Somatic cell count thresholds for the diagnostic of subclinical mastitis

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1 Introduction

When using somatic cell count (SCC) as a diagnostic test for mastitis, a threshold is generally chosen above which cows are categorised as infected and below which they are considered to be mastitis free. This is a very rough approximation since there seems to be a large overlap in the distributions of SCC between infected and uninfected, as diagnosed by bacteriology which is considered to be the gold standard for mastitis. Although we think that, within a certain range, the choice of a particular threshold is relatively unimportant as long as everybody uses the same, the cut-off 200,000 cells/mL is sometimes objected to by reviewers or academics in conferences. This is a legitimate question and we would like to justify our choice.

The aim of the choice of a particular threshold deserves to be asked. It can be argued that the elimination of any infection regardless of the consequences for the cow or the farmer may not always be suitable. Instead, the detrimental consequences of infections by different pathogens should be considered. The consequences of an IMI for the cow are the local and general symptoms resulting in discomfort or pain. Further to these, there are consequences for the farm’s profitability such as reduction in milk yield, cost of treatment . . . On the other hand, the cost of prevention depends on what we want to prevent. If as
has been found in some studies, infections by minor pathogens could prevent certain types of mastitis, specifically preventing and curing infections by minor pathogens would be responsible for the cost of prevention for these infections as well as to the cost of cure of additional infections by major pathogens. In this respect, whether infections by all minor pathogens should be eliminated remains unresolved. This of importance since minor pathogens typically elicit weaker immune responses than major pathogens and therefore lower SCCs.

2 Infection and somatic cell count

2.1 Somatic cell count and bacteriology

The presence of a pathogen in the mammary gland triggers an influx of immune cells, mostly neutrophils, to combat the infection. Therefore, an elevated cell concentration can be used as an indicator of infection. The first problem is that the cell concentration in uninfected quarters is not known precisely. In a review of 21 articles published 1971 and 2000, Djabri et al. (2002) estimated the geometric mean somatic cell count of uninfected quarters to be of 68,000 cells/mL (Figure 1). But in the UK and the Netherlands, the same mode of 22,000 cells/mL has been found for the distribution of SCC, regardless of the bacteriological status (Madouasse et al., 2010; ten Napel et al., 2009). Therefore, the most frequent SCC value is approximately 3 times lower than the estimated mean of 68,000 cells/mL. The difficulty in estimating SCC in uninfected cows comes from the limitation of bacteriology as a gold standard. Here are some possible reasons for that: no bacteria will grow when the bacteria has been successfully eliminated but the inflammatory process has not been terminated, the media used does not allow to grow the bacteria causing the infection, there is no bacteria in the milk sample collected although an IMI is present as in the case of intermittent shedding (Sears et al., 1990).

The implicit assumption is that following infection, SCC increases quickly and then remains elevated as long as the bacteria is present. It is not entirely
clear as to what extent this is the case. For example, coliform infections are known to be cleared very quickly and SCC could be elevated when the bacteria cannot be grown from the milk. This is likely to lead to misdiagnose quarters that have just recovered from an infection or that shed bacteria intermittently as non infected on the basis of bacteriology thereby overestimating SCC in healthy cows and could explain some of the bacteriology negative cases of clinical mastitis (Morin & Constable, 1998). Infection certainly increases SCC, and to a different extent depending of the aetiological agent. This was also investigated in Djabri et al. (2002) (Figure 1).

2.2 Somatic cell count distribution(s)

But mean SCC is not the whole story. There is a certain variability around this mean, and the spread of this variability could be non trivial. It appears that SCC is not normally distributed. The distribution reaches its mode very quickly,
between 20,000 and 25,000 cells/mL, and then the number of readings per class decreases, first very rapidly and then slowly to values that can be higher than 10 million cells/mL. It has been found to be better described by a log normal distribution (Ali & Shook, 1980). This is also the justification for the use of the geometric mean instead of the arithmetic mean.

In essence, the distribution we observe can be seen as a mixture of recordings from uninfected cows as well as cows infected by various pathogens in 1 to 4 quarters in various combinations. This can be described by a type of models called mixture models. The idea behind these models is to model a certain distribution as the combination of other parametric distributions. One can therefore imagine describing the distribution of SCC as the combination of the SCC distribution of SCC for uninfected quarters or cows depending on the level of study and of one or more distributions for different categories of infections. There have been various attempts at using this technique for SCC and mastitis. One of the latest published articles on the subject was by ten Napel et al. (2009). They found a combination of normal, log-normal and exponential distributions to explain most of the observed variation. This combination described the observed distribution rather well. However, the individual distributions did not correspond to the different infection status. Another combination of 4 normal distributions and an exponential distribution which seemed to cluster infected and uninfected cows was preferred (Figure 2). Infections by major pathogens were found mostly in the 4th normal and exponential distributions.

3 The minor pathogens problem

Mastitis causing agents can be divided in 2 broad categories that are minor and major pathogens. Minor pathogens include Corynebacterium bovis and coagulase negative staphylococci (CNS) while all other bacteria are considered to be major pathogens. This classification is based on the degree of inflammation induced by the infection by bacteria of these categories. Minor pathogens generally induce less severe inflammation than major pathogens as measured by SCC
Figure 2: Mixture of distributions describing cow SCC in the Netherlands as identified in ten Napel et al. (2009). The overall distribution is a combination of 4 normal and one exponential distributions. Infected cows are mostly in the 4th normal and exponential distributions.
(Schepers et al., 1997; Laevens et al., 1997; Sargeant et al., 2001) and rarely result in clinical mastitis (Supr et al., 2011). Several authors have found infection by minor pathogens to be protective against infection by major pathogens (Pankey et al., 1985; Rainard & Poutrel, 1988; Lam et al., 1997). Davidson et al. (1992) found no difference in the risk of subsequent infection by S. aureus between cows that were uninfected or infected by CNS. On the other hand, Suriyasathaporn et al. (2000) found cows with a very low SCC to be at an increased risk of clinical mastitis.

Higher SCCs are associated with lower milk productions (Hortet, 1999; Halasa et al., 2009) and this effect has been found for SCC levels as low as 50,000 cells/mL (Hortet & Seegers, 1998). However studies that looked at milk production in cows infected with minor pathogens found no difference in milk production with uninfected cows (Paradis et al., 2010) or even a slightly higher production (Schukken et al., 2009). The apparent discrepancy could be due to the fact that studies that rely on SCC and yield alone usually use much more data than studies with bacteriological data which could increase their power and thereby their ability to detect small differences. Another difficulty with all studies is to account for dilution.

4 Factors of variation of SCC

In a national UK dataset, we found individual cow SCC to vary importantly with stage of lactation and parity. This has been observed repeatedly. The lactation curve for SCC seems to have an inverse shape to the curve for milk yield. There is a sharp decrease at the start of lactation (Dohoo, 1993; Sargeant et al., 2001) which is followed by a gradual increase towards the end of lactation. On the other hand, the geometric mean SCC increases with successive parities. There are two possible explanations for these variations: either they are physiologic or they result from variations in infection prevalence that in turn affects SCC. There is no consensus as to the relative importance of these 2 competing explanations. For example, of 2 studies using Dutch data published in 1997,
one found no significant effect of parity, stage of lactation and their interaction
on SCC in bacteriology negative cows (Laevens et al., 1997), the other reported
the interaction between stage of lactation and parity to be significant (Schepers
et al., 1997). Sargeant et al. (2001) observed a decrease in SCC between calving
and 10 days in milk. This decrease was more pronounced in cows infected with
a major pathogen than in other cows.

One could assume that clinical mastitis can be considered to have a specific-
ity close to 100% and use its incidence as a way of measuring the variation in
mastitis prevalence. But at the herd level, the prevalence of clinical mastitis is
not strictly correlated with the prevalence of high SCC. Beaudeau et al. (2002)
found herds with a high prevalence of SCC > 50,000 cells/mL to have a higher
risk of clinical mastitis. Therefore, some care must be taken when looking at
the data in this way. It remains that the incidence of clinical mastitis is higher
at the start of lactation (Andersen et al., 2011; Green et al., 2004; Valde et al.,
2004) and increases with parity. It is likely that part of the high values observed
at the beginning of lactation can be attributed to IMI.

A non infectious factor that has consistently been associated with SCC is
dilution. Emanuelson & Funke (1991) noted a decrease of 11% in BMSCC
per 1,000 kg increase in milk production. Green et al. (2006) modelled the
dilution effect as linear and the decrease in yield associated with SCC resulting
from infection as log linear. More recently, this effect has been modelled using
mixture models by Jamrozik & Schaeffer (2010). They observed a decrease of
0.15 point of somatic cell score per kg of milk. Therefore, the fact that the
lactation curve for SCC has a shape that is inverse to the shape of the yield
curve could be partly explained by the dilution effect.

5 Thresholds for SCC

Although IMI is the main factor of increase for SCC, there appears to be an
important variability for this parameter in uninfected cows. It is however likely
that SCC in truly uninfected cows is below 100,000 cells/mL. But detrimental
Figure 3: Geometric mean SCC per day in milk in the first 10 days post-calving in 131 dairy cows from 3 North American herds (Sargeant et al., 2001).
consequences associated with infection are only potent for infections by major pathogens. Although very common, minor pathogens contribute very little to the bulk milk SCC, and milk production in affected cows does not seem to be impaired. Therefore, having a target threshold that is too low could result in costs of cure and prevention that are unnecessarily high.

At the cow level a different threshold can be chosen depending on specific objectives. The main advantage for the use of a particular value at the herd level lies in the possibility it offers to make comparisons. These comparisons can be made within herd in order to follow the evolution of IMI over time, but also between herds. A threshold of 200,000 cells/mL, although not universally right, has the advantage of having been used repeatedly in the literature. Thus, there are numerous studies on the sensitivities and specificities of this threshold in various contexts, references for the proportion of various cow populations on each side of this threshold, susceptibility of cows to mastitis according to whether they are below or above this threshold.

References


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