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Short Communication

The Zoonotic Crohn's Disease Pandemic an Infectious Disease Incorporated Perspective

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Isolated, rare reports of Crohn's disease had long existed. Morgagni wrote what maybe the first description of Crohn's disease in 1769. Braun in 1909 and Dalziel in 1913 describe cases that could have also been Crohn's disease. These cases had little traction in attracting critical focus.

Crohn's disease became a clinically defined entity in 1932. Crohn, Ginzbury and Oppenhiemer described a disease of the terminal ileum affecting primarily young adults. Central to their observations is the fact that the terminal ileum was the primary initial location of disease.

Over a nine-decade period, the natural history of Crohn's disease has emerged.

- Crohn's disease affects the gastrointestinal tract.
- Once it was a rare disease that has become progressively more common among industrialized nations.
- The initial site of disease is the end portion of the small intestines called the ileocecum.
- The disease strikes primarily teenagers and young adults.
- In the course of the disease, fistulous tracts into the cul-de-sac, adjacent bowel, bowel perforation, and strictures may occur.
- An estimated quarter of afflicted individuals stand to have one or more operations to remove diseased small bowel.
- It is still rare with economically disadvantaged subpopulations.
- Breastfeeding confers apparent immunity against the baby subsequently developing Crohn's disease later in life.,
- Drugs (selected antimetabolites, steroids, biologics) that interfere with the body's immune system's ability to respond to being challenge may produce temporary amelioration of the signs and symptom of the disease.
- An estimated one-third of afflicted individuals will either leave the workforce in their prime reproductive years or modify their employment to accommodate their disease.
- The direct costs of Crohn's disease exceed 15 billion dollars. Its indirect costs are calculated at 25% of direct medical costs.
- It is an immune-mediate disease Crohn's disease which is the product of two separate immune system interactions involving *Mycobacterium avium* subspecies *paratuberculosis* (MAP).

What it not identified is how a limited veterinary problem transformed itself into a global pandemic affecting an estimated four million individuals. Disease creation has zoonotic roots.

Mycobacterium Avium subspecies Paratuberculosis

Mycobacterium avium subspecies *paratuberculosis* (MAP) is a significant pathogen for herbivores. MAP produces a chronic

granulomatous infection of the gastrointestinal tract (Johne's disease) in herbivores. In beef cattle, MAP infection results in lower cow fertility, lower calf weight, and lower weaning calf weight adjusted to 295 days [1]. MAP ELISA positive animals have a 10-17% reduction in slaughter weight. If the animal's fecal culture contains MAP, the reduction in slaughter weight can be as high as 31% [2]. MAP-infected cows exhibit a decrease in milk production ranges from 0.02-1 kg/day. Heavily infected cows decrease their milk production by 4 kg/day [3-5]. A large Danish study documented that declines in reproduction, milk production, and fat content attributable to MAP occur over such a long period of time that they tend not to be identified by producers: the so-called MAP Milk Tax [6].

The gastrointestinal tract of herbivores and humans contain complimentary receptor sites that allow MAP to attach to the mucosa [10]. MAP isolates derived from goats or elk can infect cows and humans. Human MAP isolates have similar genetic markers to animal MAP isolates.

In the mid-1990s, the presence of a bovine pathogen, *Mycobacterium avium* subspecies *paratuberculosis*, was identified in both raw and pasteurized milk [7,8]. Historically, another pathogenic bovine mycobacterium, *Mycobacterium bovis* (MB) had used the same zoonotic bridge, adulterated milk, to infect humans. Between 1912 and 1937 an estimated 65,000 individuals in England and Wales died from gastrointestinal disease contracted through consumption of MB adulterated milk. The presence of a documented bovine pathogen within the nation's food supply altered USDA's primary mission from lessening MAP's negative economic impact on herd health and milk production to protection of the quality reputation of milk and milk products. In response to the early documentation of viable MAP isolated from pasteurized milk, USDA published a study that claimed that U.S. high temperature/short duration pasteurization effectively destroyed MAP [9]. When milk was then taken from the grocery shelves of the five-leading milk-producing states, viable MAP isolates were recovered from 2.8% of milk cartons (The Marshfield Retail Milk Study).

In 2000, the U.S. Congress undertook in earnest the task of assessing whether MAP in pasteurized milk constituted a public health hazard. In the ensuing hearings, the USDA publication was introduced into evidence. Congress gave USDA ultimately upwards of 90 million dollars and stewardship of determining whether MAP constituted a risk to the public welfare.

In 2001, USDA-APHIS implemented the Uniform Program Standards for the Voluntary Bovine Johne's Disease Control Program. In 2002, USDA instituted the five-year Johne's Disease Prevention Dairy Herd Demonstration Program [10,11]. At that time, 20-30% of all U.S. dairy herds had MAP infected animals. Despite evidence that other mycobacteria on the evolutionary transition from *Mycobacterium avium* subspecies *avium* (MAA) to MAP caused a Johne's-like disease in domestic animals [12-19] and despite the literature having identified necropsy documented cases of Johne's disease with positive agar immunodiffusion (AGID) tests that serologically tested negative in MAP ELISA tests, USDA obligated the diagnostic test manufacturers to construct their tests to specifically identify MAP's IS900 genomic insertion. Owing to their high threshold for positivity, USDA sanctioned MAP IS900 ELISA tests primarily functioned to identify the probability of an animal developing Johne's disease. A negative MAP ELISA test designation did not address whether a given animal had ever been infected by MAP [23]. McKenna., *et al*. showed that the commercial MAP ELISA tests identified only 6.8% to 8.8% of tissue positive cattle [24]. Pinedo., *et al*. demonstrated that MAP ELISA tests had a poor correlation with the documented presence of MAP in the corresponding milk [25]. Cows whose milk tested positive for MAP had negative or inconclusive MAP titers in 23.5% and 11.8% of the cases respectfully.

By certifying MAP ELISA tests to be but a statement of probability of developing Johne's disease rather than a valid measurement of the presence or absence of MAP antibody, the USDA certified MAP ELISA tests underestimated the number of MAP infected animals allowing the introduction of infected animals into uninfected herds.

USDA is responsible for the U.S. national standards for animal product warranty. Quality of merchandise is primarily addressed through the animal's health certificate. The Code's language in 9 CFR chapter 1 subchapter C restricts the inter-state movement of infected livestock. Revisions to part 71 and 80 of the Code of Federal Regulations (CFR) were intended to specifically restrict the interstate movement of MAP-infected animals, except to recognized slaughter establishments. Despite being confronted

with unchecked MAP infection in dairy and beef cattle, USDA opted to not require a statement on the health certificate as to whether an animal is or has not been infected by MAP and thereby circumvented the Animal Health Protection Act (7 U.S.C. 8301 et seq.). USDA permitted possibly infected animals to be shipped across state and national borders.

By 2005, analysis of 49% of 51 brands of infant formula manufactured in seven different countries by 10 different producers demonstrated the presence of MAP DNA [26]. The USDA's 2007 survey identified 70% of dairy herds possessed MAP-infected animals [27]. In 2007, the National Health Monitoring System identified that 31.2% of 515 dairy farms had bulk tank milk that tested positive for MAP. In 2008, USDA announced a Johne's Disease Control Problem whose three goals were to reduce the prevalence of MAP/Johne's disease in cattle, reduce the impact of Johne's disease on individual herds and reduce introducing Johne's disease to uninfected herds [28]. By 2012, the incidence of MAP infected dairy cows in milking herds on a global level had achieved such a density that the World Organization for Animal Health (OIE) proposed having paratuberculosis (caused by MAP) removed as a disease entity from the Terrestrial Animal Health Code. The rationale put forth by OIE was that "because MAP infection is so widespread, continued recognition of MAP as an animal pathogen would only cause economic losses through the restrictions in international animal trade". In 2012, 54 of MAP infected/diseased animals imported into Japan came from the United States [29].

Epidemiologic studies had indicated that MAP dissemination within milking-herds appeared to precede the appearance of CD in the general population. Prior to the co-habitation of Iceland, disease caused by MAP in domestic milk-producing animals was undetected. In 1933, Germans brought sheep to Iceland. Johne's disease became well established in sheep and cattle. The incidence of Crohn's disease in Iceland from 1950-1959 was 0.4 per 100,000 individuals per year; from 1960-1969 0.9; from 1970-1979 3.1; from 1980-1989 3.11 and from 1990-1995 (5.6). Prior to 1950, MAP disease was virtually unknown in the Czech Republic. Economic hardship necessitated that most mothers breastfeed their babies. Following the fall of the Iron Curtain, some 30,000 heifers were imported from the west. As the local economy improved, women began abandoning breastfeeding in favor of milk and infant formula. Between 1995 and 2004, the incidence of Crohn's disease in the Czech Republic increased 4.5-fold among 19+ year old and 6.5-fold in 65+ year old individuals [30]. The epidemiology of CD in the Czech Republic had a built-in control population. Breastfeeding is culturally based among Roma (gypsy) women. They were significantly slower to embrace infant formula. The rate of CD among Roma has been consistently half the incidence of that of the general population.

Mycobacterium avium subspecies *paratuberculosis* and the public welfare

The global CD pandemic has answered the question of whether MAP is a threat to public welfare. With MAP being so extensively embedded in the food supply of industrialized nations, an individual's probability of having MAP infection is a function of diet and time. Given the comparatively small number of CD afflicted individuals compared to world population, a case can be made that MAP is a non-potential pathogen for individuals with intact immunity, but not for those with significant impairment of their immune system. At birth, a newborn is immunologically comparable to a germ-free animal. To attain this perspective required permitting a rare disease entity to expand into a full global zoonotic pandemic whose ongoing legacy is CD.

Crohn's disease has now been demonstrated to be a zoonotic induced immune-mediated disease which is the product of two separate immune system interactions involving *Mycobacterium avium* subspecies *paratuberculosis* (MAP).

- Confronted by MAP infectious challenge, the baby's inherent immunity may become so stressed in arresting continued mycobacterium replication that its pro-inflammatory response to MAP becomes fixed within immunological memory. Whenever re-challenged by MAP's presence in milk-based commercial products, its immune system responds by again unleashing a Th1 immune response against MAP at its site of mucosal attachment rather than exhibiting immunological tolerance. Dealing with MAP as an infectious agent, a baby's inherent immunity may become so stressed in arresting continued mycobacterium replication that its pro-inflammatory response to MAP becomes fixed within immunological memory. Whenever re-challenged by MAP's presence in milk-based commercial products, the immune system always responds by unleashing a Th1 immune cytokines against MAP at its site of mucosal attachment.

- The requisite for disease requires that MAP and its interaction with anti-MAP directed cytokine be both repetitive and concentrated to overwhelm the regenerative capacity of the small bowel gastrointestinal mucosa. The focal loss of mucosal integrity allows the gastrointestinal microbiota to establish submucosa residence. If not addressed, the failure to treat the resultant polymicrobial infection created becomes the second driving mechanism of CD.

This unchallenged pathogenesis of CD answers all of the key epidemiological facts embedded in CD's natural history: why breast feeding confers protection against the future development of CD, why CD is a new disease, why CD has attained global epidemic status only in industrialized nations, why the ileocecum is the site of initial disease and why MAP infection must become prevalent in the milking herds before CD manifests in the general population [2,3].

In retrospect, it is Infectious Diseases Incorporated's perspective that the global pandemic of CD is the product of USDA's unwillingness and ultimate failure to limit the dissemination of MAO among milking herds.

Bibliography

1. "Association between cow reproduction and calf growth traits and ELISA scores for paratuberculosis in multi-breed herds of beef animals". *Tropical Animal Health and Production* 41 (2009): 851-858.

2. Kudahl AB and Nielsen SS. "Effect of paratuberculosis on slaughter weight and slaughter value of dairy cows". *Journal of Dairy Science* (2009): 92-4340-4346.

3. Tiwari A., *et al*. "Estimate of the direct production loss in Canadian dairy herds with subclinical Mycobacterium avium subspecies paratuberculosis infection". *Canadian Veterinary Journal* 49 (2008): 569-576.

4. Smith RL., *et al*. "A longitudinal study on the impact of Johne's disease status on milk production in individual cows". *Journal of Dairy Science* 92 (2009): 2653-2661.

5. Raizman EA., *et al*. "Loss of income from cows shedding Mycobacterium avium subspecies paratuberculosis prior to calving compared to cows not shedding the organism on two Minnesota dairy farms". *Journal of Dairy Science* 92 (2009): 4929-4936.

6. Nielsen SS., *et al*. "Time to occurrence of a decline in milk production in cows with various paratuberculosis antibody profiles". *Journal of Dairy Science* 92 (2009): 149-155.

7. Schleig PM., *et al*. "Attachment of Mycobacterium avium subspecies paratuberculosis to bovine intestinal organ cultures: method of development and strain differences". *Veterinary Microbiology* 108 (2005): 271-279.

8. Millar D., *et al*. "IS 900 PCR to detect Mycobacterium avium subspecies paratuberculosis in retail supplies of whole pasteurized milk in England and Wales". *Applied and Environmental Microbiology* 62 (1996): 3446-3454.

9. Grant IR., *et al*. "Inactivation of Mycobacterium paratuberculosis in cow's milk at pasteurization temperatures". *Applied and Environmental Microbiology* 62 (1996): 631-636.

10. Stabel JR., *et al*. "Heat inactivation of Mycobacterium paratuberculosis in raw milk: are current pasteurization conditions effective?" *Applied and Environmental Microbiology* 63 (1997): 975-977.

11. "United States voluntary Johne's disease herd status program for cattle." United States Animal Health Association (2000).

12. "Uniform program standards for the voluntary Johne's disease control program". United States Department of Agriculture Animal and Plant Health Inspection Service". APIS 91-45-014.

13. Cousins D V., *et al*. "Mycobacteria distinct from Mycobacterium avium subspecies paratuberculosis isolated from the faeces of ruminants possess IS900-like sequences detectable IS900 polymerase chain reaction: implications for diagnosis". *Molecular Cell Probes* 13 (1999): 431-442.

14. McIntyre G., *et al*. "Immunodiffusion analysis showed that M. paratuberculosis and other mycobactin-dependant mycobacteria are variants of M. avium". *Journal of General Microbiology* 26 (1966): 21120-2123

15. Englund S., *et al*. "An IS900-like sequence found in a Mycobacterium sp. other than Mycobacterium avium subspecies paratuberculosis". *FEMS Microbiology Letter* 209 (2002): 267-271.

16. Yoder S., *et al*. "PCR comparison of Mycobacterium avium isolated obtained from patients and foods". *Applied and Environmental Microbiology* 65 (1999): 2650-2653

17. O'Grady D., *et al*. "Restriction fragment length polymorphism analysis of Mycobacterium avium isolates from animal and human sources". *International Journal of Tuberculosis and Lung Disease* 4 (2000): 278-281.

18. Tuenne CY., *et al*. "Mycobacterium avium in the postgenomic era". *Clinical Microbiology Review* 20 (2007): 205-229.

19. Collins DM., *et al*. "Identification of two groups of Mycobacterium paratuberculosis by restriction endonuclease analysis and DNA hybridization". *Journal of Clinical Microbiology* 28 (1990): 1591-1596.

20. Monif GRG and Williams JE. "The significance of a negative Map ELISA test for Mycobacterium avium subspecies paratuberculosis". *International Journal of Applied Research in Veterinary Medicine* 11 (2013): 171-1722.

21. McKenna SL., *et al*. "Evaluation of three ELISAs for Mycobacterium avium subsp. paratuberculosis using tissue and fecal culture comparison standards". *Veterinary Microbiology* 110 (2005): 105-111.

22. Pinedo PJ., *et al*. "Mycobacterium paratuberculosis shedding into milk: association of ELISA seroreactivity with DNA detection in milk". *International Journal of Applied Research in Veterinary Medicine* 6 (2008): 137-144.

23. Hruska K., *et al*. "Mycobacterium avium subsp. paratuberculosis in powdered infant milk: paratuberculosis in cattle – public health problem to be solved". *Veterinarni Medicina* (2005): 327-335.

24. USDA-APHIS Johne's Disease in U.S. Dairies 1991-2007.

25. Schwartz A. "National Johne's Disease Control Program Strategic Plan". U.S/Animal Health Association. October 23, (2008).

26. Monotami E. "Epidemiological situation and control strategies for paratuberculsis in Japan". *Japanese Journal of Veterinary Research* 60 (2012): 19s-29s.

27. Hruska K and Pavlik I. "Crohn's disease and related inflammatory diseases: from a single hypothesis to one "superhypothesis". *Veterinarni Medicina* 59 (2011): 583-630.

28. Monif G R G. "The WHY? of Crohn's disease". *Advanced Research in Gastroenterology and Hepatology* 10 (2018): 1-4.

29. Monif G R G. "The The Crohn's disease: The Infectious Disease Incorporated's Perspective". *Gastrointestinal Disorder* 3 (2021): 138-141.