Invited review: Abomasal damage in veal calves

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ABSTRACT

Within all cattle production systems, veal calves are the most severely affected by abomasal damage, with current prevalence at slaughter ranging from 70 to 93% of all animals affected. Although most damage is found in the pyloric region of the abomasum, fundic lesions are also found. Despite past research into the etiology of abomasal damage and the many risk factors that have been proposed, consensus on the causal factors of abomasal damage in veal calves has not yet been reached. The aim of this review was to integrate and analyze available information on the etiology of, and possible risk factors for, abomasal damage in veal calves. We describe various proposed pathways through which risk factors may contribute to damage formation and conclude that the etiology of abomasal damage is most likely multifactorial, with diet being a main contributor. Pyloric lesions, the most common type of damage in veal calves, are likely the result of large and infrequent milk and solid feed meals, whereas fundic lesions may be caused by stress, although the evidence for this is inconclusive. Providing calves with multiple smaller milk and solid feed meals (or ad libitum provision) may decrease abomasal damage. In future research, ulcers, erosions, and scars as well as fundic and pyloric lesions should be recorded separately, because etiologies of these may differ. Further research is required to understand the exact pathway(s) by which milk replacer causes abomasal damage in veal calves; that is, whether low abomasal pH, overloading, or composition are important. Further research is also required to elucidate whether rapid intake of milk replacer and solid feed, which is influenced by restricted amounts fed, inter-calf competition, and calf breed, increases abomasal damage. Research is also needed into the effect of medication and nutrient deficiencies other than iron. The types of experimental designs that can be used for future research could be enhanced if a means to

assess abomasal damage antemortem is developed. We conclude that it is unlikely that abomasal or ruminal hairballs, iron deficiency, water provision, and various infections and diseases are significant contributors to abomasal damage in veal calves.

Key words: abomasal damage, veal calf, etiology, risk factor

INTRODUCTION

Abomasal damage involves lesions of the inner wall of the ruminant abomasum, which include minor perturbations or more severe damage causing bleeding or perforation of the wall and subsequent peritonitis. Abomasal damage is a problem in cattle of all ages and all production systems, with “white” veal calves (hereafter, veal calves) being most affected (e.g., Brscic et al., 2011; Kureljušić et al., 2013; Hund et al., 2016). Abomasal damage in the form of lesions can cause high mortality rates of, for example, between 0.53 and 0.11% in veal calves in Switzerland and Belgium (Bähler et al., 2012; Pardon et al., 2012a). The mortality rates only reflect the most extreme forms of abomasal damage—perforating ulcers—and hence represent only the tip of the iceberg. Average reported prevalence of nonfatal damage at slaughter ranged from 70 to 93% of veal calves in Europe (Bähler et al., 2010; Brscic et al., 2011). Certain veal farms in Europe have reported 100% prevalence for abomasal damage (Brscic et al., 2011). Veal calves are reared on a diet made up of milk replacer (**MR**), supplemented by moderate amounts of solid feed (**SF**) with a high percentage of concentrate (at least in Europe since 1997), until a slaughter age of approximately 6 mo and a BW of approximately 200 to 250 kg. The MR is typically fed in buckets or troughs twice a day, although some farms use automated milk dispensers, which allow more frequent feedings (typically 3 meals per day; Bokkers and Koene, 2001; Brscic et al., 2011). The SF is generally only fed after the morning MR meal, in the same container as the MR, once the MR has been consumed. This diet of mostly iron-poor MR and concentrate ensures low blood hemoglobin levels and the pale color of the veal.

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The exact implications of abomasal damage for calf welfare are not fully understood. Whether calves experience pain due to nonperforating abomasal damage has not been determined. In most cases, the presence of abomasal damage is not associated with clinical signs (Veissier et al., 1998; Marshall, 2009; Hund et al., 2016), unless the lesions are severe enough to perforate the abomasal wall or cause hemorrhage (Smith et al., 1983, 1986). Commonly, affected veal calves are found dead in the stable or lesions are only identified at slaughter (Marshall, 2009). Mortality following abomasal perforation, of course, does present a welfare issue. Although it has been proposed that (nonperforating) abomasal damage may reduce feed intake and thus lead to decreased growth and economic losses (Tajik et al., 2012), most studies have been unable to identify a reduction in growth (Welchman and Baust, 1987; Breukink et al., 1989; Bähler et al., 2010).

Many articles have addressed the causes and predisposing factors of abomasal damage in veal calves and, although no consensus has yet been reached, it is generally accepted that the etiology is multifactorial. The aim of this systematic review is to integrate and analyze the available information on the etiology of, and possible risk factors for, abomasal damage in veal calves. The literature search was conducted from January to April 2017 using the search engine Web of Science, and included the following search terms: Abomas* AND (damage OR ulcer* OR lesion* OR scar*). In addition, the technique of snowballing references (using the reference list of a paper to identify additional papers) was applied. Titles and abstracts were scanned, during which papers referring to non-bovine species or not in English or Dutch were discarded. When no articles on bovines could be identified on a specific mechanism of interest, other ruminant and monogastric articles were used. These other articles were selected based on relevance to the mechanisms of interest only, given that often very few papers were written on the topics we sought, with a preference for ruminant species over monogastric species. Because the fourth stomach compartment of the ruminant, the abomasum, is functionally similar to the monogastric stomach, it may be that knowledge of gastric ulcers extends to abomasal ulcers, although this should be approached cautiously. For some articles, only the abstract could be obtained. This led to 122 articles being evaluated for this review. Despite the fact that veal production systems have changed substantially over the past decades, older literature was included, because experimental studies from that time are often still relevant. This review is divided in 4 parts, covering the association between abomasal damage in veal calves and (1) nutritional factors, (2) stress, (3) diseases, and (4) other miscel-

laneous factors such as breed and season. We will begin with an overview of lesion type and localization and end by proposing paths for future research.

ANATOMICAL LOCALIZATION AND LESION TYPE

In veal calves, abomasal damage has been commonly described as consisting of 3 types of lesions: erosions, ulcers, and scars (e.g., Wiepkema et al., 1987; Veissier et al., 1998; Webb et al., 2013), although other methods, such as estimated surface area, have recently been applied to record lesion severity (Berends et al., 2014). In this review, we will distinguish between erosions, ulcers, and scars, as these may have slightly different etiologies because of differences in location. Erosions are local defects of the mucosal layer that have not yet penetrated the lamina muscularis mucosae, the thin layer of smooth muscle that separates the lamina propria from the submucosa (Mattiello et al., 2002; Marshall, 2009; Webb et al., 2013). They are small compared with ulcers, usually only 1 to 20 mm in diameter (Sun, 1974; Smith et al., 1983), with more recent studies reporting mean erosion sizes of 0.7 cm² (Webb et al., 2013). In addition, erosions are likely to have a lower prevalence than ulcers (Webb et al., 2013). Ulcers are lesions of the abomasal mucosa that penetrate into the submucosa and range from a few millimeters to several centimeters in size (Mattiello et al., 2002; Marshall, 2009; Webb et al., 2013). Ulcers can cause perforation of the abomasal wall, which can lead to inflammation and infection of the peritoneum (peritonitis), the membrane that forms the lining of the abdominal cavity (Jensen et al., 1976; Tanwar et al., 2009). Ulcers have been classified into 4 types. Type 1 ulcers are nonperforating ulcers that come without extensive bleeding; type 2 ulcers are nonperforating and involve (severe) blood loss; type 3 ulcers are perforating with local peritonitis; and type 4 ulcers are perforating with diffuse peritonitis (Smith et al., 1983; Marshall, 2009; Van Immerseel et al., 2010). Most experimental studies, however, did not use the latter classification to distinguish between ulcer types. Scars are thought to be healed ulcers, partially because they are found in a similar location and are fibrous contractions of the mucosa (Degen, 1982, as cited by Wiepkema et al., 1987; Webb et al., 2013). When abomasal ulcers heal, the wound contracts and new scar tissue is synthesized (Smith et al., 1983). No scar tissue is formed in the healing process of erosions because erosions heal using epithelial regeneration, which does not involve the formation of scar tissue (Sun, 1974, as cited by Smith et al., 1983). In veal calves, abomasal damage is mostly found in the pyloric region of the abomasum (Lourens et al., 1985; De Wilt, 1985; Welchman, 1986; Pearson et al., 1987; Breukink et al., 1989; Veissier et

al., 1998; Marshall, 2009). Nevertheless, erosions can also be found scattered throughout the abomasum (Wiepkema et al., 1987) and in the fundic region, though with lower prevalence and severity (Groth and Berner, 1971 as cited by Welchman and Baust, 1987; Bähler et al., 2010; Valgaeren et al., 2013).

FACTORS ASSOCIATED WITH NUTRITION

A complete overview of all factors associated with nutrition and their possible role in the development of abomasal damage in veal calves is presented in Table 1.

Milk Replacer

In the past, European veal calves were fed only MR, until European legislation mandated the provision of fibrous feed in addition to MR in 1997 (European Union Directive 97/2/EC). At slaughter, it was observed that many of the calves fed only MR were suffering from abomasal lesions, with a prevalence of up to 70% (Wensing et al., 1986; Wiepkema et al., 1987). An MR-only diet was typically fed in 2 meals per day, and increased linearly in volume throughout the fattening period, ending with provision of approximately 3 kg of MR powder per day (Prevedello et al., 2012; Webb et al., 2013). The provision of MR has now decreased because of the mandatory provision of SF, the latter being generally provided above the European Union's requirement of 50 to 250 g/d (Brscic et al., 2011). The theory is that an MR-only diet causes abomasal lesions via abomasal overloading or low abomasal pH; however, this has never been studied experimentally and, without further research, it is impossible to know whether these pathways are indeed accurate. Below we present the proposed mechanisms behind these 2 theories and some indirect evidence in support of, or contradicting, these theories. The term "milk" will be used when both MR and whole milk are discussed together or when the distinction is not important. Although overloading and pH are the most mentioned theories in relation to the effect of MR on abomasal damage, the specific milk composition might also affect abomasal damage. No research could be found on the effect of MR composition on abomasal damage; nonetheless, composition will affect the clotting potential of the MR, which might, in some way, affect abomasal damage. This area warrants future attention.

Abomasal Overloading. As explained above, most lesions in veal calves are found near the torus pylorus, which controls the passage of abomasal contents into the duodenum and which is a site of peristalsis and segmentation. Overloading of the abomasum could cause localized hypoxia in the pyloric region (Lourens et al.,

1985; Breukink et al., 1991): the pathway is proposed to start with an increase in the tonus of the abomasal muscles, leading to peristaltic contractions that are strongest around the pylorus. Both these contractions and direct pressure exerted on the abomasal wall by a large milk volume could lead to compression of the mucosa and blood vessels and subsequent oxygen shortage. Over time, damaged sites could develop into erosions and ulcers, although the exact pathway for this is unclear. Current evidence is insufficient to support this theory. The only findings in favor of the overloading theory are 3 articles providing some indirect evidence: Veissier et al. (1998) found that group-housed calves that (probably) drank their MR meal faster had more pyloric lesions than individually housed calves that (probably) drank their MR slower; and Bähler et al. (2010) and Welchman and Baust (1987) found that the heaviest calves, hence possibly dominant, faster-drinking calves, developed the most pyloric (but not fundic) lesions. Two articles opposing the overloading theory are Berends et al. (2014), who found that decreasing milk meal size while simultaneously increasing the concentrate part of the diet caused worse damage (experimental study), and Brscic et al. (2011), who found that calves receiving relatively low amounts of MR had a higher risk for lesions (risk assessment study).

Low Abomasal pH. In rats, horses, and humans, a low gastric pH has been associated with a higher frequency of gastric ulceration or eroding (Nagamachi and Skoryna, 1977; Murray, 1999; Uchida et al., 1999). In adult beef cattle, a lower pH has been associated with more abomasal erosion (Jensen et al., 1992), which has led to the proposition that pH may also be an important factor in abomasal damage in calves (Ahmed et al., 2002; Marshall, 2009). There is no direct evidence currently for this, however. In fact, Hund et al. (2016) reported no difference in lumen pH between damaged and intact abomasa of slaughtered bulls, cows, and (non-veal) calves. Pathways explaining the possible relationship between low abomasal pH and abomasal damage are as follows: (1) excessive activation of the proenzyme pepsinogen into pepsin, whereby the proteolytic activity of pepsin may break through the barriers protecting the abomasal wall and cause lesion of mucosal proteins (Nagamachi and Skoryna, 1977; Ahmed et al., 2002; Mesarič et al., 2002); and (2) compromised functioning of the mucus layer that protects the abomasal mucosa, which leads to decreased hydrogen carbonate production and increased back-diffusion of hydrogen ions into the abomasal wall, because fewer ions are neutralized by hydrogen carbonate before coming into contact with the wall (Nagamachi and Skoryna, 1977; Lourens et al., 1985; Yandrapu and Sarosiek, 2015). In support of the latter, mucin concentration was reported to be lower

Table 1. Nutritional factors put forward as likely to worsen abomasal damage in veal calves and associated number of studies in support (for) or not in support (against) of these proposed factors

Factor	For	Against	Summary of findings	Conclusion ¹
Milk replacer (MR)	4	2	No direct evidence, except that potentially faster drinking calves have worse damage.	Unknown
Abomasal overloading	0	0	No study has assessed this link.	Unknown
Low abomasal pH				
Solid feed				
Roughages vs. MR only	9	2	Roughages tend to exacerbate existing damage caused by MR, except when provided ad libitum.	Yes, in restricted amounts
Coarse vs. less coarse roughages	6	5	In restricted amounts, straw tends to worsen damage compared with only MR or other types of roughage.	Yes, in restricted amounts
Roughages vs. concentrate	2	3	Concentrates cause less damage when small amounts are provided. In large amounts, concentrates are worse than roughage.	Yes, in small amounts
Larger particles of roughage	1 ²	4	This is not supported by experimental evidence.	No
Increasing amounts of solid feed	2	3	Larger amounts of solid feed worsen damage when amounts are relatively low (500 g/d) or when a large portion is concentrate (80:20). However, when roughage is provided ad libitum, existing damage is not exacerbated.	No, if ad libitum roughage; yes, if mostly concentrate
Poor rumen development	3	1 ³	Rumen development may protect against abomasal damage when large quantities of concentrate are not provided.	Probably
Ruminal hairballs	0	1	This is not supported by experimental evidence.	No
Abomasal hairballs	0	0	No study has assessed this link in veal calves.	Unknown
Nutrient deficiency				
Iron deficiency	0	1	This is not supported by experimental evidence.	No
Copper deficiency	0	0	No study has assessed this link in veal calves.	Unknown
Water				
Water provision	1	1	Evidence is inconclusive, but the only experimental study is against.	Unlikely

¹The bold typeface indicates those factors that might contribute most to abomasal damage.

²Larger particles = more but less severe lesions.

³Large quantities of concentrate led to better rumen development but worse damage.

at damaged sites (Pearson et al., 1987; Breukink et al., 1991) and in the pyloric region (Lourens et al., 1985), the region in which most damage occurs in veal calves.

Indirect evidence that pH may play a role in the development of abomasal damage is the successful treatment of abomasal and gastric ulcers using medication that increases abomasal pH, either by neutralizing secreted HCl or by decreasing HCl secretion, in other mammals (adult cattle: Tharwat and Ahmed, 2012; sheep: Morgado et al., 2014; musk-ox, moose, deer, and wapiti: Haigh, 1982; humans: Maton and Burton, 1999; Holle, 2010). Although medication can be used in the treatment of ulcers, lack of knowledge on their long-term (health) consequences limits its application as a preventive measure. Moreover, preventive administration of medication could be considered unethical. It should be noted that the HCl-secretory cells, whose secretions cause abomasal acidity, develop only after a few days of life, possibly to prevent colostral antigens from being broken down (Lourens et al., 1985; Weiner, 1996; Guilloteau et al., 2009).

Abomasal luminal pH in calves depends on meal volume, sucking rate, abomasal emptying rate, acidity of the milk, and the buffering and clotting capacity of the milk (Woodford et al., 1987; Ahmed et al., 2002; Constable et al., 2005, 2006). Smaller milk volumes provided multiple times a day maintain a higher and more stable abomasal pH than infrequent large meals (Woodford et al., 1987; Ahmed et al., 2002). Normally, acidified MR would lead to a decreased abomasal pH compared with normal MR (Vajda et al., 2007); however, acidified MR can be provided *ad libitum*, leading to more frequent consumption (Webb et al., 2014), although not all studies support this (Hill et al., 2013). The clotting properties of the milk can affect abomasal pH, because whole milk, which has a fast clotting capacity, allows for a lower pH than nonclotting MR (Constable et al., 2005). Hence, adjusting the milk regimen can affect abomasal pH, and potentially abomasal damage, although no direct evidence has yet been presented for the latter.

Solid Feed

If SF is supplemented to an MR diet, the incidence of abomasal lesions is often observed to worsen (Wensing et al., 1986; Welchman and Baust, 1987; Breukink et al., 1991; Veissier et al., 2001; Mattiello et al., 2002; Cozzi et al., 2010; Berends et al., 2012; Prevedello et al., 2012; Webb et al., 2013). However, such an effect has not been found with all roughage types, quantities, and particle sizes. The current theory is that SF can exacerbate damage that has already been caused by large quantities of MR in two ways. The first pathway is by causing trauma, often referred to in the literature

as abrasion, to the abomasal wall. The second pathway is by blocking the pylorus, thereby delaying digesta from leaving the abomasum and exacerbating abomasal overloading by extending the time during which large quantities remain in the abomasum (Welchman and Baust, 1987; Mattiello et al., 2002; Webb et al., 2013). The traumatizing capacity of SF is thought to be enhanced by the earlier mentioned increased peristaltic contractions caused by abomasal overloading with MR because those contractions lead to increased contact between the coarse SF and the abomasal wall (Lourens et al., 1985). The effects of SF on abomasal damage may depend on the SF type provided, its physical form, and the amount of SF fed.

Solid Feed Type. Roughage types fed to veal calves include wheat and barley straw, lucerne (or alfalfa), beet pulp, maize silage, and maize cob silage, although the high starch content of maize cob silage makes it similar to concentrate rather than roughage. With the exception of lucerne, these roughage types are chosen because they have a low iron content and thus minimally affect the hemoglobin level of the blood, which helps to preserve the pale color of veal. In research, hay is sometimes fed to veal calves, but this is rarely done on farms, because hay has a high iron content and will cause the meat to darken (Blokhuys, 2000). The effects of roughage on abomasal damage are not entirely clear. Studies have generally found that the feeding of straw, a very coarse roughage, exacerbates abomasal lesions (Van der Mei, 1985; Welchman and Baust, 1987; Breukink et al., 1991; Webb et al., 2013), although 5 studies do not support this (Van Putten, 1982; De Wilt, 1985; Veissier et al., 1998; Prevedello et al., 2012; Webb et al., 2015). The order of roughages from least to most deleterious appears to be hay < maize cob silage/beet pulp < lucerne < maize silage < straw (Wensing et al., 1986; Breukink et al., 1991; Mattiello et al., 2002; Råber et al., 2013b; Webb et al., 2013). However, comparison between studies is difficult due to different amounts and particle sizes of roughage being fed. Interestingly, Råber et al. (2013a,b) found no significant difference in the pylorus between maize silage and straw but did find more lesions in the fundus of straw-fed calves.

Cereal grains, barley grains, whole-plant maize pellets, and pellet mixes (containing, for example, oat hulls, maize or barley grain, soy flakes or plant oils, and a pellet binder) are the types of concentrate that have been researched in veal calves for their effects on abomasal damage. However, most studies combined both concentrate and roughages in the diet. Only one study added solely concentrate to an MR diet and found a decreasing trend for lesion incidence compared with straw (Råber et al., 2013a). In addition, one study

compared 2 concentrate types, and found no difference in lesion incidence between them (Räber et al., 2013b). Furthermore, feeding pellets of roughage and concentrate with 4 different compositions (differences were in the starch, fiber, crude protein, and ash contents) did not affect lesions >0.5 cm (Morisse et al., 2000). Adding concentrate to roughage may prevent an increase in the lesion incidence that would normally happen with roughage (Morisse et al., 1999). However, some studies found no improvement or even a worsening of the damage with a combination of roughage and concentrate compared with only roughage (Berends et al., 2012; Prevedello et al., 2012). Very high levels of concentrate (concentrate:roughage $\geq 80:20$) have been seen to lead to acute ulceration in beef calves (Tharwat and Ahmed, 2012) and can increase abomasal damage in veal calves as the amount fed increases, even when MR is decreased simultaneously (Berends et al., 2014). It should be noted that no studies used a larger relative proportion of roughage than concentrate, and therefore no general conclusion for all combinations of roughage and concentrate can be made.

The addition of specific feedstuffs, such as extruded pea, extruded soybean, or urea, has been researched to determine their value in adding protein or nitrogen to the diet without compromising veal color and quality. No effect on the incidence or severity of abomasal lesions was found with the addition of these feedstuffs (Prevedello et al., 2012; Brscic et al., 2014).

Physical Form of Solid Feed. A feedstuff can be provided in various physical forms. Roughage can be fed as large particles, chopped to a smaller particle size, ground, or included in a pellet. Different physical forms may have different traumatizing or blocking effects, and one might expect that larger particles are more blocking than shorter ones and therefore cause more abomasal lesions. Shorter particles, however, may be sharper and may become stuck in the abomasal wall more easily. Chopping wheat straw to particle sizes equal to those of barley grain lowered lesion incidence to approximately equal levels for the 2 feedstuffs, supporting the theory that longer particles cause more damage, although the severity of the lesions was higher for barley grain (Cozzi et al., 2002b). This implies that both the size and type of feed affect abomasal damage. In contrast, Webb et al. (2013) found no effect of providing roughages in long-chopped, short-chopped, or ground form.

For pelleted feeds, it would be expected that their finer particles would have a lesser traumatizing or blocking effect on the abomasal mucosa or pyloric sphincter, respectively, and thereby cause less damage to the abomasum. However, no difference in abomasal damage was found between straw and straw pellets (Van Putten, 1982), which contradicts this hypothesis. In addition,

pelleted maize silage was observed to cause more lesions than short-chopped or ground maize (cob) silage (Wensing et al., 1986; Breukink et al., 1991). Whether this difference is actually due to the pelleted form or to the different roughage types cannot be determined from these studies. Nevertheless, the lack of difference between straw and pelleted straw implies that roughage type is the main determinant here.

Amount of Solid Feed. In addition to type and physical form of roughage, the amount of SF fed may affect abomasal damage. Larger amounts are expected to increase trauma to the abomasal wall and worsen blocking of the pyloric sphincter, thereby increasing abomasal damage. Indeed, larger, but still restricted, amounts of roughage have been shown to increase the prevalence and size of ulcers (Brscic et al., 2011; Webb et al., 2013). However, increasing amounts of cereal grains and straw were not seen to increase lesion incidence (Morisse et al., 1999), which may be linked to the inclusion of concentrate in the diet, as theorized before. In contrast, inclusion of a high level of concentrate (concentrate:roughage 80:20) does increase abomasal damage when the amount fed increases (Berends et al., 2014). For some roughage sources, an interaction between the amount fed and the type of roughage was found, where the source was only severely damaging when fed in larger amounts. For example, maize (cob) silage caused fewer lesions than straw at small amounts (250 g/d) and more lesions than straw in larger amounts (500 g/d; Webb et al., 2013). A finding that contradicts the hypothesis that larger amounts of SF lead to more abomasal damage is that provision of straw or hay *ad libitum* does not exacerbate lesions caused by MR (Webb et al., 2013, 2015). We may speculate that *ad libitum* provision allows the individual calf to select a diet that is quantitatively optimal for its body, including its abomasum. Furthermore, it minimizes competition between pen mates and allows meals throughout the day, likely reducing feeding rate and meal size. Alternatively, rumen development may play a role (see the Rumen Development section). Finally, Prevedello et al. (2012) proposed that the moment at which the SF is consumed might matter. Consumption of SF immediately after the abomasum has been filled with a large quantity of MR may exacerbate overloading and increase abomasal damage.

Rumen Development

Before entering the abomasum, SF must pass the 3 other stomach compartments. In the first, the rumen, SF will be fermented. It has been hypothesized that feed will be less coarse when it enters the abomasum if this feed is well fermented (Berends et al., 2012). In

addition, because of the smaller particle size resulting from good or better fermentation, SF should also block the pylorus less frequently than when fermented incompletely. Because calves are born with a nonfunctional rumen, its development affects the extent to which SF is fermented. Beef calves and lambs are more susceptible to abomasal perforation caused by ulcers during the development from preruminant to ruminant, which normally occurs at approximately 4 to 8 wk of age (Jelinski et al., 1996a; Dirksen et al., 1997; Vatn and Ulvund, 2000). Stimulating rumen fermentation and development at an early age has been proposed as a way of minimizing abomasal damage (Berends et al., 2012). This can be achieved using a feeding regimen aimed at early rumen development (**ERD**), which includes feeding both roughage and concentrate from an early age onward. An increase in rumen volume and weight is stimulated by the feeding of fibrous feeds, whereas the development of rumen papillae is stimulated by volatile fatty acids and therefore by less fibrous feeds (Berends et al., 2014; Suarez-Mena et al., 2016).

In support of this theory, one study found that calves with better developed rumens had fewer abomasal lesions than calves with less developed rumens (Webb et al., 2013), and others found that stimulating early development with concentrate or hay meant that future feeding of coarse straw did not exacerbate damage (Veissier et al., 1998; Webb et al., 2015). However, when the ERD theory was tested by adjusting calf diet before the age of 12 wk, it was found that ERD only decreases the incidence of scars (Berends et al., 2012). These findings suggest that ERD protects calves from developing abomasal ulcers during the early weeks of life, leading to less scarring later on, but that it has no effect on ulcer or erosion development in later life. Two years later, the same authors confirmed that better rumen development does not protect against abomasal damage at a later age (Berends et al., 2014), based on the finding that both rumen development score and abomasal damage increased for increasing SF amounts (when the proportion of concentrate was high).

Hairballs

Hairballs (or trichobezoars) are round masses composed of ingested hair (Çatik et al., 2015) that develop in the rumen and sometimes in the abomasum (Osborne, 1976). Webb et al. (2013) proposed that ruminal hairballs might prevent proper digestion in the rumen, which would allow large feed particles to pass through the first 3 stomach compartments into the abomasum. These underfermented particles may cause trauma to the abomasal mucosa or block the pyloric exit. Rumen motility, which can be improved by feeding SF

in addition to MR, aids in the removal of hair from the rumen, thereby preventing the development of hairballs (Morisse et al., 1999, 2000; Cozzi et al., 2002a). Alternatively, calves fed SF may ingest less hair than calves fed MR only, as they display fewer abnormal oral behaviors (Veissier et al., 1998; Mattiello et al., 2002), during which hair can be ingested. Calves fed straw or hay have less hair in their rumen than calves fed maize (cob) silage, with those fed maize silage being intermediate, and the amount of hair was further reduced as roughage particle size was increased (Webb et al., 2010, 2013). Nevertheless, Webb et al. (2013) found that calves fed only milk had more ruminal hairballs and fewer abomasal lesions than calves fed additional roughage (with the exception of *ad libitum* hay, for which ulcer incidence was not increased). This implies that ruminal hairballs are at least not a prerequisite for the development of abomasal lesions.

Hairballs can also be found inside the abomasum, although this is only true in veal calves fed only MR. Abomasal hairballs have been hypothesized to cause trauma to the abomasal mucosa or to block the pylorus, both of which may lead to abomasal damage (Jelinski et al., 1996b; Marshall, 2009; Sasaki et al., 2012). Especially during abomasal surgeries performed on suckling calves, large amounts of hair were found in ulcerated or perforated abomasa (Tulleners and Hamilton, 1980; Katchuik, 1992; Çatik et al., 2015). Only one study on veal calves measured the presence of both hairballs and lesions in the abomasum, but a relationship between the two was not evaluated (Osborne, 1976). Studies in beef calves suggest that hairballs do not cause trauma to the abomasal mucosa and do not block the pylorus enough to cause ulceration (Katchuik, 1992; Jelinski et al., 1996b). In lambs, significantly more bezoars were found in lambs with abomasal ulcers than in those without ulcers (Vatn and Ulvund, 2000). Despite these inconclusive results, veterinarians and researchers tend to assume a relationship between hairball presence in the abomasum and abomasal lesions (Stokka and Perino, 2000; Marshall, 2009; Çatik et al., 2015).

Nutrient Deficiencies

Although it is often suggested that nutrient, most often mineral, deficiencies can cause or facilitate the formation of abomasal ulcers (Jelinski et al., 1996b; Stokka and Perino, 2000; Ahmed et al., 2002; Marshall, 2009; Van Immerseel et al., 2010), only one study on veal calves researched part of this relationship. That study found no effect of iron supplementation on abomasal damage, in calves fed MR only (Webb et al., 2013). When beef calves were supplied with a free-choice mineral mix, a nonsignificant trend for a

decreased need for abomasal surgery due to ulceration was observed (Katchuik, 1992). This implies a role for nutrient deficiencies in abomasal ulceration (composition of the mineral mix is unknown but can be assumed to differ between the farms the calves originated from). In another study, deficiencies in copper or selenium occurred more often in beef calves with (perforating) abomasal damage (Mills et al., 1990). Supplementation of copper to both cows and their calves immediately decreased the occurrence of ulceration close to zero (Lilley et al., 1985).

It has been theorized that a low serum copper concentration can lead to a derangement of elastin cross-linkages in the abomasal wall, compromising the abomasal mucosa and microvasculature and leaving the abomasal wall prone to damage (Lilley et al., 1985; Marshall, 2009). In addition, copper deficiency can lead to decreased neutrophil function and subsequently to an increased risk of infection, as occurs when the abomasum is damaged (Lilley et al., 1985; Mills et al., 1990; Marshall, 2009). Because a high concentration of zinc, molybdenum, or sulfur reduces the availability of copper, surplus of these minerals can exacerbate the problem of copper shortage. Thus, in beef calves, nutrient deficiencies, at least for copper and selenium, appear to affect abomasal damage. Whether the same occurs in veal calves has not yet been studied.

Water

Veal calves receive fluids from milk, from other feeds provided, from the drinking of free water, and from the oxidation of food and body tissue. Whereas water originating from feed and free water is deposited in the rumen, water originating from milk generally bypasses the rumen and is deposited in the abomasum (Hepola et al., 2008). Although some studies report that calves fed MR *ad libitum* drink hardly any water (Hepola et al., 2008), others found that calves may ingest large amounts of up to 36 L/d, with an average consumption of 11.3 L/d (Ruis-Heutinck and Van Reenen, 2000; Webb et al., 2014). Water intake increases when calves start consuming SF (Kertz et al., 1984) and is higher in calves fed more SF (Webb et al., 2014). Supplying calves with an increasing amount of free water (from 3 to 8 L/d) did not affect abomasal ulcers, erosions, or inflammations (Gottardo et al., 2002). However, one risk assessment showed that calves receiving water *ad libitum* were at higher risk for lesions than those receiving none at all (Brscic et al., 2011). However, consumption of water was, in that study, strongly linked to the provision of SF and ruminal plaque, indicating that SF may have caused the damage in that case.

THE FACTOR OF STRESS

In many monogastric species, acute stress has been experimentally shown to cause ulceration of the stomach mucosa (rat: Goldman and Rosoff, 1968; Weiner, 1996; guinea pig: Ludwig and Lipkin, 1969; piglet, but not pig: Norton et al., 1972). In ruminants, such as calves, the abomasum acts similarly to the monogastric stomach. In some cases, the stress-induced lesions of monogastrics resemble the ulcers found in the calf fundus (Welchman and Baust, 1987). Therefore, many authors have noted that stress may be a predisposing or even causal factor for ulceration in calves as well (Tuleners and Hamilton, 1980; Lourens et al., 1985; Wiepkema, 1985; Welchman and Baust, 1987; Wiepkema et al., 1987; Breukink et al., 1989; Mills et al., 1990; Lallès and Toullec, 1998; Stokka and Perino, 2000; Ahmed et al., 2002; Constable et al., 2005; Marshall, 2009; Van Immerseel et al., 2010; Sasaki et al., 2012; Valgaeren et al., 2013; Webb et al., 2013; Berends et al., 2014; Çatik et al., 2015). Proposed pathways through which stress could cause abomasal damage have a common starting point, whose involvement has been demonstrated only in rats. In rats, ulcers caused by stress only develop after a drop in body temperature (Weiner, 1996), which initiates 2 main pathways: (1) increased production of gastric acids, whose effects were described previously (see section titled Low Abomasal pH); and (2) a decrease in the rhythm of stomach contractions from 6 to 7 to only 0.5 to 2 times per minute, which leads to decreased mucosal blood flow and subsequent damage through local hypoxia, mechanical damage, and decreased function of the mucosa-protecting mucus and cytoprotective prostaglandins (Weiner, 1996; Marshall, 2009; Kureljušić et al., 2013).

It has been proposed that abomasal ulcers in veal calves are not related to stress, because the location of these ulcers is not similar to that of ulcers caused by stress in adult cattle (Breukink et al., 1991). In veal calves, ulcers are predominantly found in the pyloric region, whereas in adult cattle they are predominantly found in the fundic region. It is unclear what this assumption of stress being a causal factor in adult cattle is based on. Bähler et al. (2010) found that calves in conventional veal systems had more fundic but not pyloric lesions than animals kept in a potentially less stressful system in which they had more square meters per individual, could go outdoors and received water and roughage *ad libitum*. Bähler et al. (2010) proposed, therefore, that stress might be involved in the development of fundic but not pyloric lesions. In that study, however, diet was also an important difference between these 2 rearing systems. Other studies did not

identify links between stress and abomasal damage in veal calves, regardless of the treatments that were used: individual housing versus group housing (Veissier et al., 1997; Bokkers and Koene, 2001), repeated regrouping (Veissier et al., 2001), or environmental enrichment (Veissier et al., 1997). Furthermore, calves used to human–calf interactions (gentled calves), which involved the stockperson talking to and stroking the calves and letting the calf suck the person's fingers for 90 s following feeding, had fewer pyloric lesions at slaughter (Lensink et al., 2000). Because the human–calf interaction allowed calves to suck on the stockperson's fingers, enhanced saliva production might also have decreased abomasal acidity and consequently ulceration. In that case, the difference found would be unrelated to stress, as also suggested by the absence of differences in stress measurements (behavioral observations, response to ACTH challenge) between gentled and control calves.

Calves performing more of the abnormal oral behavior tongue rolling or playing were found to have significantly fewer abomasal ulcers and scars but not fewer erosions (Wiepkema et al., 1987). In addition, in a more recent study, the same relationship was found between abomasal lesions and tongue playing as well as oral manipulation of the environment (Webb, 2014). Stereotypies, such as these abnormal oral behaviors, are defined as repetitive and invariant behavioral patterns that lack an obvious goal or function (Rushen and Mason, 2006), and they may provide captive animals with a way to cope with a suboptimal environment (Würbel et al., 2006). Calves that tongue roll may develop less abomasal damage due to reduced stress through better coping. Similarly, rats that were exposed to acute stress, in the form of electric shocks, developed more gastric ulcers when punished for attempting to escape these shocks, which denies the rats a way to cope with the acute stress (Weiner, 1996). Other mechanisms might be that extra saliva produced when performing abnormal oral behaviors would increase abomasal pH, although it could be argued that saliva produced during object manipulation may not enter the gastrointestinal tract, or that increased satisfaction of oral eating behaviors decreased milk intake and thereby abomasal overloading.

FACTORS ASSOCIATED WITH DISEASE

Microorganisms

Infections caused by fungi, bacteria, parasites, and diseases caused by viruses are hypothesized to lead to the development of abomasal damage in calves (Ross, 1963; Smith, 1966; Stokka and Perino, 2000; Marshall,

2009; Moeller et al., 2013); see Tables 2, 3, 4, and 5 for an overview of these studies. This hypothesis is partly because microorganisms are known to cause peptic ulcers in humans (Overmier and Murison, 2013). In calves, fungi have been isolated from abomasal lesions but evidence for a causal role is currently insufficient (Table 2). Studies investigating bacterial involvement are more abundant; however, bacteria isolated from lesions may originate from postmortem colonization. Additionally, administration of bacteria leads to a different type of damage than is commonly observed in veal calves; namely, numerous small ulcers spread throughout the abomasum (Table 3). Furthermore, although some parasites are capable of causing ulcers in calves (Ross, 1963; Ross and Dow, 1965; Snider et al., 1981, 1985; Taylor et al., 1989; Yang et al., 1993), cattle (Snider et al., 1985), and elk (Woodbury and Parry, 2009), it is unlikely and not reported that indoor-housed calves, fed on concentrate and silages, are exposed to these parasites. Moreover, the type of abomasal damage caused by parasites, referred to as nodules, is quite different from that described most commonly in veal calves (Table 4). Finally, although some viruses can cause lesions in several organs (including the abomasum) in dairy calves (Moeller et al., 2013), beef calves (Bianchi et al., 2017), and adult cattle (Assis et al., 2002), the prevalence of viral diseases is much lower than the prevalence of abomasal damage (2% compared with >70%; Brscic et al., 2011; Bianchi et al., 2017). Viruses, although possibly causing some of the cases of abomasal damage found in veal calves (Table 5), are unlikely to be a main factor in the majority of damage found. However, given the small amount of research in this area, we encourage further focus on this area. Noninfectious diseases, left abomasal displacement, and certain types of tumors might also cause abomasal ulcers in adult cattle and, in very rare cases, calves (Smith et al., 1983; Mueller et al., 1999; Sasaki et al., 2012) but are deemed irrelevant in veal calves.

Medication

Veal calves in Europe (Belgium and the Netherlands) have been reported to be the group of farm animals receiving the most antimicrobial (AM) treatments (Bondt et al., 2012; Pardon et al., 2012b), likely as a direct consequence of the mixing of young, low-immunity calves from many different origins. In Belgium, over 40% of calves were treated with AM every day of the production cycle (Pardon et al., 2012b). Although much less frequently used than AM drugs, (non)steroidal anti-inflammatory drugs [(N)SAID] are also given to veal calves; of all treatments in Belgian veal calves

in 2009, 88% was AM and 12% was NSAID (Pardon et al., 2012b). Compared with the 40% use of AM drugs, NSAID were given to 0.6% of veal calves per day of production in Belgium (Pardon et al., 2012b). Most NSAID are likely given as part of the treatment for respiratory diseases, similar to AM drugs (Pardon et al., 2012b). Ibuprofen (Walsh et al., 2016) and other NSAID (Semrad and Dubielzig, 1994; Sasaki et al., 2012) have been found to cause abomasal lesions in calves. Additionally, NSAID are a known cause of peptic ulcers in humans (Yeomans and Næsdal, 2008). Medication is widely used in veal calves and could be an important factor in the development of abomasal damage; however, the current evidence for this is non-existent and future research is warranted. Nonetheless, the use of NSAID lies far below the prevalence of abomasal damage in veal calves and is unlikely to be one of the main factors.

OTHER FACTORS

Breed

It has been proposed by some authors that the breed of calf may affect abomasal damage. For example, Montbéliarde calves develop more pyloric scars than Holstein Friesian calves when kept in similar systems (Veissier et al., 1997). However, it should be noted that Montbéliarde calves are also capable of growing faster, which is accompanied by a higher MR and SF intake, and thus possibly more severe overloading of the abomasum. This was confirmed by both Bähler et al. (2010) and Brscic et al. (2011), who found no effect of breed on pyloric lesions although, in these studies, breeds were categorized in 3 groups (dairy breeds, cross-breeds, and other breeds) and only those groups were compared, not individual breeds. Breed might have an important impact on abomasal lesions when a particular breed is able to consume more MR and SF more rapidly.

Seasonal Effects

In adult cattle and in beef cattle, occurrence of abomasal damage differs between seasons. In adult dairy cattle, this could be related to the seasonality of milk production, because most ulcers develop around parturition, a period marked by stress and a severe change in diet (Smith et al., 1983; Sanford and Josephson, 1988; Ok et al., 2001; Tharwat and Ahmed, 2012). In beef calves, bad weather has often been proposed as a contributing factor (Jensen et al., 1976; Lilley et al., 1985; Mills et al., 1990; Marshall, 2009). It is theorized that calves do not nurse when the weather is bad, which leads to a drop in abomasal pH, leaving the abomasum vulnerable to ulceration. When the weather is better, calves overconsume milk, which leads to abomasal overloading. In one study, the seasonal effect was fully explained by the use of a seasonal beef production system, indicating that other season-related effects, such as pasture growth, were not causal (Jelinski et al., 1996a).

Because veal calves originate mostly from a nonseasonal dairy system, are kept inside, and do not nurse their dams, bad weather should not have an effect on abomasal damage. Nevertheless, a risk assessment conducted by Brscic et al. (2011) showed that veal calves have a higher risk of developing pyloric lesions when they are raised in the summer or autumn and the lowest risk when reared in spring, both compared with winter. Why this effect exists is unknown. We may speculate that it correlates with other (as yet unidentified) factors that differ seasonally or that differences in living conditions between seasons on the dairy farm of origin have a predisposing effect. Temperature fluctuations in the stable, which can occur if temperature is not regulated year round, might have an effect; for example, through cold or heat stress. As mentioned before, fluctuations in body temperature of rats can lead to the development of stomach ulcers (Weiner, 1996). Alternatively, calves may be fed more or ingest feed faster in certain seasons.

Table 2. Evidence for the role of fungi in the etiology of abomasal damage in calves

Study conclusion	Lesion type	Fungus species	No. of calves	Reference	Study type ¹
Isolation of fungal hyphae from damage	Numerous ulcers, edema	Not identified	3/5	Wray and Thomlinson, 1968	O
Isolation of fungus from damage	Ulcerative abomasitis	<i>Saksenaea erythrospora</i>	1	Lawhon et al., 2012	O
Isolation of fungal hyphae from damage	Ulcers	<i>Absidia remosa</i> <i>Absidia corymbifera</i> <i>Mucor pusillus</i>	7	Gitter and Austwick, 1957	O

¹Observational.

Table 3. Evidence for the role of bacteria in the etiology of abomasal damage in calves

Study conclusion	Lesion type	Bacterium species	No. of calves ¹	Reference	Study type ²
Isolation of bacteria from 6 calves	Many small (1–2 mm) ulcers: ulcerative abomasitis	<i>Clostridium perfringens</i> type D <i>Escherichia coli</i> (likely postmortem infection)	6	Assis et al., 2002	O
No difference in bacteria incidence between damaged and intact abomasa	Type-1 ulcer	All	215 fattening bulls, cows, and calves	Hund et al., 2015	O
No relation between bacteria and damage	Fundic type-1 ulcers	<i>C. perfringens</i> <i>Helicobacter</i> spp.	604	Valgaeren et al., 2013	O
No relation between bacteria and damage	Fundic and pyloric ulcers and erosions	<i>E. coli</i> <i>Streptococcus faecalis</i> <i>Streptococcus bovis</i> <i>Bacillus</i> spp. <i>Corynebacterium</i> spp. <i>Moraxella</i> spp. <i>Acinetobacter</i> spp.	304	Welchman and Baust, 1987	O
Bacteria likely postmortem contaminant	Fatal ulcers	<i>C. perfringens</i> type A	30 beef calves	Jelinski et al., 1995	O
Isolation of bacteria from one calf	Hundreds of small type-1 ulcers	<i>C. perfringens</i>	1	Van Immerseel et al., 2010	O
Isolation of bacteria from one calf	Many small ulcers: ulcerative abomasitis	<i>C. perfringens</i> type A	1 Asian gaur calf	Songer and Miskimins, 2005	O
Administration of bacteria caused damage	Ulcerative abomasitis	<i>C. perfringens</i> type A	10 bull calves	Roeder et al., 1988	E
Administration of bacteria caused damage	Gross lesions, abomasitis and sometimes peritonitis	<i>Salmonella enterica</i>	6	Carlson et al., 2002	E
Administration of bacteria caused damage	Gross lesions throughout the gastrointestinal tract	<i>Chlamydiae</i> strain LW-613	12	Doughri et al., 1974	E

¹Veal calves unless otherwise specified.²O = observational; E = experimental.

Table 4. Evidence for the role of parasites in the etiology of abomasal damage in calves

Study conclusion	Lesion type	Parasite species	No. of calves	Reference	Study type ¹
Administration of parasite caused damage	Abomasitis with ulcers	<i>Ostertagia ostertagi</i>	27	Ross, 1963	E
Administration of parasite caused damage	Larvae-containing nodules, edema	<i>O. ostertagi</i>	10	Ross and Dow, 1965	E
Administration of parasite caused damage	Small nodules	<i>O. ostertagi</i>	10	Snider et al., 1981	E
Administration of parasite caused damage	Small nodules	<i>O. ostertagi</i> , <i>Trichostrongylus axei</i> , or both	20	Snider et al., 1985	E
Administration of parasite caused damage	Fundic and pyloric nodules	<i>O. ostertagi</i>	25	Taylor et al., 1989	E
Administration of parasite caused damage	Nodules	<i>O. ostertagi</i> and <i>Cooperia oncophora</i>	24	Yang et al., 1993	E

¹E = experimental.

Table 5. Evidence for the role of viruses in the etiology of abomasal damage in calves

Study conclusion	Lesion type	Virus species	No. of calves	Reference	Study type ¹
Viral infection caused lesions in various organs, including the abomasum	Ulcers, edema	Bovine herpesvirus 1	2/62 calves showed ulcers in the abomasum	Moeller et al., 2013	O
Viral infection caused lesions in various organs, including the abomasum	Ulcers	Bovine viral diarrhea virus	1/7 calves showed ulcers in the abomasum	Bianchi et al., 2017	O

¹O = observational.

Housing and Management

Certain aspects of housing and management have also been associated with lesion prevalence via surveys. Absence of a heating system and regular visits of a veterinarian appear to be linked to the occurrence of pyloric lesions (Brscic et al., 2011). Calves living in a stable with an open-front building had fewer pyloric lesions than those in stables with manual ventilation (Bähler et al., 2010). It is unlikely that these factors have a direct effect on abomasal lesions; instead, they likely correlate with other factors that do have a direct effect, such as stress or fluctuations in temperature.

Individual Susceptibility

Finally, it has been proposed that calves differ in individual susceptibility to abomasal lesions, because calves kept in similar systems, either on the same or on

another farm, can show very different degrees of abomasal lesion. This is also observed within pens (Wensing et al., 1986; Wiepkema et al., 1987), although contradictory findings exist (Räber et al., 2013b). Where some calves show severe ulceration, other calves kept under the same conditions may have completely undamaged abomasa. Thus, calves are not equally susceptible to abomasal ulceration (Welchman and Baust, 1987), possibly because of the different mechanisms with which calves cope with stress (and stress is likely associated with fundic lesions). In addition, faster-growing calves are more susceptible to damage (Bähler et al., 2010), although this was not seen in fattening bulls, in which carcass weight and fat distribution were not found to affect abomasal ulceration (Hund et al., 2016). This implies that the difference is not due to individual susceptibility but perhaps to more abomasal overloading in faster-growing calves. It has been suggested that only by offering calves free choice of diet can an appropriate

Table 6. Overview of current knowledge on all proposed risk factors of abomasal damage in veal calves, with associated number of studies in support (for) or not in support (against) of these proposed factors¹

Factor	For	Against	Summary of findings	Important
Milk replacer (MR)	4	2	Despite little actual experimental study in this field, MR is likely to play an important role. The exact pathways are unknown.	Yes
Solid feed	9	2	Solid feed provision on top of MR is likely to affect the level of damage, unless roughage is provided ad libitum.	Yes
Rumen development	3	1	The evidence for rumen development protecting against abomasal damage is limited, but studies showing that ad libitum provision of roughage does not exacerbate damage support this hypothesis.	Probably
Hairballs	0	1	Both hairballs in the rumen and abomasum do not show a clear association with abomasal lesions.	No
Nutrients	0	1	Only iron was tested in veal calves.	No
Water	1 ²	1	The experimental study against has stronger findings than the observational study, which involves confounders.	Unlikely
Stress	1 ³	4	Despite studies showing a link between stereotypies and lower damage (not included here), the link with stress is not strong in veal calves, except for fundic lesions, which are not the most common in this group of animals.	Unlikely
Bacteria	3 ⁴	3	Studies that found a link between bacteria and abomasal damage found very different patterns of damage; that is, many small lesions widespread across the abomasum.	Unlikely
Viruses	0	0	No research in veal calves. The prevalence of viruses that cause abomasal damage is much lower than the prevalence of abomasal damage, making a viral factor unlikely to be a main contributor to lesions veal calves.	Unlikely
Fungi	0	0	No research in veal calves.	Unknown
Parasites	0	0	No research in veal calves. In dairy calves, damage caused by parasites are nodules, which are very different from damage commonly observed in veal calves.	No
Breed	1	2	Breed is probably only relevant when it affects growth rate: hence feeding speed and amounts ingested.	Unknown
Medication	0	1	Too little medication specifically tested. Medication is widespread enough to be linked to damage.	Unknown
Season	1	0	Too little work on this. Probably only has an indirect effect.	Unknown
Housing	2	0	Too little work on this. Probably only has an indirect effect.	Unknown
Growth rate	1	0	Although there is little work on this, our own unpublished work suggests that calves that grow faster have more damage.	Yes
Genetics	0	0	No study.	Unknown

¹Only studies specifically studying veal calves are included here.

²A cross-country survey by Brscic et al. (2011), where water provision was correlated with solid feed provision.

³Substantial confounding factor of nutrition (Bähler et al., 2010).

⁴Two of these studies found damage very different from that commonly found in veal calf abomasa.

diet be provided for each individual calf (Webb et al., 2014).

In humans, stomach ulcers are thought to have a heritable component (Holle, 2010). Whether this is the case in calves has, to our knowledge, never been studied. Nevertheless, if abomasal damage in calves indeed has a heritable component, the application of this finding is likely limited, because veal calves originate from the dairy sector, in which other breeding factors are important.

SUMMARY AND CONCLUSIONS

The aim of this review was to integrate the information currently available on the etiology of, and risk factors for, abomasal damage in the form of ulcers, erosions, and scars. Some information may have been excluded because it was in a (for us) foreign language (articles included were in Dutch or English). In addition, extrapolation of results from the older literature may not be fully accurate, because the growing systems in which veal calves are kept have changed substantially over time. Nevertheless, experimental studies from earlier periods can still provide useful information. An overview of all proposed factors and associated litera-

ture support or evidence is presented in Table 6. An overview of the most likely factors and the associated mechanisms is presented in Figure 1. Although a clear effect on the development of abomasal damage was not identified for all proposed factors, it is clear that the etiology is multifactorial, with various dietary factors contributing to pyloric lesion formation extensively and fundic lesions probably being linked to stress. Pyloric lesion incidence can likely be reduced by feeding smaller quantities of milk replacer in more frequent meals that, from an early age, should be combined with SF in the form of both concentrate and roughages. In addition, decreasing the level of stress experienced by veal calves may decrease the occurrence of fundic lesions and improve overall animal welfare by minimizing negative experiences.

In future research, pyloric and fundic lesions, as well as lesion types (i.e., ulcer types 1–4, erosions, or scars), should be scored separately. More research is required to understand the precise pathways by which MR causes such a high prevalence of abomasal lesions in veal calves. Further research is also warranted on the effect of rapid intake of MR and SF caused by restricted amounts, competition, and breed. More research is needed on the impact of medication, the chemical composition of the

Figure 1. Schematic overview of the most likely risk factors for abomasal damage and the pathways through which these operate. Early rumen development can mitigate trauma, at least during early life.

MR, and into deficiencies of other nutrients than iron, especially copper and selenium. Finally, no studies have yet focused on the effects of the abomasal emptying rate on abomasal damage; in adult cattle, delayed abomasal emptying has been proposed as a risk (Constable et al., 2006) and in humans, peptic ulcer disease has been associated with delayed gastric emptying (Minami and McCallum, 1984). Another important path of research would be to develop a method that can assess abomasal damage antemortem. Currently, calves must be killed to assess and measure the extent of abomasal damage, which limits the experimental design options. The finding that calves that grow fastest develop most abomasal lesions (Bähler et al., 2010) could be used for this purpose. Finally, the link between abomasal damage and animal welfare, or more specifically pain, is not well understood and has received no research attention to our knowledge. Whether these lesions are painful, and if so, which types are painful and how severe the pain is, is of crucial importance because this health problem is widespread in the veal industry. One complication here is that commonly used indicators of pain in calves, such as growth rate and feeding rate, are linked to the etiology of the problem at hand. Other indicators of pain (e.g., facial expressions) will have to be investigated.

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