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Brief Communication

Milking-Related Changes in the Surface Temperature of the Bovine Teat Skin

The surface temperature of the teat at rest is about 30°C in healthy dairy cows kept indoor under ordinary conditions. It usually decreases by about 1°C during manual premilking teat stimulation *(Hamann & Dück* 1984, *Eichel* 1986, *Hamann* 1988, 1992). And it usually increases above the level prior to the stimulation by up to about 2.5°C during the milk removal phase of conventional machine milking *(Schneider* 1981, *Hamann & Dück* 1984, *Hamann* 1985, 1988, 1989, *Mayntz* 1990, *Eichel* 1992). The statements made are supported by the results of our own investigations, i.e. – 0.8 and + 2.1°C, respectively.

Our communication is meant to be a contribution to current world wide discussion about milking related teat reactions and their possible implications with regard to udder function and health. It presents interpretations of the temperature changes in concern which deviate from those proposed in the literature.

The temperature decrease. The interpretation originally launched by *Hamann & Dück* (1984) – to which *Eichel* (1986) agrees – is repeated by *Hamann* (1992) and summarized by him as follows. "Thus, the combined effects of removing the blood in the teat veins, reducing the rate of muscle contractions and an increased area of the teat skin are probably responsible for the decrease in teat skin temperature immediately after pre-milking udder preparation".

By "veins" is quite obviously meant the specific teat venous system of Fürstenberg in the inner part of the teat wall, by "muscle" the extravascular teat smooth muscle, and the temperature decrease is apparently supposed not to occur until the initial stimulation response in this muscle (transient contraction) has been replaced by relaxation (cf. *Isaksson & Sjöstrand* 1984).

Our opinion on the temperature decrease is principally based on the statements – in relevant details adapted by us to the bovine teat skin – concerning the temperature conditions in the skin in general that are presented in the textbooks of physiology edited by *West* (1991), *Swenson & Reece* (1993), and others. The cutaneous vascular plexuses are integrated in the whole-body thermoregulatory system and – what is more important in the present context – they also respond locally to various kinds of local extrinsic stimuli, not least mechanical and thermic ones. In the main the responses to these stimuli are mediated by adrenergic vasoconstrictor nerves (contraction or relaxation of vascular muscle, respectively; α-adrenergic responses), or are directly evoked by the stimulus in concern.

Manual premilking teat stimulation is certainly an adequate mechanical stimulus and can be presupposed to be able to elicit a series of events in the teat skin starting with contraction in the smooth muscle layer of the local cutaneous vascular plexuses (α-adrenergic re-

sponse). The contraction causes vasoconstriction which diminishes the blood and heat flow through the plexuses. This reduces the heat dissipation from the teat skin into the surroundings. Thereby the temperature gap between the skin surface and the ambient air becomes narrower, i.e., the temperature of the skin surface decreases. The small decrease (about 1°C) should lack physiological, as well as other, significance.

It may here be interposed that exposition of the teat skin (as well as glabrous skin in general) to cold usually elicits the same reactions – vasoconstriction and diminished flowrate in the cutaneous vascular plexuses with subsequent reduction of heat dissipation from the skin surface and lowered surface temperature. In hairy skin contraction of the arrector muscles of the hairs also occurs. By this means loss of body heat is reduced.

It may also be mentioned that the contraction in the cutaneous vascular muscle of the teat in all probability is closely related to the simultaneously occurring transient contraction in the extravascular teat muscle. This results in teat shortening and stiffening, and teat skin wrinkling. It is described by *Isaksson & Sjöstrand* (1984) as most likely being caused by reflex discharges in adrenergic nerves (α-adrenergic response).

The temperature increase. According to *Hamann & Dück* (1984) and *Hamann* (1985, 1987, 1989), the temperature increase in the teat surface during milking is due to circulatory changes (possibly disturbances) in the teat wall induced by the mechanical teat treatment in the teatcup. *Eichel* (1992) means that the small increases (about 1°C) observed by him under, according to him, favourable milking conditions "did not provide any clue to excessive stress on teats by mechanical milking".

There are in our opinion at least 3 circumstances which definitely influence the temperature conditions during milking: (1) the milk flow through the teat lumen, (2) the enclosure of the teat in the teatcup, and (3) reactions in the cutaneous vascular plexuses.

Ad 1. As can be deduced from the following experiment, the teat wall is warmed up by the milk flow during milking. The lumen of excised teats was passed by water, the temperature of which was 38° – 39°C, i.e., equal to the temperature of central parts of the udder corpus directly after slaughter and to the temperature of the milk when it leaves the streak canal during milking (own measurements). The water was let in by a plastic tube into the uppermost part of the teat cistern and let out through the streak canal which was cannulated. The flowrate of the water was appr. 250 ml/min. The temperature of the teats before the water passage was adjusted to 30°C (skin and cisternal membrane surfaces measured). The temperature of the skin surface increased to 38.0°C in less than 6 min. The teats were enclosed in a teatcup-like model (shell, no liner) during the water passage.

Ad 2. For obvious reasons the enclosure of the teat in the teatcup effectively diminishes heat loss from the teat surface by dissipation.

Ad 3. Contrary to the manual premilking stimulation (and, usually, stimulation by cold), the temperature increase in the teat wall caused by the milk flow elicits relaxation in the smooth muscle layer of the cutaneous vascular plexuses (in the main α-adrenergic response) with vasodilation and enhanced blood flowrate (active hyperaemia). Most likely similar reactions also take place in the corresponding vessels under the cisternal membrane.

Teat wall temperatures above a physiologically acceptable level are prevented by transmission of surplus heat across the vascular walls and removal of it from the teat by the blood stream. Cf. the textbook literature referred to above.

In the live cow (blood circulation functioning) heat gain is largely balanced by heat loss to the blood stream; the temperature increase in the teat skin surface usually being up to 2.5°C. In the experiment with excised teats (no circulation), on the other hand, heat from the water was largely accumulated in the teat wall; the temperature increase in the teat skin surface being 8.0°C. This gives an idea of the effectiveness of the thermoregulatory mechanisms in the teat skin with respect to removal of excess heat from the teat. It also explains the fact that the temperature increase is not correlated with the machine on-time (own observation). The physiological significance of the involved mechanisms should be obvious.

The active cutaneous hyperaemia mentioned above makes the ordinary pallor of unpigmented teat skin at rest change into a more or less reddish hue during milking (as noticed by observant milkers). Cf. the textbook literature referred to above.

Enhanced flowrate in the superficial teat skin vessels has been demonstrated by *Persson* (1991) using the laser Doppler flowmetry method; the flowrate increased during milking by a factor of 1.8 – 2.6.

The diagnosis repeatedly made in the literature (e.g., *Hamann* 1987) of milking-induced congestion (passive hyperaemia) and oedema in the teat wall can, with respect to the teat skin, be excluded in this context. Congestion would have been characterized by decreased (not increased) flowrate in the vascular plexuses, and lowered (not raised) temperature, and bluish (not reddish) hue in the skin surface. And oedema would have given the teat skin a doughy consistency (what has never been observed in our experimental work with conventional milking machines). Cf. *Isaksson & Lind* (1992) where physiologically significant active hyperaemia in the specific teat venous system of Fürstenberg has been dealt with.

A fourth circumstance deserving some attention in this connection is the role that might be played by the pulsating liner movements. Heat is certainly produced in the liner wall both by the liner movements per se and – in the inner liner surface – by occurring friction between the liner and the teat surface. Most likely, however, the teat does not receive any liner heat during milking but rather emits some to the liner because the teat skin surface is, in our experience, invariably warmer than the inner surface of the liner at temperature measurements immediately after finished milking – even when the liner has been used for a series of cows, one after another (according to customary milking practice).

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