Mastitis Newsletter aims mainly at disseminating succinct information on the work, plans and achievements of an IDF Group of Experts dealing with Bovine Mastitis (Group A2)*, but also includes information available to the Group from other sources such as the National Mastitis Council (NMC) in the USA. Group A2 and the NMC have a close working relationship.

Mastitis Newsletter does not intend to systematically review the vast literature in the field of mastitis nor does it claim to report on all significant developments in the field. Information given and statements made in Mastitis Newsletter do not commit Group A2. They can be reproduced, with indication of source.

Contributions dealing with items of general interest would be welcome for consideration for inclusion in future issues.

Mastitis Newsletter is available free of charge from International Dairy Federation (IDF), General Secretariat, Square Vergote 41, B-1040 Brussels (Belgium).

It is produced in English only and it is expected to be of interest to persons studying the many aspects of mastitis, to veterinarians, research institutes, students, etc.

* Current membership of Group A2
J.M. Booth (GB), Chairman, D. Østergaard (NO), W.M. Schallibaum (CH), A. Saran (IL), D. Ryan (AU), B. Poutrel (FR), M. Woolford (NZ), P. Casado (ES), K. Leslie (CA), U. Vecht (NL), T. Ichikawa (JP), W. Heeschen (DE), J. García-Lopez (ES), (corresponding member), E. Glawisch (AT), R. S. Singh (IN), P. Schmidt Madsen (DK), H. Saloniemi (FI), Ch. Burvenich (BE), K. L. Smith (US), G. Rufio (IT), A. Contini (IT), W.H. Giesek (ZA), O. Odgeirsson (IS), A. Zecconi (IT), W. Meaney (IE), K. Pym-Forsheil (SE), Observers: B.G. Cané (AR), M. Laborde (UY), G. Kalatzopoulos (GR).

ANNUAL REPORT OF THE IDF GROUP OF EXPERTS ON MASTITIS (1992)

The IDF Group of Experts on Mastitis held well attended meetings of the main group (A2) and its subgroups in Milan and Brussels during 1992. The Group consists of a representative from 23 IDF member countries, with two corresponding members from South America. Two long-standing members, Prof. D. Barnum of Canada and Dr R. Hoare of Australia, retired from the Group and were replaced by Dr K. Leslie and Dr D. Ryan, respectively. Prof. Barnum had made major contributions in several areas of the work of the Group, and the Group recorded its appreciation to both Prof. Barnum and Dr Hoare for their work over more than a decade.

The 10th edition of Mastitis Research Index was published under the editorship of Prof. H. Saloniemi (FI). This bulletin lists 267 projects currently in progress at 71 research centres in 24 countries. For ease of use, projects are also indexed by responsible worker and by subject. Mastitis Newsletter No. 17 was published in June 1992 under the editorship of Prof. M. Schallibaum (CH).

The Machine Milking and Mastitis Subgroup A2D (Chairman: Prof. J. Hamann, DE) was particularly productive during the year. The three main chapters of the monograph “Machine milking induced teat tissue reactions and new infection risk” have been completed and work is proceeding on various special aspects. The intention is to publish the monograph during 1993. The Subgroup will attempt subsequently to develop standard guidelines for testing conductivity instruments used in the detection of mastitis.

Cell Count Subgroup A2B (Chairman: Mr J. Booth, GB) received several comments to Provisional IDF Standard 148:1991 on “Enumeration of somatic cells” and these are now being considered before publication of the final standard. Two international ring trials, Nos 20 and 21, were organized and reported on by Prof. W. Heeschen (DE). Results of the Kiel standard samples in Trial 21 were (mean ± s.d.) 278 ± 20 and 680 ± 69 thousand cells/ml (n = 17) for the low and high cell count samples using Fossomatic instruments. The Subgroup planned to evaluate newly available cell counting instruments but to date no data have been made available by the manufacturers. The lack of a scientific basis for the cell count limits used in payment schemes was also discussed. The question of standardization of the reporting of somatic cell counts was raised again and a proposal is being prepared. The paper by Prof. D. Barnum (CA) on single cow cell counts is to be published in the next Mastitis Newsletter. A summary of the replies from the 23 countries submitting data in response to Mastitis Cell Count Questionnaire 1792/A is given in the present publication.

The Group has set up an organizing committee (Chairman: Dr A. Saran, IL) to prepare the scientific programme for the third IDF Mastitis Seminar which will be held in Israel in May 1995.

A range of other subjects was considered by the Group. Following the discussion of a paper produced by Prof. J. Hamann (DE) on the homeopathic
treatment of bovine mastitis, it was agreed that scientific information on
the effectiveness of homoeopathics in
the control of bovine mastitis is too
limited to justify a definite conclusion.
The use of vaccines to prevent masti-
titis was discussed and promising
results were reported for a staphylo-
coccocal vaccine, although the numbers
were too small for statistical signifi-
cance.

A regular update on the activities
of the US National Mastitis Council
was given by Prof. L. Smith (US). Dr
K. Plym-Forshell (SE) reported on
work in Sweden which indicated that
a part of the decrease in average bulk
milk cell counts during recent years
may be a dilution effect due to the
concurrent increase in milk produc-
tion. Dr A. Saran (IL) reported on
Israeli trials on the use of systemic
dry cow therapy and on the apparent
lack of success to date in treating
Staphylococcus aureus infections.
Other topics included the need to
determine the repeatability and repro-
ducibility of cell counting, the partial
insertion technique for intramammary
antibiotic administration, the value of
currently available cowside tests for
antibiotic residues, possible effects of
robotic milking, and aspects of rele-
vant work of related IDF groups.

J.M. Booth, Chairman
Genus Animal Health, Veterinary
Laboratory
Cleeve House, Lower Wick, Worcester,
WR2 4 NS, England
February 1993

**

MASTITIS CELL COUNT DATA

In February 1992 the IDF Group of Experts on Mastitis (Group A2) issued a
further questionnaire (No. 1792/A) to the National Committees of the 32 IDF
member countries requesting cell count data for the years 1990 and 1991. This
followed the publication of data for previous years in IDF Mastitis Newsletters 14

Twenty-seven countries replied to the questionnaire although four of these
(Greece, Italy, Poland and Spain) stated that no national data were available.
No replies were received from five countries (Bulgaria, India, Kenya, Kuwait and
Luxembourg).

The collated replies from the 23 countries having data in one or both years
are summarized here. The IDF abbreviations used for the countries are:

AT Austria
AU Australia
BE Belgium
CA Canada
CH Switzerland
CS Czechoslovakia
DE Germany
DK Denmark
FI Finland
FR France
GB United Kingdom
HU Hungary
IE Ireland
IL Israel
IS Iceland
JP Japan
NL Netherlands
NO Norway
NZ New Zealand
SE Sweden
SU Russia
US United States of America
ZA South Africa

The replies to the questionnaire highlighted the large differences in the struc-
ture of dairy farming in different countries. Nevertheless, the IDF Mastitis Group
was pleased to note a trend towards greater uniformity in cell counting and in
the presentation of data. For the future the Group hopes to make recommenda-
tions on the presentation and analysis of data based upon common usage. In
the meantime it is important to bear in mind that there is still a wide variation in
presentation and much of the data are not directly comparable, as many of the
footnotes attest.

The IDF Mastitis Group wishes to place on record its thanks to the National
Committees of all the member countries which replied to this questionnaire.

J.M. Booth, Chairman Group A2
Genus Animal Health, Veterinary Laboratory
Cleeve House, Lower Wick, Worcester, WR2 4 NS, England
January 1993
IDF Questionnaire on national herd milk cell count data: 1990 data

1. Herds and samples:

<table>
<thead>
<tr>
<th></th>
<th>AT</th>
<th>BE</th>
<th>CA</th>
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<th>DE</th>
<th>DK</th>
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<th>FR</th>
<th>GB/EW</th>
<th>GB/NI</th>
<th>GB/SC</th>
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<tbody>
<tr>
<td>Total number of dairy herds in country</td>
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<td>27126</td>
<td>28575</td>
<td>53400</td>
<td>275100</td>
<td>20800</td>
<td>45500</td>
<td>241200</td>
<td>31492</td>
<td>6338</td>
<td>2314</td>
</tr>
<tr>
<td>Average herd size (cows only)</td>
<td>8</td>
<td>31</td>
<td>43.8</td>
<td>14.3</td>
<td>17.3</td>
<td>34</td>
<td>10.8</td>
<td>22.8</td>
<td>71</td>
<td>39</td>
<td>88</td>
</tr>
<tr>
<td>Number of herds being cell counted regularly</td>
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<td>25000</td>
<td>28252</td>
<td>53400</td>
<td>275100</td>
<td>20800</td>
<td>45500</td>
<td>241200</td>
<td>31492</td>
<td>6338</td>
<td>2314</td>
</tr>
<tr>
<td>Percentage of country's herds (1.3 divided by 1.1, x 100)</td>
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<td>92</td>
<td>98.9</td>
<td>100</td>
<td>100</td>
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<td>100</td>
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<tr>
<td>Number of bulk milk cell counts on each herd per year</td>
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<td>21</td>
<td>12.2</td>
<td>12</td>
<td>12</td>
<td>12</td>
<td>12</td>
<td>12</td>
<td>52</td>
<td>48</td>
<td>48</td>
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2. Cell counts (in thousand cells/ml):

<table>
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<th>CA</th>
<th>CH</th>
<th>DE</th>
<th>DK</th>
<th>FI</th>
<th>FR</th>
<th>GB/EW</th>
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<tr>
<td>280&lt;sup&gt;1&lt;/sup&gt;</td>
<td>280&lt;sup&gt;1&lt;/sup&gt;</td>
<td>360</td>
<td>304.7&lt;sup&gt;4&lt;/sup&gt;</td>
<td>117</td>
<td>274</td>
<td>313</td>
<td>282/205&lt;sup&gt;9&lt;/sup&gt;</td>
<td>NA&lt;sup&gt;2&lt;/sup&gt;</td>
<td>323</td>
<td>325&lt;sup&gt;15&lt;/sup&gt;</td>
<td>322</td>
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<td>2.1 Annual mean cell count</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.2 Arithmetic (A) or Geometric (G) mean</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.3 Distribution of herds according to their annual mean cell count: (thousand cells/ml)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
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<td>31.7</td>
<td>10.3</td>
<td>36</td>
<td>5.4</td>
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<td>5.4</td>
<td></td>
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<td>31.7</td>
<td>10.3</td>
<td>36</td>
<td>5.4</td>
<td></td>
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<td>33.9</td>
<td>36.1</td>
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<td>31.7</td>
<td>10.3</td>
<td>36</td>
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<td>36.1</td>
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<td>31.7</td>
<td>10.3</td>
<td>36</td>
<td>5.4</td>
<td></td>
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<td>33.9</td>
<td>36.1</td>
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<td>31.7</td>
<td>10.3</td>
<td>36</td>
<td>5.4</td>
<td></td>
</tr>
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<td>700-999</td>
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<td>33.9</td>
<td>36.1</td>
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<td>31.7</td>
<td>10.3</td>
<td>36</td>
<td>5.4</td>
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<td>33.9</td>
<td>36.1</td>
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<td>31.7</td>
<td>10.3</td>
<td>36</td>
<td>5.4</td>
<td></td>
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<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
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<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
</tr>
</tbody>
</table>

2.4 Cell count instruments used
F=Fossomatic, C=Coulter, O=Other

Footnotes:
7. Categories: <125
8. Categories: <200
9. Categories: 125 - 249
10. Categories: 250 - 399
11. Categories: 400 - 500
12. Categories: 500 - 599
13. Categories: >500
14. Categories: >750
15. By reference to lowest 3 of 4 counts available in month

1. Approximately.
2. Data not available.
3. Categories: 500-570
4. Data from 9 of 10 provinces, 98% of herds.
5. Data from 5 provinces, 60% of herds.
6. Data from West Germany.
7. Fossomatic 98%, Coulter 1%, Somascope 1%.
8. Co-operatives only, approx. 90% of milk.
9. Geometric averages over periods of three months.
IDF Questionnaire on national herd milk cell count data: 1990 data

1. Herds and samples:

<table>
<thead>
<tr>
<th></th>
<th>HU</th>
<th>IE</th>
<th>IL</th>
<th>IS</th>
<th>JP</th>
<th>NL</th>
<th>NO</th>
<th>NZ</th>
<th>SE</th>
<th>US</th>
<th>ZA</th>
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</thead>
<tbody>
<tr>
<td>1.1 Total number of dairy herds in country</td>
<td>1500</td>
<td>57000</td>
<td>1470</td>
<td>1541</td>
<td>63300</td>
<td>4700</td>
<td>27633</td>
<td>14595</td>
<td>23600</td>
<td>193700</td>
<td>8800</td>
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<tr>
<td>1.2 Average herd size (cows only)</td>
<td>270</td>
<td>25</td>
<td>297/54</td>
<td>23</td>
<td>20</td>
<td>25</td>
<td>13.0</td>
<td>159</td>
<td>21.4</td>
<td>52</td>
<td>78</td>
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<tr>
<td>1.3 Number of herds being cell counted regularly</td>
<td>750</td>
<td>57000</td>
<td>650</td>
<td>1541</td>
<td>56900</td>
<td>4700</td>
<td>27633</td>
<td>7250</td>
<td>23600</td>
<td>174000</td>
<td>7040</td>
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<tr>
<td>1.4 Percentage of country's herds (1.3 divided by 1.1, x 100)</td>
<td>50</td>
<td>100</td>
<td>44.2</td>
<td>100</td>
<td>90</td>
<td>100</td>
<td>100</td>
<td>50</td>
<td>100</td>
<td>90</td>
<td>80</td>
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<td>1.5 Number of bulk milk cell counts on each herd per year</td>
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<td>12</td>
<td>31</td>
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<td>24</td>
<td>10</td>
<td>12</td>
<td>8</td>
<td>6</td>
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</tbody>
</table>

2. Cell counts (in thousand cells/ml):

<table>
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<tr>
<th></th>
<th>% herds</th>
<th>% herds²</th>
<th>% herds</th>
<th>% herds</th>
<th>% herds</th>
<th>% herds</th>
<th>% herds</th>
<th>% herds</th>
<th>% herds</th>
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<tbody>
<tr>
<td>2.1 Annual mean cell count</td>
<td>420</td>
<td>2</td>
<td>395</td>
<td>471</td>
<td>260</td>
<td>311</td>
<td>206/182/158</td>
<td>358</td>
<td>230</td>
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<tr>
<td>2.2 Arithmetic (A) or Geometric (G) mean</td>
<td>A</td>
<td>-</td>
<td>A</td>
<td>A</td>
<td>A</td>
<td>G</td>
<td>A/AG/G</td>
<td>A</td>
<td>G</td>
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</table>

2.3 Distribution of herds according to their annual mean cell count: (thousand cells/ml)

<table>
<thead>
<tr>
<th></th>
<th>% herds</th>
<th>% herds²</th>
<th>% herds</th>
<th>% herds</th>
<th>% herds</th>
<th>% herds</th>
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<td>&lt;100</td>
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<td>0.6</td>
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</tr>
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<td>100.0</td>
<td>-</td>
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</tbody>
</table>

2.4 Cell count instruments used

- F=Fossomatic, C=Coulter, O=Other

Footnotes:

1. Data refer to the 75% of dairy cows in big estates.

2. Data not available.

3. Kibbutz (collective farm)/Moshav (family farm).

4. Categories: 500 - 749
   750 - 999

5. A = Arithmetic mean of samples.
   AG = Arithmetic mean of herds' geometric means.
   G = Geometric mean of herds' geometric means.

6. No national statistics.
IDF Questionnaire on national herd milk cell count data: 1991 data

<table>
<thead>
<tr>
<th>1. Herds and samples:</th>
<th>AT</th>
<th>AU</th>
<th>BE</th>
<th>CA</th>
<th>CH</th>
<th>CS</th>
<th>DE</th>
<th>DK</th>
<th>FI</th>
<th>FR</th>
<th>GB/EW(^\text{1})</th>
<th>GB/NI(^\text{1})</th>
<th>GB/SC(^\text{1})</th>
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</thead>
<tbody>
<tr>
<td>1.1 Total number of dairy herds in country</td>
<td>95000</td>
<td>14900</td>
<td>25608</td>
<td>27573</td>
<td>52300</td>
<td>11871</td>
<td>249200</td>
<td>19550</td>
<td>39600</td>
<td>212600</td>
<td>30219</td>
<td>6060</td>
<td>2289</td>
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<tr>
<td>1.2 Average herd size (cows only)</td>
<td>10</td>
<td>110</td>
<td>32</td>
<td>43.9</td>
<td>14.4</td>
<td>140</td>
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<td>11.1</td>
<td>24.8</td>
<td>71</td>
<td>39</td>
<td>88</td>
</tr>
<tr>
<td>1.3 Number of herds being cell counted regularly</td>
<td>95000</td>
<td>14900</td>
<td>24100(^\text{1})</td>
<td>27329</td>
<td>52300</td>
<td>11871</td>
<td>249200</td>
<td>19550</td>
<td>39600</td>
<td>212600</td>
<td>30219</td>
<td>6060</td>
<td>2289</td>
</tr>
<tr>
<td>1.4 Percentage of country's herds (1.3 divided by 1.1 x 100)</td>
<td>100</td>
<td>100</td>
<td>94</td>
<td>99.1</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>1.5 Number of bulk milk cell counts on each herd per year</td>
<td>12</td>
<td>12</td>
<td>21</td>
<td>12.2</td>
<td>12</td>
<td>24</td>
<td>12.36</td>
<td>13</td>
<td>24</td>
<td>12</td>
<td>52</td>
<td>48</td>
<td>48</td>
</tr>
</tbody>
</table>

| 2. Cell counts (in thousand cells/ml): |
|----------------------|----|----|----|----|----|----|----|----|----|----|-----------------|-----------------|-----------------|
| 2.1 Annual mean cell count | 260\(^\text{1}\) | 335\(^\text{3}\) | 301 | 280.1\(^\text{6}\) | 114 | 450 | 230 | 295 | 247/179 | NA\(^\text{2}\) | 310/288 | 235\(^\text{15}\) | 314 |
| 2.2 Arithmetic (A) or Geometric (G) mean | A | A | G | A | G | G | G | G | A/G\(^\text{14}\) | G | A | A |

<table>
<thead>
<tr>
<th>2.3 Distribution of herds according to their annual mean cell count: (thousand cells/ml)</th>
<th>% herds(^{2})</th>
<th>% herds(^{2})</th>
<th>% herds(^{2})</th>
<th>% herds(^{2})</th>
<th>% herds(^{10})</th>
<th>% herds(^{2})</th>
<th>% herds(^{2})</th>
<th>% herds(^{2})</th>
<th>% herds(^{2})</th>
<th>% herds(^{2})</th>
<th>% herds(^{2})</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;100</td>
<td>-</td>
<td>-</td>
<td>3</td>
<td>10.1</td>
<td>42.7</td>
<td>24.4</td>
<td>1</td>
<td>19.3</td>
<td>-</td>
<td>2.4</td>
<td>18</td>
</tr>
<tr>
<td>100-199</td>
<td>-</td>
<td>-</td>
<td>18</td>
<td>29.9</td>
<td>34.6</td>
<td>26</td>
<td>13</td>
<td>35.7</td>
<td>-</td>
<td>21.4</td>
<td>29</td>
</tr>
<tr>
<td>200-299</td>
<td>-</td>
<td>-</td>
<td>27</td>
<td>24.4</td>
<td>12.3</td>
<td>-</td>
<td>27</td>
<td>32.2</td>
<td>-</td>
<td>25.5</td>
<td>29</td>
</tr>
<tr>
<td>300-399</td>
<td>-</td>
<td>-</td>
<td>22</td>
<td>15.1</td>
<td>5.0</td>
<td>57</td>
<td>22.4</td>
<td>30</td>
<td>11.5</td>
<td>-</td>
<td>18.0</td>
</tr>
<tr>
<td>400-499</td>
<td>-</td>
<td>-</td>
<td>14</td>
<td>8.7</td>
<td>2.4</td>
<td>-</td>
<td>6.9</td>
<td>12</td>
<td>5.1</td>
<td>-</td>
<td>11.2</td>
</tr>
<tr>
<td>500-699</td>
<td>-</td>
<td>-</td>
<td>12(^{5})</td>
<td>8.5</td>
<td>2.0</td>
<td>5</td>
<td>6</td>
<td>3.9</td>
<td>-</td>
<td>11.9</td>
<td>7(^{16})</td>
</tr>
<tr>
<td>700-999</td>
<td>-</td>
<td>-</td>
<td>4(^{5})</td>
<td>0.9</td>
<td>0.3</td>
<td>17</td>
<td>9.5</td>
<td>0</td>
<td>1.4</td>
<td>-</td>
<td>6.4</td>
</tr>
<tr>
<td>&gt;1000</td>
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<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>0</td>
<td>0.5</td>
<td>-</td>
<td>3.2</td>
<td>2</td>
</tr>
<tr>
<td><strong>TOTALS</strong></td>
<td>-</td>
<td>-</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>-</td>
<td>100.0</td>
<td>100.0</td>
</tr>
</tbody>
</table>

2.4 Cell count instruments used
F=Fossomatic, C=Coulter, O=Other.

3. Payment on cell count
3.1 Farmers paid on cell count in 1991
   All: Some: All: Some: All: Some\(^{8}\): All: All: All: All: All: All: All: All: All
   All: All: All: All: All: All: All: All: All: All: All: All: All: All: All: All

3.2 Lowest cell count category (000 cells/ml)
   Highest cell count category (000 cells/ml)
   Data from 9 of 10 provinces, 98% of herds.
   Data from 6 provinces, 60% of herds.

Footnotes:
1. Approximately.
5. Categories: 500-750
8. All from 1 July 1992
11. Fossomatic 98%,
13. EW: England and Wales
2. Data not available.
750-1000
9. Data from West Germany
14. GA=Geometric mean of herds' arithmetic means.
3. See below.
6. Data from 9 of 10 provinces,
10. Categories: 25-125-249, 250-399, 400-500, 500-1000
12. Geometric mean over 3 months.
16. Categories: 400-599
4. O = Bentley Foss.
198% of herds.
7. Data from 6 provinces, 60% of herds.
### IDF Questionnaire on national herd milk cell count data: 1991 data

#### 1. Herds and samples:

<table>
<thead>
<tr>
<th>Country</th>
<th>HU</th>
<th>IE</th>
<th>IL</th>
<th>IS</th>
<th>JP</th>
<th>NL</th>
<th>NO</th>
<th>NZ</th>
<th>SE</th>
<th>SU</th>
<th>US</th>
<th>ZA</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Total number of dairy herds in country</td>
<td>1380</td>
<td>57000</td>
<td>1478</td>
<td>1501</td>
<td>59800</td>
<td>4500</td>
<td>27439</td>
<td>14685</td>
<td>20200</td>
<td>71200</td>
<td>18900</td>
<td>8100</td>
</tr>
<tr>
<td>2. Average herd size (cows only)</td>
<td>261</td>
<td>25</td>
<td>296/50^2</td>
<td>23</td>
<td>21</td>
<td>25</td>
<td>12.9</td>
<td>159</td>
<td>22.1</td>
<td>290</td>
<td>52</td>
<td>92</td>
</tr>
<tr>
<td>3. Number of herds being cell counted regularly</td>
<td>1120</td>
<td>57000</td>
<td>1250</td>
<td>1501</td>
<td>53800</td>
<td>4500</td>
<td>27439</td>
<td>7250</td>
<td>20200</td>
<td>712</td>
<td>17000</td>
<td>6885</td>
</tr>
<tr>
<td>4. Percentage of country's herds (1.3 divided by 1.1, x100)</td>
<td>81</td>
<td>100</td>
<td>84.6</td>
<td>100</td>
<td>90</td>
<td>100</td>
<td>100</td>
<td>50</td>
<td>100</td>
<td>10^8</td>
<td>90</td>
<td>85</td>
</tr>
<tr>
<td>5. Number of bulk milk cell counts on each herd per year</td>
<td>36</td>
<td>10</td>
<td>24</td>
<td>38</td>
<td>24-36</td>
<td>13</td>
<td>24</td>
<td>10</td>
<td>12</td>
<td>5</td>
<td>810</td>
<td>9</td>
</tr>
</tbody>
</table>

#### 2. Cell counts (in thousand cells/ml):

<table>
<thead>
<tr>
<th>Country</th>
<th>HU</th>
<th>IE</th>
<th>IL</th>
<th>IS</th>
<th>JP</th>
<th>NL</th>
<th>NO</th>
<th>NZ</th>
<th>SE</th>
<th>SU</th>
<th>US</th>
<th>ZA</th>
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</thead>
<tbody>
<tr>
<td>2.1 Annual mean cell count</td>
<td>433</td>
<td>398^4</td>
<td>481</td>
<td>280</td>
<td>301</td>
<td>204/179/157</td>
<td>298</td>
<td>233</td>
<td>300-600</td>
<td>300-400^11</td>
<td>465</td>
<td></td>
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<tr>
<td>2.2 Arithmetic (A) or Geometric (G) mean</td>
<td>A</td>
<td>A</td>
<td>A</td>
<td>A</td>
<td>G</td>
<td>A/AG/G^2</td>
<td>A</td>
<td>G</td>
<td>A</td>
<td>-</td>
<td>A</td>
<td></td>
</tr>
<tr>
<td>2.3 Distribution of herds according to their annual mean cell count: (thousand cells/ml)</td>
<td>% herds</td>
<td>% herds</td>
<td>% herds</td>
<td>% herds</td>
<td>% herds</td>
<td>% herds</td>
<td>% herds</td>
<td>% herds</td>
<td>% herds</td>
<td>% herds</td>
<td>% herds</td>
<td></td>
</tr>
<tr>
<td>&lt;100</td>
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<td>3.9</td>
<td>17.5</td>
<td>-</td>
<td>6.3</td>
<td>-</td>
<td>-</td>
<td>4.3</td>
</tr>
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<td>100-199</td>
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<td>3.5</td>
<td>4.4</td>
<td>31.8</td>
<td>28.9</td>
<td>50.8</td>
<td>-</td>
<td>36.6</td>
<td>-</td>
<td>-</td>
<td>14.1</td>
</tr>
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<td>200-299</td>
<td>29^1</td>
<td>-</td>
<td>19.4</td>
<td>16.3</td>
<td>27.2</td>
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<td>12</td>
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<td>19.3</td>
</tr>
<tr>
<td>300-399</td>
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<td>30.0</td>
<td>20.3</td>
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<td>17.6</td>
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<td>-</td>
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<td>18.0</td>
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<td>2.1</td>
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<td>-</td>
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<td>35</td>
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<td>500-699</td>
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<td>24.9</td>
<td>7.7</td>
<td>7.9^6</td>
<td>1.1</td>
<td>-</td>
<td>1.2</td>
<td>5</td>
<td>-</td>
<td>16.4</td>
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<td>-</td>
<td>5.7</td>
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<td>1.9^6</td>
<td>0.1</td>
<td>-</td>
<td>0.1</td>
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<tr>
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<td>3.1</td>
<td>2.3</td>
<td>1.1</td>
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<td>-</td>
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</tr>
<tr>
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<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>-</td>
<td>100.0</td>
<td>100.0</td>
<td>-</td>
<td>100.0</td>
</tr>
</tbody>
</table>

#### 2.4 Cell count instruments used

- F = Fossomatic, C = Coulter, D = Other

#### 3. Payment on cell count

<table>
<thead>
<tr>
<th>Country</th>
<th>HU</th>
<th>IE</th>
<th>IL</th>
<th>IS</th>
<th>JP</th>
<th>NL</th>
<th>NO</th>
<th>NZ</th>
<th>SE</th>
<th>SU</th>
<th>US</th>
<th>ZA</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.1 Farmers paid on cell count in 1991</td>
<td>Some</td>
<td>Some</td>
<td>None^5</td>
<td>None</td>
<td>Some</td>
<td>All</td>
<td>All</td>
<td>None</td>
<td>Some</td>
<td>None</td>
<td>Some</td>
<td>Some</td>
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<tr>
<td>3.2 Lowest cell count category (000 cells/ml)</td>
<td>400</td>
<td>250</td>
<td>-</td>
<td>-</td>
<td>300</td>
<td>&lt;400</td>
<td>250</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>&lt;100^13</td>
<td>-</td>
</tr>
<tr>
<td>Highest cell count category (000 cells/ml)</td>
<td>1000</td>
<td>750</td>
<td>-</td>
<td>-</td>
<td>1000</td>
<td>&gt;500</td>
<td>750</td>
<td>-</td>
<td>500</td>
<td>-</td>
<td>&gt;300^13</td>
<td>750</td>
</tr>
</tbody>
</table>

#### Footnotes:

1. Category: <300
2. Data not available.
3. Kibbutz (collective farm)/Moshev (family farm)
4. Refers to Herd-Book registered herds only (>80% of all herds)
6. Categories: 500 - 749
7. A = Arithmetic mean of samples.
8. As stated
10. Varieties of milk processors.
12. Majority.
14. Variable, depending on milk buyer.

^1 Listed when not available.
^2 Calculations of national herds.
^3 Calculations of national herds.
^4 Calculations of national herds.
^5 Calculations of national herds.
^6 Calculations of national herds.
^7 Calculations of national herds.
^8 Calculations of national herds.
^9 Calculations of national herds.
^10 Calculations of national herds.
^11 Calculations of national herds.
^12 Calculations of national herds.
^13 Calculations of national herds.
^14 Calculations of national herds.

### Additional Notes:

- **HU** = Herd Unit
- **IE** = Intra-Epizootic
- **IL** = Inter-Land
- **IS** = Intra-State
- **JP** = Japanese Production
- **NL** = National Laboratory
- **NO** = National Office
- **NZ** = New Zealand
- **SE** = Southeast
- **SU** = Southwest
- **US** = Kansas
- **ZA** = Zimbabwe
### IDF Questionnaire on national herd milk cell count data

<table>
<thead>
<tr>
<th>AT</th>
<th>AU</th>
<th>BE</th>
<th>CA</th>
<th>CH</th>
<th>CS</th>
<th>DE</th>
<th>DK</th>
<th>FI</th>
<th>FR</th>
<th>GB/EW</th>
<th>GB/NI</th>
<th>GB/SC</th>
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<tr>
<td><strong>4 Single cow cell counts:</strong></td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.1 Are these carried out on some herds in your country? (Y = Yes)</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>4.2 If yes, most recent figures on:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.2.1 Number of herds</td>
<td>5000</td>
<td>2500</td>
<td>5100&lt;sup&gt;2&lt;/sup&gt;</td>
<td>12899</td>
<td>4100</td>
<td>200&lt;sup&gt;5&lt;/sup&gt;</td>
<td>105000</td>
<td>13405</td>
<td>21900</td>
<td>75000</td>
<td>7488</td>
<td>1243</td>
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<tr>
<td>4.2.2 Percentage of all dairy herds in the country</td>
<td>30</td>
<td>20</td>
<td>20&lt;sup&gt;2&lt;/sup&gt;</td>
<td>46</td>
<td>7.8</td>
<td>1.68</td>
<td>42.1</td>
<td>69</td>
<td>54</td>
<td>35&lt;sup&gt;2&lt;/sup&gt;</td>
<td>25.2</td>
<td>21</td>
</tr>
<tr>
<td>4.2.3 Number of cows (approx)</td>
<td>185000</td>
<td>300000</td>
<td>180000&lt;sup&gt;2&lt;/sup&gt;</td>
<td>541193</td>
<td>74000</td>
<td>28000&lt;sup&gt;5&lt;/sup&gt;</td>
<td>2724400</td>
<td>585770</td>
<td>290000</td>
<td>235000</td>
<td>704500</td>
<td>55540</td>
</tr>
<tr>
<td>4.2.4 Number of tests per cow per year</td>
<td>12</td>
<td>1·8&lt;sup&gt;3&lt;/sup&gt;</td>
<td>12&lt;sup&gt;2&lt;/sup&gt;</td>
<td>10.1</td>
<td>12</td>
<td>4</td>
<td>11·12</td>
<td>12</td>
<td>4·6</td>
<td>10</td>
<td>10</td>
<td>1·12&lt;sup&gt;7&lt;/sup&gt;</td>
</tr>
<tr>
<td>4.2.5 Cost to farmer of each test</td>
<td>&lt;70c&lt;sup&gt;3&lt;/sup&gt;</td>
<td>10 FB&lt;sup&gt;5&lt;/sup&gt;</td>
<td>$2·73</td>
<td>SFr6·00</td>
<td>6·15Kcs</td>
<td>0·4·0·5</td>
<td>1 DKR</td>
<td>FIM2·60</td>
<td>5·6</td>
<td>£0·11</td>
<td>£1·2&lt;sup&gt;8&lt;/sup&gt;</td>
<td>£0·12</td>
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<tr>
<td><strong>4.2.6 Purpose (Y = Yes, N = No):</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 To improve farmers’ awareness of subclinical mastitis</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>2 To identify herds with mastitis problems</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y&lt;sup&gt;3&lt;/sup&gt;</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>Y</td>
</tr>
<tr>
<td>3 To investigate herds with symptoms of mastitis</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y&lt;sup&gt;3&lt;/sup&gt;</td>
<td>N</td>
<td>Y</td>
<td>-</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>4 To assess a herd’s economic losses due to mastitis</td>
<td>Y</td>
<td>-</td>
<td>N</td>
<td>Y&lt;sup&gt;4&lt;/sup&gt;</td>
<td>N</td>
<td>N</td>
<td>-</td>
<td>Y</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>5 To identify cows with mastitis problems</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>6 To identify cows for bacteriological examination</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y&lt;sup&gt;3&lt;/sup&gt;</td>
<td>N</td>
<td>N</td>
<td>-</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>Y</td>
</tr>
<tr>
<td>7 To identify cows for treatment during lactation</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>Y&lt;sup&gt;4&lt;/sup&gt;</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>-</td>
<td>Y</td>
</tr>
<tr>
<td>8 To identify cows for treatment during dry period</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y&lt;sup&gt;3&lt;/sup&gt;</td>
<td>N</td>
<td>Y</td>
<td>-</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>9 To assess the effectiveness of therapy</td>
<td>Y</td>
<td>-</td>
<td>Y</td>
<td>Y&lt;sup&gt;4&lt;/sup&gt;</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>10 To select cows for culling</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>11 For genetic studies or sire selection</td>
<td>N</td>
<td>-</td>
<td>N</td>
<td>Y&lt;sup&gt;4&lt;/sup&gt;</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>12 Other</td>
<td>-</td>
<td>-</td>
<td>N</td>
<td>-</td>
<td>N</td>
<td>-</td>
<td>-</td>
<td>N</td>
<td>-</td>
<td>-</td>
<td>Y&lt;sup&gt;9&lt;/sup&gt;</td>
<td></td>
</tr>
</tbody>
</table>

**5 Can this information be published in the IDF Mastitis Newsletter? (Y = Yes)**

**Footnotes:**

1. Varies.
2. Approximately.
3. Most.
4. Some.
5. Estimates.
6. Included in milk recording fee.
7. 12 in milk recorded herds, one in other herds.
8. £1 in milk recorded herds, £2 in other herds.
9. To enable producers to meet prospective EEC standards.
IDF Questionnaire on national herd milk cell count data

<table>
<thead>
<tr>
<th>4 Single cow cell counts:</th>
</tr>
</thead>
<tbody>
<tr>
<td>4.1 Are these carried out on some herds in your country? (Y = Yes)</td>
</tr>
<tr>
<td>HU</td>
</tr>
<tr>
<td>Y</td>
</tr>
<tr>
<td>4.2 If yes, most recent figures on:</td>
</tr>
<tr>
<td>4.2.1 Number of herds</td>
</tr>
<tr>
<td>750</td>
</tr>
<tr>
<td>4.2.2 Percentage of all dairy herds in the country</td>
</tr>
<tr>
<td>54</td>
</tr>
<tr>
<td>4.2.3 Number of cows (approx)</td>
</tr>
<tr>
<td>260000</td>
</tr>
<tr>
<td>4.2.4 Number of tests per cow per year</td>
</tr>
<tr>
<td>6-12</td>
</tr>
<tr>
<td>4.2.5 Cost to farmer of each test</td>
</tr>
<tr>
<td>12 HUF</td>
</tr>
<tr>
<td>4.2.6 Purpose (Y = Yes, N = No)</td>
</tr>
<tr>
<td>1 To improve farmers' awareness of subclinical mastitis</td>
</tr>
<tr>
<td>Y</td>
</tr>
<tr>
<td>2 To identify herds with mastitis problems</td>
</tr>
<tr>
<td>Y</td>
</tr>
<tr>
<td>3 To investigate herds with mastitis problems</td>
</tr>
<tr>
<td>Y</td>
</tr>
<tr>
<td>4 To assess a herd's economic losses due to mastitis</td>
</tr>
<tr>
<td>N</td>
</tr>
<tr>
<td>5 To identify cows with mastitis problems</td>
</tr>
<tr>
<td>Y</td>
</tr>
<tr>
<td>6 To identify cows for bacteriological examination</td>
</tr>
<tr>
<td>Y</td>
</tr>
<tr>
<td>7 To identify cows for treatment during lactation</td>
</tr>
<tr>
<td>Y</td>
</tr>
<tr>
<td>8 To identify cows for treatment during dry period</td>
</tr>
<tr>
<td>N</td>
</tr>
<tr>
<td>9 To assess the effectiveness of therapy</td>
</tr>
<tr>
<td>N</td>
</tr>
<tr>
<td>10 To select cows for culling</td>
</tr>
<tr>
<td>Y</td>
</tr>
<tr>
<td>11 For genetic studies or sire selection</td>
</tr>
<tr>
<td>N</td>
</tr>
<tr>
<td>12 Other</td>
</tr>
<tr>
<td>-</td>
</tr>
</tbody>
</table>

5 Can this information be published in the IDF Mastitis Newsletter? (Y = Yes)

| Y  | Y  | Y  | Y  | Y  | Y  | Y  | Y  | Y  | Y  |

Footnotes:
1. Data not available.
2. Included in annual fees to Cattle Breeders Association.
3. To assess milking and management practices.
4. Estimated from 1990 data.
5. To identify cows for exclusion from vat to avoid penalty.
6. Included in milk recording analyses.
7. To estimate prevalence and incidence of infectious subclinical mastitis.
8. Not paid.
10. Very seldom.
SOMATIC CELLS IN MILK - ASPECTS OF QUALITY, HYGIENE AND MASTITIS CONTROL

The Council Directive 92/46/EEC of 16 June 1992 laying down the health rules for the production and placing on the market of raw milk, heat-treated milk and milk-based products gives in its Annex A (Chapter IV) detailed standards to be met for the collection of raw milk from the production holding or for the acceptance at the treatment or processing establishments. The hygienic requirements including somatic cells are summarized in Table 1.

The regulation EEC No. 2377/90 refers to Maximum Residue Limits (MRLs) for antimicrobial drugs. Within 3 months of notification of the results milk from the holding in question does not meet these standards, that holding shall no longer be authorized to supply raw milk until such milk again meets the standards mentioned.

In addition in Annex C (Chapter II) of this directive, raw cow's milk for drinking in that state must meet standards, which are higher than those for raw milk used for further processing.

The figures given for somatic cells (finally 400,000/ml) raise the question of how far these standards would meet the requirements under aspects of quality, hygiene and mastitis control.

It has to be clearly stated, that the somatic cell counts in the EEC Milk Hygiene Directive 92/46 of 16 June 1992 are not directly related to the mastitis situation in the production holding. On the other hand, it became obvious during the discussions in a working group of the council that increased cell counts reflect compositional changes of the milk and the possible occurrence of pathogenic mastitis organisms. To minimize compositional changes cell counts as low as possible are required. As “food hygiene” is defined to include all measures for the production of food which is safe, wholesome and of high quality and which ensure a free trade between countries, the standards given in the EEC Milk Hygiene Directive 92/46 are indirectly related to the mastitis problem. The word “mastitis” is not mentioned in the whole directive.

This background gives the information that with the occurrence of somatic cells in milk three different objectives have to be differentiated:

(1) protection of the consumer (safety of the food),
(2) product quality,
(3) mastitis control.

With cell counts of more than 400,000/ml in most cases a mastitis problem in the herd will be obvious. Low cell counts do not exclude the occurrence of mastitis cases in a given herd. This is due to the fact that milk from acutely diseased cows is not delivered. Moreover, a high dilution rate has to be regarded. In other cases, cell counts are low despite the occurrence of pathogenic microorganisms in the mammary gland. As there is a basic relation between the number of somatic cells and the compositional quality of the milk, in these cases the quality aspect of the milk might be of inferior significance.

It has never been the idea of standards for cell counts in the EEC Milk Hygiene Directive 92/46 to be used for purposes of mastitis control. They are "hygienic parameters" in the definition given above. Nevertheless, in cases where the standards are not met, mastitis problems in the herd will have a very high probability. Low cell counts, however, do not exclude the existence of mastitis cows.

### Table 1: EEC Milk Hygiene Directive 92/46/EEC of 16 June 1992

<table>
<thead>
<tr>
<th>Annex A</th>
<th>Chapter IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Standards to be met for collection of raw milk</td>
<td></td>
</tr>
</tbody>
</table>

A. Raw cow's milk
Without prejudice to the limits laid down in Annexes I and III to Regulation (EEC) No 2377/90:

1. Raw cow’s milk intended for the production of heat-treated drinking milk, fermented milk, junket, jellied or flavoured milk and cream must meet the following standards:
   - Plate count 30°C (per ml) ≤ 100,000* 
   - Somatic cell count (per ml) ≤ 400,000**

2. Raw cow's milk for the manufacture of milk-based products other than those referred to in point 1 must meet the following standards:
   - Plate count 30°C (per ml) ≤ 400,000* ≤ 100,000* 
   - Somatic cell count (per ml) ≤ 500,000** ≤ 400,000**

3. Raw cow's milk intended for direct human consumption and raw cow's milk for the manufacture of products “made with raw milk” whose manufacturing process does not involve any heat treatment must:
   - a) meet the standards of point 1
   - b) in addition meet the following standard:
     - Staphylococcus aureus (per ml)
     - n = 5, m = 500, M = 2000, c = 2

* Geometric average over a period of 2 months, with at least two samples a month.
** Geometric average over a period of 3 months, with at least one sample a month, or where production levels vary considerably according to season, method of calculating results to be adjusted in accordance with the procedure laid down in Article 31 of this Directive.

n = number of samples, m = threshold value, M = maximum value, c = number of samples < m

### Table 2: EEC Milk Hygiene Directive 92/46/EEC of 16 June 1992

<table>
<thead>
<tr>
<th>Annex C</th>
<th>Chapter II</th>
</tr>
</thead>
<tbody>
<tr>
<td>Microbiological criteria for milk-based products and drinking milk</td>
<td></td>
</tr>
</tbody>
</table>

B. Microbiological criteria for drinking milk

1. Raw cow's milk for drinking in that state must meet the following standards after wrapping:
   - Plate count at 30°C (per ml) ≤ 50,000* 
   - Somatic cell count (per ml) ≤ 400,000
   - Staphylococcus aureus (per ml)
   - Salmonella
     - m = 100 M = 500 n = 5 c = 2
     - absent in 25 g
     - n = 5 c = 2

   In addition, pathogenic microorganisms and their toxins must not be present in quantities such as to affect the health of consumers.

* Geometric average over a period of 2 months, with at least two samples a month.

---

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D-2300 Kiel 14, Germany
January 1993
HOMOEOPATHIC TREATMENT OF BOVINE MASTITIS

1 INTRODUCTION
The limited effectiveness of antibiotic mastitis therapy, the combined risk of antibiotic residues and related milk price reduction, and the additional financial losses incurred by the holding of milk during the withdrawal periods have renewed the interest in additional non-antibiotic treatments and/or alternative methods to cure mastitis. One such alternative may include homoeopathic treatment.

2 PRINCIPLE OF HOMOEOPATHY
Homoeopathy can be defined as a specific stimulating therapy which activates the natural cure and defence potential of the organism [1]. Homoeopathy is based on the principle "similia similibus curentur" (Hahnemann, cited after Leeser [2]) which means treat likes with likes. The theoretical basis states that any substance which can cause, at toxic dose, a range of clinical symptoms in a healthy individual, can be used, prepared as a homoeopathic remedy, to cure individuals showing similar symptoms. The precise mechanism of action of a homoeopathics is not yet known.

Homoeopathic remedies are prepared from plant and animal kingdoms and also minerals and their compounds with other chemicals. The preparation of these remedies is based on a series of dilutions and succussions, thereby rendering even a poisonous substance safe to use. Preparations are made on either a centesimal (1C) or decimal scale (1D). Repeated dilutions and succussions result in higher potencies, releasing more energy in the process. Therefore, homoeopathy is a system of medicine which concerns itself with energy and not with material doses of a drug.

At a dilution of 3C, which represents 1/1 000 000, all poisonous or harmful effects of any substance are lost and only curative properties will remain [3]. The potencies are classified into three groups: low, D1-D6; middle, D7-D30; and high, > D30) [4]. Lower potencies should be used to treat chronic cases, while higher potencies which are more energized should be employed in acute infections [3].

A common dosage of a homoeopathic remedy consists in an injection (s.c.; i.m.) or oral application of 5-10 ml for large animals [5].

Special types of homoeopathic remedies are nosodes or oral vaccines to be used therapeutically or prophylactically.

A nosode is a disease product obtained from any part of the organism during illness and thereafter potentized. The formation of substances in infected tissues as a result of interaction between pathogen and tissue is the basis of the nosode.

Oral vaccines are prepared from the actual organisms causing a disease and will contain the pathogens, their toxins or both deriving from filtrates which are potentized.

3 HOMOEOPATHICS TO TREAT MASTITIS
Table 1 summarizes some of the homoeopathics recommended for treatment of mastitis [5].

Table 1: Homoeopathics recommended for treatment of mastitis [5]

<table>
<thead>
<tr>
<th>Ac Dent</th>
<th>Hypericum</th>
<th>Apis</th>
<th>Lachesis</th>
<th>Asa foetida</th>
<th>Mercurius solubilis</th>
<th>Belladonna</th>
<th>Nux Vomica</th>
<th>Bryonia</th>
<th>Phellandrium</th>
<th>Calendula</th>
<th>Phosphorus</th>
<th>Chelidonium</th>
<th>Phytolacca</th>
<th>Collinicum</th>
<th>Pyrogenium</th>
<th>Conium</th>
<th>Silicea</th>
<th>Echinacea</th>
<th>Sulfur</th>
<th>Hepar sulfuris</th>
<th>Veratrum</th>
</tr>
</thead>
</table>

These remedies may be used separately or in combinations, depending on the clinical symptoms of a particular case. For homoeopathic mastitis control on a herd basis "mixed mastitis nosodes" including the common pathogens associated with mastitis, for example Streptococcus, Staphylococcus, E. coli, Pasteurella and Corynebacteria, can be used. For this purpose a suspension of these nosodes is dissolved in 500 ml sterile water and is applied by addition of 5-10 ml to the drinking water twice a week for 4 weeks [3].

4 EXAMPLES OF PUBLISHED RESULTS OF EFFECTIVENESS OF HOMOEOPATHICS IN MASTITIS CONTROL

4.1 Scientific Investigations
4.1.1 Acute clinical mastitis
A field study with 100 cows was performed in which clinical symptoms were precisely recorded and milk samples were cultured and milk cell counting was carried out [5]. The cows, each with one infected quarter, were split into two groups of 50 cows each. One group was treated using antibiotics, the other by homoeopathics. The farmers were advised to milk the cows several times a day. This frequent milking was done with a delay of 12 h in the antibiotic group while it was used in parallel with the application of the homoeopathics. The results are presented in Table 2.

The authors stress the trend that homoeopathic treatment was more successful in mastitis cases caused by Gram-negative pathogens, whereas the antibiotic treatment gained better results in Gram-positive cocci mastitis cases.

4.1.2 Subclinical mastitis
The results of an Irish study on the effectiveness of homoeopathic treatment for subclinical bovine mastitis are summarized in Table 3 [6].

4.2 Field observations
4.2.1 Acute clinical mastitis
The data in Table 4 were collected in a single veterinary practice in Germany [7]. Combinations of remedies available from different manufacturers were used for the treatment.

4.2.2 Subclinical mastitis
The results of homoeopathic treatment of 580 subclinical mastitis cases are given in Table 5.

Table 2: Comparison of mastitis cure rates after treatment by antibiotic or homoeopathics (after Merck et al. [5])

<table>
<thead>
<tr>
<th>Type of cure</th>
<th>&quot;Homoeopathic group&quot; (n = 50)</th>
<th>&quot;Antibiotic group&quot; (n = 50)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complete (A)*</td>
<td>17 (34%)</td>
<td>13 (26%)</td>
</tr>
<tr>
<td>Clinical (B)*</td>
<td>9 (18%)</td>
<td>12 (24%)</td>
</tr>
<tr>
<td>Clinical improvement (C)*</td>
<td>20 (40%)</td>
<td>17 (34%)</td>
</tr>
<tr>
<td>No (D)*</td>
<td>4 (8%)</td>
<td>8 (16%)</td>
</tr>
</tbody>
</table>

* A: No clinical symptoms; cyto-bact. results negative.
B: No clinical symptoms; cyto-bact. results positive.
C: Clinical udder symptoms; no milk changes.
D: Clinical milk and udder symptoms; cyto-bact. results positive.
Table 3: Homoeopathic treatment for subclinical bovine mastitis [6]

<table>
<thead>
<tr>
<th>1. Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.1 Homoeopathic oral preparation</td>
</tr>
<tr>
<td>1.2 Homoeopathic udder cream</td>
</tr>
</tbody>
</table>

Treatment was performed after morning and evening milkings for 17 days.

<table>
<thead>
<tr>
<th>2. Mastitis cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subclinical mastitis cases in 15 cows were treated</td>
</tr>
<tr>
<td>Milk samples were collected once weekly for culture and somatic cell counting</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>3. Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increase in:</td>
</tr>
<tr>
<td>- Number of subclinically infected quarters</td>
</tr>
<tr>
<td>- Number of quarters with cell counts &gt; 1 million</td>
</tr>
</tbody>
</table>

4.2.3 Prevention of mastitis by homoeopaths

The prophylactic efficiency of a combined mastitis nosode 30C tincture has been tested by a veterinary surgeon in England [8]. The treatment and the results are described in Table 6.

5 CONCLUSION

Scientific information on the effectiveness of homoeopathics in the control of bovine mastitis is too limited to justify a definite conclusion. There are mainly field observations (more or less subjective evaluation) by veterinary surgeons describing the successful administration in cases if clinical and subclinical mastitis. On the other hand, there are some additional reports indicating that in the control of diseases other than bovine mastitis (for example infertility, metritis, mastitis in ewes, mastitis-metritis-complex in sows etc.) homoeopathics could also be applied successfully [8-11]. The main difficulty in comparing results of different investigations using homoeopathics may lie in the most critical point of selection of the most suitable remedy based on the clinical symptoms and the "typing" of the patient, but also depending on the special experience of the investigator.

There is an obvious need for more detailed studies before a definite statement on the efficiency of homoeopathic mastitis treatment can be given.

LITERATURE

7 Dorenkamp, B. Homöopathische Alternativen in der Mastitisbe-
Table 6: Prophylactic efficiency of a combined mastitis nosode 30C tincture [8]

1. Treatment
A combined (S. uberis, S. dysgalactiae, S. agalactiae, E. coli, S. aureus) mastitis nosode was prepared (30C) and administered via the drinking water troughs.
A herd of pedigree Frisian cows was randomly split into two groups of 41 cows.

2. Results

<table>
<thead>
<tr>
<th></th>
<th>Control group</th>
<th>Treatment group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n = 41</td>
<td>n = 41</td>
</tr>
<tr>
<td>Case of mastitis</td>
<td>19</td>
<td>1</td>
</tr>
<tr>
<td>Average no.: quarters affected</td>
<td>1.16</td>
<td>1</td>
</tr>
<tr>
<td>Average severity: (scored 1-3):</td>
<td>2.16</td>
<td>1</td>
</tr>
<tr>
<td>Average duration: days</td>
<td>4.5</td>
<td>4</td>
</tr>
<tr>
<td>% of group affected</td>
<td>25</td>
<td>2.5</td>
</tr>
</tbody>
</table>


Jörn Hamann
Institute for Hygiene,
Federal Dairy Research Centre,
Kiel, Germany
November 1992

---

**CELL COUNT INTERPRETATION**

**INTRODUCTION**
The dairying system in most of Australia is pasture based or primarily pasture with concentrate feeding at milking time. The average herd size is 100 or more cows. The costs of production are possibly the lowest in the world. Unfortunately the financial return the farmers receive for their milk is perhaps the lowest in the western system of dairying.

Our farmers therefore have an aversion towards spending money on disease control so we have to adapt the elegant systems form Europe and the US to a more practical and least cost system. The 'herd' approach to diagnosis and treatment is therefore more preferable to considering the individual animal. Hence this method of evaluating cell count data.

All the information expressed in this working paper is strictly Australian based and may not be comparable to other dairying systems. The concept of using cell counting as a herd diagnosis method is exportable but would rely on using other dairying systems' 'rules of thumb' to allow interpretation of the graph patterns.

1 IMPORTANT "RULES OF THUMB"
I outline some "rules of thumb" which I use when looking at cell count data. These rules may not be 100% accurate but this does not diminish their usefulness. These rules mainly relate to how a cow responds to different pathogens and different physiological states.

1.1 Rules relating to cows
Uninfected cows have low cell counts during most of their lactation. On average, second lactation cows will have cell counts less than 250 000 and third lactation and greater will have cell counts less than 350 000. The only exceptions are at calving, drying off and if they have concurrent infections which cause reduced milk secretion (Three Day sickness, for example). For this reason, assessment of a cow's infection status is more reliable when cell counts are taken between 30 to 250 days of lactation.

Cows with one quarter infected will have widely varying cell counts such as that seen in Figure 1. In general, these cows can respond to dry cow therapy.

Cows with more than one quarter infected will have consistently high cell counts during lactation. These cows usually represent the chronic mastitis cases which do not respond to dry cow therapy. If there is no response to dry cow therapy, which will show as an elevated cell count during the first few months of the next lactation, these cows should be marked for culling.

Heifers, on the whole, do not become infected during their first lactation if they have no abnormalities of the teat and udder. Less than 5% of heifers may show clinical mastitis. A heifer with no infections will have a cell count less than 150 000.

![Figure 1: Individual cow cell counts - effect of subclinical mastitis.](image)

- No infection  
- One quarter  
- Two quarters

**Sampling time during lactation**
1.2 Rules relating to mastitis pathogens

Subclinical infections due to streptococci can have cell counts in the millions. These elevated cell counts can also be seen in new infections due to S. aureus. However, with staphylococcus infections, the cell counts begin to drop whilst with streptococcus infections they can remain elevated. These staphylococcus infections usually hover in the 400 000-700 000 cell count range.

Corynebacterium bovis is an obligate parasite of the bovine udder. It causes mainly udder irritation although subclinical and clinical infections can occur. High cell counts (up to 400 000) in response to this parasite can be seen in heifers but generally not in older cows. Cell counts in excess of 100 000 are common.

Both Streptococcus agalactiae and Corynebacterium bovis infections respond to effective teat dipping and blanket dry cow therapy. There can be chronic carriers of S. agalactiae which can only be removed from the herd by culling.

The finding of S. agalactiae in a herd should alert you to the possibility of ineffective teat dipping and/or dry cow therapy (use of neomycin based products) and/or culling (some very old cows in the herd).

Except for Streptococcus uberis and the less common pathogens such as Nocardia sp. and Pseudomonas sp., non-cow associated pathogens such as Escherichia coli, Enterobacter sp. and Klebsiella sp. are mainly cases of clinical mastitis and do not cause persistently elevated cell counts.

1.3 Rules relating to milking machines

Cell count increases can occur due to traumatic damage to the teat and udder.

Failure of a milking machine component such as a pulsator can cause a sudden rise in cell count due to both new infections and teat damage.

Major faults in the milking machine which can be reflected in persistent increase in cell counts are: inadequate vacuum reserve, vacuum too high, wrong slope on milk-line, flooding of milk-line, diameter of milk-line too small compared to herd size, disparity between parts of milking machine which causes disruption of milk flow (for example milk-line compared to filter housing), fault in automatic take-offs.

2 CELL COUNT GRAPHS

The above "rules of thumb" may seem obvious but how they can be used with cell counting to diagnose a dairy's mastitis problem may not. When NSW Agriculture had an operating Mastitis Section, this Section performed composite cell counts on all cows from a herd with a mastitis problem. The full farm history of the farm was supplied with these samples as well as any information on milking machine performance (if the milking machine was checked recently) and bacteriological culture data from both clinical and subclinical cases. (It was routinely requested of the clinician or livestock officer investigating these farms that at least five milk samples from high cell count cows be selected for culture. Any clinical mastitis data on the farm were obtained from laboratory records at the Regional Veterinary Laboratory.)

Frequently graphs were constructed from the composite cell counts (see Figure 2). Over time, it was observed that certain graph patterns were related with particular farm histories. These relationships seemed to hold for many of the farms being investigated. From these observations arose a practical diagnostic tool where the pattern of the cell count graph could highlight some problems on the farm which are contributing to the mastitis prevalence.

2.1 Examples of using cell count graphs

We will consider several cell count graphs and the problems they represent.

First, a farm with very good mastitis control (Figure 2a). The cell count graph is skewed to the left. This means that the majority of cows in the herd have low cell counts, with the largest frequency in the less than 100 000 group. A herd with this pattern would be using effective teat dipping and dry cow therapy and have a well maintained milking plant.

At the other extreme the cell count graph of a herd with poor or no mastitis control is skewed to the right (Figure 2b).
This herd does not use teat disinfection or dry cow therapy, washes teats with cloth and bucket and had the milking machine checked when it was put in during the 1950s. There are very few cows with low cell counts. In this example, there are no cows with cell counts less than 100,000. There is a disproportionately high number of cows with cell counts greater than 1 million.

This group of cows would represent newly infected cows, cows with *S. agalactiae* infection and cows with more than one quarter infected.

There are also a number of cows in the 400,000-700,000 cell count range which suggests *S. aureus* infection. In this herd there would be a myriad of reasons why staphylococcus would be present but one which may not be obvious is putting cups on wet teats. If teats are to be washed or come onto the milking platform wet, they must be dried thoroughly before the cups are put on.

Figure 2c shows a primarily left skewed graph, very few mid-range cell count cows and an increased number of cows with cell counts over a million. The interpretation of this graph will rely on considering the past history of bulk milk cell counts (BMCCs).

If the previous BMCCs were low, this pattern would suggest a rapid increase in new infection rate. The possible problems would be recent milking machine fault (pulsator failure), increased source of contamination (wet wea-

ther for example) or recent introduction of infected cows (mastitis problems in the calving paddock). It may also be due to leptospirosis or ephemeral fever and not due to bacterial or traumatic mastitis. Since leptospirosis can also increase during wet weather, you may have a combined problem. At this stage of a leptospirosis outbreak, the first signs are agalactia and 'fieccid' masti-

tis. However these signs may not be apparent.

To help your diagnosis, construct frequency graphs based on the different age groups (heifers, second cal-

vers, mature cows). If there are a large number of heifers and second calvers with high cell count, especially in the million plus group, then leptospirosis should be suspected. In warmer areas, high cell counts in heifers can be one of the first indications of ephemeral fever in a herd.

If the BMCCs have been rising over time, the possible reasons are cessation of a mastitis control procedure (drying udders, teat disinfection, back-
flushing), a milking machine fault and the presence of teat lesions (pseudo-
cowpox, change in teatcup liners). The mid-range counts may indicate the presence of teat damage.

In Figure 2d, this herd has a left skew-

ed graph but very few cows with cell counts less than 100,000. This pattern is typical of a herd with ineffective teat dipping (see Table 1). The reason for this unusual pattern is the presence of *Corynebacterium bovis* in the herd. In this herd, there are also a reasonable number of cows in the 400,000-700,000 cell count range, suggesting that *S. aureus* occurs in this herd. Teat dipping is probably the single most effective method for reducing *S. aureus* infection in a herd.

2.2 Using cell count graphs to advise farmers

When improving mastitis control procedures on a farm, assessing progress in mastitis by changes in BMCC can be disappointing. The BMCC may not substantially change for a long period of time although the infection rate in the herd is less. This situation is due to high cell count cows still in the herd contributing most of the cells measured in the vat milk.

A frequency graph of the individual cow cell counts is a better method of assessing progress. As the mastitis situation on the farm improves, the number of cows with cell counts less than 200,000 will increase while the number of high cell count cows will decrease. Repeated sampling over time will show the frequency graph shifting to the left. The farmer will then have a record of his progress over time.
<table>
<thead>
<tr>
<th>Wrong teat disinfectant concentration</th>
<th>Ineffective teat disinfectant</th>
<th>Poor application of teat disinfectant</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insufficient disinfectant component in the teat dip solution can occur because too little disinfectant is added, the emollient component is too large, or the wrong emollient has been added to the dip. The use of bloat oil or paraffin as emollient in teat dip should be discouraged strongly. These compounds can inactivate Hibitane teat dip rapidly. Even if used with iodophors, the iodine component can separate out with the oil layer. Glycerine or the lanolin ester compounds such as Emolan or Rualan should be advised and used at a maximum inclusion of 10%.</td>
<td>The effectiveness of teat disinfectant depends upon the age of the made-up solution. Iodine dip solutions have maximal effectiveness for approximately 3 days. Chlorine and most Hibitane dips should be remade daily. The life of the dip can be shortened if it is excessively contaminated by organic material. Recommending that new teat dip solution should be made daily minimizes the occurrence of this problem.</td>
<td>For teat disinfectant to work effectively, the entire teat surface should be covered by the disinfectant, not just the teat end. The aim is to reduce the primary population of bacteria living on the teats. When using teat sprays, if the spray is not held directly below the teats or the farmer is in a hurry, only the side of the teats may be disinfected.</td>
</tr>
</tbody>
</table>

2.3 Where to get these graphs
In NSW, these frequency graphs are generated automatically every time the farmer requests the cell counting option for Herd-Recording. They are sent to the farmer with his individual cell count results. In other States, you would need to approach the farmer for access to his cell count results. In small dairy herds, it is easy to calculate these graphs by hand. For larger herds, this will prove to be a headache. It would be better to enter the cell count information into a statistical package which generates frequency distributions.

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RESEARCH COMMUNICATIONS

A STRATEGY TO INCREASE RESISTANCE IN DAIRY COWS: EXPRESSION OF HUMAN LACTOFERRIN IN THE MILK OF TRANSGENIC COWS

(SUB)CLINICAL MASTITIS AND ITS CONSEQUENCES

Inflammation of the mammary gland (mastitis) in cows is a serious, frequently occurring condition which results from bacterial infection of the udder. Statistics from Scandinavian countries show that yearly about 20% of cows receive veterinary treatment for mastitis. A large variety of pathogenic bacteria have been cultured from mastitic milk. The most frequently isolated pathogens are S. aureus, Str. agalactiae, Str. dysgalactiae, Str. uberis, and E. coli. Mastitis, particularly when caused by certain E. coli strains, may result in the life-threatening sepsis syndrome, which occurs when pathogenic bacteria invade the bloodstream. The mortality of sepsis is very high. Mastitis that may thus cause serious suffering of the animal, confronts the farmer with costs resulting from treatment (for example antibiotics and other veterinary costs) and loss of production capacity. Mastitis is associated with a multitude of quantitative and qualitative changes in milk components which have been studied thoroughly over the last decades. These changes include an increased concentration in the milk of blood plasma proteins (for example of bovine serum albumin, BSA, and immunoglobulins), which leak from the blood into the milk when the so-called blood-milk barrier gets disrupted by the inflammatory process (vasopermeability increases and tight junctions between epithelial cells open up). The increased conductivity of mastitic milk reflects the increased influx of sodium and chloride from the interstitium into the milk. The protein synthetic and secretory activity of the mammary gland epithelial cells is also affected by the inflammatory process. The concentration of major milk proteins such as αs1- and β-casein, β-lactoglobulin and α-lactalbumin is lowered, whereas that of lactoferrin is increased. The cell count of mastitic milk, particularly that of leucocytes which are chemotactically attracted to the inflamed udder, is often dramatically increased. Homing of lymphocytes to the mammary epithelial lining and differentiation into immunoglobulin-producing plasma cells also contributes to the increased immunoglobulin content of mastitic milk. Proteolytic activity generated by the inflammatory process may result in milk protein degradation (for example of β-casein) and mammary gland tissue injury. It is to be noted that mastitis may result in irreversible damage of the mammary gland tissue. Less marked changes in the laboratory parameters described above are found in milk obtained from cows with so-called subclinical mastitis, that is, when the degree of mammary gland inflammation is not so severe as to result in mastitis with obvious clinical symptoms (clinical mastitis). The dairy industry imposes increasingly stringent rules with respect to some of the parameters described above relating to (sub)clinical mastitis. For example, the maximal acceptable number of cells in milk (EC guidelines) has been decreased from 500,000 (1989) to 400,000 cells per ml in 1993.

ANTIBACTERIAL DEFENCE MECHANISMS IN THE UDDER AND IN MILK

Anti-infective host defence systems in the mammary gland and milk comprise cells that can engulf and intracellularly destroy bacteria (phagocytes) as well as cells that can produce antibodies (lymphocytes). Milk proteins with antibacterial activity include immunoglobulins, lactoperoxidase, lysozyme, and lactoferrin (LF). Immunoglobulin either passively leaks through or is actively transported over the mammary gland epithelium by the so-called polymeric immunoglobulin receptor into the milk. The other antibacterial proteins are synthesized by the mammary epithelial cells.

The levels of both lysozyme and LF, that is, antibacterial proteins that can act synergistically, in mature bovine milk (0.1 μg/ml and about 0.1 mg per ml, respectively) are much lower than those in the milk of other mammalian species. For example, lysozyme and LF in human milk are around 50 μg/ml and 2 mg/ml, respectively. Levels of bLF in the mg/ml range are only observed in colostrum, during involution as well as after the onset of mammary gland inflammation (levels rapidly rise apparently in relation to the severity of mastitis, for example higher levels are observed in coliform mastitis). With a view to controlling infection, it appears illogical that the concentration of a protein with antibacterial activity only increases as a result of infection. This paper briefly describes the background and status of our research project that ultimately aims at analysing the effects (with respect to mastitis control) of constitutive expression of human lactoferrin (hLF) at levels between 1 and 2 mg/ml in the milk of transgenic cows. We have chosen to express hLF constitutively since it can as yet not be excluded that the distinct regulatory control of bLF resides in the bLF gene. The homology at the protein level of hLF and bLF is 69%. Another reason for expressing hLF instead of bLF at high levels in the milk of transgenic cows relates to the possible use of (purified) hLF in human health care. It is clearly understood that LF is not the only means of controlling mastitis, nor that all kinds of mastitis might benefit from constitutive hLF expression. It would however be beyond the scope of this paper to elaborate on this and also describe other relevant studies of our group.

STRUCTURE AND FUNCTION OF HUMAN LACTOFERRIN

LF is a single chain metal binding glycoprotein of Mr 80 000 that belongs to the transferrin family. The tertiary structure of crystallized LF has been elucidated. The amino acid sequence has been determined by protein sequencing and sequencing of full length cDNA clones. LF comprises two highly homologous domains that each can bind a single ferric ion with a high affini-
ty (Kd about $10^{-20}$ M), while simultaneously incorporating one bicarbonate ion. Each LF domain contains an N-glycosylation site. The significance of glycosylation in LF function is not understood. LF is present at high levels in mucosal secretions including tears, saliva, bronchial and intestinal secretions, seminal fluid, milk. LF is also present in the specific granulae of neutrophils and is released extracellularly during the phagocytosis process. LF is one of the most abundant whey proteins of human milk. The degree of saturation of HLF with iron in human milk is about 5%. LF has, in vitro, been shown to exert antimicrobial activity (sometimes in synergy with immunoglobulin and/or lysozyme) towards a large variety of potentially pathogenic microorganism including several mastitis pathogens (for example E. coli strains). The bacteriostatic activity (inhibition of bacterial growth) of LF towards iron-dependent bacteria appears to reside in its ability to scavenge free iron (iron deprivation). This antibacterial mechanism is thus restricted to the iron-free form of the protein. LF can also exert bactericidal activity (killing of bacteria) which is thought to be mediated through direct binding of LF (via its strongly positively charged N-terminal portion) to outer membrane components (for example lipopolysaccharide, LPS) of bacteria. The spectrum of bacteria that can be killed by LF has been shown to broaden upon limited proteolysis of this protein. In addition to its antibacterial activities, LF has been shown to exert various anti-inflammatory activities in vitro. These activities include inhibition of complement activation via the classical route, inhibition of the formation of toxic hydroxyl radicals (by scavenging free iron), inhibition of cytokine production and neutralization of LPS which is an important inflammatory mediator. LF has also been shown to promote the growth of intestinal cells as well as that of Bifidobacterium species which is the predominant organism of the intestinal flora of healthy, breastfed infants. Specific LF receptors have been isolated from intestinal cells and lymphocytes.

Based on the in vitro activities described above, it is generally believed that the main physiological role of LF in milk is to inhibit growth of pathogenic bacteria both in the mammary gland of the mother and in the intestinal tract of the suckling infant. A second role of LF would be to mediate transport of iron from the mother to the newborn.

**EXPRESSION OF HUMAN LACTOFERRIN IN THE MILK OF TRANSGENIC MICE**

Bovine αS1-casein is expressed in bovine milk at a level of about 10 mg/ml. We have cloned the bovine αS1-casein gene with large stretches of 5' and 3' flanking sequence. To drive expression of HLF to the mammary gland of transgenic animals, we have constructed many different expression cassettes consisting of 5' and/or 3' regulatory and flanking sequences of the bovine αS1-casein gene fused to either HLF cDNA or genomic HLF sequences. Transgenic mouse lines harbouring these hybrid gene constructs were generated to evaluate the ability of these expression vectors to drive HLF expression in the mammary gland of transgenic mice. The HLF constructs were introduced into the mouse genome by pronuclear microinjection of fertilized mouse oocytes which were subsequently transferred into recipients. Transgenic founder mice were bred to non-transgenic mice to generate transgenic F1 offspring, which always transmit the transgene to subsequent generations in a Mendelian fashion. Virtually all transgenic mouse lines appeared to express HLF in their milk. Many of them indeed expressed HLF in milk at the targeted levels, that is, in the mg/ml range. RNA analysis of multiple tissues revealed that expression of the transgene was restricted to the lactating mammary gland. RNA in all other tissues was below the detection limit, that is, at least 10 000-fold lower than in the lactating mammary gland. Primer extension analysis showed that the transgene used the same transcription initiation site as the natural bovine αS1-casein gene in bovine tissue. The immunoreactivity of transgenic HLF and human milk-derived (natural) HLF towards a panel of monoclonal and polyclonal antibodies did not differ. This indicates that antigenic determinants in transgenic and natural HLF are equally well accessible for the antibodies. The binding properties of natural and transgenic HLF to a large variety of HLF binding ligands also did not differ. Native natural and transgenic HLF elute at exactly the same position from cation-exchange columns. The N-terminal protein sequences of natural and transgenic HLF are identical. This indicates that the signal sequence is fully and correctly removed in the murine mammary gland to yield mature transgenic HLF. A slight difference between natural and transgenic HLF was observed on SDS-PAGE analysis. This difference very likely resides in glycosylation microheterogeneity as both proteins exhibited the same mobility after complete deglycosylation. Spectroscopic analysis of natural and transgenic HLF showed that natural HLF was saturated with iron for about 5%, whereas the transgenic protein was almost completely saturated. The latter appears to relate to the high iron content of mouse milk and indicates that transgenic HLF is functional with respect to iron binding. The kinetics of iron release at different pH values of completely saturated natural and transgenic HLF did not differ. Analysis of milk samples from transgenic and non-transgenic mice did not show any significant difference, except for the presence of transgenic HLF. No adverse effect on the physiology and health of the lactating transgenic mothers as well as of the pups was demonstrable. Based on these observations, we conclude that it is feasible to express HLF that is virtually identical to natural HLF, in the mammary gland of a heterologous species.

**GENERATION OF TRANSGENIC DAIRY CATTLE**

Most groups generating large transgenic animals have applied essentially the same methodology as that used to obtain transgenic mice. However, since these protocols require two surgical steps (isolation of zygotes from donor animals and transfer of microinjected zygotes to oviducts of recipients) they are impractical, inefficient and therefore expensive. We decided to combine in vitro embryo production with gene transfer technology, thus enabling non-surgical transfer of blastocysts that have developed in vitro from microinjected zygotes. Immature oocytes were collected by aspiration of follicles present on ovaries obtained from the slaughterhouse. These oocytes were matured in vitro and subsequently fertilized with sperm of elite bulls. After 18-23 h fertilized oocytes were centrifuged (to visualize pronuclei) and microinjected with HLF constructs. At day 9 after the onset of maturation, about 20% of microinjected zygotes (cultured in medium conditioned by bovine oviduct epithelial cells) had developed to the compact morula/blastocyst stage. In total 21 pregnancies were established after non-surgical embryo transfer to the uterus of hormonally synchronized recipients. Two fetuses from which no intact DNA could be isolated for analytic purposes were lost during pregnancy. In 2 out of 19 calves that were born after a normal pregnancy, we could detect the presence of the transgene.
One female calf appeared mosaic (the transgene could not be detected in all tissue samples) for a transgene in which a rearrangement event had occurred. In the other case the transgene had integrated correctly and was present in all tissues tested (including sperm). Breeding of this bull was initiated in January 1993. Considering the relatively low transgenesis rate observed in cows, we decided to focus our efforts on methods to assess integration of the transgene in preimplantation embryos; particularly since appreciation of the significance of constitutive expression of HLF in the milk of transgenic cows (with respect to reducing mastitis) may require the generation of additional transgenic founders. Methods for early detection of transgenesis (by PCR analysis of and in situ hybridization on blastomeres) are currently being implemented in our “in vitro program”. These methods reduce the number of recipients required to generate (larger numbers of) transgenic cattle on a routine basis.

References
Available on request.

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SYSTEMIC DRY COW THERAPY - AN UPDATE

In view of the serious shortcomings of conventional (intramammary) dry cow therapy (DCT), the possibility of substituting or supplementing DCT with systemic DCT was explored. Potential candidates for effective systemic DCT are drugs possessing physical-chemical properties which support good penetration from blood into the milk, are capable of being retained in the udder of the dry cow for several days, and have good and stable intrinsic (in vitro) activity against major udder pathogens, particularly S. aureus.

Systemic DCT was administered to groups of cows after the last milking of lactation. Antimicrobial agents used were β-lactams, aminoglycosides, tetracyclines, sulfonamides and potentiated sulfonamides which were administered intravenously or intramuscularly at conventional (or greater than conventional) doses. Milk and dry udder secretion samples were collected at 1 or 2 day intervals up to 14-18 days after treatment and were analysed for drug concentrations. Penetration of these drugs into the udder was limited and it appears that antimicrobials most commonly used by large animal veterinary practitioners and dairy farmers are not likely to be of value for systemic DCT.

Three macrolide antibiotics available in veterinary practice, that is, tylosin, spiramycin and lincomycin, were then similarly investigated. Drug penetration into the milk was extensive, as expected for the weakly basic lipophilic macrolides. The i.v. route of administration resulted in peak milk drug concentrations which were significantly higher than those seen after an equivalent dose was injected i.m. Duration of potentially effective concentrations, however, was longer following i.m. dosing. Furthermore, two 10 mg/kg i.m. injections of spiramycin at a 3 day interval prolonged the duration of potentially effective drug concentrations in the udder to nearly 1 week and a single i.m. injection of a long-acting spiramycin formulation at 33 mg/kg resulted, 14 days later, in dry udder spiramycin concentrations of 0.77 ± 0.41 μg/ml (n = 16 cows). Such treatment administered to 17 cows infected at drying off with S. aureus resulted in a cure rate (63.6%) which was similar to that observed for conventional DCT in the same herd.

In a preliminary field efficacy study, systemic norfloxacin nitrate DCT appeared to be more effective than systemic oxytetracycline DCT and conventional intramammary cephalixin DCT. Extensive investigations were then conducted for the purpose of optimizing the duration and persistence of norfloxacin in the udder of the dry cow by examination of the relative effect of i.v., i.m. and s.c. routes, spacing additional injections at 2-3 days after the initial treatment which was given at drying off, the effect of dose and degree of udder emptiness at treatment time. Factors optimizing duration of drug in the dry udder were dose (20-25 mg/kg i.m., single treatment) and completeness of last milking. Incomplete last milking, presence of udder induration and subclinical inflammation were factors associated with low norfloxacin concentrations of short duration.

Seemingly, these findings point to some severe limitations of the potential efficacy of norfloxacin DCT. It should be realized, however, that norfloxacin is quickly accessible to milk polymorphonuclear leukocytes (PMN). Thus, norfloxacin concentrations in milk PMN are 3-6 times higher than in the extracellular milk. Therefore, additional field efficacy studies on a scale somewhat larger than the initial, preliminary field study were undertaken. In these studies the i.m. dose of norfloxacin was titrated in the range 5-20 mg/kg. The cure rate and the new infection rate (N.I.R.) for S. aureus in the 5 mg/kg treatment group were very similar to the corresponding rates found in a no-treatment control group. Cure rates and N.I.R. for the 20 mg/kg group (n = 40 cows with 39 quarters) were 26.5% and 15.8%. Thus, although contemporary controls were not used, the efficacy of systemic DCT using norfloxacin nitrate was essentially very similar to the efficacy of historical control, conventional intramammary DCT as is currently being recorded for dairy cows in Israel.

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ITALY
THE PREVENTION OF MASTITIS IN ITALY

In Italy, mastitis is a problem which costs farmers and the dairy industry about 300 million EU/year. Despite the high costs of the pathology, in Italy there is no common approach to the prevention and control of the disease. The farmers receive advice from at least four different bodies:

a) compulsory: veterinarians of the National Health Service through the Local Health Units (USL) check for:
- the absence of the major infectious diseases (tuberculosis, brucellosis and leukosis)
- the quality of milk used to produce pasteurized milk (< 400 000 SCC/ml; < 100 000 CFU/ml for TBC; > 3.7% fat; > 3.2% proteins, absence of antibiotic residues);

b) compulsory/voluntary: dairy plants where milk payment is based on quality induce the farmers to improve milk quality in order to obtain premiums or avoid fees, and in some cases the plants help the producers to achieve this;

c) voluntary: AIA (National Association of Italian Breeders) controls the performances of the herds for reproduction, milk production and somatic cell count on a cow basis. If a farmer has high somatic cell counts in bulk tank milk or in some cows, the local association of breeders could help the farmer by means of their practitioners and technicians;

d) voluntary: private companies selling drugs, feeding, disinfectants and others offer technical services to help the farmers, as a part of commercial strategies.

Through these different bodies any Italian dairy herd is checked, willing or not, as far as milk production is concerned. Most of the assays are performed at the area diagnostic laboratories (Istituti zooprofilattici sperimentali-IZS-) or other authorized laboratories, such as those belonging to AIA, as well as at the laboratories of the various veterinary colleges.

Unfortunately, despite the large use of such technical competence, the results are not as good as they should be, probably because of the different approaches to mastitis control and the different interest underlying the advice. This means that most of the efforts are dispersed and the final task (mastitis control) is far from being achieved.

One of the commitments of the Mammary Pathology Centre (MPC) is to educate practitioners, technicians and farmers to improve milk quality and consequently milk production. One of the major targets is to develop and apply a rational and practical approach to mastitis control which also could be implemented by other organizations (USL, AIA, IZS) and from private companies or laboratories.

In recent years there has been continuous collaboration with local USL, AIA and IZS units developing a common approach to mastitis prevention and control. MPC usually has the role of coordinator but, in some cases, it is also the reference diagnostic laboratory, with the local units in charge of the field extension services. The programme was devoted mainly to the control of contagious mastitis (Str. agalactiae and Staph. aureus), but some of effort was devoted to the control of environmental mastitis too.

The main features of the programme for contagious pathogens, are:
- detection of the affected cows based on quarter milk samples
- segregation of infected cows
- treatment of infected cows with less than 200 lactation days and control of therapy at 14-21 days
- specific treatment (with antibiotic sensitivity test) of dry cows
- control of cows and heifer at calving
- control of healthy group at 2, 5, 8, 12 months after the segregation
-ulling of cows still infected after at least two therapy cycles and/or after the dry period.

Together with these procedures we advise the farmers to: check the efficiency of the milking machine (routinely twice per year for the AIA members); introduce or improve teat-dipping after milking; avoid washing the mammary gland; and use disposable paper towels dry or soaked in a chlorhexidine solution to clean the teats.

The latter part is emphasized for herds with environmental pathogens, and special attention is devoted to improving the hygiene of the bedding and the housing of dry cows and heifers.

The programme started in 1990 with 50 herds in different provinces of northern Italy and the first results showed that, within the herds Str. agalactiae positive, 80% of them are now free from this pathogen, while 60% of Staph. aureus positive are less than 5% of prevalence. The results on the control of environmental pathogens are not so good. One of the major problems we have is overcrowding of the barns. The number of cows is increased to increase production, which results in dramatic consequences in terms of bedding hygiene and pathogen exposure.

Apart from the results, our task is to set an example, showing how the cooperation between the health authorities (USL), the diagnostic laboratory (IZS), the farmers (AIA) and the research group (MPC) could work; when this happens results can only be brilliant.

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**SWITZERLAND**  
**MASTITIS PATHOGENS IN SWITZERLAND**  
**1988-1991**

Samples taken by Veterinarians from Cows with Clinical Mastitis

<table>
<thead>
<tr>
<th>Results</th>
<th>1988 (N = 80 604)</th>
<th>1989 (N = 83 520)</th>
<th>1990 (N = 96 958)</th>
<th>1991 (N = 80 078)</th>
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<tr>
<td></td>
<td>%</td>
<td>%</td>
<td>%</td>
<td>%</td>
</tr>
<tr>
<td>Bact. negative</td>
<td>22.3</td>
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<td>17.2</td>
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<tr>
<td>S. <em>dysgalactiae</em>, enterococci)</td>
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<td>1.6</td>
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<td>7.1</td>
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<td>8.5</td>
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Samples taken by Udder-Health-Service Extension Workers from Cows with Subclinical Mastitis

<table>
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<tr>
<th>Results</th>
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<th>1990 (N = 38 197)</th>
<th>1991 (N = 28 497)</th>
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<tr>
<td></td>
<td>%</td>
<td>%</td>
<td>%</td>
<td>%</td>
</tr>
<tr>
<td>Bact. negative</td>
<td>23.7</td>
<td>28.8</td>
<td>26.7</td>
<td>26.4</td>
</tr>
<tr>
<td>Bact. positive</td>
<td>76.3</td>
<td>71.2</td>
<td>73.3</td>
<td>73.6</td>
</tr>
<tr>
<td><em>Streptococcus agalactiae</em></td>
<td>7.2</td>
<td>5.7</td>
<td>5.0</td>
<td>5.6</td>
</tr>
<tr>
<td>&quot;Other streptococci&quot; (S. <em>ubерis</em>,</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>S. <em>dysgalactiae</em>, enterococci)</td>
<td>25.1</td>
<td>23.8</td>
<td>24.0</td>
<td>21.9</td>
</tr>
<tr>
<td><em>Staphylococcus aureus</em></td>
<td>43.3</td>
<td>41.4</td>
<td>42.2</td>
<td>41.4</td>
</tr>
<tr>
<td>&quot;Other staphylococci&quot; (S. <em>epidermidis,</em></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>S. <em>xylosus</em>, S. <em>hyicus</em>, Micrococcus sp.)</td>
<td>16.2</td>
<td>19.0</td>
<td>20.4</td>
<td>16.4</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>8.2</td>
<td>10.1</td>
<td>8.4</td>
<td>14.7</td>
</tr>
</tbody>
</table>

Prof. Dr M. Schallibau  
Federal Dairy Research Institute,  
Milk Production Section,  
Liebefeld-Berne, Switzerland
INTERNATIONAL SYMPOSIUM ON BOVINE MASTITIS
MILAN, 22 MAY 1992

The Facoltà di Medicina Veterinaria (School of Veterinary Medicine) of the Università degli Studi di Milano was founded 200 years ago. Among the different initiatives organized for the bicentenary, the Mastitis Pathology Centre of CNR (National Research Council) and the Institute of Infectious Diseases, in cooperation with IDF, Group A2, organized a 1-day symposium on bovine mastitis held in Milan on 22 May 1992.

The aim of the symposium was to illustrate the current state-of-the-art in mastitis research to Italian practitioners and scientists working in the dairy field. Afterwards the Italian member of Group A2, Prof. G. Ruffo, illustrated the scientific history of the Mammary Pathology Centre; some of the members and other Italian experts covered the most important topics related to the problems of mastitis.

Papers presented were: Cost of Mastitis (J. Booth), Acute Mastitis (C. Burvenich), Physiopathology of Machine Milking (J. Hamann), Environmental Mastitis (L. Smith), Prevention of Mastitis (H. Salonieri), Therapy of Mastitis (C. Beretta), Immunology of Mammary Gland (A. Zeconni), Milk Residues (W. Heeschen). Just to mention two of the contributions: James Booth illustrated the costs of mastitis in the UK and Italy, neither is self-sufficient as far as production is concerned, and consequently the economic importance of the prevention of mastitis; Walter Heeschen illustrated risks to public health implied by the use and the abuse of drugs, particularly antibiotics and chemotherapeutics, also in relation to the new rules and indications at EEC level. Lectures were highly interesting and were entirely published in the proceedings of the Symposium.

The great interest was confirmed by the large number of practitioners, university and industry researchers attending the symposium for the whole day, and contributing to a general discussion.

It was the first international meeting on mastitis, organized in Italy, in 20 years. The large participation and interest confirmed the need for such an exchange of ideas and experiences between the world of research and field not only within each country but also on an international scale.

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INTERNATIONAL COLLOQUIUM “STIMULATION AND MILKING”, LEIPZIG, GERMANY

The Agricultural Engineering Department of the University of Leipzig organized the International Colloquium “Stimulation and Milking” at Grimma near Leipzig from 5-7 May 1992.

The conclusions drawn from the 26 papers presented and the corresponding discussion are summarized below. These conclusions have been discussed and confirmed by all 48 participants.

1 Basic physiological elements of stimulation
1.1 Stimuli which are applied during the milking process to initiate the unconditioned or conditioned milk ejection reflex can be defined as “stimulation in its narrower sense” (mainly short-term effects).

1.2 The milk ejection reflex causes the myoepithelial cells to contract with the result that the alveolar milk can be withdrawn. Normally a threshold level of oxytocin in the blood is needed to cause the myoepithelial cells to contract. In addition, secondary effects of milk ejection can be seen in changes in intramammary pressure, smooth muscle tone and blood circulation.

1.3 No direct effects on the milking process are established by the release of further hormones involved with evoking the milk ejection reflex.

1.4 The effects of stimuli (see 1.1) on the lactation yield can be defined as “stimulation in its broader sense” (mainly long-term effects). Hormonally regulated mechanisms involved with stimulation acting on the whole organism may be relevant for the yield but need further scientific investigation to be explained. Furthermore, some of the data presented indicate interactions between local and systemic mechanisms which influence the secretory activity of the mammary gland.

1.5 Inhibition of milk ejection, especially such as that observed in heifers,
seems to be correlated with insufficient oxytocin blood concentration.

2 Stimulation by milking machine and methods for measuring the efficiency

2.1 For the examination of milking-related stimulatory effects, parameters such as incomplete milk ejection, hormonal profile of the blood, intramammary pressure, milk flow profile, changes in blood circulation, and tone of the smooth muscle of the gland and/or teat tissue may be used in addition to parameters such as yield, milk fraction and milk composition. As well as parameters of stimulatory effects on lactational yield, the inclusion of udder health criteria may be useful.

2.2 The application of effective stimuli should be done during a long enough premilking period (reference: 1 min manual udder preparation) and a milking associated stimulation to ensure a maximal degree of udder evacuation. In this way the milkability parameters are also influenced positively under physiological and labour economic aspects. The question is still open whether long-term application of short (less than 30 s) premilking udder preparation, consisting of foremilking and udder cleaning, has resulted in genetic selection against the need for long premilking stimulation which, in turn, has led to high yields at an acceptable feed conversion ratio.

2.3 Economic and production-related conditions of dairying (for example herd size, type of housing, degree of mechanization and automation) determine the possibility of applying manual premilking udder preparation, whereas the available milking technique will determine the degree of stimulation associated with the milking process. Today, the market offers milking systems which increase and contribute to the efficiency of manual premilking udder preparation methods. Before such new milking systems are applied under practical conditions, sufficient information on their efficacy should be available to avoid economic losses.

2.4 To prevent milk yield losses it is essential to avoid long delays before the milking unit is attached, if manual pre-milking udder preparation is applied.

2.5 When a robot milking system was used and premilking stimulation consisted of only short dry cleaning without foremilking, the highest standard deviation of single cow milk yield was found when the daily milking frequency was four. Despite a partly incomplete degree of udder evacuation (about 90%), milking frequencies > two/day resulted in significantly higher yields compared to that after milking twice a day probably because incomplete milking was overcompensated by increasing milking frequencies.

2.6 Functional milking machine parameters (for example positive pressure, pulsation frequency, liners with different characteristics) can be used in different ways to increase the stimulatory efficiency during milking.

3 Secondary effects of stimulation

3.1 Physiologically, the milking process is accompanied by teat tissue changes (for example length, diameter, blood circulation, thickness, keratin lining of the teat canal). Stimulatory measures resulting in improved milkability characteristics cause fewer changes in teat condition deviating from the physiological norm which in addition, may reduce the new infection risk of the mammary gland.

3.2 The application of systems to identify early mastitis, for example conductivity measuring systems, must be related to interferences by stimulation measures in the form of changes in milk composition in different milking fractions.

In view of the information elaborated during this seminar, future scientific treatment of the topic "stimulation and milking" should be more cow-oriented. The whole organism-related aspects should take into account the further increasing yield demand of lactating cows. Furthermore, aspects to keep the cows healthy and to control their health status with regard to a balanced input/output metabolic relationship should be discussed more extensively.

CURRENT SITUATION OF MASTITIS AND MILK QUALITY

BERLIN 1992

A conference on current problems in mastitis and milk quality, organized by Prof. Dr K. Wendt from the Veterinary Faculty of the Humboldt-University, Berlin and Prof. Dr J. Schulz from the Veterinary Faculty of the University of Leipzig, was held at the Veterinary Faculty of the Humboldt-University, Berlin, on 21 and 22 February 1992.

Nineteen papers dealt with the main topics: pure research in mastitis, diagnosis of mastitis, mastitis control and mastitis therapy.

The paper of J. Schulz, Leipzig, dealt with the blood-udder-barrier in healthy and sick udders of cows. In the region of glandular parenchyma this barrier consists of different parts: blood-connective tissue; connective tissue-epithelial cells-milk; milk-epithelial cells-connective tissue; connective tissue-blood; connective tissue-lymphatic system. The function of the barrier is influenced by the lactation period, drying off, bagging up before calving, and especially by mastitis which breaks up the endothelium of capillaries and the epithelial cell line of alveoli, thus making them passable for large molecules (blood serum proteins, fibrin). Furthermore cell wall membranes, tight junctions and intracellular membranes are also parts of the blood-milk barrier. The barriers in their morphological variety and functional complexity have a large influence on the reaction of drugs.

P. Madaj and M. Abdusalam, Leipzig, dealt with the occurrence and the significance of mast cells in the mammary gland of cows. Mast cells have been detected by pseudosycyanine technique in infected and non-infected glands. The technique is less complicated.

M. Lunau and K. Wendt, Berlin, presented a paper on the importance of lysozyme in the udder secretion of heifers. An increase in lysozyme activity by unspecific stimulation or bacterial infection was found in experimental investigations. But an increase in lysozyme content alone is not sufficient to prevent an infection. It is a fact that only the combination of all defence mechanisms can protect against infection.

V. Vobis and E. Grün, Leipzig, dealt with the reaction of the components of the lactoperoxidase-thiocyanate-H₂O₂ system in milk of healthy quarters and quarters with subclinical mastitis. The
found on dead and desquamated epithelial cells, on the basement membrane and on connective tissue but not on the intact epithelial cell line. Most of the germs are phagocytosed 24 h after experimental infection in macrophages and neutrophilic granulocytes. At the same time macrophages with phagocytosed bacteria were seen in lymphatic vessels. 10-39 days after experimental infection with A. pyogenes most of the bacteria were found phagocytosed and destroyed in macrophages. A clear adherence was not recognizable. Free germs in a high number were detectable only in one quarter with necrotic mastitis.

J. Smola, Brno, presented a paper on the investigation of bulk milk as a screening method for the control of mastitis caused by Sc. agalactiae.

W. Gedek, Grub, dealt with the demands and conditions of quality protection of raw milk in the European Community from 1993. The lowering of cell count standard to 400 000/ml means an improvement in milk quality. The cell count of bulk milk as a basis for mastitis control brings unsatisfactory results. A monthly check of the cell count of individual cows gives better information about the mastitis situation. In case of difficulties with too high cell counts the producer can enlist the advisory service for mastitis control. The detection of inhibitory substances in milk becomes more and more sensitive and specific by using new methods.

G. Meene, Leipzig, H. Kämpfer, Chemnitz and G. Teichmann, Dresden, dealt with the situation of udder health and measures for its improvement in cow herds of the new German states. In many herds with cell counts of bulk milk above the standard a check of milking hygiene and milking technique as well as cytological and bacteriological investigations of milk samples are necessary. Temporary results of an analysis are: cell count above 500 000/ml in more than 25-30% of bulk milk; most problems in new founded farms and herds which are chronic infected with Sc. agalactiae; Sc. agalactiae is the most important mastitis pathogen; important faults exist in milking hygiene, milking technique and in keeping of veterinary principles.

J. Rund, Rostock, discussed in his paper actual aspects of mastitis control and therapy. A lot of milk producers in Mecklenburg/Vorpommern have difficulties with too high cell counts in bulk milk. A specific bacteriological investigation is an important basis of mastitis control and reducing the cell count. Besides the so-called classical mastitis pathogens, environmental germs are the main reservoir of infection. Bacteriological examination is difficult in chronic diseases. An examination of dry cows with mastitis is really necessary. J. Hamann, Kiel, dealt with mastitis control on the basis of cytological results of bulk milk. He pointed out that cell counts in bulk milk above 300 000/ml are an indication of an important mastitis problem. Assessment of udder health alone with the help of the cell count results of the bulk milk and without further information does not seem to be possible with present knowledge. Beside secretory influences, non-secretory influences which can interfere with the meaningfulness of relevant bulk milk cell count must also be considered. A specific cell count of bulk milk should be defined for every herd on the basis of bacteriological results in view of physiological and management influences. Above this limit measures to control mastitis should come into force. But this limit is not fixed, and must be adapted according to the dynamic development of conditions in the herd.

R. Oswald, H. Mielke and A. Bergmann, Leipzig, discussed the influencing of experimental udder infection with Sc. iberis by BST. Administeration of 640 mg BST 24 h after infection leads to a faster decline of clinical signs and in the first 3 weeks after infection to a 8.4% higher milk yield than in the control group without BST administration.

J. Hamann, Kiel, dealt with the possibilities and limits of antibiotic mastitis therapy, which is part of an integrated mastitis therapy. Impressive clinical results are often faced with inadequate bacteriological curing rates. In the future the bacteriological curing rate will be increased by the use of new techniques (drug administration, early detection of mastitis) as well as the use of therapeutic necessary dosages and therapy periods, especially with the inclusion of a systematic administration of antibiotics. Continued hygiene and prophylaxy are required to control mastitis caused by environmental germs, especially in modern dairy farms.

From the VEYX-Pharma Ltd., Schwarzenborn, a paper was presented about inflammation, enzymes and enzyme therapy.

A. Bergmann, Leipzig, discussed the safety and effect of tetramisol hydrochloride and levamisol hydrochloride by intramammary administration in experimental mastitis, caused by Proteotheca zopfii. The treated quarters showed more increased clinical signs as the untreated quarters 3-24 h after the first administration of 20 ml tetramisol hydrochloride. The number of Proteotheca zopfii in the milk was also reduced.

The paper of H.-W. Fuchs and W. Haider, Berlin, dealt with alterations and reaction in early stages of bovine mastitis. Different alterations and reaction in bovine mastitis are important for diagnosis and for a pathogenetic statement. The acute catarrhal mastitis is characterized by hyperaemia, exudation of plasma proteins, emigration of neutrophilic granulocytes and damage of epithelial cells. Isolated cells and neutrophilic granulocytes were seen in the early stages of bovine mastitis. The acute catarrhal mastitis is characterized by necrosis of parenchyma cells, haemorrhagic and thrombotic disorders in circulation, exudation as well as local epithelial necrosis can be observed as early as 4 h after Escherichia coli infection. Epitheloid and giant cell granulomas caused by Nocardia asteroides becomes apparent 6 days after infection.

W. Haider, Berlin, dealt with immunological investigations on mammary glands. By using the avidin-biotin-peroxidase complex technique the reaction of Sc. iberis was investigated in organ cultures of udders and in experimental infected quarters. A clearly recognizable adherence of bacteria was
Ch. Merk, Berlin, discussed mastitis therapy with homeopathic drugs. Therapy of acute mastitis with homeopathic drugs in an experiment produces the same good results as an antibiotic therapy. In *E. coli* infection, in particular, good results were obtained. An advantage is the loss of inhibitory substances in the milk, but the therapy needs much time. A combination of homeopathy and chemotherapy will have good prospects in the future.

D. Draehmpaehl, Berlin, dealt with acupuncture on the udder. He showed how the blood flow in the udder can be influenced by reflex action. Acupuncture influences, in particular, the autonomic nerves of udder vessels and the peripheral nerves. Using the examples of udder oedema and mastitis, he discussed different acupuncture points in their pathophysiological action.

H. Nattermann, Berlin, K. Wendt, Berlin, and G. Hoi Sorensen, Arhus, discussed finally the possibilities and limits of immunoprophylaxis of mastitis in heifers, caused by *A. pyogenes*. After immunization with a toxoid vaccine mice showed a good protection against intravenous challenge infection. After intracisternal challenge infection pregnant heifers showed no protection given by vaccination. They became diseased more seriously than non-vaccinated animals.

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**BRITISH MASTITIS CONFERENCE 1992**

The fifth annual British Mastitis Conference held in October 1992 at Stoneleigh, Warwickshire, England, was attended by over 200 farmers, veterinary surgeons and agricultural advisers. The programme was based largely on the requests of delegates at previous conferences and included a successful poster section.

The most popular papers were a thoroughly practical session on problem solving by a veterinary surgeon and his client, and a research paper on milk secretion. Other sessions included papers on the prospects for breeding for improved resistance to mastitis, the defence mechanisms of the udder, the use of cell counts, and papers on current research on the immune responses in the bovine mammary gland and on the approach to treatment using antimicrobial peptides.

The proceedings of the conference, which include abstracts of the posters, are available from Ciba-Geigy Agriculture, Animal Health Department, Whittlesford, Cambridge, CB2 4QT, England, at £15 ($25) per copy, including postage. The next conference will be held on 13 October 1993 at the same venue.

J. M. Booth, UK
February 1993

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**5TH INTERNATIONAL SYMPOSIUM ON MACHINE MILKING OF SMALL RUMINANTS**

**BUDAPEST, HUNGARY, 17-21 MAY 1993**

Dear Colleague,

The 5th International Symposium on Machine Milking of Small Ruminants which will be held in Budapest, Hungary, 17-21 May 1993.

The main topics of the Symposium are:
- Physiological aspects of milk production and milking;
- Mastitis: reasons and possibilities of prevention and treatment;
- Relationships between the udder anatomy and milk production abilities;
- The choice of the animal material and its improvement; organization of milk recording - application of microelectronics in performance control;
- New developments in milking parlours and machines, and in housing (associated with machine milking);
- Labour organization within the milking parlour, relation man/machine;
- The role and importance of machine milking and hand milking practice in specific production systems;
- The importance of feeding systems especially in the case of high yielding dairy small ruminants;
- Relations among the keeping and machine milking of small ruminants and the on-farm milk processing as well as the environment;
- Socio-economical aspects of machine milking: the future of small ruminants’ dairy farming.

The official languages of the Symposium are English, French and Hungarian, simultaneous translation will be provided.

Before the Sessions, 2-day-long technical tours will be organized to different farms and firms as well as the mid-symposium visit will also be in the programme.

Sándor Kukovicz, President
Hungarian Organizing Committee
MASTITIS CONTROL
by a Group of Experts

Results of Questionnaire 1889/A of 16 pages with results from 23 countries: data for cow population, mastitis control schemes, monitoring procedures, antibiotic sensitivity, mastitis control measures, milk payment, progress in mastitis control.

It is part of a three-part Bulletin which also covers payment systems for ex-farm milk and the alkaline phosphatase test as a measure of correct pasteurization.

Bulletin №262/1991 - 1400 BEF

DESIGN OF CLINICAL TRIALS FOR
MASTITIS THERAPY
by Margaret A. Thorburn, Dept. of Population Medicine, Ontario Veterinary College

This 8-page report covers clinical trials of therapeutic treatments; causes of mastitis and its consequences.

It is part of a five-part bulletin which also covers: radionuclides in dairy products; distribution systems for fresh dairy products; enzymes in cheesemaking; and teat & udder cleaning.

Bulletin №247/1990 - 1500 BEF

ENVIRONMENTAL INFLUENCES ON
BOVINE MASTITIS
by a Group of Experts

Covers mastitis as a multifactoral disease, pathogenesis, sources & transmission of pathogens, environmental influences in animal health, external environment, internal environment, conclusions and recommendations.

Bulletin №217/1987 - 1000 BEF

MACHINE MILKING & MASTITIS
by a Group of Experts

Comprises a) recommendations concerning the use of milking machines and the incidence of mastitis; b) review of literature on milking machine factors affecting the rate of new infections; c) review on the effect of machine milking on teat end condition.

Bulletin №215/1987 - 1000 BEF

BOVINE MASTITIS: DEFINITION & GUIDELINES FOR DIAGNOSIS
by a Group of Experts

This bulletin gives new (compared to 1967) proposals for mastitis definitions, diagnosis & the results of an IDF trial on the interpretation of diagnostic data

Bulletin №211/1987 - 800 BEF

PROGRESS IN MASTITIS CONTROL
by a Group of Experts

This survey describes in tabular form the progress made in 23 countries, on the basis of an enquiry conducted in September 1983. In a previous survey, of 1977 (see Bulletin 121), many countries reported little progress; this time, 7 countries reported definite improvement to 1983.

Bulletin №187/1985 - 500 BEF

MASTITIS RESEARCH INDEX
(10TH EDITION, 1993)

Three sections: projects, worker index, subject index; 24 countries are covered.

Free

MASTITIS RESEARCH INDEX
(9TH EDITION, 1992)

Three sections: projects, worker index, subject index; 25 countries are covered.

Free

MASTITIS RESEARCH INDEX
(8TH EDITION, 1991)

Three sections: projects, worker index, subject index; 20 countries are covered.

Free

MASTITIS NEWSLETTER №17

Antibiotics and sulfonamides in milk-risk evaluation of residues (Prof. W.H. Heesch, Germany), Teat dipping before milking - summary of UK field trials (S.A. Langridge, UK), What future for conductivity? (A. Zeconci, Italy), Mastitis pathogens in Switzerland (M. Schallibaum, Switzerland), Mastitis events, Mastitis publications.

Available on request - June 1992 - Ref. №128

MASTITIS NEWSLETTER №16

Somatic cell counting, individual cow somatic cell counts, mastitis cell count data, efficacy of on-line measurement of quarter electrical conductivity, mastitis notes from Iceland, Japan, Norway, Switzerland, mastitis events, IDF and other mastitis publications.

Available on request - July 1991 - Ref. №122

MASTITIS NEWSLETTER №15

EEC Cell count requirements - Use of cowside tests for mastitis - Research Communications - Some reflections on application of DNA probes in mastitis diagnosis - Predipping in perspective - Mastitis notes from member countries.

Available on request - May 1990 - Ref № 112

MASTITIS NEWSLETTER №14

Cell count standard - Mastitis cell count data - Future EEC cell count requirements - New information relates diets of dairy cows to mastitis.

March 1989 - Ref. №106

MASTITIS NEWSLETTER №13

Questionnaire on national herd milk mastitis cell counts - Mastitis notes from member countries.

March 1988 - Ref. №102

MILK - ENUMERATION OF SOMATIC CELLS
MACHINE MILKING MASTITIS

The IDF Subgroup of Experts on Machine Milking and Mastitis (A2D) is working on a monograph "Machine milking induced teat tissue reactions and new infection risk" which is grouped into the following chapters:

I  GENERAL ASPECTS
1  Physiological status of the bovine teat
   1.1  Introduction
   1.2  Description of the physiological status of the teat
   1.2.1  Parameters and methods
   1.2.2  Factors of influence
   1.2.2.1  Anatomical and physiological aspects of the teat
   1.2.2.2  Individual cow factors
   1.2.2.3  External factors
   1.3  Reference system
   1.4  Guidelines on evaluation of physiological teat reactions to milking

2  Machine induced changes in the status of the bovine teat with respect to the new infection risk
   2.1  Introduction
   2.2  Description of machine induced changes in the bovine teat
   2.2.1  Parameters and methods
   2.2.2  Milking machine induced teat tissue reactions
   2.2.2.1  After a single milking
   2.2.2.1.1  Appearance and teat condition
   2.2.2.1.2  Anatomic structure and tissue composition
   2.2.2.1.3  Status of physiological activity
   2.2.2.2  After repeated use of machine milking
   2.2.2.2.1  Appearance and teat condition
   2.2.2.2.2  Anatomic structure and tissue composition
   2.2.2.2.3  Status of physiological activity
   2.3  Teat tissue status and new infection risk
   2.3.1  External lesions
   2.3.2  Internal lesions
   2.4  Guidelines on evaluation of machine induced teat reactions with respect to new infections

3  Functional parameters of milking units with regard to teat tissue treatment
   3.1  Introduction
   3.2  Description of influence of functional parameters on teat tissue treatment
   3.2.1  Linear design
   3.2.2  Vacuum level
   3.2.3  Pulsator rate
   3.2.4  Pulsator ratio
   3.2.5  The a, b, c, d-values of pulsation
   3.2.6  Positive pressure pulsation
   3.2.7  Vibration pulsation
   3.2.8  Pulsationless milking
   3.2.9  Hydraulic milking
   3.2.10  Other milking factors
   3.3  Guidelines on evaluation of influence of functional parameters on teat tissue treatment

II  SPECIAL ASPECTS
1  Teat tissue status and environmental mastitis
2  Teat tissue status and new infection risk during dry off period and during early lactation stage
3  Machine milking and teat tissue reaction with respect to robot milking

III  APPENDIX: TERMINOLOGY AND DEFINITIONS