases:
	Exclude until microbiological clearance obtained. Review risk
	assessment to determine whether restriction/redeployment/
	supervised return to childcare may be appropriate whilst awaiting
	results of clearance
	Non-O157 STEC
	Exclude all symptomatic cases until 48 hours symptom free.
	Exclusion of cases in risk groups may be required as guided by
	results of microbiological testing and risk assessment as per
	national guidance

Microbiological	England:
clearance	CASES
	STEC 0157
	Cases not in a risk group:
	No microbiological clearance required
	Cases in risk groups A-D:
	Two consecutive negative faecal specimens taken at least 24 hours apart, once the case is symptom free for at least 48 hours
	Non-O157 STEC Microbiological clearance for cases in risk groups may be required as guided by results of microbiological testing and risk assessment as per national guidance
	CONTACTS STEC 0157
	Contacts not in risk group: No microbiological clearance required
	Contacts in risk groups A, C and D: No microbiological clearance routinely required
	<u>Contacts in risk group B:</u> Two consecutive negative faecal specimens taken at least 24 hours apart and undertake risk assessment
Case definitions:	Non-O157 STEC Microbiological clearance for contacts in risk groups may be required as guided by results of microbiological testing and risk assessment as per national guidance

Case definitions:

Confirmed case: positive STEC culture or PCR shiga toxin positive result from PHE GBRU (with or without clinical features, with or without epidemiological link to a confirmed case) **Confirmed STEC-related HUS**: clinical features if HUS AND positive STEC culture or PCR shiga toxin positive result or serological confirmation of STEC from PHE GBRU OR

Clinical features of HUS AND diagnostic/local laboratory PCR shiga toxin positive result

Probable case:

<u>Local O157 culture positive</u> – diagnostic/local laboratory positive culture presumptive STEC O157 with or without clinical features, with or without epidemiological link to a confirmed case <u>Probable STEC-related HUS</u> – clinical features of HUS, with or without epidemiological link to a confirmed case, awaiting results of microbiological testing

<u>Epidemiological link</u> – epidemiological link to a confirmed case, awaiting results of microbiological testing OR diagnostic/local laboratory PCR shiga toxin positive, with or without clinical features of STEC

<u>PCR probable</u> - diagnostic/local laboratory PCR shiga toxin positive BUT negative culture for STEC O157, bloody diarrhoea/hospitalisation for acute diarrhoea, without epidemiological link to a confirmed case

Causative agent:	
Cause	Shiga toxin/verocytotoxin producing Escherichia coli
Reservoir	Gastrointestinal tract of ruminants (in the UK mainly cattle, sheep
	and goats). Other animals and birds acts as transmission vectors
Epidemiology	E. coli O157 is the most common serogroup of STEC causing
	infections in the UK.
	In England and Wales, almost 50% of STEC O157 cases are in
	children under 16 and rates of infection are highest in children
	under 5 years with the peak incidence in the 1-4 age group
	O157 is the only strain that can be routinely tested for in the
	majority of UK diagnostic laboratories by current methods. The
	use of PCR methods by diagnostic/local laboratories is
	increasing, leading to increased detection of non-O157 cases
	(confirmed by the PHE GBRU reference laboratory)
	STEC O157 and non-O157 (such as O104, O26 and O55) have
	been associated with outbreaks of HUS in the UK and
- <u> </u>	internationally
Transmission	Faecal-oral route
	- ingestion of contaminated food (particularly undercooked meat,
	minced beef, salad products including water cress) water or
	unpasteurized milk
	- person-to-person spread
	- direct/indirect contact with an infected animal or their faeces
	 environmental exposure e.g. swimming/playing in contaminated water, streams or ponds
	Seasonal outbreaks have been associated with farm visits to feed
	and handle calves and lambs
Incubation period	Usually 2-4 days for STEC O157 and similar for most strains of
	non-O157 STEC
Common clinical	Gastroenteritis:
features	- May be asymptomatic
loataroo	- Non-bloody diarrhoea, fever, abdominal cramps and vomiting
	- Bloody diarrhoea and severe abdominal pain in more severe
	disease
	- Usually self-limiting with recovery in around 10 days
	Haemolytic uraemic syndrome (HUS):
	 Approximately 10% of STEC O157 cases develop HUS
	- Characterised by acute renal failure, thrombocytopenia and
	microangiopathic haemolytic anaemia
	- Children aged less than 5 years are at greatest risk of developing
	HUS, usually 1 week after onset of bloody diarrhoea
	- Around 50% may develop chronic renal complications. Mortality
	is between 3-5%
	Non-0157 infactions show a similar spectrum of illness but
	Non-O157 infections show a similar spectrum of illness but several strains have been associated with more severe disease
	(bloody diarrhoea and HUS) including O26, O45 and others

Period of infectiousness	Shedding of organisms depending on strain of STEC and age of patient may be prolonged. It tends to be shorter in adults but there have been reports of children shedding for over 6 weeks		
Further relevant guidance			
	for Shiga Toxin producing Escherichia coli (STEC) –		
https://www.gov.uk/governm health-management	nent/publications/shiga-toxin-producing-escherichia-coli-public-		
	February 2011) VTEC Operational Manual: Operational guidance asses and incidents of VTEC infection		
Available at: www.gov.uk/go	Available at: www.gov.uk/government/publications/vero-cytotoxin-producing-escherichia-coli- operational-guidelines-for-public-health-management		
Health Protection Agency (F evidence for the Public Heal coli (VTEC) Available at: www escherichia-coli-advice-for-p Public Health Wales: www.w Health Protection Agency (J caused by vero cytotoxin pro Available at: www.gov.uk/go caused-by-vero-cytotoxin-pro Food Standards Agency: www industry/guidancenotes/hygo US Food and Drug Administ	February 2011) The VTEC Support Document. Background and Ith management of infection with verocytotoxigenic Escherichia ww.gov.uk/government/publications/vero-cytotoxin-producing- public-health-management-teams vales.nhs.uk/sitesplus/888/page/43884 July 2011) The management of acute bloody diarrhoea potentially oducing Escherichia coli in children overnment/publications/acute-bloody-diarrhoea-potentially- roducing-escherichia-coli-managing-cases-in-children ww.food.gov.uk/business-		
www.fda.gov/Food/Foodbor Preventing or controlling ill h	nellInessContaminants/CausesOfIIInessBadBugBook/ nealth from animal contact at visitor attractions. Industry COP, 2015) Available at: www.visitmyfarm.org/component/k2/item/339-		

Shigellosis/ Shigella species

Control of human source	
	follow usual enteric precautions. Specific advice on exclusion from
nursery, school or work set	
Public health follow-up	YES
required	Public Health action for confirmed and probable cases of <i>Shigella</i>
required	
	flexneri/boydii/dysenteria and Shigella spp.
	Specific guidance exists. See 'Further relevant guidance and key
	references' below.
Cases	Shigella sonnei
	Emphasise hygiene advice. Manage according to local
	arrangements.
	Shigella flexneri/boydii/dysenteriae (except type
	1)/unspecified
	Complete national non-sonnei questionnaire to identify
	travel/food/activity history, risk groups and contacts
	Shigella dysenteriae type 1
	Complete national non-sonnei questionnaire to identify
	travel/food/activity history, risk groups and contacts
Contacts	Shigella sonnei
	Symptomatic contacts: emphasise hygiene advice and 48-hour
	exclusion after first normal stool. Seek medical advice and testing
	for diagnostic purposes.
	Asymptomatic contacts: no action required
	Shigella flexneril boydiil dysenteriae/unspecified
	England and Wales: All contacts to be given link to NHS Choices
	information
	Group C contacts should be excluded and seek medical
	clearance
	NI: All contacts to be given information
	Symptomatic contacts: seek medical advice and testing for
	diagnostic purposes
Exclusions	Shigella sonnei
Exclusions	A minimum of 48 hours after first normal stool
	Shigella flexneri/boydii/dysenteriae (except type
	1)/unspecified
	Cases in risk groups: until microbiological clearance completed
	Cases not in risk groups: A minimum of 48 hours after first normal
	stool and medical clearance
	Symptomatic contacts: manage as a probable case
	Asymptomatic contacts: no exclusion
	Shigella dysenteriae (type 1)
	Cases in risk groups: until microbiological clearance completed
	Cases not in risk groups: A minimum of 48 hours after first normal
	stool
	Symptomatic contacts: manage as a probable case
	Asymptomatic contacts in risk groups: until microbiological
	clearance completed

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	Asymptomatic contacts not in risk groups: no exclusion
Microbiological	Shigella sonnei
clearance	None required
	Shigella flexneri /boydii / dysenteriae (except type 1)/unspecified
	Cases in risk group: one negative sample a minimum of 48 hours after first normal stool or 48 hours after completing antibiotics, whichever is later
	Cases not in risk groups: no microbiological clearance samples required
	Shigella dysenteriae (type 1)
	Cases in risk group: 2 consecutive negative samples a minimum of 48 hours after first normal stool or 48 hours after completing antibiotics. Samples to be taken at least 24 hours apart Cases not in risk groups: A minimum of 48 hours after first normal stool
	Symptomatic contacts: manage as a probable case Asymptomatic contacts in risk groups: 2 consecutive negative
	samples taken at least 24 hours apart
	Asymptomatic contacts not in risk groups: no microbiological clearance samples required
Coop definitional	

Case definitions:

Confirmed case:

A person with speciated shigella infection determined by a local laboratory or the PHE Reference Laboratory

Probable case:

A person with a culture positive *Shigella spp.,* determined by a local laboratory in the UK or overseas

OR

A person with a clinical history compatible with bacterial dysentery and/or a Shigella PCR positive (ipaH) result AND an epidemiological link to a confirmed or probable case

Causative agent:	
Cause	4 species of shigella: Shigella sonnei, Shigella flexneri, Shigella boydii, Shigella dysenteriae
Reservoir	Humans
Epidemiology	Infections peak in late summer in the UK Highest rates of infection occur in children aged < 5 years, followed by 5-14 year age group <i>S. sonnei</i> is the most common species in Western Europe and both <i>S. sonnei</i> and <i>S. flexneri</i> are endemic in UK Most cases of <i>S. boydii</i> and <i>S. dysenteriae</i> are imported but all strains may be travel-associated
Transmission	 Faeco-oral transmission directly amongst households, nursery and infant schools is most common. Foodborne infections occur but are rare. Direct transmission between men-who-have-sex-with-men (MSM) is also an important transmission route

	Environmental contamination during enjagdes of coute dis where a
	Environmental contamination during episodes of acute diarrhoea
	can occur, where bacilli may be aerosolised during toilet flushing
	and settle on surrounding surfaces and survive for weeks in cool
	and humid locations.
Incubation period	12 hours – 4 days (usually 1-3 days) but up to 1 week for S.
•	dysenteriae
Common clinical	Clinical features vary depending on Shigella species
features	S. sonnei causes mild illness in most cases with symptoms of
loataloo	diarrhoea (may be bloody in 10-50%) and abdominal pain
	with/without nausea, vomiting, headache and malaise lasting
	average of 4-5 days (range 1 day – 2 weeks)
	S. flexneri causes similar symptoms to S. sonnei but illness may
	be more severe with dysentery more prominent, longer duration
	of illness and hospitalisation rates higher. Complications include
	reactive arthritis and Reiter's syndrome
	S. boydii causes diarrhoeal illness like that of S. flexneri
	S. dysenteriae type 1 infection causes more severe illness, with
	dysentery in most cases and complications including haemolytic
	uraemic syndrome (HUS)
Period of infectiousness	Cases are most infectious when diarrhoea is present but
	considered infectious as long as organisms are excreted in stool
	(average of 2-4 weeks but prolonged carriage of several months
	has been reported)
Other relevant	See risk groups for transmission of gastrointestinal pathogens
information	(Table 1)
internation	Other contacts for consideration:
	- sexual contacts of MSM while case was infectious and for a
	week after symptoms have ceased
	•
	symptomatic whilst at a childcare setting or whilst working at a
	healthcare/food establishment
	Notifying clinicians should be reminded that any child <16 years
	presenting with infectious bloody diarrhoea should be managed
	as per the 2011 Royal College of Paediatrics and Child Health,
	RCGP and HPA guidelines "The management of acute bloody
	diarrhoea potentially caused by Vero cytotoxin producing
	Escherichia coli in children"
Further relevant guidance and key references:	
Public Health England Interim public health operational guidelines for shigellosis	
9	overnment/collections/shigella-guidance-data-and-analysis
Hawker J, Begg N, Blair I, Reintjes R, Weinberg J & Ekdahl K. (2012) Communicable	

Disease Control and Health Protection Handbook – Third Edition. Wiley-Blackwell. US Food and Drug Administration (2012) Bad Bug Book - Foodborne Pathogenic Microorganisms and Natural Toxins Handbook. Second Edition Available at:

www.fda.gov/Food/FoodbornellInessContaminants/CausesOfIlInessBadBugBook

Vibriosis/ Vibrionaceae species excluding Vibrio cholera

Control of human source:	
	follow usual enteric precautions. Specific advice on exclusion from
nursery, school or work settings is given below.	
Public health follow-up	
required	
Cases	Clinical treatment as appropriate
	Enteric precautions
Contacts	No actions required
Exclusions	A minimum of 48 hours symptom free/no loose stools
Microbiological	None required
clearance	
Case definitions:	
	non-cholera Vibrio spp. in faeces, blood or wound specimen.
Causative agent:	
Cause	Vibrio spp. excluding Vibrio cholera
Reservoir	Approximately 12 known pathogenic species. They are halophilic
	organisms, widely and naturally found within estuarine and
	marine waters.
	Vibrio vulnificus and Vibrio parahaemolyticus are the most
	commonly identified organisms.
Epidemiology	Greater number of cases occur during summer months
	associated with increased proliferation of Vibrio spp. during
	warmer weather.
Transmission	Consumption of raw or undercooked shellfish (particularly oysters
	harvested from warmer waters).
	Skin and wound infections may occur when wounds or soft tissues
	are exposed to warm seawater.
Incubation period	Most commonly 12-24 hours but extremes of 4-96 hours have
	been reported
Common clinical	V. parahaemolyticus:
features	- Watery diarrhoea, abdominal cramps, nausea, vomiting, fever,
	and (rarely) primary septicaemia. Wound and soft tissue
	infections are less common.
	- duration: 1-7 days (median 3 days) <i>V. vulnificus:</i>
	- diarrhoea, vomiting, abdominal pain
	- skin infection possibly leading to skin breakdown, ulceration,
	bullae, fever, septicaemia and death
	- Approximately 50% case fatality among cases with pre-existing
	immunocompromise who develop septicaemia.
	Persons with underlying medical conditions, such as alcoholism
	and chronic liver disease may be at increased risk of infection and
	serious complications.
Period of infectiousness	No evidence of person-to-person spread.
	Other people may have been exposed to the same risk factor(s).
Other relevant	In a suspected cluster/outbreak situation, obtain detailed travel,
information	activity and food history

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Further relevant guidance and key references:

Centers for Disease Control and Prevention: www.cdc.gov/vibrio/index.html US Food and Drug Administration (2012) Bad Bug Book - Foodborne Pathogenic Microorganisms and Natural Toxins Handbook. Second Edition Available at:

www.fda.gov/Food/FoodbornellInessContaminants/CausesOfIlInessBadBugBook

Worm infestation

Control of human source:	
Cases should be advised to	o follow usual enteric precautions.
Public health follow-up	NO
required	
Cases	Clinical treatment as appropriate
	Enteric precautions: in particular good personal hygiene including
	keeping fingernails short and regular changing of clothes, bedding
	and towels
	Supervised handwashing of children
Contacts	Screen symptomatic contacts
	Some infections require coordinated treatment for all household
	contacts (e.g. Enterobius vermicularis)
Exclusions	None required
Microbiological	None required
clearance	
Case definitions:	
	arasite by microscopic examination of stool or other recognised
laboratory methods	
Causative agent:	
Cause	Nematodes:
	Roundworm (Ascaris lumbricoides, Strongyloides stercoralis and
	Toxocara canis)
	Whipworm (Trichuris trichiura)
	Threadworm (Enterobius vermicularis)
	Hookworm (Ancylostoma duodenale and Necator americanus)
	Cestodes:
	Tapeworm (Taenia solium and T. saginata, Diphyllobothrium
	spp., Echinococcus granulosus, Hymenolepsis nana)
	Trematodes:
	Flukes (Schistosoma species, Paragonimus westermani,
	Fasciolopsis. buski, etc.)
Reservoir	Variable
Epidemiology	Variable
	Prevalent in areas with poor sanitation and food safety systems
Transmission	Variable; often direct person-to-person spread.
	Others are transmitted via contaminated foodborne sources
Incubation period	Variable
Common clinical	Cases may be asymptomatic or symptomatic
features	Typical symptoms include abdominal pain, diarrhoea, loss of
	appetite and passing worms in faeces
Period of infectiousness	Variable
Further relevant guidance	
	Reintjes R, Weinberg J & Ekdahl K. (2012) Communicable
	Protection Handbook – Third Edition. Wiley-Blackwell.

US Food and Drug Administration (2012) *Bad Bug Book - Foodborne Pathogenic Microorganisms and Natural Toxins Handbook.* Second Edition Available at: www.fda.gov/Food/FoodbornellInessContaminants/CausesOfIIInessBadBugBook

Yersiniosis/ Yersinia enterocolitica (and Yersinia pseudotuberculosis)

Control of human source	
	follow usual enteric precautions. Specific advice on exclusion from
nursery, school or work set	
-	NO – unless in an outbreak situation
required	
Cases	Obtain food and other potential risk factor history
	Enteric precautions
Contacts	Clinical surveillance
Exclusions	Case: A minimum of 48 hours symptom free/no loose stools
	Symptomatic contacts in risk groups: A minimum of 48 hours after
	diarrhoea has stopped
Microbiological	None required
clearance	
Case definitions:	
	laboratory identification of Yersinia spp. in a stool specimen.
Causative agent:	
Cause	Yersinia enterocolitica and Yersinia pseudotuberculosis
Reservoir	Asymptomatic carriage in the gastrointestinal tract of wild and
Reservon	domesticated animals and birds, particularly pigs for Y.
	enterocolitica
Enidomiology	
Epidemiology	Most commonly seen in those less than 15 years of age.
	Seasonal variation occurs - Y. pseudotuberculosis is more
	common in winter and Y. enterocolitica is more common from
	June to November
Transmission	Faecal-oral:
	Consumption of contaminated food or water, particularly pork or
	pork products. A wide variety of foodstuffs have been implicated
	in cases and outbreaks.
	Person-to-person: particularly within nurseries, schools and
	healthcare settings
	Direct contact with animals
	Via contaminated blood products
Incubation period	Y. enterocolitica: usually 3-7 days with extremes of 1-12 days
	reported
	Y. pseudotuberculosis: range of 2-25 days reported (median 5-8
	days)
Common clinical	Y. enterocolitica:
features	- watery diarrhoea, abdominal pain, fever
	- duration: 2 days to 6 weeks
	- complications: reactive arthritis, erythema nodosum,
	septicaemia
	Y. pseudotuberculosis:
	- mesenteric adenitis, fever, abdominal pain often mimicking
	appendicitis
	- duration: 1-37 days (average 18 days)
	- complications: reactive arthritis, erythema nodosum, acute renal
	failure

Period of infectiousness	Excretion of the organism in stool may persist for several months after infection but infectivity decreases substantially after the first 4 days or so
Further relevant guidance and key references:	
Hawker J, Begg N, Blair I, Reintjes R, Weinberg J & Ekdahl K. (2012) Communicable Disease Control and Health Protection Handbook – Third Edition. Wiley-Blackwell. US Food and Drug Administration (2012) Bad Bug Book - Foodborne Pathogenic Microorganisms and Natural Toxins Handbook. Second Edition Available at: www.fda.gov/Food/FoodbornellInessContaminants/CausesOfIlInessBadBugBook	
Membership of the guidelines working group

Main authors

Toyin Ejidokun (Consultant in Communicable Disease Control, PHE) **(Joint Chair)** Jeremy Hawker (Consultant Epidemiologist, PHE) Lorraine Lighton (Consultant in Communicable Disease Control, PHE- Retired) **(Joint Chair)** Karthik Paranthaman (Consultant in Communicable Disease Control, PHE) Rhianwen Stiff (Consultant in Communicable Disease Control, Public Health Wales)

Gemma Ward (Specialty Registrar in Public Health, PHE)

Additional core members of the guidelines working group

Bob Adak (Head of Gastrointestinal Disease Surveillance, NIS PHE) Neil Anstey (Health Protection Practitioner, PHE) Matthieu Pegorie (Consultant in Communicable Disease Control, PHE) Ian Gray (The Chartered Institute of Environmental Health (retired))

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Kate McPhedran (Senior Health Protection Practitioner, PHE) Juli Treacey (Health Protection Practitioner, PHE)

Exclusions

Tony Lewis (Head of Policy, The Chartered Institute of Environmental Health) Bernadette Nazareth (Consultant in Communicable Disease Control, PHE) Deborah Wilson (Consultant in Communicable Disease Control, PHE) Kevin Caroll (Consultant in Communicable Disease Control, PHE) Roger Gajraj (Consultant in Communicable Disease Control, PHE) Lisa Harvey-Vince (Senior Health Protection Practitioner, PHE)

Amoebiasis

Girija Dabke (Consultant in Communicable Disease Control, PHE) Lisa Harvey-Vince (Senior Health Protection Practitioner, PHE) Claire Alexander (Consultant Clinical Scientist and Honorary Clinical Senior Lecturer, Scottish Microbiology Reference Laboratories)

Bacillus

Jeremy Hawker (Consultant Epidemiologist, PHE) Jim McLauchlin (Lead Public Health Microbiologist, PHE) Karthik Paranthanam (Consultant in Communicable Disease Control, PHE)

Botulism

Kathie Grant (Head of Gastrointestinal Bacteria Reference Unit, PHE) Rohini Manuel (Consultant Clinical Microbiologist, NIS PHE)

Campylobacteriosis

Jeremy Hawker (Consultant Epidemiologist, PHE) Rohini Manuel (Consultant Clinical Microbiologist, PHE)

Cholera

Kathie Grant (Head of Gastrointestinal Bacteria Reference Unit, PHE) Joanne Edge (Senior Scientific officer, Food Standards Agency) Gauri Godbole (Consultant Medical Microbiologist and Parasitologist, NIS, PHE) Girija Dabke (Consultant in Communicable Disease Control, PHE)

Clostridium botulinum

Jim McLauchlin (Lead Public Health Microbiologist, PHE) Rohini Manuel (Consultant Clinical Microbiologist, PHE) Kathie Grant (Head of Gastrointestinal Bacteria Reference Unit, PHE) Gauri Godbole (Consultant Medical Microbiologist and Parasitologist, NIS, PHE) Joanne Edge (Senior Scientific officer, Food Standards Agency)

Clostridium difficile infection

Rohini Manuel (Consultant Clinical Microbiologist, NIS PHE)

Gauri Godbole (Consultant Medical Microbiologist and Parasitologist, NIS, PHE) Kathie Grant (Head of Gastrointestinal Bacteria Reference Unit, PHE)

Clostridium perfringens food poisoning

Kathie Grant (Head of Gastrointestinal Bacteria Reference Unit, PHE) Rohini Manuel (Consultant Clinical Microbiologist, NIS PHE) Gauri Godbole (Consultant Medical Microbiologist and Parasitologist, NIS, PHE) Jim McLauchlin (Lead Public Health Microbiologist, PHE)

Cryptosporidiosis

Rachel Chalmers (Consultant in Communicable Disease Control, Public Health Wales) Jim McLauchlin (Lead Public Health Microbiologist, PHE) Emma Crawley-Bovey (Consultant in Communicable Disease Control, PHE)

Cyclosporiasis

Gauri Godbole (Consultant Medical Microbiologist and Parasitologist, NIS, PHE) Karthik Paranthanam (Consultant in Communicable Disease Control, PHE) Kathie Grant (Head, Gastrointestinal Bacteria Reference unit, PHE) Jim McLauchlin (Lead Public Health Microbiologist, FWE Microbiology Services, PHE)

Enteric fever: typhoid and paratyphoid fevers

Members of the PHE Enteric Fever guidelines working group Sooria Balasegaram (Consultant Epidemiologist, PHE) Joanne Freedman (Senior Scientist, Travel and Migrant Health section, PHE) Katherine Russell (Consultant Epidemiologist, PHE) Kathie Grant (Head, Gastrointestinal Bacteria Reference unit, PHE)

Giardia

Jim McLauchlin (Lead Public Health Microbiologist, FWE Microbiology Services, PHE) Jeremy Hawker (Consultant Epidemiologist, PHE) Gauri Godbole (Consultant Microbiologist and Parasitologist, NIS, PHE) Claire Alexander (Consultant Clinical Scientist and Honorary Clinical Senior Lecturer, Scottish Microbiology Reference Laboratories)

Hepatitis A infection

Members of the PHE Hepatitis A guidelines working group Grainne Nixon (Nurse consultant, PHE) Sema Mandal (Consultant epidemiologist, PHE) Koye Balogun (Clinical Scientist, PHE) Richard Tedder (Head Joint NHSBT/PHE Blood Borne Virus Unit/Consultant Virologist, PHE) Smita Kapadia (Consultant in Communicable Disease Control, PHE) Jim McLauchlin (Lead, Public Health Microbiologist, FWE Microbiology services, PHE)

Hepatitis E infection

Members of the PHE Hepatitis E guidelines working group Miranda Mindlin (Consultant in Health Protection, PHE) Grainne Nixon (Nurse consultant, PHE) Smita Kapadia (Consultant in Communicable Disease Control, PHE) Koye Balogun (Clinical Scientist, PHE) Richard Tedder (Head, Joint NHSBT/PHE Blood Borne Virus Unit/Consultant Virologist, PHE) Sema Mandal (Consultant epidemiologist, PHE) Jim McLauchlin (Lead Public Health Microbiologist, FWE Microbiology Services, PHE)

Histamine poisoning

Jim McLauchlin (Lead Public Health Microbiologist, FWE Microbiology Services, PHE) Neil Anstey (Health Protection practitioner, PHE)

Listeriosis

Kathie Grant (Head of Gastrointestinal Bacteria Reference Unit, PHE) Jim McLauchlin (Lead Public Health Microbiologist, FWE Microbiology services, PHE) Karthik Paranthanam (Consultant in Communicable Disease Control, PHE) Jeremy Hawker (Consultant Epidemiologist, PHE) Matthieu Pegorie (Consultant in Communicable Disease Control, PHE)

Marine algal shellfish poisoning syndromes and ciguatera poisoning

Jim McLauchin (Lead Public Health Microbiologist, FWE Microbiology Services) Kathie Grant (Head, Gastrointestinal Disease Bacteria Reference unit, PHE)

Norovirus gastroenteritis

Kate McPhedran (Senior Health Protection Practitioner, PHE Juli Treacy (Health Protection Practitioner, PHE) Marie Chattaway (Pathogen lead for Salmonella Services, Gastrointestinal Bacteria Reference unit, PHE)

Rotavirus infection

Natalie Adams (Epidemiologist, Gastrointestinal, Emerging and Zoonoses Infections, PHE) David James Allan (Unit Head, Enteric Virus Unit, Virus Reference Department)

Sapovirus gastroenteritis

Girija Dabke (Consultant in Communicable Disease Control, PHE) Jeremy Hawker (Consultant Epidemiologist, PHE)

Shiga toxin producing Escherichia coli gastroenteritis (STEC), HUS

Members of the PHE STEC guidelines working group Lisa Harvey-Vince (Senior Health Protection Practitioner, PHE) Neil Anstey (Health Protection Practitioner, PHE) Kathie Grant (Head, Gastrointestinal Bacteria Reference unit, PHE) Jim McLauchlin (Lead Public Health microbiologist, FWE Microbiology Services, PHE)

Shigellosis

Bernadette Nazareth (Consultant in Communicable Disease Control, PHE) Lisa Harvey-Vince (Senior Health Protection Practitioner, PHE)

Vibriosis

Jeremy Hawker (Consultant epidemiologist, PHE) Kathie Grant (Head, Gastrointestinal Bacteria Reference unit, PHE) Jim McLauchlin (Lead, Public Health Microbiologist, FWE Microbiology Services, PHE)

Worms

Karthik Paranthanam (Consultant in Communicable Disease Control, PHE) Gauri Godbole (Consultant Medical Microbiologist and Parasitologist, NIS, PHE)

Yersiniosis

Jeremy Hawker (Consultant Epidemiologist, PHE) Marie Chattaway (Pathogen lead for Salmonella services, Gastrointestinal bacteria reference Services, PHE) Jim McLauchlin (Lead, Public Health Microbiologist, FWE Microbiology Services, PHE)

Guidelines as a whole

Deborah Wilson (Consultant in Communicable Disease Control, PHE) Tony Lewis (Head of Policy, The Chartered Institute of Environmental Health) Caroline Willis (Unit Head, Porton Food Water and Environmental microbiology lab) Rohini Manuel (Consultant Medical Microbiologist, PHE) Karthik Paranthanam (Consultant in Communicable Disease Control, PHE) Joanne Edge (Senior Scientific officer, Microbiological risk assessment, Food Standards Agency) Philip Randles (Head of Incident branch and members of the microbiological risk assessment branch, Food Standards Agency) Bernadette Nazareth (Consultant in Communicable Disease Control, PHE) Kevin Caroll (Consultant in Communicable Disease Control, PHE) Roger Gajraj (Consultant in Communicable Disease Control, PHE) Lisa Harvey-Vince (Senior Health Protection practitioner, PHE) Neil Anstey (Health Protection practitioner, PHE)

Abbreviations

Anti HAV IgM	Hepatitis A virus antibodies IgM
CDC	Centre for Disease Control
CSF	Cerebro Spinal Fluid
DNA	Deoxy Ribonucleic Acid
EIA	Enzyme Immuno Assay
FSA	Food Standards Agency
GDH	Glucose Dehydrogenase
HAV	Hepatitis A Virus
HDC	Histidine Decarboxylase
HEV	Hepatitis E Virus
HPA	Health Protection Agency
HUS	Haemolytic Uraemic Syndrome
lgG	Immunoglobulin G
IgM	Immunoglobulin M
ipaH	Invasion plasmid antigen H (gene found in Shigella and some enteroinvasive E.coli)
MSM	Men who have sex with Men
NAAT	Nucleic Acid Amplification Test
NBTS	National Blood Transfusion Service
NHS	National Health Service
NIS	National Infection Service
PCR	Polymerase Chain Reaction
PHE	Public Health England
PPV	Positive Predictive Value

RNA	Ribo Nucleic Acid
RCGP	Royal College of General Practitioners
STEC	Shiga Toxin producing Escherichia Coli
TTP	Thrombotic Thrombocytopenic Purpura
UK	United Kingdom
US	United States

Appendix I – Notifiable diseases and links to law

www.gov.uk/notifiable-diseases-and-causative-organisms-how-to-report

The Health Protection (Notification) Regulations 2010. Available at: www.legislation.gov.uk/uksi/2010/659/contents/made

The Health Protection (Notification) (Wales) Regulations 2010. Available at: www.legislation.gov.uk/wsi/2010/1546/made

The Health Protection (Local Authority Powers) Regulations 2010. Available at: www.legislation.gov.uk/uksi/2010/657/contents/made

The Health Protection (Local Authority Powers) (Wales) Regulations 2010. Available at: www.legislation.gov.uk/wsi/2010/1545/contents/made

The Health Protection (Part 2A Orders) Regulations 2010. Available at: www.legislation.gov.uk/uksi/2010/658/contents/made

The Health Protection (Part 2A Orders) (Wales) Regulations 2010. Available at: www.legislation.gov.uk/wsi/2010/1544/contents/made

Health Protection legislation guidance 2010. Available at: webarchive.nationalarchives.gov.uk/20130107105354/http://www.dh.gov.uk/en/Publicationsand statistics/Publications/PublicationsPolicyAndGuidance/DH_114510

Health Protection Regulations 2010 Toolkit. Available at: www.cieh.org/policy/health-protection-regulations-toolkit.html

NI Legislation is: Public Health Act (Northern Ireland) 1967 www.legislation.gov.uk/apni/1967/36

Appendix II – Information leaflet – Minimising the spread of gastrointestinal infection

This is particula	ted to stay away from work to help reduce the risk of spreading gastrointestinal illness to other people. In important if you work with food or people who are particularly vulnerable. The asked to stay away from crèche, nursery, school or other social activities to help reduce the spread of
Personal	Hand washing is the single most important method of preventing and controlling the spread of infection
hygiene	 Hands should be washed thoroughly with warm running water and soap: Deferse pating
	 Before eating Before handling, preparing or serving food
	3. After visiting the toilet
	4. After attending to any person who has diarrhoea or vomiting
	5. After changing a baby's nappy
	 After handling or washing soiled clothing or bedding or after cleaning the toilet or child's potty After handling pets, including reptiles, or non-domestic animals
	Dry hands thoroughly after every wash using disposable paper towels, or ensure that each person has their own towel
	Hand washing should be supervised for young children and other people for who, hand washing may be difficult
	 Do not share towels with someone who has diarrhoea or vomiting
	• Do not share, or allow children to share, a bath with someone with diarrhoea or vomiting
	• Where possible, avoid close contact, including sexual contact, with someone with diarrhoea or vomiting
	Avoid preparing or handling food for other people until symptoms have resolved for at least 48 hours
Environmental	Toilet and Bathroom areas
cleaning	Clean hard surfaces at least daily (or more frequently dependent on use) with separate disposable cloth using hot water and diluted bleach solution

	 Pay particular attention to potentially contaminated surfaces such as the toilet bowl and seat (surface and underneath), taps, flush handle (and surrounding area), and door handles Spillages Deal with any spillage or contamination with faeces, vomit or urine immediately Absorbent material such as paper towels, tissues, or sawdust may be used to limit the spread of liquid soiling and can be disposed of afterwards Cleaning the soiled area with hot water and detergent is usually adequate Always rinse with clean water and allow to dry before using the area again After clearing a spillage from carpet, ideally use a proprietary carpet shampoo or steam cleaner to further clean the area
	 Soiled linen or clothing Before washing, carefully remove as much solid material as possible into the toilet bowl and flush away Wash separately in the washing machine, using a pre-wash if possible, and on the hottest temperature possible for the fabric Use a biological washing powder Do not use the half wash button or the rapid wash function and do not overload the washing machine Wipe down the outside surface of the washing machine after loading using a disposable cloth and hot soapy water If heavily soiled items have been washed, consider running an empty hot (90°C) cycle before washing other items
	 General: Do not clean any soiled items in areas where food is prepared (e.g. do not use a domestic kitchen sink to clean soiled garments) Ideally, wear disposable gloves and use disposable cloths/mop-heads whilst cleaning
Disposal of soiled materials	 Always wash hands thoroughly after cleaning is completed, including when gloves have been worn Wherever possible, cases should use a toilet If bedpans, commodes or urinals are required, empty these into the toilet bowl, wash the vessel with hot
	 In bedpans, commodes of unnais are required, empty these into the tonet bowl, wash the vessel with not water and detergent, rinse and allow to dry Ideally, use disposable plastic aprons when dealing with diarrhoea or vomit and soiled materials

• Used gloves, aprons, cleaning cloths, mop-heads etc. may be disposed of by placing them in a plastic bag,
sealing the neck and placing with household refuse
• If rubber gloves or non-disposable cloths are used, thoroughly wash in hot water and detergent after use,
rinse and allow drying. Ideally, these should be disposed of at the end of the episode of illness

Appendix III – Stool sample collection instructions leaflet

http://patient.info/health/poo-stool-sample