

Bovine Mastitis Pathogens in Smallholder Dairy Herds: A Review of Prevalence and Antimicrobial Susceptibility Patterns

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ABSTRACT

Mastitis is one of the costliest diseases of dairy animals, and in smallholder herds, where a household may milk only a few cows, it is both common and poorly documented. Reports of which bacteria cause it, how often, and which antimicrobials still work against them are widely quoted, yet they are rarely comparable. This narrative review sets out what is known about the main groups of mastitis pathogens, how prevalence is measured, and how susceptibility is tested and reported, and it asks how much weight published figures can carry for small herds. It draws on published work on pathogen ecology, diagnostic tests, surveillance and laboratory methods, and it reports no new data. Prevalence estimates differ because case definitions, sampling frames and screening tests differ, so a figure from one survey cannot be set beside another without knowing how each was produced. Susceptibility results differ because methods, interpretive breakpoints and drug panels differ, and because resistance data often come from samples sent to a laboratory after treatment has already failed. The review concludes that the broad ecology of mastitis pathogens is well understood, that local prevalence and susceptibility in smallholder herds are not, and it lists the reporting details that would make future studies comparable and useful to the farms they describe.

Keywords: Bovine mastitis; Smallholder dairying; Prevalence; Antimicrobial susceptibility; Staphylococcus aureus; Surveillance; Narrative review

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1. Introduction

Mastitis is inflammation of the mammary gland, and in dairy animals it is usually the result of bacteria entering the udder through the teat canal. It is among the most costly diseases of dairying, because it reduces the quantity and quality of milk, adds to veterinary and labour costs, and shortens the productive life of affected animals [1, 2]. Much of what is known about its cost comes

from commercial herds in countries with well-developed advisory services. For a household that keeps two or three cows and sells milk to a neighbour or a cooperative, the same disease carries a different weight: there is less margin to absorb a bad month, and less access to a laboratory that could say what is growing in the milk. That gap in information is the subject of this review.

Two questions come up again and again whenever mastitis in such herds is discussed. Which bacteria are responsible, and how often? And which antimicrobials still work against them? Both questions have answers in the literature, in the sense that many surveys have been published, and both answers are quoted freely in extension leaflets, student theses and project proposals. What is much less often asked is whether the numbers in those surveys can be compared, and whether a figure from a research station or a referral laboratory says anything about a smallholder's cow.

This review argues that, for the most part, they cannot be compared without first reading how each was produced. A prevalence figure is the product of a case definition, a sampling decision and a test, and each of those can move the result more than any real difference between herds [3, 4]. A susceptibility pattern is the product of a laboratory method, an interpretive standard, a choice of drugs and, very often, a decision by someone to send a sample at all [5, 6]. A reader who sees only the final percentage cannot tell which of these dominated. The aim is a fair account of what is known and what is merely repeated.

The article is a narrative review. It draws on published work on the ecology of mastitis pathogens, the diagnostic tests used to find them, the methods of susceptibility testing and the epidemiology of surveillance, and it deliberately reports no new data and quotes no prevalence figures. Where evidence from smallholder herds is thin, we say so, and we describe what a study would need to report for its numbers to be useful, instead of repeating figures we could not check against their sources.

2. Literature Review

2.1 Mastitis and Its Economic Weight

The costs of mastitis are spread across many items: milk that is discarded or sold at a lower price, lower yield, treatment, culling and extra labour. Reviews of the economics describe these items and show that the totals depend heavily on the production system and on how costs are counted [1, 2]. Studies of what farmers believe about these costs have found that perception and calculation can be some distance apart, which matters because a farmer's decisions follow the perceived cost [7].

Clinical mastitis, with visible changes in the milk or the udder, is the form that farmers notice. Subclinical mastitis, in which the milk looks

normal but cell counts are raised and infection is present, is widely held to be the more common form and to account for much of the loss because it goes unseen [8, 9]. A survey that records only clinical cases therefore describes a different slice of the problem from one that tests every quarter, a point that returns repeatedly below.

2.2 Pathogen Groups and Their Ecology

The bacteria that cause mastitis are conventionally sorted into contagious and environmental groups, a division that is imperfect but useful. Contagious pathogens, of which *Staphylococcus aureus* and *Streptococcus agalactiae* are the standard examples, live mainly in the udder and spread between cows during milking; they are controlled by hygiene at milking and by managing infected animals [8, 10]. Environmental pathogens, such as *Escherichia coli* and *Streptococcus uberis*, come from bedding, soil and manure, and the exposure depends on housing and cleanliness [8, 11].

Species labels hide a good deal of variation. Molecular typing has shown that strains within a single species can behave quite differently, with some spreading from cow to cow and others arising from the surroundings, and that some mastitis bacteria are shared with humans [12]. For a survey this means that a count of isolates by species is a convenient simplification, not a description of how infection moves through a herd. Table 1 sets out the groups and what each means for anyone designing or reading a survey.

Table 1. Main groups of mastitis pathogens and what each means for a survey.

Group	Usual behaviour	Consequence for surveys
Contagious (e.g. <i>Staphylococcus aureus</i> , <i>Streptococcus agalactiae</i>)	Spreads from cow to cow, mostly around milking; infections often persist [10, 27]	Clustered by herd; a few herds can dominate a pooled estimate
Environmental (e.g. <i>Escherichia coli</i> , <i>Streptococcus uberis</i>)	Comes from bedding, soil and manure; infections are often short [8, 11]	Varies with season and housing; one sample can miss it
Coagulase-negative staphylococci	Very commonly isolated, often with mild effect [13, 14]	Counts depend on how isolates are classed and whether they are called infections
Other and mixed	Includes less frequent bacteria and mixed growth [12]	Contamination and mixed growth blur the case definition

Note: Qualitative summary of widely described behaviour. Real herds mix the groups, and a single species can act in more than one way.

Coagulase-negative staphylococci deserve separate mention. They are among the most frequently isolated bacteria from milk in many surveys, they are generally associated with milder disease, and there is a long-running argument over how often a positive culture represents infection and how often contamination from the skin of the teat [13, 14]. Surveys that group them with the major pathogens, or leave them out, give totals that cannot be reconciled with one another.

2.3 Smallholder Herds as a Setting

Published work on mastitis in smallholder herds is thinner than for commercial systems, but it exists. Studies in East Africa have examined clinical and subclinical mastitis on small farms and have combined description with risk analysis and, in one case, with an intervention and an attempt at knowledge transfer [15, 16]. A study in the Hawassa milk shed of southern Ethiopia reported on prevalence, risk factors and the isolation of *S. aureus* in dairy herds [17]. These papers are useful precisely because they come from the setting in question, and none of them should be stretched to cover other regions or other production systems.

Several features of smallholder dairying are likely to shape what a survey finds: hand milking, shared cloths and buckets, mixed housing, variable access to clean water, and sales of milk that bypass any quality test. They also shape what a survey can find, since few such households can afford culture, and many farms are reached only when a project, a student or a veterinary visit brings someone to the door. These are reasons for caution about extending findings from large commercial herds, and equally about extending a single regional survey. A farmer reading a survey needs the answer to a plain question: is this what I am likely to find in my own shed?

2.4 Finding Mastitis: Tests and Case Definitions

Detection methods vary in what they measure. A cow-side test of the milk responds to the cell content of the milk and can be done by the farmer; automated cell counts are done in a laboratory and are used to monitor herds [18, 19]. Culture identifies the bacteria but takes time and laboratory facilities, and molecular methods identify them more precisely at higher cost. Figure 1 compares these approaches in qualitative terms, and a longer review of how mastitis is detected, managed and prevented is available [20].

	On the farm	Names the bacteria	Needs a laboratory	Relative cost
Cow-side test	Yes	No	No	Low
Cell count	Rarely	No	Usually	Moderate
Culture	Rarely	Yes	Yes	Moderate
Molecular test	No	Yes	Yes	Higher

Qualitative comparison; schematic matrix, not data

Figure 1. Qualitative comparison of common ways to detect mastitis (schematic matrix, not data).

None of these tests is a perfect measure of infection. Cell-based tests may flag a quarter that is inflamed but culture-negative and may miss an infected quarter whose cell count has not yet risen [21, 22]. Culture can miss bacteria that are shed intermittently, which is why definitions based on a single milk sample are less reliable than those that use more than one [3]. Inflammation itself can be measured in several ways, and the markers do not always agree [9].

The consequence is that “prevalence of mastitis” is not one quantity. It may mean cows with any positive culture, quarters with a high cell count, cows with a positive cow-side test, or cows treated by a farmer in the last year, and each gives a different answer. Epidemiologists distinguish apparent prevalence, which is what the test shows, from true prevalence, which is what is present, and the relation between the two depends on the test’s sensitivity and specificity [4, 23]. Such differences look small on paper, but they decide whether two numbers can sit in the same table.

2.5 Measuring Antimicrobial Susceptibility

Susceptibility testing asks whether a bacterium isolated from a sample is inhibited by a drug at a concentration that could in principle be reached in the tissue. The two broad approaches are diffusion of the drug from a disc into agar, giving a zone of inhibition, and dilution in broth or agar, giving a minimum inhibitory concentration [5, 24]. The detailed procedures of dilution testing are well documented, and their results depend on conditions that are easy to vary without noticing [25].

Turning a zone or a concentration into “susceptible” or “resistant” requires a breakpoint, and breakpoints are set by committees that weigh pharmacology, microbiology and clinical data; different committees have not always reached the

same answer, and efforts at harmonisation are recent [6, 26]. A study that does not say which breakpoints it used, and for which edition, leaves the reader unable to interpret its percentages. For mastitis there is a further difficulty, since breakpoints were often developed for infections at other body sites and their fit to the udder is debated.

A laboratory result is also not a prediction of cure. The likelihood that a given infection resolves depends on the cow, the pathogen and the way the animal is managed, as well as on the drug [27], and reviews of treatment stress that in-vitro susceptibility and clinical outcome do not always agree [28]. Some habits of treatment are better described as tradition than as evidence [29]. This review concerns susceptibility patterns as a description of bacteria and gives no advice on therapy.

2.6 Resistance: Mechanisms, Selection and Transfer

Resistance in mastitis bacteria is a story of selection as well as of mechanism. Reviews of antimicrobial resistance in mastitis pathogens describe the species and drug classes of concern and the ways resistance is acquired [30]. In *S. aureus*, the production of an enzyme that inactivates certain drugs is a long-established route, and methicillin resistance through an altered target is another. A methicillin-resistant lineage carrying a divergent form of the resistance gene was described in both human and bovine populations, a reminder that tests designed around the familiar mechanism may not recognise an unfamiliar one [31].

The link between how much antimicrobial is used on a farm and how resistant its bacteria are is plausible but hard to prove. Studies of drug use on dairy farms document how much practices vary [32], and work relating herd-level use to the susceptibility of Gram-positive mastitis bacteria shows how difficult it is to detect a clean relationship [33]. Longer series have looked at how susceptibility changes through time [34]. Surveillance programmes in Germany and Finland have reported both pathogen distributions and resistance side by side, giving a template for what a national picture can look like [35, 36].

Bacteria in a chronically infected quarter may not behave like the laboratory culture that is later tested. Biofilm-like growth has been proposed as one reason infections recur and resist cure [37], and the host's own defences determine whether a bacterium that enters the udder establishes infection at all [38]. Each of these means that an

in-vitro susceptibility figure describes an isolate on a plate and says less about an infection in a cow. Alternatives and adjuncts to conventional drugs are under study, but their evidence base is separate from the surveillance question considered here [39]. This is why the same herd can look troubled in one report and unremarkable in the next.

3. Materials and Methods

3.1 Scope and Question

The review asks two linked questions: what do published studies say about the prevalence of the main mastitis pathogens in smallholder dairy herds, and what do they say about the susceptibility of those pathogens to antimicrobials? It then asks a third, which is the one that determines how the first two should be read: how comparable are the figures, and how much does the way they were produced limit what a reader can infer for a particular herd? None of these steps is difficult, but each one is easy to skip.

3.2 Literature Assembly

The review draws on peer-reviewed work in four areas: the ecology and epidemiology of mastitis pathogens; diagnostic tests and case definitions; susceptibility testing and its interpretation; and general methodological literature on bias in surveys and in the evaluation of tests. Sources were chosen for relevance, citation in the field and the clarity of their methods. Studies from commercial herds and from other regions are used for what they teach about biology and method, not as measurements of smallholder prevalence.

We did not run a formal systematic search and we do not claim one. This is a narrative synthesis, and the selection reflects the authors' judgement. The practical consequence is that the review is a better guide to how a survey should be read than to the exact level of mastitis in any region, and it avoids quoting prevalence or resistance percentages from individual surveys, which could not be checked against the primary papers for this article.

3.3 Appraisal Framework

To judge how far a published figure can be carried, we used four questions. How was a case defined and by what test, and what is known about that test's errors [4, 23]? How were herds and animals chosen, and could the choice have favoured sick animals or sick herds [40, 41]? For susceptibility, what method and which breakpoints were used, and for which drugs [24, 26]? And did the report give enough detail for another group to repeat it? Table 2 lists the sources of variation we looked for

in prevalence studies.

4. Results

4.1 What a Prevalence Figure Can and Cannot Say

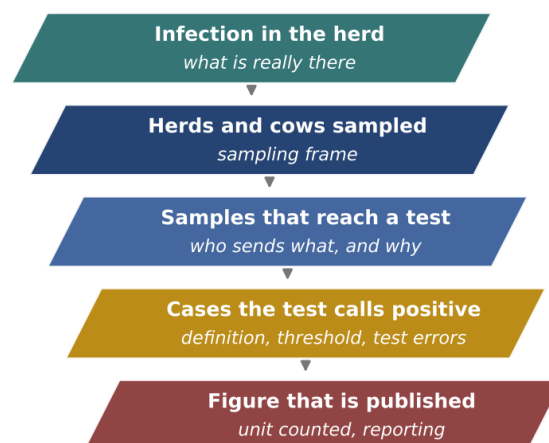
Read against these questions, published prevalence figures turn out to depend on choices that are often unstated. Subclinical infection recorded by a cell-based test, infection recorded by culture, and clinical cases recorded by farmers are different outcomes and they are reported under the same name [3, 9]. The unit matters too, since a percentage of cows, of quarters and of herds are three different things, and the herd-level figure can be high when only a few cows in each herd are affected.

Table 2. Why reported mastitis prevalence differs between studies.

Source of variation	Effect on the estimate	What a report should state
Case definition	Clinical, subclinical, any infection, or any positive test give different totals [3, 9]	The exact definition used
Unit counted	Cow, quarter, herd or milk sample are not interchangeable	Unit of analysis and denominator
Screening test	Cow-side and cell-count tests miss some infections and flag some uninfected quarters [21, 22]	Test, threshold and any culture confirmation
Apparent versus true prevalence	Test errors shift the observed value away from the real one [4, 23]	Whether any correction was applied
Sampling frame	Herds that volunteer or are visited by a vet are not a random draw [40, 41]	How herds and cows were chosen
Timing	Stage of lactation, season and recent treatment change what is found [42]	Dates and stage of lactation

Note: Drawn from the cited methodological literature; the table does not rank sources of variation by size.

The sampling frame matters as much. A survey of herds that have joined a project, or of cows presented to a clinic, is likely to include more disease than one of herds chosen at random. The statistical literature on selection bias, written for hospital studies, describes the same trap that arises when the sample is made up of those who came forward [40, 41]. In smallholder research it is common to sample where access is easiest, which is understandable and should be stated. Figure 2 sketches the layers between an infected quarter and a published figure.



Each layer can change the number; schematic, not data

Figure 2. Layers between an infected quarter and a published prevalence figure (schematic, not data).

Timing completes the picture. The occurrence of clinical mastitis differs between herds and over the year, and measuring it as new cases over time, as large surveys have done, gives something different from counting positive animals on a single visit [42]. A survey carried out in one season in one district speaks for that season and district. Read this way, a low count from a small survey is not evidence that the problem is small.

4.2 Patterns Among the Pathogens

Despite the difficulties, some broad patterns recur in the literature and can be stated qualitatively. *S. aureus* is consistently found in surveys of mastitis from many regions, including those from smallholder herds [10, 17]. Streptococci and coliform bacteria appear in varying proportions that seem to reflect housing and hygiene [8, 11]. Coagulase-negative staphylococci are widely isolated in surveys of subclinical infection [14, 43].

Which of these dominates in a particular herd is a matter of local management and chance, and a pooled picture can mislead. A few herds with a persistent contagious infection can lift a regional average, while many herds with sporadic environmental cases can lower it. Smallholder surveys with few herds are especially exposed to this effect, since one unusual herd carries a large share of the data. The honest description is that the main groups are well known and the local mix is not.

4.3 Patterns of Susceptibility

Susceptibility patterns share the same difficulty with an additional one. The surveillance reports that exist, from countries with organised programmes, give a template: isolates identified to species, drugs listed, method and standard stated

[35, 36]. Even there, resistance varies by species and by drug, and conclusions about “mastitis bacteria” in general hide the differences that matter [30, 34]. Table 3 sets out why results from different studies seldom line up.

The origin of isolates deserves emphasis. In many places, a milk sample reaches a laboratory only when treatment has failed or the case is severe. Isolates from such cases are likely to be more resistant than those from untreated, mild or subclinical infection, so a laboratory’s resistance percentages describe the cases that were sent, not the herds from which they came [40, 41]. Where the only available data come from a referral laboratory, they set an upper reference, not an estimate of what is circulating in farms. A laboratory that is told why a sample was taken can often help more than one that is not.

Table 3. Why reported susceptibility patterns differ between studies.

Source of variation	Consequence	Remedy
Test method	Disc diffusion and dilution methods answer related but not identical questions [5, 25]	Name the method and the guideline edition
Breakpoints	Different bodies set different cut-offs, so the same zone or MIC can be read differently [6, 26]	State the breakpoint source for each drug
Drug panel	Drugs tested vary, and absent drugs cannot be compared	List every drug tested
Sample origin	Isolates sent after treatment failure overstate resistance [40, 41]	Describe why samples were taken
Species mix	Pooling species hides large differences between them [30, 34]	Report by species
Link to cure	Laboratory result and clinical outcome often disagree [27, 28]	Do not read a result as a prediction of cure

Note: Qualitative. The table lists reasons for caution when reading a figure and does not say which laboratories are more or less reliable.

A further complication is that drug use and resistance are intertwined. Where drugs are used heavily, selection favours resistant strains, but the relationship is rarely linear and is hard to separate from differences in herds and in sampling [32, 33]. In smallholder settings, where drugs may be bought without a prescription and given without a diagnosis, the use data needed to interpret resistance data may not exist at all. Without them, a high figure for resistance can neither be attributed to use nor excluded as a result of it.

4.4 Quality of the Evidence

Taken as a body, the evidence on smallholder mastitis shares a set of limitations that are not unique to any one paper. Studies are few relative to the number of production systems; sample sizes are modest; case definitions and tests are heterogeneous; and the methods for susceptibility testing are not always described to a standard that would let another laboratory reproduce them [4, 26]. These limitations are common across veterinary epidemiology and are not a criticism of the cited authors, who often worked with scarce resources in difficult conditions.

Some of the strongest evidence in the field comes from sources that were not designed for smallholders at all, which is a reason to treat the biological claims (how pathogens spread, how tests misclassify) as more secure than the quantitative ones (how much and how often). Put plainly, the literature supports a cautious statement. The main pathogen groups are well described, the reasons that figures differ are well understood, and the local prevalence and susceptibility in smallholder herds are not established by evidence that can be compared across studies. That is a modest claim, and it is the one the evidence will currently bear.

4.5 Reading a Reported Figure

A reader meeting a prevalence or resistance figure can ask a short sequence of questions. Who was sampled, and how? What counted as a case? Which test, at what threshold? For susceptibility, which method, which breakpoints, which drugs, and why were the isolates collected? Figure 3 arranges these questions as a decision tree. A report that answers all of them is rare, and one that answers none should be read as an observation and not as an estimate.

The same questions are useful for the authors of a survey. If a report can state the case definition, the unit counted, the test and its threshold, the method of selecting herds, the laboratory method and the breakpoints, then another group can set its own results beside it. If it cannot, the figures may still be useful locally, but they cannot be combined with others. A short checklist of this kind costs a reviewer a few minutes, and it separates the surveys that can be built on from those that can only be admired. A figure that cannot be traced to a stated method is a rumour with a decimal point, however carefully it is printed.

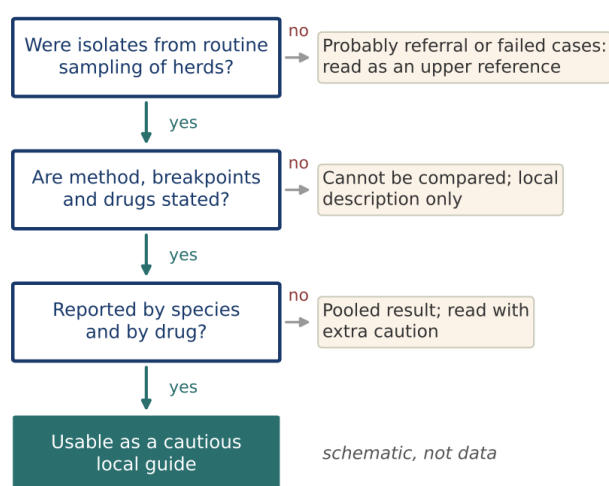


Figure 3. Questions to ask of a reported susceptibility result (schematic decision tree, not data).

5. Discussion

5.1 Interpretation and Practical Reading

The main finding of this review is a conditional one. The ecology of mastitis pathogens is well described, the logic of testing is well established, and the sources of disagreement between surveys are known. What is missing is not understanding but comparable local data. A figure for prevalence or resistance should therefore be read as a statement about a particular sample, tested in a particular way, and not as a property of smallholder dairying in general. Nothing in this reading argues against surveillance; it argues for surveillance that says how it was done.

The second conditional concerns use. Local figures matter most to the people least able to produce them: the household that needs to know whether a cow with a hard quarter is likely to have a contagious pathogen, and the adviser who must decide what to recommend. Both are better served by a modest, clearly described local survey than by a borrowed national figure, and by a laboratory that reports methods along with results [20, 39].

A third point is about interpretation of resistance. A resistance percentage can be read as an alarm or as reassurance, and either reading may be unjustified. The referral bias that inflates it, the breakpoints that move it and the species pooling that blurs it all point the same way: the number needs context before it can guide any decision, and the decision itself belongs to a veterinarian who has examined the animal. These cautions apply equally to any bacterial disease reported from a few small herds, not only to mastitis.

Three kinds of evidence would change this reading. A well-designed survey of randomly

selected smallholder herds, using stated definitions and a stated method, would give a genuinely local baseline. A paired comparison of culture-based and cell-based case definitions in the same herds would show how much of the apparent variation between studies is a matter of definition [3, 21]. And a surveillance series of susceptibility data from routine, not only failed, cases would show how far referral bias distorts the usual picture [34, 35].

5.2 Limitations

This is a narrative review and shares the limitations of that form. The selection of sources reflects judgement, no formal search or quality scoring was done, and the review does not quote prevalence or resistance figures from individual surveys, because these could not be checked against the primary reports for this article. A reader who needs a number should consult the original studies directly, applying the appraisal questions above, or wait for a formal systematic review with meta-analysis of studies that are sufficiently alike to be combined.

The review also leans on evidence from commercial herds and from other regions to explain how pathogens and tests behave. Those studies are imperfect proxies for smallholder herds, where housing, milking practice, feeding and access to services differ. We have used them to frame hypotheses and to explain method, and the conclusions should be read in that light. Reviews of this kind are more useful when they say where the evidence stops than when they smooth it over.

5.3 Implications for Study Design

A survey that could serve both science and the farms it describes would define its case at the outset and report the definition in full, count at a stated unit, and select herds in a way that can be described, with random selection where it is feasible and an honest account of the compromise where it is not. It would record the stage of lactation and recent treatment, and sample more than once where possible, since a single sample misses intermittent shedders [3]. Where the test is imperfect, it would say so and, if the performance of the test is known, report the corrected estimate alongside the apparent one [23].

On the laboratory side, a study would identify isolates to species, state the method and the interpretive standard with its edition, list every drug tested, report results by species, and say why each sample was taken [5, 26]. It would also record, where it can, the drugs the herd has used

recently, since that is the context in which susceptibility makes sense [32]. These requirements are demanding but not exotic, and the best of the existing surveillance reports already meet most of them.

5.4 Directions for Future Work

Three needs stand out. First, a formal systematic review of mastitis prevalence and susceptibility in smallholder herds, with a transparent search and a structured account of how each included study defined its outcome. Second, well-designed local surveys, ideally repeated, that use comparable definitions so that the studies can be combined. Third, work on the economics and decision-making of smallholders themselves, because the value of better data depends on what households can do with them [2, 7].

5.5 Practical Reading for Advisers and Smallholders

For an adviser, the sensible position is neither to dismiss published figures nor to treat them as local truth. When a farmer asks what is likely to be causing the mastitis in a herd, the useful answer begins with what is known about the main pathogen groups and about how they are spread, and goes on to recommend finding out what is present on this farm. Even a small number of properly collected samples, accompanied by a note on why they were collected, can be more informative locally than a regional percentage.

Such local sampling is not a substitute for a survey, and it should not be reported as one. It can nonetheless tell a household which pathogens are present, which groups of cows are affected and whether a problem is spread by milking or arises from the surroundings [10, 44]. Its value lies in local decision-making, and the broader question of regional prevalence and susceptibility will be settled only by studies and syntheses built to be compared.

6. Conclusion

Bovine mastitis in smallholder dairy herds is common, costly and poorly measured. The pathogens responsible are well known as groups, and the reasons that prevalence and susceptibility figures differ are well understood, but neither the local level of infection nor the local pattern of resistance is established by evidence that can be compared across studies. What the literature does show is how a figure is produced, how it can mislead, and what a report would need to state for its figures to be used with confidence. Readers should treat headline percentages with caution

until they can see the definition, the sample and the method behind them.

For the practitioner the message is practical rather than discouraging. Published patterns can frame the questions to ask, and a modest local investigation can answer them for one farm. For the researcher, the opportunity is a set of carefully described surveys of smallholder herds that report their definitions, samples and laboratory methods in enough detail to be set side by side, turning scattered observations into a body of evidence that smallholders can rely on.

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9. Conflict of Interest

The authors declare no conflict of interest.

10. Author Contributions

C.L. conceived the review and wrote the sections on herd epidemiology and study design. A.v.d.H. wrote the sections on laboratory methods and susceptibility testing. M.C. wrote the sections on pathogen ecology and diagnostics. All authors revised the manuscript and approved the final version.

11. Ethics Statement

This is a review of published literature. No animals were used and no ethical approval was required.

12. Data Availability

No new data were generated. All sources are cited in the reference list.

13. References

- [1] Halasa, T., Huijps, K., Østerås, O., & Hogeveen, H. (2007). Economic effects of bovine mastitis and mastitis management: A review. *Veterinary Quarterly*, 29(1), 18-31. DOI: [10.1080/01652176.2007.9695224](https://doi.org/10.1080/01652176.2007.9695224)
- [2] Hogeveen, H., Huijps, K., & Lam, T. J. G. M. (2011). Economic aspects of mastitis: New developments. *New Zealand Veterinary Journal*, 59(1), 16-23. DOI: [10.1080/00480169.2011.547165](https://doi.org/10.1080/00480169.2011.547165)
- [3] Dohoo, I., Smith, J., Andersen, S., Kelton, D., & Godden, S. (2011). Diagnosing intramammary infections: Evaluation of definitions based on a single milk sample. *Journal of Dairy Science*,

- 94(1), 250-261. DOI: [10.3168/jds.2010-3559](https://doi.org/10.3168/jds.2010-3559)
- [4] Greiner, M., & Gardner, I. A. (2000). Epidemiologic issues in the validation of veterinary diagnostic tests. *Preventive Veterinary Medicine*, 45(1-2), 3-22. DOI: [10.1016/S0167-5877\(00\)00114-8](https://doi.org/10.1016/S0167-5877(00)00114-8)
- [5] Jorgensen, J. H., & Ferraro, M. J. (2009). Antimicrobial susceptibility testing: A review of general principles and contemporary practices. *Clinical Infectious Diseases*, 49(11), 1749-1755. DOI: [10.1086/647952](https://doi.org/10.1086/647952)
- [6] Turnidge, J., & Paterson, D. L. (2007). Setting and revising antibacterial susceptibility breakpoints. *Clinical Microbiology Reviews*, 20(3), 391-408. DOI: [10.1128/CMR.00047-06](https://doi.org/10.1128/CMR.00047-06)
- [7] Huijps, K., Lam, T. J., & Hogeveen, H. (2008). Costs of mastitis: Facts and perception. *Journal of Dairy Research*, 75(1), 113-120. DOI: [10.1017/S0022029907002932](https://doi.org/10.1017/S0022029907002932)
- [8] Bradley, A. J. (2002). Bovine mastitis: An evolving disease. *The Veterinary Journal*, 164(2), 116-128. DOI: [10.1053/tvjl.2002.0724](https://doi.org/10.1053/tvjl.2002.0724)
- [9] Pyörälä, S. (2003). Indicators of inflammation in the diagnosis of mastitis. *Veterinary Research*, 34(5), 565-578. DOI: [10.1051/vetres:2003026](https://doi.org/10.1051/vetres:2003026)
- [10] Keefe, G. (2012). Update on control of *Staphylococcus aureus* and *Streptococcus agalactiae* for management of mastitis. *Veterinary Clinics of North America: Food Animal Practice*, 28(2), 203-216. DOI: [10.1016/j.cvfa.2012.03.010](https://doi.org/10.1016/j.cvfa.2012.03.010)
- [11] Hogan, J., & Smith, K. L. (2003). Coliform mastitis. *Veterinary Research*, 34(5), 507-519. DOI: [10.1051/vetres:2003019](https://doi.org/10.1051/vetres:2003019)
- [12] Zadoks, R. N., Middleton, J. R., McDougall, S., Katholm, J., & Schukken, Y. H. (2011). Molecular epidemiology of mastitis pathogens of dairy cattle and comparative relevance to humans. *Journal of Mammary Gland Biology and Neoplasia*, 16(4), 357-372. DOI: [10.1007/s10911-011-9236-y](https://doi.org/10.1007/s10911-011-9236-y)
- [13] Pyörälä, S., & Taponen, S. (2009). Coagulase-negative staphylococci—Emerging mastitis pathogens. *Veterinary Microbiology*, 134(1-2), 3-8. DOI: [10.1016/j.vetmic.2008.09.015](https://doi.org/10.1016/j.vetmic.2008.09.015)
- [14] Sampimon, O. C., Barkema, H. W., Berends, I. M. G. A., Sol, J., & Lam, T. J. G. M. (2009). Prevalence and herd-level risk factors for intramammary infection with coagulase-negative staphylococci in Dutch dairy herds. *Veterinary Microbiology*, 134(1-2), 37-44. DOI: [10.1016/j.vetmic.2008.09.010](https://doi.org/10.1016/j.vetmic.2008.09.010)
- [15] Karimuribo, E. D., Fitzpatrick, J. L., Bell, C. E., Swai, E. S., Kambarage, D. M., Ogden, N. H., ... French, N. P. (2006). Clinical and subclinical mastitis in smallholder dairy farms in Tanzania: Risk, intervention and knowledge transfer. *Preventive Veterinary Medicine*, 74(1), 84-98. DOI: [10.1016/j.prevetmed.2005.10.006](https://doi.org/10.1016/j.prevetmed.2005.10.006)
- [16] Mdegela, R. H., Ryoba, R., Karimuribo, E. D., Phiri, E. J., Løken, T., Reksen, O., ... Urio, N. A. (2009). Prevalence of clinical and subclinical mastitis and quality of milk on smallholder dairy farms in Tanzania. *Journal of the South African Veterinary Association*, 80(3), 163-168. DOI: [10.4102/jsava.v80i3.195](https://doi.org/10.4102/jsava.v80i3.195)
- [17] Abebe, R., Hatiya, H., Abera, M., Megersa, B., & Asmare, K. (2016). Bovine mastitis: Prevalence, risk factors and isolation of *Staphylococcus aureus* in dairy herds at Hawassa milk shed, South Ethiopia. *BMC Veterinary Research*, 12, 270. DOI: [10.1186/s12917-016-0905-3](https://doi.org/10.1186/s12917-016-0905-3)
- [18] Schukken, Y. H., Wilson, D. J., Welcome, F., Garrison-Tikofsky, L., & Gonzalez, R. N. (2003). Monitoring udder health and milk quality using somatic cell counts. *Veterinary Research*, 34(5), 579-596. DOI: [10.1051/vetres:2003028](https://doi.org/10.1051/vetres:2003028)
- [19] Viguier, C., Arora, S., Gilmartin, N., Welbeck, K., & O'Kennedy, R. (2009). Mastitis detection: Current trends and future perspectives. *Trends in Biotechnology*, 27(8), 486-493. DOI: [10.1016/j.tibtech.2009.05.004](https://doi.org/10.1016/j.tibtech.2009.05.004)
- [20] Ruegg, P. L. (2017). A 100-Year Review: Mastitis detection, management, and prevention. *Journal of Dairy Science*, 100(12), 10381-10397. DOI: [10.3168/jds.2017-13023](https://doi.org/10.3168/jds.2017-13023)
- [21] Sargeant, J. M., Leslie, K. E., Shirley, J. E., Pulkrabek, B. J., & Lim, G. H. (2001). Sensitivity and specificity of somatic cell count and California Mastitis Test for identifying intramammary infection in early lactation. *Journal of Dairy Science*, 84(9), 2018-2024. DOI: [10.3168/jds.S0022-0302\(01\)74645-0](https://doi.org/10.3168/jds.S0022-0302(01)74645-0)
- [22] Dohoo, I. R., & Leslie, K. E. (1991). Evaluation of changes in somatic cell counts as indicators of new intramammary infections. *Preventive Veterinary Medicine*, 10(3), 225-237. DOI: [10.1016/0167-5877\(91\)90006-N](https://doi.org/10.1016/0167-5877(91)90006-N)
- [23] Rogan, W. J., & Gladen, B. (1978). Estimating prevalence from the results of a screening test. *American Journal of Epidemiology*, 107(1), 71-76. DOI: [10.1093/oxfordjournals.aje.a112510](https://doi.org/10.1093/oxfordjournals.aje.a112510)
- [24] Andrews, J. M. (2001). Determination of minimum inhibitory concentrations. *Journal of Antimicrobial Chemotherapy*, 48(Suppl. 1), 5-16. DOI: [10.1093/jac/48.suppl_1.5](https://doi.org/10.1093/jac/48.suppl_1.5)
- [25] Wiegand, I., Hilpert, K., & Hancock, R. E. W. (2008). Agar and broth dilution methods to determine the minimal inhibitory concentration (MIC) of antimicrobial substances. *Nature Protocols*, 3(2), 163-175. DOI: [10.1038/nprot.2007.521](https://doi.org/10.1038/nprot.2007.521)
- [26] Kahlmeter, G., Brown, D. F. J., Goldstein, F. W., MacGowan, A. P., Mouton, J. W., Österlund, A., ... Vatopoulos, A. (2003). European harmonization of MIC breakpoints for antimicrobial susceptibility testing of bacteria. *Journal of Antimicrobial Chemotherapy*, 52(2), 145-148. DOI: [10.1093/jac/dkg312](https://doi.org/10.1093/jac/dkg312)
- [27] Barkema, H. W., Schukken, Y. H., & Zadoks, R. N. (2006). Invited review: The role of cow, pathogen, and treatment regimen in the therapeutic success of bovine *Staphylococcus aureus* mastitis. *Journal of Dairy Science*, 89(6), 1877-1895. DOI:

- [10.3168/jds.S0022-0302\(06\)72256-1](https://doi.org/10.3168/jds.S0022-0302(06)72256-1)
- [28] Barlow, J. (2011). Mastitis therapy and antimicrobial susceptibility: A multispecies review with a focus on antibiotic treatment of mastitis in dairy cattle. *Journal of Mammary Gland Biology and Neoplasia*, 16(4), 383–407. DOI: [10.1007/s10911-011-9235-z](https://doi.org/10.1007/s10911-011-9235-z)
- [29] Hillerton, J. E., & Berry, E. A. (2005). Treating mastitis in the cow—a tradition or an archaism. *Journal of Applied Microbiology*, 98(6), 1250–1255. DOI: [10.1111/j.1365-2672.2005.02560.x](https://doi.org/10.1111/j.1365-2672.2005.02560.x)
- [30] Oliver, S. P., & Murinda, S. E. (2012). Antimicrobial resistance of mastitis pathogens. *Veterinary Clinics of North America: Food Animal Practice*, 28(2), 165–185. DOI: [10.1016/j.cvfa.2012.03.005](https://doi.org/10.1016/j.cvfa.2012.03.005)
- [31] García-Álvarez, L., Holden, M. T. G., Lindsay, H., Webb, C. R., Brown, D. F. J., Curran, M. D., ... Holmes, M. A. (2011). Meticillin-resistant *Staphylococcus aureus* with a novel *mecA* homologue in human and bovine populations in the UK and Denmark: A descriptive study. *The Lancet Infectious Diseases*, 11(8), 595–603. DOI: [10.1016/S1473-3099\(11\)70126-8](https://doi.org/10.1016/S1473-3099(11)70126-8)
- [32] Saini, V., McClure, J. T., Léger, D., Dufour, S., Sheldon, A. G., Scholl, D. T., & Barkema, H. W. (2012). Antimicrobial use on Canadian dairy farms. *Journal of Dairy Science*, 95(3), 1209–1221. DOI: [10.3168/jds.2011-4527](https://doi.org/10.3168/jds.2011-4527)
- [33] Pol, M., & Ruegg, P. L. (2007). Relationship between antimicrobial drug usage and antimicrobial susceptibility of gram-positive mastitis pathogens. *Journal of Dairy Science*, 90(1), 262–273. DOI: [10.3168/jds.S0022-0302\(07\)72627-9](https://doi.org/10.3168/jds.S0022-0302(07)72627-9)
- [34] Erskine, R. J., Walker, R. D., Bolin, C. A., Bartlett, P. C., & White, D. G. (2002). Trends in antibacterial susceptibility of mastitis pathogens during a seven-year period. *Journal of Dairy Science*, 85(5), 1111–1118. DOI: [10.3168/jds.S0022-0302\(02\)74172-6](https://doi.org/10.3168/jds.S0022-0302(02)74172-6)
- [35] Tenhagen, B.-A., Köster, G., Wallmann, J., & Heuwieser, W. (2006). Prevalence of mastitis pathogens and their resistance against antimicrobial agents in dairy cows in Brandenburg, Germany. *Journal of Dairy Science*, 89(7), 2542–2551. DOI: [10.3168/jds.S0022-0302\(06\)72330-X](https://doi.org/10.3168/jds.S0022-0302(06)72330-X)
- [36] Pitkälä, A., Haveri, M., Pyörälä, S., Myllys, V., & Honkanen-Buzalski, T. (2004). Bovine mastitis in Finland 2001—prevalence, distribution of bacteria, and antimicrobial resistance. *Journal of Dairy Science*, 87(8), 2433–2441. DOI: [10.3168/jds.S0022-0302\(04\)73366-4](https://doi.org/10.3168/jds.S0022-0302(04)73366-4)
- [37] Melchior, M. B., Vaarkamp, H., & Fink-Gremmels, J. (2006). Biofilms: A role in recurrent mastitis infections? *The Veterinary Journal*, 171(3), 398–407. DOI: [10.1016/j.tvjl.2005.01.006](https://doi.org/10.1016/j.tvjl.2005.01.006)
- [38] Rainard, P., & Riollot, C. (2006). Innate immunity of the bovine mammary gland. *Veterinary Research*, 37(3), 369–400. DOI: [10.1051/vetres:2006007](https://doi.org/10.1051/vetres:2006007)
- [39] Gomes, F., & Henriques, M. (2016). Control of bovine mastitis: Old and recent therapeutic approaches. *Current Microbiology*, 72(4), 377–382. DOI: [10.1007/s00284-015-0958-8](https://doi.org/10.1007/s00284-015-0958-8)
- [40] Berkson, J. (1946). Limitations of the application of fourfold table analysis to hospital data. *Biometrics Bulletin*, 2(3), 47–53. DOI: [10.2307/3002000](https://doi.org/10.2307/3002000)
- [41] Sackett, D. L. (1979). Bias in analytic research. *Journal of Chronic Diseases*, 32(1–2), 51–63. DOI: [10.1016/0021-9681\(79\)90012-2](https://doi.org/10.1016/0021-9681(79)90012-2)
- [42] Olde Riekerink, R. G. M., Barkema, H. W., Kelton, D. F., & Scholl, D. T. (2008). Incidence rate of clinical mastitis on Canadian dairy farms. *Journal of Dairy Science*, 91(4), 1366–1377. DOI: [10.3168/jds.2007-0757](https://doi.org/10.3168/jds.2007-0757)
- [43] Piepers, S., De Meulemeester, L., de Kruif, A., Opsomer, G., Barkema, H. W., & De Vlieghe, S. (2007). Prevalence and distribution of mastitis pathogens in subclinically infected dairy cows in Flanders, Belgium. *Journal of Dairy Research*, 74(4), 478–483. DOI: [10.1017/S0022029907002841](https://doi.org/10.1017/S0022029907002841)
- [44] Dufour, S., Fréchette, A., Barkema, H. W., Mussell, A., & Scholl, D. T. (2011). Invited review: Effect of udder health management practices on herd somatic cell count. *Journal of Dairy Science*, 94(2), 563–579. DOI: [10.3168/jds.2010-3715](https://doi.org/10.3168/jds.2010-3715)