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Appendix Table. Evidence for seasonal associations between factors linked to human *Campylobacter* infections or outbreaks

Risk factor	Outbreaks	Evidence for factor causing seasonal increase	Evidence against factor causing seasonal increase
Chicken/turkey	(1–7)	Chicken is the food most commonly contaminated with *Campylobacter.* A substantial portion of infection probably derives from this source (1–6,8–10). Some evidence shows that *Campylobacter* contamination of chickens is seasonal.	Chicken is not the vehicle for most sporadic *Campylobacter* infections (8,11,12). Little evidence exists that the seasonal differences in *Campylobacter* in chickens are sufficient to drive the seasonality of human disease (13–18).
Salads and fruit	(19–21)	Untreated leaf salads and soft fruits might be potential sources of human campylobacteriosis (9,19–21) because these raw products are eaten without any heat treatment.	In most of the outbreaks involving salad items, cross-contamination from contaminated raw foods was thought to be involved. While seasonal import of fresh fruit or vegetables from different countries might represent a potential source of infection it would be surprising if this manifested itself as an annual nationwide outbreak across the whole of England and Wales while remaining refractory to epidemiologic investigation. Fly transmission from animal feces may be important.
Cross-contamination from raw meats to ready-to-eat foods	(9)	Cross-contamination from raw meats to ready to eat foods within kitchens and retail premises probably contributes significantly to *Campylobacter* infection.	Why cross-contamination should be strongly influenced by the season is unclear, unless levels of raw meat contamination change with the seasons.
Unpasteurized or inadequately pasteurized milk	(6,22–33)	Unpasteurized or badly pasteurized milk can be a source of *Campylobacter* infection (6,23,26,29,33–36). Milk could cause the seasonality if the numbers of *Campylobacter* in raw milk changed with the season and other critical control points in milk production (pasteurization) are not tightly maintained. Infections related to consumption of unpasteurized milk appear to be seasonal, with a peak in May, which suggests seasonal changes in the *Campylobacter* contamination of unpasteurized milk.	No evidence shows that the seasonality of human disease is largely due to unpasteurized milk because this product is not commonly consumed. No evidence shows that pasteurization varies substantially by season.
Birds	(37,38)	*Campylobacter* is common in birds. Migratory birds result in large seasonal changes in the inputs to the environment from bird feces and could contribute to human *Campylobacter* exposure (39). Migratory birds could be a seasonally changing driver to human disease (40). The main likely exposure route if this were the case would be direct contact with contaminated bird feces in the garden, contamination of field-grown fruit and vegetables and contamination of source waters for drinking. Bird-pecked milk is a recognized route by which *Campylobacter* infection can be acquired (37,38). The contamination is thought to result from birds feeding consecutively on cow feces and milk in bottles. The infections related to bird-pecked milk appear to be seasonal in distribution with a marked increase in May (41).	Bird-pecked milk is unlikely to be the cause of the worldwide seasonal distribution of *Campylobacter* infections. Fly transmission from bird feces, particularly farmed poultry, may be important. Evidence from extensive monitoring of ready-to-eat foods sampled at retail businesses suggests little evidence of *Campylobacter* contamination (Little, pers. comm.).
Barbecue	(1)	Barbecue use might be a contributing factor to the total *Campylobacter* infection because standards of food safety associated with barbecue use are likely to be poorer (1,42,43). Case-control studies have found associations between barbecue use and sporadic *Campylobacter* infection (44,45).	Barbecue use on its own is unlikely a big enough, or seasonal enough, driver of disease to account for seasonal changes in incidence.

Risk factor	Outbreaks	Evidence for factor causing seasonal increase	Evidence against factor causing seasonal increase
Food packaging		The packaging around chickens is commonly contaminated with *Campylobacter*, which may represent a source of some infections through cross-contamination.	Strong seasonal changes in the extent of this contamination would have to exist for this factor to affect the disease epidemiology, and no evidence for these changes exists.
Food handlers/hygiene	(46–50)	Infected food handlers might represent a source of infection in catering premises.	Infections in food handlers probably are seasonal, reflecting the seasonality of *Campylobacter* in general, but they are probably not the driver for the overall seasonality.
Food, stir-fried	(2)	Stir-fried food may be contaminated through inadequately cooking raw ingredients or cross-contamination.	A seasonal change in the contamination of raw ingredients would need to exist to explain the epidemiology.
Flies		Flies provide a biological explanation for the spring increase in *Campylobacter* cases through the increase in fly numbers. *Campylobacter* has been isolated from flies, and the low infectious dose required to cause human disease would make this route credible. Historical records link “summer diarrhea” to flies.	Little hard evidence exists for this transmission route.
Mains drinking water	(28,51–60)		With mains water supplies, the relatively even distribution of seasonal changes in the distribution of *Campylobacter* cases suggests that any contamination of public supplies must be systemic (a generic problem with all supplies) or a much bigger regional difference in the incidence would be seen. Potential seasonal differences in water quality that could explain why treatment might not prevent sporadic *Campylobacter* infection through mains water (e.g., viable noncultivable *Campylobacter* in chlorine-resistant protozoa) are not supported by evidence. The rarity of outbreaks associated with public water supplies suggests that drinking water is not a substantial source of *Campylobacter* infection.
Private drinking water supplies/untreated surface water, rain water, or well water	(6,59;61–70)	Waterborne infection associated with private water supplies can result in outbreaks of infection because many people drink the contaminated water (71). *Campylobacter* is the most common organism causing these outbreaks. A seasonal change in water quality could occur.	Seasonal changes in water contamination should trigger outbreaks rather than a national increase in sporadic disease. The comparative rarity of outbreaks associated with private supplies suggests that this source does not substantially contribute to the total illness that is seen to change dramatically with the season. Given the influence of surface water on the microbiologic quality of private water supplies, we expect that the seasonal occurrence of *Campylobacter* might be more influenced by rainfall than time of year, which does not appear to happen.
Bottled water		In a case-case study of *Campylobacter*, people with *C. coli* infection were more likely to have drunk bottled water than were those with *C. jejuni* infection (72). Natural mineral water is not disinfected and could be a widely dispersed product that experiences seasonal changes in contamination.	Sources of water that are used to produce natural mineral water and other bottled waters are relatively well protected. These groundwaters are unlikely to be contaminated with *Campylobacter*. If bottled water consumption is a risk factor, it should come up as such in analytic epidemiologic studies of *Campylobacter* infection. It is unclear why the seasonal pattern of infection should be so constant both geographically and annually if bottled water contamination is such a substantial contributor to human disease.
Pools, lakes, and streams		Potential exists for illness after swallowing contaminated recreational water (73–76). Water sports in natural waters can be a source of exposure. If the contamination of water with *Campylobacter* is seasonal, then any seasonality in this group could be linked to either changes in water quality or behavior.	Illness associated with recreational water activity has not been established, and this is unlikely to be the source of the spring increase in campylobacteriosis. Little evidence shows that the change in recreational water activity in the spring is enough to explain the seasonal change in *Campylobacter* cases.

Risk factor	Outbreaks	Evidence for factor causing seasonal increase	Evidence against factor causing seasonal increase
Within-family transmission	(77)	Person-to-person transmission can occur.	No obvious reason explains why within-household transmission of *Campylobacter* should be seasonal, given that personal hygiene practices are not likely to change substantially over a matter of weeks.
Domestic catering		Domestic food preparation may contribute to human *Campylobacter* disease.	Fly transmission within kitchens may contribute to transmission, and this would likely be seasonal. Little else within the kitchen environment, other than the contamination of raw food ingredients, is likely to vary seasonally.
Nursery/childcare/school	(78,79)	As *Campylobacter* is common in children, transmission may occur within the childcare setting.	No evidence shows that infections in childcare are common or that they vary through the year.
Nosocomial transmission	(80)		Nosocomial transmission cannot account for the national seasonal increase in cases.
Pets		Pets, particularly kittens and puppies, have been postulated as a source of *Campylobacter*. Canine births, as recorded in Kennel Club and Guide Dogs for the Blind Association records, show a strong seasonal distribution, and this factor has been proposed as a driver for human disease (81).	Little evidence shows that the seasonal change in *Campylobacter* is directly related to pets, although fly transmission from animal feces may be important.
Farm animals	(82)	*Campylobacter* strains isolated from cattle have been linked to strains from human infections (83,84). Cattle and sheep represent a reservoir of *Campylobacter* (85,86), and milkborne outbreaks (6,23,26,29,33–36) suggest that other routes may occur. Fecal shedding by sheep may be more frequent around lambing (87). Seasonal differences in *Campylobacter* infections have also been demonstrated in rhesus monkeys, other agricultural animals, and birds (15,16,88–91).	Any seasonality of *Campylobacter* infection or colonization in animals could cause seasonality in humans, but this seasonality is most likely to result from the contamination of food. Fly transmission from animal feces may be important.
Farm visits	(92)	Visits to farms can expose children to common zoonotic enteric pathogens, including *Campylobacter*.	Any seasonality of farm visits is unlikely to contribute to the seasonal distribution of all cases.
The countryside		Direct environmental exposure could occur through walking in the country.	This activity may be seasonal but is unlikely to contribute to the strong seasonal distribution of cases.
Travel		*Campylobacter* has been linked to overseas travel (93–95), including military service (96,97), and probably represents a significant percentage of all cases of travelers' diarrhea (98–101). In some countries, >50% of *Campylobacter* cases may be linked to foreign travel (102)	The seasonality of *Campylobacter* does not follow the seasonality of travel abroad.
Weather/climate		In some developing countries a higher incidence was seen in the rainy season (103,104), which suggests flies might be contributory. Although *Campylobacter* is more common during the summer months and has been linked to temperature (105), no direct relationship was seen between temperature and cases of human disease. The different seasonal distribution in different countries appears to be partly temperature-related	Little evidence shows that *Campylobacter* is associated with rainfall. There was no association between thermophilic *Campylobacter* in lambs at slaughter and rainfall (89). The main seasonal driver for *Campylobacter* infection is not likely to be rainfall itself, since the increase appears to occur annually, irrespective of when most rain falls.
Immunologic response		The immunologic response to *Campylobacter* exposure could change throughout the year. This hypothesis has been studied in male rhesus monkeys (88). A marked seasonality was seen ,with the frequency of TH1-type cytokine synthesis in the summer being markedly greater	Current evidence suggests that seasonal changes in immunologic response to *Campylobacter* infection are unlikely to account for the major seasonal changes in *Campylobacter* incidence.

Risk factor	Outbreaks	Evidence for factor causing seasonal increase	Evidence against factor causing seasonal increase
		than in the winter, whereas TH2-type cytokine expression did not vary between the seasons.	

Appendix References

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