40 YEARS ON, IT'S GRATIFYING TO SEE MY IDEA BECOMING REALITY

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now retired, looks back at the realisation of an idea he had 40 years ago to prevent IMI

STUCK at home due to bad weather, I took a trip down memory lane when I came across a 2010 paper[1] that aroused my interest because of its links with research work I did 40 years ago.

In the early 1960s, I worked at the Central Veterinary Labatory (now VLA) in Weybridge and, like the authors of the paper, I was interested in the mechanisms and predisposing factors that caused intramammary infections (IMI) in cattle. My work culminated in a PhD thesis[2].

Not all the work was published and, after getting my degree, my career moved in another direction and I never again worked in mastitis. All these years later, and now happily retired, reading the paper made me realise how little has changed during 40 years.

In the intervening years I did not even keep up with the literature in any diligent way, but I notice that, apart from three "standard" references, the authors' bibliography in general goes back to 1980. My bibliography[3] obviously preceded theirs by many years, but I think if the reader made a comparison, he or she might be surprised how much similarity existed between the research objectives in the two eras.

Murphy and Stuart (1954)[4] had described a method for collecting milk by syringe via teat wall puncture. Using this technique, I was able to confirm their findings to show that milk collected conventionally through the teat canal (with the usual aseptic precautions) could be infected, while milk taken direct from the teat sinus on the same occasion could be sterile. What is more, a pure culture with colony counts in the conventional milk could be consistent with those interpreted to be an IMI.

My early ideas led me to suspect that organisms might be transported from the teat canal into the teat sinus by mechanical forces.

It was my intention to investigate how the mammary gland became infected under normal management and milking conditions, and I wanted to avoid undue experimental interference or the introduction of new organisms as markers. I had examined the bacteriology of:

- naturally infected teat canals by swabs inserted 3mm into the canal;
- conventional hand-drawn milk; and
- milk collected aseptically by syringe through the teat wall. The latter procedure may sound unduly invasive, but with acquired skill and with time – and the perhaps surprising cooperation of the cows – it caused no untoward effect.

The predominant organisms in the teat canals were *Staphylococcus aureus*, coagulase-negative staphylococci and micrococci. All these organisms are commensal bacteria of the skin but could also establish IMI.

It was the relationship of teat canal infections to the development of IMI that was my main interest. As all the bacteria are non-motile cocci, I believed the mechanism by which they penetrated the teat canal was probably the same. The organisms were identified by strain, allowing their epidemiology to be followed within the teat canals and udders in the herd. By including all the *Micrococcaceae* that were present in my samples, I had a much bigger database to investigate the passage of bacteria through the teat canal than I would if I had limited the investigation to *S aureus*.

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Infections in the teat canals could persist for several weeks before either becoming an IMI or disappearing from the canal. This was on the basis of a comparison of weekly samples taken conventionally and those taken from the teat sinus by syringe.

My conclusion was that the only definitive diagnosis of an IMI was infected milk taken directly from the teat sinus. Conventionally collected milk infected with a recognised pathogen is also a very strong indicator when associated with an elevated somatic cell count (SCC). However, in the case of non-recognised pathogens the elevation may be minimal, making diagnosis of an IMI equivocal.

In investigating the passage of organisms through the teat canal and the consequent effect, it is important to recognise the differing inflammatory response, because invasion through the canal is unrelated to the pathogenicity as an IMI.

My work also showed that the susceptibility of the gland to an invasion of a new bacterium was dependent on the milk's SCC, which was itself dependent on the presence or absence of current infections, many of which would never cause clinical mastitis. However, let us restrict our attention to the passage of organisms through the teat canal.

Mechanical stresses at the teat end from the teat cup liner

I am not aware of any fundamental change in the design of the teat cup liner since machine milking has been practised.

During the rest phase of the pulsation cycle the liner collapses to its maximum, approximately at its mid-length. The stresses this imposes on the teat depends not only on the shape of the teat end, but also the length of the teat and its protrusion into the liner. Short teats will have relatively minor "crushing", being in the space above mid-length, while for long teats, the teat end will experience much more pressure. However, with liner crawl towards the end of milking, all teats may be subject to the same stresses. Nevertheless, depending on shape and length, one can imagine the different possible distortions induced at the level of the teat canal.

The squamous epithelium lining the teat canal produces keratin and, because of the limited space in the canal, it can cause an extrusion of surplus keratin beyond the natural orifice, which I think may be the origins of the hyperkeratotic lesions often described. I can imagine the same phenomenon occurring at both ends of the canal and causing a keratin intrusion into the lumen of the teat sinus. If this keratin was carrying an infection it could be a cause of IMI.

Also, if the keratin at the teat end was infected, I can imagine the action of the liner during milking gradually intruding keratin along the teat canal towards the teat sinus lumen, perhaps particularly in teat ends that were plate-shaped or inverted funnel-shaped – both shapes that have been shown to be susceptible to mastitis.

Of course, the infection could also grow through the length of the canal, but either one, or a combination of both, could carry the infection through to establish an IMI.

I believe these explanations provide a plausible hypothesis of how the udder can become infected, particularly for those organisms commensal to the skin that reside in keratin. In my work, I concentrated on the Micrococcaceae and recognise the mechanism may not be true for other IMI pathogens. The herd I worked on did have *Streptococcus uberis*, but *S agalactiae* and *S dysgalactiae* were absent.

Incidentally, while I was writing up my work, some of the herd were allowed to suckle their calves for their next lactation. I investigated their IMI status (again by teat wall puncture) towards the end of lactation and from recollection; all the quarters were then infected with *Corynebacterium bovis* in pure culture. The SCCs were only moderately raised and some previously existent IMIs with other infections had been eliminated. What I do remember is that the teats were pristinely healthy.

When I had finished my work and written it up, I realised that to explore further my hypothesis, I would need to produce a new teat cup design that avoided the mechanical stresses at the end of the teat, a design for which, in mechanical terms, I had neither the ideas how to implement, nor access to the engineering skills and engineering facilities required.

My best idea was to produce a milking machine that simulated the sucking and tongue action of the calf. The calf occludes the proximal end of the teat with the tip of its tongue against the dental pad. With a combination of suction and tongue massage, it draws the milk from the teat without exerting much pressure on the teat canal. In contrast, the teat cup liner occludes the teat canal and massages the teat wall expanding the lumen towards the proximal end.

A wistful paragraph in the final discussion of my thesis reads: "It is hoped to continue this work by developing teat cups that do not allow marked fluctuations of vacuum at the teat end or complete collapse of the liner.

"Controlled experiments could then establish if there was any difference in the rate of new infections between these cows and those milked with conventional models."

The author's PhD was on causes of intramammary infections.

After the work was completed, I had the opportunity to do a sabbatical year and that led me into a different science, so I never did return to follow up my ideas.

However, about a year ago, I did an internet search on bovine milking systems to see what changes had occurred over the years. I came across an American invention marketed as CoPulsation Milking System.

The description satisfies the criteria I would have looked at and seems to have been met, according to the company's data. The improvement in the condition of the teats after milking when compared to conventional systems is very evident and mastitis incidence is reported to be reduced. Where the system has been introduced, records show continuing reductions in the herd SCC. This is augmented by reduced infections.

Teat stays dry

The stresses at the proximal end of the teat have been removed. Whether this difference has reduced bacterial invasion through the canal in the way I have postulated above cannot be proved, but I consider that to be the most likely reason for the reduced incidence of mastitis.

Another benefit is that the tip of the teat remains dry, therefore, without any column of milk bridging the canal, there should be no possibility of milk being refluxed back into the teat sinus; another cause of bacterial invasion through the teat canal that has been investigated.

The company claims to have "the only product in the world to provide a true massaging rest phase".

When I read about it, I must admit to a gratifying satisfaction at what appears to have been achieved, almost as if it had been a personal fulfilment. Unknown to me, someone has completed a design that I would love to have achieved.

I have received a message from the inventors of the system complimenting my research as uniquely supporting what their product has done. It is amazing that two totally independent research programmes, done in two different continents and separated by 20 years, should complement each other so well.

Improvement

I recommend the website at www.copulsation.com for more information, particularly the explanation of teat end stresses. Two of the firm's anecdotal observations impress me: "Cows are substantially calmer and will choose our system over a conventional when one is on one side of a parlour and the other system on the other side," and "the milking parlours are noticeably cleaner, as the cows no longer automatically defecate upon entering because the conventional system induces pain and ours does not".

Those who know cow behaviour will appreciate the improvement in welfare evident in those remarks. Apart from one unit in Ireland, the system has not yet crossed the Atlantic. I wait with interest to find how successfully the system becomes established. Certainly, I wish it every success.

References

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