

Interaction of pigs and chickens with their environment and zoonotic disease
 Tom Humphrey
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Production environments and host stress

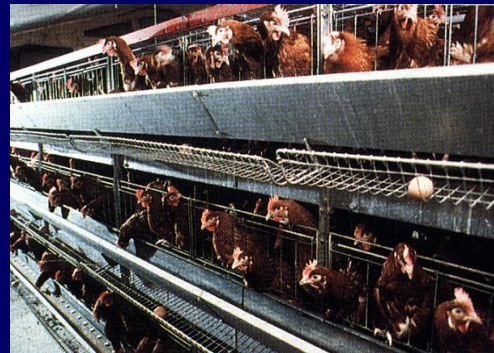
- Major physiological events
 - Chickens coming into lay
 - Birth
- Mixing of different and unnatural social groups
 - Chickens moved from rearing to laying
 - Pigs mixed, post-weaning
- Production-related conditions
 - Hock and pad burn in broilers
 - Transport and thinning
- Poor diets inflame gut mucosa
- Gut health and commensal flora compromised
- Is there evidence that food safety is compromised?



Increased media interest in welfare



Traditional cages for egg production will soon be illegal



Broiler production will still be legal and in EU, stocking densities can rise from 38 to 42 Kg/M²



Hypotheses to be explored in the talk

- Intensive production methods for pigs and chickens render the animals susceptible to infection
- In the "stressed" animal, pathogens like *Salmonella* and *Campylobacter* spp. are more likely to be invasive
- In broilers primary colonisation with *Campylobacter* is in "stressed" or "sick" birds
- Lowering broiler stocking may enhance food safety



Pig production, transport and salmonella



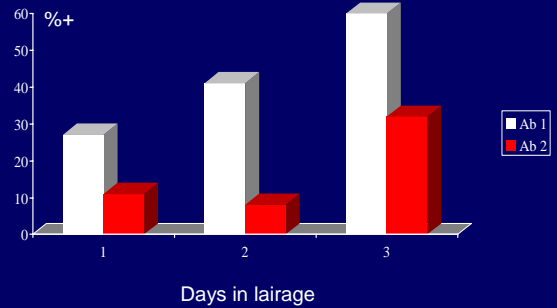
The "stressed" pig and pathogen excretion

- Salmonella levels in faeces higher after transport (Williams and Newell 1970)
- Weaning, mixing and cold stress in piglets increased shedding of enterotoxigenic *E. coli* (Jones 2001)
- A 48-h fast in pigs increased numbers of *C. jejuni* in gastrointestinal tract (Harvey 2001)
- Salmonella is more invasive in "stressed" pigs (Hurd *et al.* 2002)

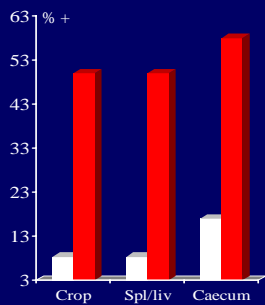


Production-related stress and salmonella

Holding time of pigs pre-slaughter and salmonella positivity



9-day feed withdrawal and SE in chickens

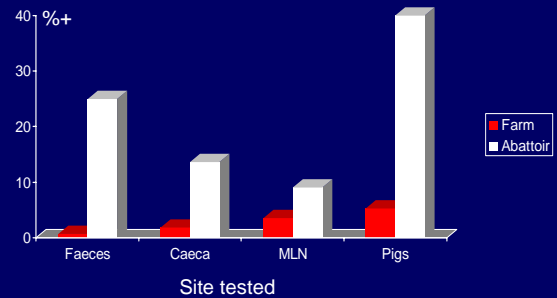


- SE levels increased at all sites
- Increased invasion from the gut
- Does changed behaviour reflect alterations in commensal flora?
- Does it reflect altered immunity
- Do host stress responses play a role?

Durant *et al.* 1999



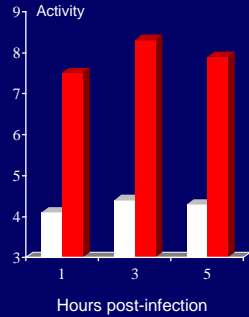
Transport and salmonella behaviour in pigs



Hurd *et al.* 2002



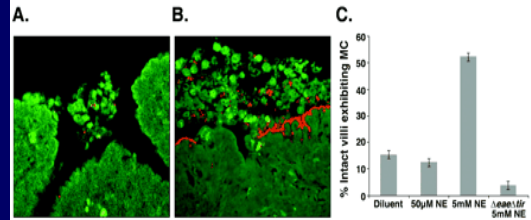
4-day feed withdrawal and *hilA* expression in SE



- SE inoculated into crop contents
- *hilA* is a global regulator involved in virulence
- Activity markedly higher in crops of starved animals
- Starvation will reduce LAB numbers
- Does this reflect a changed chemical environment?



Laser scanning micrographs of bovine ileal mucosa and *E. coli* O157:H7 with diluent (A) or 5 mM NE (B).



Vlissidou *et al.* 2004

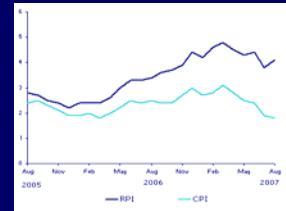


Broiler chickens and campylobacter

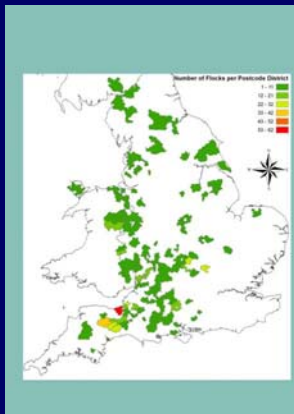


Chicken health and welfare and public health

- Government target is low inflation
- Low cost food is virtuous
- A whole chicken for ~£2
- Highly intensive production
- Poor environments
- Production-associated conditions
- Endemic/zoonotic disease
- Are chickens so dumb that environment doesn't matter?
- Do "unhappy" chickens pose a greater food safety risk than "happy" ones?
- If they do, what is the nature of the increased threat?



FSA-funded project: Factors affecting campylobacter colonisation in the absence of thinning



Outputs from an FSA-funded flock management project

- 789 housed flocks from 2003-2006
- Few housed flocks fully colonised, pre-thinning
- Significantly higher colonisation rates in summer
- Significant differences between farms
- Flocks significantly more likely to be campylobacter-positive if:
 - They have high hock/pad burn levels (p 0.009)
 - There are high levels of condemnation for infection (p 0.012; *E. coli*)
 - They are large
 - There is more than one house on the site



Main hypothesis: chicken production affects susceptibility to infection



Hypothesis is that host stress/immune responses modulate campylobacter

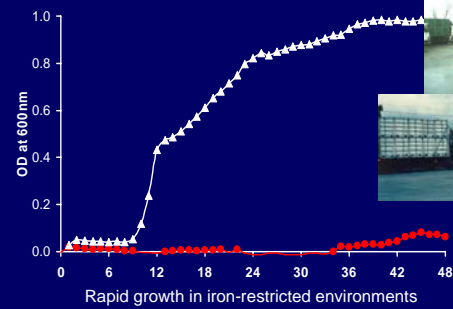
- Chickens may experience acute or chronic stress
 - Thinning
 - Over-crowding
 - Production-related conditions
 - Rapid growth
- Noradrenaline (NA) in chicken intestine increases during stress
- Inflammation of gut means that Interferon γ (IFN) also present in intestinal mucosa
- NA (pathogen) and IFN (host) act independently or synergistically to affect campylobacter colonisation



Host stress responses and campylobacter behaviour



Noradrenaline increases growth of *C. jejuni*



Cogan *et al* 2007

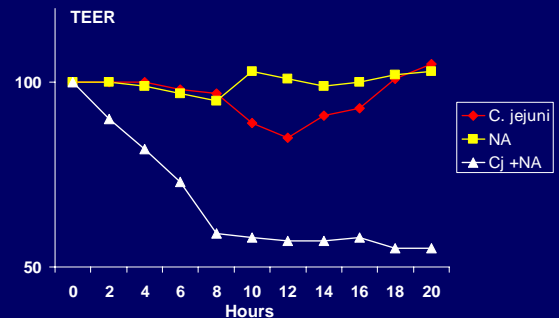


Crossing the epithelial barrier

- Up to 20% of retail chickens had campylobacter in deep muscle
- Is this due to invasion from the gut?
- Flock survey data suggested that primary infection may occur in areas of 'compromised' gut tissue
- Prompted work on campylobacter interaction with epithelial cells and effect of cytokines and neurotransmitters




NA-pre-treated *C. jejuni* rapidly damage tight junctions*



* As measured by a change in transepithelial electrical resistance

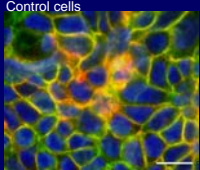



IFN γ
 interaction at the epithelium and in tissues



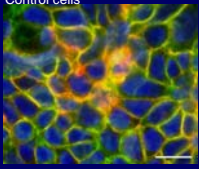
IFN and its effects on Caco2 and *C. jejuni*

Control cells

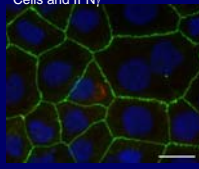




IFN and its effects on Caco2 and *C. jejuni*

Control cells

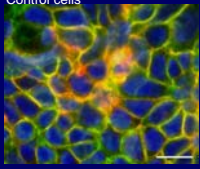


Cells and IFN γ

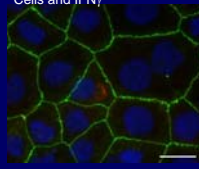



IFN and its effects on Caco2 and *C. jejuni*

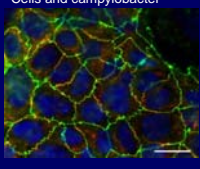

Control cells



Cells and IFN γ

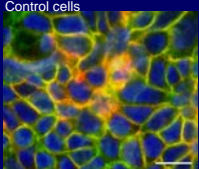


Cells and campylobacter

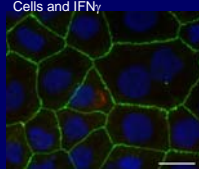



IFN and its effects on Caco2 and *C. jejuni*

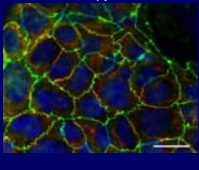
Control cells



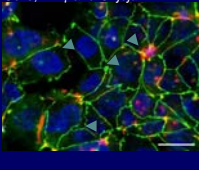

Cells and IFN γ



Cells and campylobacter



Cells, IFN γ and *C. jejuni*

Thank you to:



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