



New Zealand should control *Campylobacter* in fresh poultry before worrying about flies

The possible role of flies in the aetiology of human campylobacteriosis in New Zealand was the focus of a recent *Journal* article by Nelson and Harris.¹ This is not a new hypothesis,² so we were surprised to see it being raised again without specific data to support it.

Such speculation contrasts with the well established role of contaminated food products (and particularly fresh poultry) as the major risk factor for campylobacteriosis in New Zealand as detailed in recent reviews.^{3 4} This evidence is based on New Zealand case-control studies of sporadic disease, one of which was a large multi-centre study.⁵

Other published and unpublished New Zealand epidemiological studies of sporadic campylobacteriosis, and of outbreaks, provide additional support for the importance of food-borne transmission.⁴ Further to the epidemiological evidence, serotyping has revealed strains common to cases and poultry from New Zealand stores (including those stores at which cases shopped),⁶⁻⁹ and links with sausage,¹⁰ sheep liver,¹¹ and sheep and beef offal.¹² However, such serotyping work may still be of somewhat limited value owing to the likely genetic instability of the *Campylobacter* genome (e.g. uptake of extracellular DNA and DNA recombination). Even when considering campylobacteriosis outbreaks alone, food dominates over water-borne transmission.⁴

None of the 13 published outbreak and 16 unpublished outbreak reports in New Zealand (that met the quality criteria for inclusion in a review) identified flies or fly-contaminated environmental surfaces as risk factors for campylobacteriosis.⁴

Nelson and Harris suggest cow faeces as the major environmental source with a flies-fomites-fingers link to humans. If this was the case, we would expect to see much higher rates of illness in rural areas. Such a pattern is not observed.¹³

There is no need to propose a role for flies to explain the elevated summer rates of campylobacteriosis in New Zealand.

Many other factors may be more important, including:

- The seasonal load of *Campylobacter* contamination in poultry flocks;
- Higher summer consumption of contaminated food used on barbeques and in salads (when it is not hygienically handled or thoroughly cooked); and
- Higher levels of contact with outdoor environments (including contaminated water) and with livestock.

However, the impact of the latter point may be minor, since notification and hospitalisation rates are highest in New Zealand cities,¹³ again consistent with the importance of food-borne transmission rather than from contaminated environments.

Nelson and Harris' article also failed to explain how flies could account for the huge rise in campylobacteriosis over the past 20 years. Instead, it presented data showing

how the rise in campylobacteriosis notifications appeared closely correlated with increased consumption of chicken, which is a far more plausible explanation.

Informed scientific debate is highly desirable, but the kind of unsubstantiated speculation contained in the Nelson and Harris article can have negative consequences for public health. Indeed, it can reinforce a public perception that the sources of human *Campylobacter* infection are highly speculative, that every surface in their home environment is potentially contaminated, and that this disease is virtually unstoppable. This 'miasma' viewpoint is paralysing and easily exploited by interest groups which seek to divert attention from potential interventions.

Given the research evidence detailed above, the emphasis in this country should continue to be on reducing the levels of *Campylobacter* contamination in the food supply (e.g. particularly on poultry farms, in poultry processing plants, and in poultry in the distribution system). In fact, the bulk of government-funded research into campylobacteriosis control appears to relate to the food-borne transmission pathway (see the New Zealand Food Safety Authority website: <http://www.nzfsa.govt.nz/>).

Studies on the possible role of flies might be justified in the distant future, but only once the major sources of campylobacteriosis have been successfully controlled and control programmes evaluated.

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Response

Eating raw or undercooked chicken and associated food contamination is not disputed as a risk for campylobacteriosis. Neither is suggesting another transmission route to be taken as justification for the chicken industry to make no attempt to reduce or eliminate *Campylobacter* contamination of their product. If we are to get on top of the problem though, we need to have an honest evaluation of facts.

We repeat, chicken production interruption in both Belgium (1999) and the Netherlands (2003) cut reported *Campylobacter* rates significantly, a calculated 40% reduction. That still leaves 60% with an unexplained source. Our flies/fomites/fingers hypothesis offers an alternative transmission route to explain a chicken-consumption link to illness, while at the same time also taking into account the many factors strongly discounting the source as being only and directly chicken.

Chicken as a direct source is heavily entrenched in the medical mindset. The “well established direct link” claimed is, at best, circumstantial. In its favour is the common knowledge that chicken meat is frequently contaminated, very low levels of organism constitute an infectious dose, and people who get sick have commonly eaten chicken recently. Chickens can provide some of the same strains as found in human cases, but this is not evidence of **direction** of transmission.

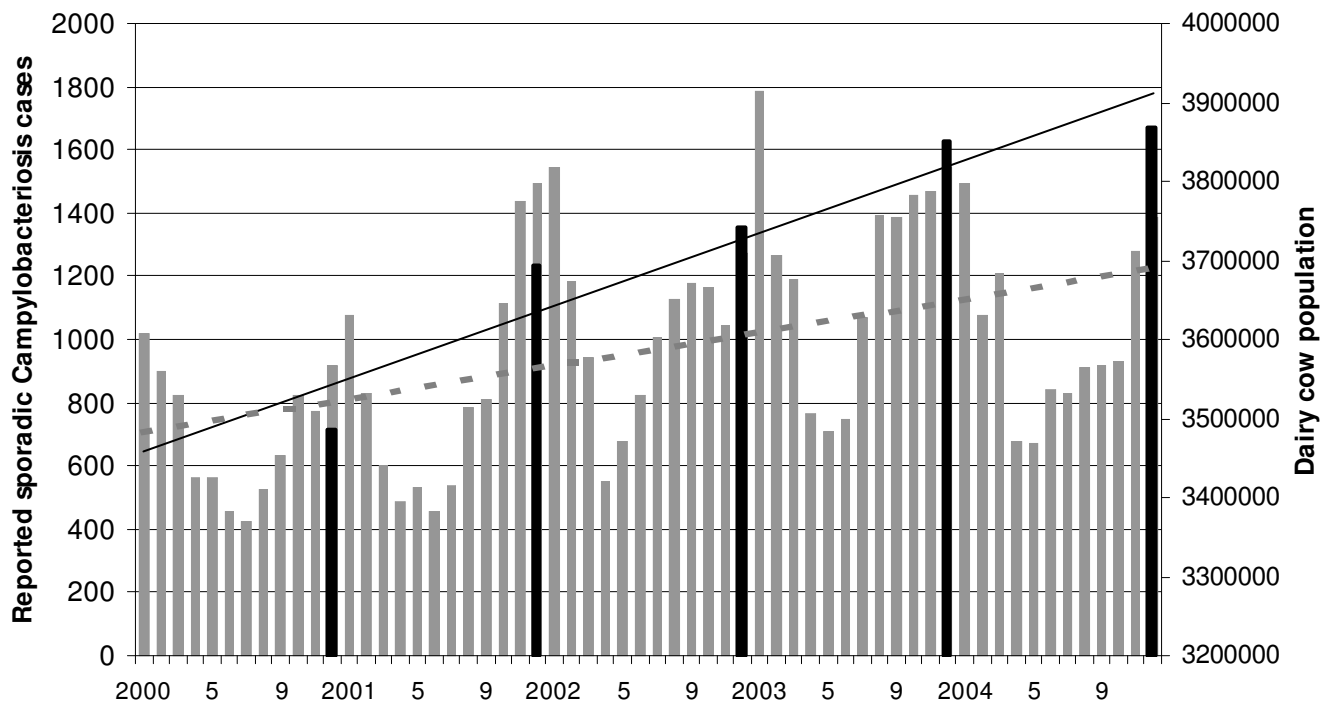
Outbreaks and their associated food or water-borne transmission routes represent about 10% of campylobacteriosis cases. Our paper focussed solely on the much more common **sporadic** cases. Food-borne causes of gastroenteritis frequently result in outbreaks of cases, not sporadic incidents as is usual with *Campylobacter*.

We made no claim to be first to involve flies in the transmission of *Campylobacter* to humans, nor for the idea that dairy cows are a significant source for *Campylobacter* in the environment. Prior credit is clearly indicated. We do however propose a fomite, finger addition to the transmission pathway.

The rural/urban aspect needs investigating, but no New Zealand city is far from a rural source of disease. Urban pets may yet prove a more common source than is currently fashionable to consider. An interesting coincidence to fomites and fingers is the hand to mouth suggestion recently postulated, although this retains chickens as source.¹

The marked increase in campylobacteriosis cases over the last 20 years was not part of our investigation. However, the increase in chicken consumption noted fits our hypothesis of food-associated transmission. The increase in dairy cow numbers also shows a somewhat similar trend (Figure 1). Far from being a paralysing viewpoint, washing hands before touching food is a key hygiene factor. We have merely tried to determine a plausible transmission route that also fits the known epidemiology and risk factors associated with eating chicken and sporadic campylobacteriosis.

Figure 1. *Campylobacter* data as before, with dairy cow numbers added



Data from Livestock Improvement Company (<http://www.lic.co.nz>). Least squares linear regression fitted.

The supposed genetic instability of *Campylobacter* is not supported by evidence. Specific subtypes are common from the Far East to Europe.² Genotype overlap

between humans and chickens has been reported as between 20%³ and 6%.⁴ Some virulence markers in *Campylobacter jejuni* show little commonality between humans and chickens.⁵ This should come as little surprise as a chicken flock is often colonised by a single or very few strains of *Campylobacter*.⁶

Flies being eaten or contaminating food are a known source of *Campylobacter* for chickens.^{7,8} Flies are therefore a highly plausible vector between other environmental sources and chickens, or between chicken houses. Thus cows are a plausible source of environmental *Campylobacter* for both chickens and humans. Flies represent a common transmission agent too, although direct consumption by humans of flies is rare, hence the indirect fomite/fingers suggestion.

Recent calls for banning the sale of fresh chicken meat in New Zealand based on our high rate of campylobacteriosis do not have experimental evidence on their side.⁹ The weekly pattern of disease in the UK supports a hypothesis of food-associated (but not necessarily food-borne) transmission.¹⁰ Unfortunately, we cannot repeat this here as New Zealand statistics are on a monthly basis.

With the bulk of research funding already going on a food-borne approach, yet with only rising rates over 20 years to show for it, perhaps a broader target is justified. *Campylobacter*-free chicken meat is a desirable aim, but this is likely to only target the outbreak (10%) side of the equation. Chicken-association appears to be about 40% of cases.

Wilson et al seem to have missed the point of our paper and the authors apologise for apparent obscurity of expression. We hope this response will also aid others who might have missed these points. However, we are delighted with the widespread uptake of our main points by the popular press as it is individual diners who control what they ingest from their own fingers.

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