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Testing Frequency, Not Assay Quality, Decides a Brucellosis Programme: A One Health Simulation of Herd Control and Human Infection

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ABSTRACT

Background. Bovine brucellosis is controlled by periodic whole-herd serological testing with removal of reactors. Human infection follows from occupational contact with infected animals and from unpasteurised dairy, so the human outcome is driven by the animal-infectious time a programme fails to remove. We asked what determines whether such a programme succeeds and what the human consequence is when it does not.

Methods. We simulated 400 replicate herds of 200 adult cattle over 10 years with a susceptible–latent–infectious structure, lifelong infection, natural turnover and periodic whole-herd testing at stated sensitivity and specificity. Occupational and milk-borne human infections were generated in proportion to infectious-animal-months. Sensitivity, testing interval, specificity and milk pasteurisation were varied.

Results. Without a programme no herd cleared infection and 12.5 human infections arose per herd over 10 years. Testing interval dominated assay quality: at 12-monthly testing a sensitivity of 0.90 left only 3.6 % of herds free, while the same assay at 6-monthly testing reached 92.0 %. Raising sensitivity from 0.40 to 0.95 within a 12-monthly programme moved herd freedom only from 0.8 % to 3.2 %. Falling specificity culled uninfected animals without epidemiological benefit: at a specificity of 0.95, 197 uninfected animals were removed per herd against 36 infected, with herd freedom unchanged. Pasteurisation alone reduced total human infection from 12.4 to 4.0 per herd with no animals culled, but left the occupational burden at 3.5, which only herd-level control reduced.

Conclusion. Programme frequency relative to the infectious period, not assay sensitivity, determined success. Testing and pasteurisation reduce risk for different populations and should not be treated as substitutes.

1. Introduction

Brucellosis remains among the most widespread zoonoses [1,2], and in most endemic settings the human burden is a direct function of infection in livestock [3–5]. Human disease arises by two distinct routes: occupational exposure among those who handle animals, birth products and carcasses, and foodborne exposure through unpasteurised milk and dairy [6–9]. Both trace back to the same quantity, the amount of time infectious animals spend in the population.

The standard veterinary control measure is periodic whole-herd serological testing with removal of reactors, applied over years until a herd qualifies as free [10,11]. Programmes of this kind have eliminated bovine brucellosis from many national herds, and where they have failed the failure is commonly attributed to test performance, to incomplete coverage or to compensation arrangements that discourage reporting [12,13].

Test performance is the attribute most often measured and most often the subject of investment. Serological assays for brucellosis differ appreciably in sensitivity, and improving that sensitivity is an intuitive route to a better programme. Testing frequency, by contrast, is usually set by cost and logistics rather than by epidemiology.

The relative importance of the two has a clear theoretical basis. A test-and-cull programme removes infectious animals only at discrete moments; between rounds, animals that have seroconverted transmit freely. If the interval is long relative to the period an animal remains infectious and in contact, a large share of

transmission occurs between rounds, and the effectiveness of each individual round has limited leverage over the outcome.

We set out to quantify that relationship in a herd model, and to carry it through to the human outcome, separating the occupational and foodborne pathways so that the beneficiaries of each intervention can be identified.

1.1 Objectives

We aimed to quantify herd-level elimination as a joint function of test sensitivity and testing interval; to determine the human infection burden by pathway under each programme configuration; to establish the cost of imperfect specificity in animals culled unnecessarily; and to compare herd-level testing with milk pasteurisation as levers on human infection.

2. Materials and Methods

2.1 Herd model

Each herd comprises 200 adult cattle in three states: susceptible; latently infected and seronegative; and infectious and seropositive. Latently infected animals become seropositive after an average of three months, reflecting the delay between infection and detectable antibody. Infection is lifelong, so animals leave the infectious class only through culling or natural turnover, which is applied at a rate corresponding to a mean productive life of four years. Replacement animals enter as susceptible.

Transmission is frequency-dependent within the herd [14,15]. Two infectious animals are introduced at the start of the simulation and each configuration is run over 10 years across 400 replicate herds.

We model a closed herd with no purchase of infected replacements and no between-herd spread. This is a simplification that isolates the mechanism of interest; Section 5 discusses what it omits.



The two interventions do not protect the same people; reporting a single human total conceals that.

Figure 1. The transmission pathway from herd infection to human disease and the two points of intervention.

2.2 Testing programme

At each scheduled round the whole herd is tested. Only seropositive animals can be detected, so latently infected animals pass regardless of assay quality. Detected animals are culled and replaced with susceptible stock. Uninfected animals testing positive are also culled, at a rate determined by specificity.

Sensitivity was varied from 0.40 to 0.95, spanning the range reported for serological assays used in field conditions [16,17], and specificity from 0.95 to 1.00. Testing intervals of 36, 24, 12 and 6 months were compared against no programme.

2.3 Human infection

Human infections are generated in proportion to infectious-animal-months, separately for the two pathways. Occupational infections accrue to the 4 people in regular animal contact per herd; milk-borne

infections accrue to consumers of unpasteurised product. Pasteurisation is modelled as removing a stated proportion of the milk-borne pathway and none of the occupational pathway.

Parameters are given in Table 1. All are illustrative, chosen to lie within published ranges and to produce an endemic herd situation, and are not estimates for any specific setting. The simulation was implemented in Python using NumPy, SciPy and Matplotlib [18–20]; the complete script is available from the author on request.

Table 1. Model parameters

Parameter	Value	Note
Herd size	200 adult cattle	Closed herd, no purchased replacements
Latent period	3 months mean	Interval to seroconversion; undetectable by serology
Infection duration	Lifelong	Removal only by culling or turnover
Turnover	Mean productive life 4 years	Replacements enter susceptible
Horizon	10 years	Typical duration of a control programme
Test sensitivity	0.40 to 0.95	Applies to seropositive animals only
Test specificity	0.95 to 1.00	Determines culling of uninfected animals
Testing interval	None, 36, 24, 12, 6 months	Whole-herd rounds
Human pathways	Occupational and milk-borne	Both proportional to infectious-animal-months
Replicate herds	400	Per configuration

3. Results

3.1 The untreated herd

Without a programme, infection established and persisted in every herd: 0.0 % were free at 10 years, and herds accumulated 16,216 infectious-animal-months.

That animal burden produced 12.49 human infections per herd over the period, comprising 3.57 occupational and 8.92 milk-borne. Expressed as an occupational rate, this is 89.2 infections per 1000 worker-years among those in regular animal contact.

The split between pathways matters for what follows. Roughly seven in ten infections in this configuration arose through consumption and three in ten through occupational exposure, and the two are reached by different interventions.

3.2 Sensitivity at a fixed interval

Table 2 and Figure 2 vary sensitivity within a 6-monthly programme. Herd freedom at 10 years rose from 4.5 % at a sensitivity of 0.40 to 95.8 % at 0.95, and human infections fell from 6.42 to 0.08 per herd.

Read alone, this table supports the intuitive conclusion that assay sensitivity is the critical parameter, and it is the analysis most likely to be performed when a programme is being designed. The next section shows why it is misleading.

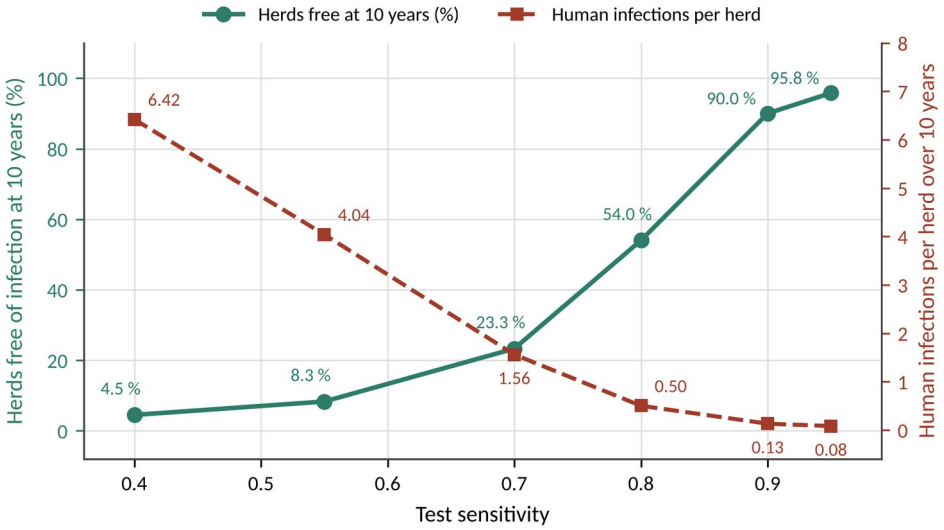


Figure 2. Herd freedom and human infection against test sensitivity under 6-monthly testing.

Table 2. Sensitivity sweep at 6-monthly testing, specificity 0.995

Sensitivity	Herds free (%)	Infectious-animal-months	Human infections	Animals culled	Occupational rate per 1000 worker-years
0.40	4.5	8335	6.42	692.6	45.84
0.55	8.3	5251	4.04	646.2	28.88
0.70	23.3	2025	1.56	352.8	11.14
0.80	54.0	651	0.50	147.6	3.58
0.90	90.0	168	0.13	57.9	0.92
0.95	95.8	105	0.08	45.4	0.58

3.3 Interval dominates assay quality

Table 3 and Figures 3 and 4 vary both parameters. The pattern is strongly asymmetric. Reading down the 12-monthly column, raising sensitivity from 0.40 to 0.95 moved herd freedom from 0.8 % to only 3.2 %. Reading across the row for a sensitivity of 0.90, moving from 12-monthly to 6-monthly testing moved it from 3.6 % to 92.0 %.

At 24-monthly testing and longer, no combination of sensitivity achieved meaningful elimination: the best result across the whole sensitivity range was 1.2 % of herds free.

The mechanism is that culling acts only at discrete moments while transmission is continuous. An animal infected shortly after a round seroconverts and transmits for the remainder of the interval regardless of how good the assay is when the next round arrives. Where the interval is long relative to the time an animal spends infectious and in contact, most transmission occurs between rounds and assay quality has little to act on.

The practical implication is that a programme whose interval is too long cannot be rescued by a better test, and that investment in assay sensitivity is justified only once the interval is short enough for it to matter. On these figures a service choosing between doubling testing frequency and adopting a substantially more sensitive assay should choose the former without hesitation.

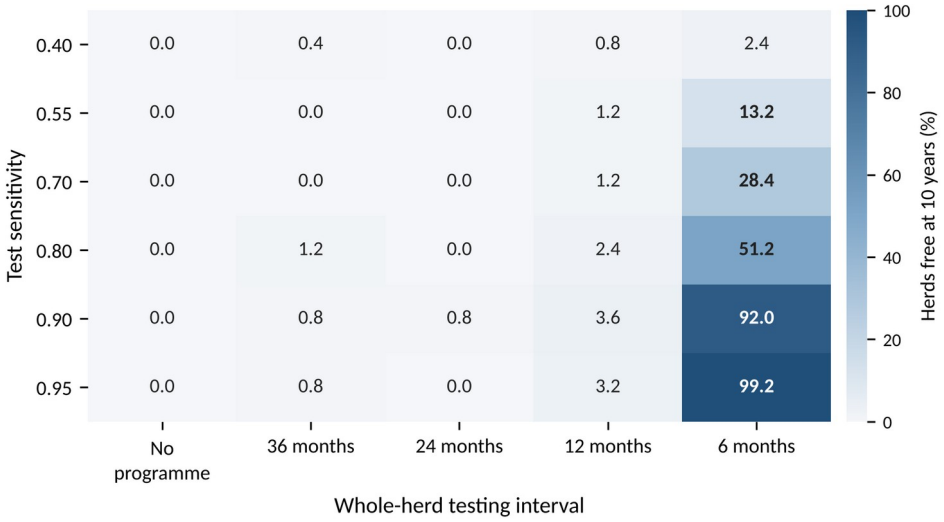


Figure 3. Herds free of infection at 10 years across sensitivity and testing interval.

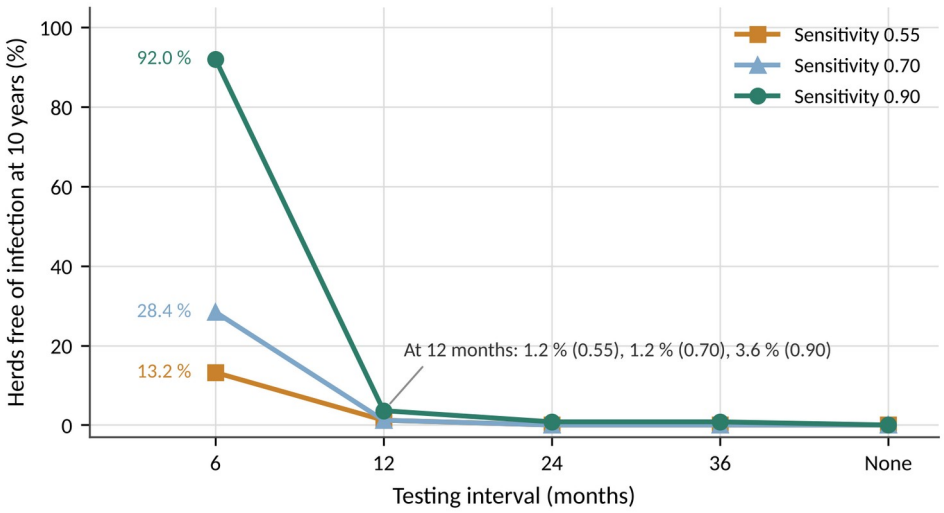


Figure 4. Herd freedom against testing interval at three levels of sensitivity.

Table 3. Herds free of infection at 10 years (%), by sensitivity and interval

Sensitivity	No programme	36 months	24 months	12 months	6 months
0.40	0.0	0.4	0.0	0.8	2.4
0.55	0.0	0.0	0.0	1.2	13.2
0.70	0.0	0.0	0.0	1.2	28.4
0.80	0.0	1.2	0.0	2.4	51.2
0.90	0.0	0.8	0.8	3.6	92.0
0.95	0.0	0.8	0.0	3.2	99.2

3.4 Specificity is an economic parameter

Table 4 and Figure 5 vary specificity at a sensitivity of 0.90 with 6-monthly testing. Herd freedom was essentially unchanged across the range, from 89.5 % at perfect specificity to 91.5 % at 0.95.

The cost fell entirely on the farmer. At a specificity of 0.995, 19.9 uninfected animals were culled per herd against 39.4 infected, so 33 % of removals were unnecessary. At 0.95 the figures were 197 against 36, with 84 % unnecessary.

Specificity therefore behaves quite differently from sensitivity in this system. Sensitivity buys epidemiological progress; specificity buys nothing epidemiologically and determines only how many healthy

animals are destroyed in the process. Where compensation is inadequate, this is also the parameter most likely to drive non-cooperation and concealment, which no model of a compliant herd can capture.

A programme reporting only the number of reactors removed conflates the two. Reporting confirmed infections separately from serological reactors is straightforward and makes the burden visible.

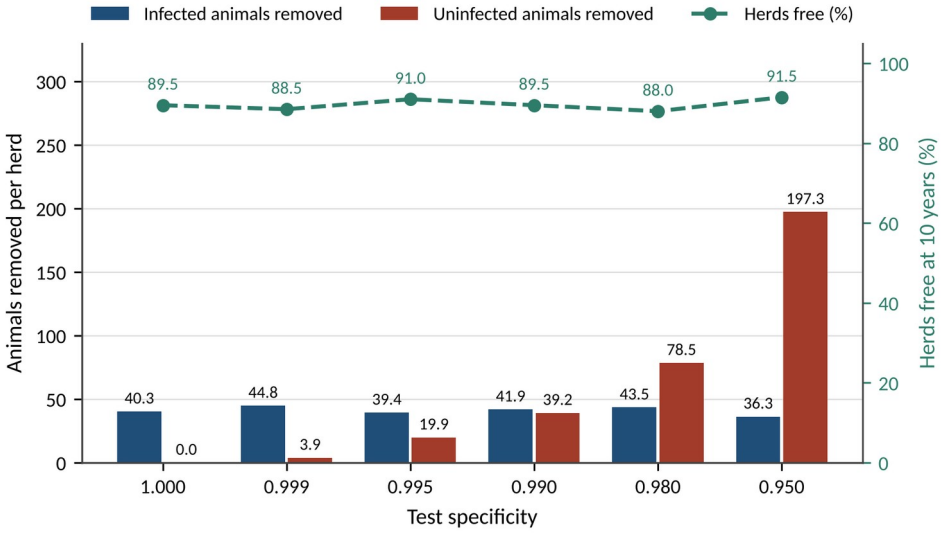


Figure 5. Animals removed and herd freedom against test specificity.

Table 4. Specificity sweep at sensitivity 0.90, 6-monthly testing

Specificity	Infected animals removed	Uninfected animals removed	Unnecessary share of removals	Herds free (%)
1.000	40.3	0.0	0.0 %	89.5
0.999	44.8	3.9	8.1 %	88.5
0.995	39.4	19.9	33.5 %	91.0
0.990	41.9	39.2	48.3 %	89.5
0.980	43.5	78.5	64.4 %	88.0
0.950	36.3	197.3	84.4 %	91.5

3.5 Human burden by pathway

Table 5 gives the human outcome under 6-monthly testing. Total infections per herd fell from 12.49 with no programme to 6.45 at a sensitivity of 0.40 and 0.08 at 0.95, corresponding to 48.3 % and 99.4 % of the burden averted.

The occupational rate fell from 89.2 to 0.56 infections per 1000 worker-years. Because both pathways scale with the same animal quantity, herd-level control reduces them together and in proportion.

This is the sense in which the veterinary programme is a human health intervention rather than an agricultural one, and the case for funding it from a public health budget rests on this proportionality [21,22].

Table 5. Human infections per herd over 10 years, 6-monthly testing

Sensitivity	Occupational	Milk-borne	Total	Occupational rate per 1000 worker-years
No programme	3.57	8.92	12.49	89.19
0.40	1.84	4.61	6.45	46.07
0.55	1.12	2.79	3.91	27.92
0.70	0.48	1.20	1.67	11.96
0.80	0.15	0.38	0.54	3.84
0.90	0.04	0.09	0.12	0.88
0.95	0.02	0.06	0.08	0.56

3.6 The two levers protect different people

Table 6 and Figure 6 compare herd testing with milk pasteurisation. Pasteurisation alone reduced total human infection from 12.41 to 3.99 per herd, a reduction of 68 %, with no animals culled and no change to the herd.

It did so entirely by removing the milk-borne component, from 8.86 to 0.44. The occupational component was unchanged at 3.54, because pasteurisation acts after the animal–human interface that generates it.

Testing reduced both, from 12.41 to 0.15, at a cost of 64.7 animals culled per herd.

The policy reading is that these are not alternatives. Pasteurisation is the cheaper and faster route to the larger share of the burden and protects consumers, who are numerous and diffuse. It offers nothing to the farmer, the herder, the abattoir worker or the veterinarian, whose exposure is occupational and who bear both the residual risk and the cost of any culling programme.

A programme evaluated on total human cases would show pasteurisation performing well and might defer herd control on that basis. Disaggregating the two pathways makes visible that the deferral transfers the entire remaining risk onto the occupational group, and that this group is also the one asked to bear the cost when herd control does proceed.

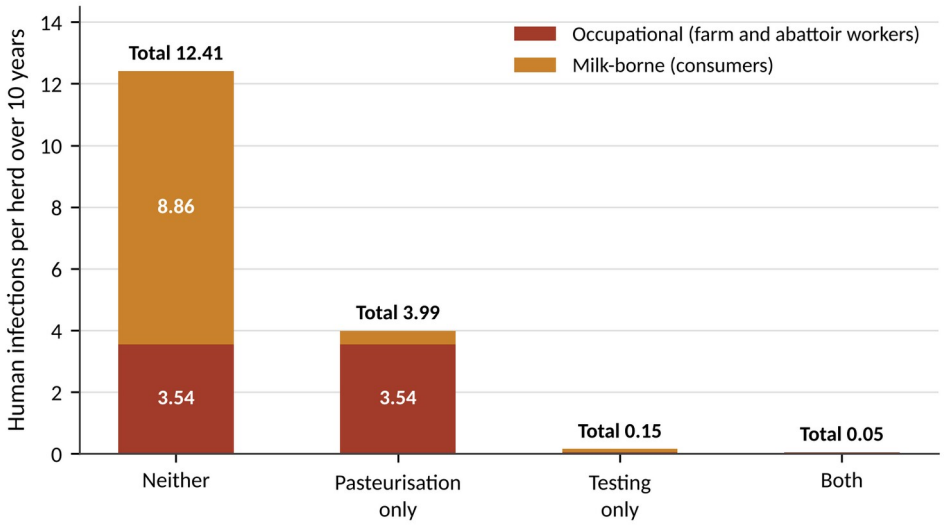


Figure 6. Human infections by pathway under testing, pasteurisation, both and neither.

Table 6. Comparison of the two levers

Configuration	Occupational	Milk-borne	Total human	Animals culled
Neither	3.54	8.86	12.41	0.0
Pasteurisation only	3.54	0.44	3.99	0.0
Testing only	0.04	0.11	0.15	64.7
Both	0.04	0.01	0.05	64.7

Table 7. Recommendations

Recommendation	Addresses	Cost	Evidence here
Set the testing interval against the infectious period	An interval too long for any assay to rescue	More frequent rounds	Section 3.3
Do not invest in assay sensitivity before shortening the interval	Effort on the parameter with less leverage	None; a sequencing decision	Section 3.3
Report confirmed infections separately from reactors	Unnecessary culling hidden in one figure	Reporting change	Section 3.4
Ensure compensation reflects false-positive culling	Cost falling on the farmer, driving non-cooperation	Programme budget	Section 3.4
Report human cases by pathway, not as a single total	Occupational risk concealed by consumer gains	Surveillance change	Section 3.6
Treat pasteurisation and herd control as complements	Two levers mistaken for alternatives	None	Section 3.6

4. Discussion

4.1 Principal findings

Four findings follow. Testing interval dominated assay sensitivity: at 12-monthly rounds the full sensitivity range moved herd freedom only from 0.8 % to 3.2 %, while halving the interval at fixed sensitivity moved it from 3.6 % to 92.0 %. Specificity had no epidemiological effect and determined only the number of healthy animals destroyed. Human infection scaled directly with the animal burden across both pathways. And pasteurisation removed the consumer share while leaving the occupational share untouched.

The first and last are the ones we would emphasise. Together they say that the design decisions that matter most in such a programme are the frequency of rounds and the choice of whose risk is being reduced, and that neither is a laboratory question.

4.2 Relation to existing work

The epidemiology of brucellosis in cattle and the performance characteristics of serological assays are well described [10,16,17], as is the human burden and its division between occupational and foodborne routes [6,7,23–25]. Modelling studies of test-and-slaughter programmes have examined the effect of testing frequency and of vaccination, generally finding frequency to be influential [26–28], and economic analyses have addressed compensation and its effect on reporting [12,29,30].

What we add is a direct comparison of the two parameters over a full grid with the human outcome carried through and disaggregated by pathway. The finding that assay sensitivity has little leverage at long intervals is consistent with the modelling literature; the explicit statement that pasteurisation and herd control protect different populations, with the numbers attached, is the part we would want carried into programme design.

4.3 Implications for practice

Table 7 sets out six recommendations. Two deserve comment.

Sequence the investment. Where a programme is failing, the first question is whether the interval is short enough for any test to work. Procuring a more sensitive assay into a 24-monthly programme is expenditure without a plausible mechanism of benefit, and our grid suggests it would not have been detectable as an improvement.

Surveil human cases by pathway. Occupational and foodborne brucellosis are frequently reported together. Separating them costs an exposure question at notification and makes visible whether a control programme is protecting the people who work with the animals or the people who consume their product, which is a distributional question that a combined total cannot answer.

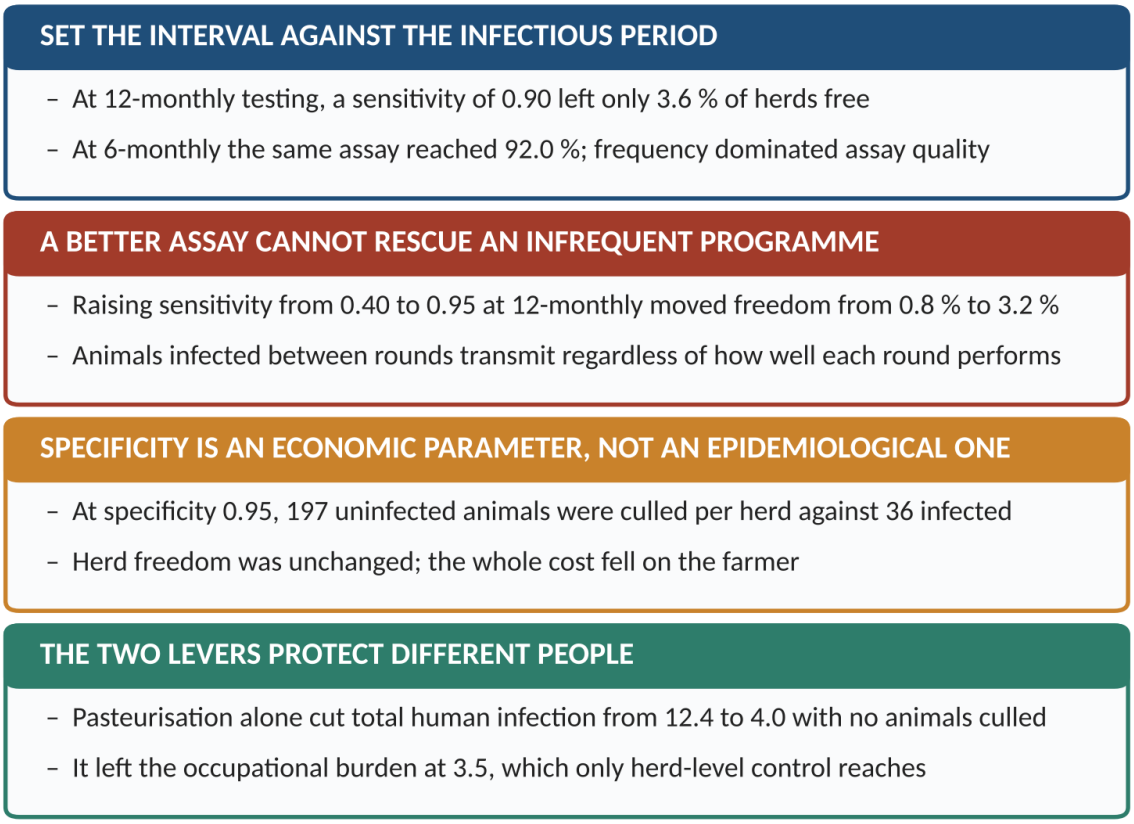


Figure 7. Proposed practice for brucellosis control programmes at the veterinary–human interface.

5. Limitations

The model is a closed herd. Purchase of infected replacement stock, shared grazing, contact at markets and wildlife reservoirs are all important routes in real settings, and all would reduce the achievable benefit of within-herd testing. A herd that clears infection and then buys it back has not been protected, and our elimination figures should be read as an upper bound.

We model serological detection of seropositive animals only, with a fixed latent period. Real assays vary in what they detect, chronically infected animals may test intermittently negative, and vaccinated animals complicate interpretation. We also do not model vaccination, which is the principal alternative control strategy in high-prevalence settings and would change the comparison substantially.

Human infection is generated as a linear function of infectious-animal-months with fixed coefficients. Real exposure is concentrated around parturition and abortion events, is strongly modified by protective equipment, handling practice and the use of milk within the household, and is not proportional to animal-time in a simple way. The human numbers should be read as indexing the animal burden rather than as incidence estimates.

Under-ascertainment of human brucellosis is substantial and variable, so the ratio between occupational and foodborne cases in surveillance data may differ considerably from the underlying ratio we have assumed. Our qualitative conclusion about who each lever protects does not depend on the split, but the relative sizes do.

Finally, we treat compliance as complete. In practice the specificity result interacts with farmer behaviour: unnecessary culling without adequate compensation discourages presentation of animals for testing, which is a feedback our model cannot represent and which would tend to make poor specificity worse than it appears here.

6. Conclusion

In simulated herds, test-and-cull control of bovine brucellosis succeeded or failed on the interval between rounds rather than on the sensitivity of the assay. At 12-monthly testing the entire sensitivity range from 0.40 to 0.95 left fewer than 4 % of herds free; at 6-monthly testing a sensitivity of 0.90 reached 92 %.

Specificity contributed nothing epidemiologically and determined only the number of uninfected animals destroyed, which reached 197 per herd at a specificity of 0.95. Human infection tracked the animal burden closely, falling from 12.5 infections per herd without a programme to 0.08 under effective control.

Pasteurisation removed 68 % of the human burden at no cost in animals, and none of the occupational burden. The two interventions are complements addressed to different populations, and a programme judged on a single human case total will not reveal which population it has left unprotected.

Declarations

Ethics approval and consent to participate. Not applicable. This study used entirely synthetic data generated by the author. No animals, human participants, tissue or records were involved, and no field or surveillance data were accessed. Institutional review board, ethics committee and animal ethics approval were therefore not required.

Consent for publication. Not applicable.

Data availability. No real-world data were used. All results were generated by the simulation described in Section 2, with every parameter stated in Table 1 and all random seeds fixed. Every figure and every value in Tables 2 to 6 can be regenerated exactly by executing the simulation script, which is available from the corresponding author upon reasonable request.

Conflicts of interest. The author declares no conflicts of interest, financial or non-financial, in relation to the subject matter of this manuscript. The author has no financial or advisory relationship with any manufacturer of veterinary diagnostic assays, vaccines or dairy processing equipment.

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Author contributions. Kamleshwari Durgam is the sole author of this work and was responsible for the conception and design of the study, development and implementation of the herd and human infection model, analysis and interpretation of the results, preparation of the figures and tables, and drafting, revision and final approval of the manuscript. The author is accountable for all aspects of the work.

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