

Metabolic disorders and their relationships to milk production traits in Austrian Fleckvieh

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Abstract

In 2010, a routine genetic evaluation for the direct health traits mastitis, early fertility disorders, ovarian cysts and milk fever was introduced for Fleckvieh as part of the joint Austrian-German genetic evaluation. In order to include direct health traits in the total merit index, a female fertility index and an udder health index were additionally implemented in 2013. For metabolic disorders, no such index has been developed yet. Thus, genetic relationships between metabolic disorders and possible auxiliary traits needed to be investigated. In total, 12,512 diagnoses for metabolic disorders and more than two million test-day records were available for validated farms. After all restrictions, 118,237 lactation records based on electronically transmitted veterinarian diagnoses collected between 2006 and January 2013 could be used. Metabolic disorders for rumen acidosis (ACI), displaced abomasum (ABO), milk fever (FEV), ketosis (KET) and culling due to a metabolic disease (CUL) were defined as binary traits (0/1 or healthy/diseased) in specific time periods. The frequencies of the metabolic disorders were 0.08%, 0.02%, 2.75%, 0.56% and 0.51 % for ACI, ABO, FEV, KET and CUL, respectively. As metabolic disorders mainly occur at the start of lactation, first test-day performance traits were considered as auxiliary traits: fat, protein, lactose and urea content (UREA) of the milk, and the calculated ratios of fat-protein (F:P1), fat-lactose (F:L1) and protein-lactose (P:L1). For the second test-day only ratios were considered. Genetic correlations and heritabilities were estimated with a linear repeatability animal model. For the low frequency traits ACI and ABO, genetic variances were close to zero with high standard errors. Heritabilities for FEV, KET and CUL were 0.034, 0.008 and 0.006, respectively. Heritabilities and genetic correlations indicated that the F:P1 ratio is a possible indicator trait for FEV, KET and CUL. Additionally, F:L1 and UREA could be considered.

Keywords: dairy cattle, metabolic disorders, genetic parameters, test-day records, auxiliary traits

Introduction

Improved animal health is getting increasingly important worldwide. Indirect measures of health or disease have been included into routine performance tests by many countries. However, directly observed measures of health or disease increase the efficiency of genetic improvement of health or health-related traits. In Austria, a health monitoring system for cattle was started in 2006 and has become part of the routine performance recording in the meantime (Egger-Danner *et al.*, 2012b). In 2010 and 2013, a routine genetic evaluation for the

direct health traits mastitis, early fertility disorders, ovarian cysts and milk fever was introduced for Fleckvieh (dual purpose Simmental; Fuerst *et al.*, 2011) and Brown Swiss (Egger-Danner *et al.*, 2012a,b; Fuerst & Egger-Danner, 2014), respectively, as part of the joint Austrian-German genetic evaluation. So far the focus is on veterinarian diagnoses, but diagnostic observations of farmers are also recorded and will be included in the routine evaluation in the near future. For both breeds, two new indices were introduced to include direct health traits in the total merit indices (TMI). These indices are a female fertility index consisting of non-return-rate, time from first to last insemination, early fertility disorders and ovarian cysts and an udder health index calculated from somatic cell score and mastitis (Fuerst & Egger-Danner, 2014).

For metabolic disorders, no such index has been developed yet. Apart from milk fever, for which a genetic evaluation already exists, the most important metabolic disorders (e.g. Koeck *et al.*, 2013; Krause & Oetzel, 2006) which are routinely recorded are ketosis, rumen acidosis, and displaced abomasum.

In order to evaluate the possibility of a development of a metabolic index in Fleckvieh cattle, the aims of this study were (1) to define trait specific observation periods, (2) to analyze frequencies of diagnoses, (3) to estimate variance components, and (4) to investigate potential indicator traits from routine milk performance testing.

Data and analyses

In Austria, diagnosis data are either transmitted electronically by the veterinarians or are recorded by the performance recording organizations during routine milk recording. In total, 12,512 diagnoses for metabolic disorders and more than two million test-day records were available for validated farms. Data validation is described by Egger-Danner *et al.* (2012b) and Egger-Danner *et al.* (2013). However, to ensure best possible data quality, only herds with electronically transmitted veterinarian diagnoses collected between 2006 and January 2013 were used for this analysis. After all further restrictions (usual plausibility checks, minimum number of cows per herd-year = 10, minimum number of cows per sire = 20, first and second test-day available), 118,237 lactation records were available for 44,184 animals.

Definition of traits

Results from diagnoses for rumen acidosis (ACI), displaced abomasum (ABO), milk fever (FEV), ketosis (KET) were defined as binary traits (0/1 or healthy/diseased) in specific time periods within lactation. According to the routine genetic evaluation for health traits (Fuerst *et al.*, 2011), only first diagnoses were taken into account. Following descriptive data analyses, the following observation periods were defined.

Rumen acidosis: the observation period for rumen acidosis was defined as the full standard lactation (305 days).

Displaced abomasum: left- and right-sided displaced abomasum occurring until 42 days in milk were subsumed as one trait.

Milk fever: FEV diagnoses were considered for the period of 10 d before and 10 d after calving.

Ketosis: the observation period for ketosis was defined from calving to 70 days in milk.

As culling reasons are also routinely recorded, culling due to a metabolic disorder (CUL) within standard lactation (305 days in milk), was also defined as a binary trait (0/1 or not culled/culled due to metabolic disorder). If culled for any other reason, cows were only included as healthy, if they had the chance to be under recording until day 200, 30, 10 and 50

for ACI, ABO, FEV and KET, respectively. Otherwise they were considered as missing for the respective trait.

From the traits routinely recorded in milk performance testing, fat, protein and lactose content (in %), and urea (mg/100ml milk) of the first test-day (8-49 days in milk) were used. Additionally, the fat-protein-ratio (F:P1; F:P2), the fat-lactose-ratio (F:L1, F:L2) and the protein-lactose-ratio (P:L1, P:L2) were calculated for the first and second (40-90 days in milk) test-day.

Model

Heritabilities were calculated by means of the software package VCE6 (Groeneveld *et al.*, 2008), based on univariate linear animal models. Genetic correlations were estimated by bivariate models. The following model was used for health traits:

$$y_{ijklmnop} = \text{lact}_i * \text{age}_j + y_k * m_l + h_m * y_k + pe_n + a_o + e_{ijklmnop}$$

where $y_{ijklmnop}$ is the observation for FEV, ABO, ACI, KET and CUL (0 = healthy, 1 = diseased and 0 = not culled, 1 = culled for metabolic disorder); $\text{lact}_i * \text{age}_j$ is the fixed effect of parity (1, 2, ..., 5+) by calving age (6 classes for 1st and 2nd parity); $y_k * m_l$ is the fixed effect of calving year and month; $h_m * y_k$ is the random herd-year effect; pe_n is the random permanent environmental effect; a_o is the random genetic effect of the animal and $e_{ijklmnop}$ is the random residual effect.

In contrast to the routine genetic evaluation of health traits, the fixed effect of type of recording (electronic by veterinarian/milk recording) by year was not taken into account as only electronically transmitted data were used in this analysis. For production traits, the same model additionally including the covariate days in milk (linear, quadratic) was applied.

Results and Discussion

Table 1: Data characteristics.

Health traits	Trait	N	Frequency (%)
	Milk fever	117,757	2.8
	Ketosis	112,874	0.6
	Acidosis	96,843	0.1
	Displaced abomasum	115,435	0.02
	Culled for metabolic disorders	105,344	0.6
Production traits	Trait	N	Mean
	Fat content (%)	97,981	4.15
	Protein content (%)	97,981	3.22
	Lactose content (%)	97,146	4.84
	Urea content (mg/100ml)	97,075	18.1
	Fat:protein ratio (1 st test-day)	97,981	1.30
	Fat:protein ratio (2 nd test-day)	93,316	1.25
	Fat:lactose ratio (1 st test-day)	97,146	0.86
	Fat:lactose ratio (2 nd test-day)	92,567	0.82
	Protein:lactose ratio (1 st test-day)	97,146	0.67
	Protein:lactose ratio (2 nd test-day)	92,567	0.67

In Table 1, frequencies of disorders and arithmetic means of production traits are provided. For FEV (2.8%), the frequency calculated in an earlier data set for the same breed but without restriction on electronically transmitted data, was 2.3% and thus rather similar (Fuerst *et al.*, 2011). This trait, for which a routine genetic evaluation already exists, has the highest frequency of all metabolic disorders followed by KET (0.6%). The frequencies of both traits, ACI and ABO (0.1% and 0.02%) were very low. Thus, these disorders seem to be of negligible importance in Fleckvieh cattle. Especially for Holstein, markedly higher frequencies were reported for KET and ABO, e.g. in Canadian Holstein with 4.1% and 2.7%, respectively (Koeck *et al.*, 2013). Average milk contents and calculated ratios were within the expected range.

Heritabilities and genetic correlations for metabolic disorders and culling due to metabolic disorders are provided in Table 2. Genetic variances for ACI und ABO were close to zero, and as a result heritabilities were markedly below 0.01. Higher heritabilities for ABO were estimated in Holstein populations (e.g. $h^2 = 0.04$ in Canadian Holstein; Koeck *et al.*, 2013). Due to the genetic variances close to zero, genetic correlations to other traits should also be treated with caution. Partly we found genetic correlations close to unity, partly high or even implausible standard errors (between ACI and FEV, Table 2). Given that the frequencies of those two disorders are very low in Fleckvieh as well, both traits will most likely not be considered for a routine genetic evaluation. In the following, genetic correlations to milk production traits will thus not be presented for ACI and ABO. The heritability for FEV is more or less the same as presented by Fuerst *et al.* (2011), genetic correlations to KET and CUL are rather high with 0.45 and 0.67, respectively. For KET and CUL, heritabilities were lower than for FEV, but significantly different from zero for both traits. In Canadian Holstein (Koeck *et al.*, 2013), a higher heritability of 0.02 was reported for KET.

Table 2: Heritabilities (on the diagonal), genetic correlations (above diagonal) and their standard errors (in brackets) for acidosis (ACI), displaced abomasum (ABO), milk fever (FEV), ketosis (KET) and culled due to metabolic disorder (CUL).

	ACI	ABO	FEV	KET	CUL
ACI	0.001 _(0.001)	0.999 _(0.109)	-0.805 _(53.006)	-0.999 _(0.004)	0.999 _(0.002)
ABO		0.001 _(0.001)	0.567 _(0.220)	-0.038 _(0.366)	0.429 _(0.218)
FEV			0.034 _(0.004)	0.450 _(0.092)	0.667 _(0.075)
KET				0.008 _(0.002)	0.617 _(0.137)
CUL					0.006 _(0.001)

Especially in high-yielding dairy cows, a pronounced negative energy balance at the beginning of the lactation may lead to health problems including metabolic disorders (Bertoni *et al.*, 2009). Traits from routine performance testing may serve as indicators for energy balance, with fat:protein ratio being among the most appropriate ones (Buttchereit *et al.*, 2011). As a consequence, such traits may also be eligible indicator traits for metabolic disorders, if they are reasonably heritable and have rather high genetic correlations. Heritabilities for milk contents range from 0.095 (urea content) to 0.337 (protein-%), those for the ratios range from 0.155 (F:P1) to 0.384 (P:L2; Table 3). For fat- and protein-% and F:P1 ratio, similar heritabilities (0.19, 0.27, and 0.15, respectively) were estimated in Canadian Holstein

Table 3: Heritabilities (h^2) and standard errors (SE) for milk production traits from the first test day (fat content (Fat-%), protein content (Prot-%), lactose content (LACT-%) and urea content (UREA)) and ratios calculated for the first and second test-day (fat:protein ratio (F:P1, F:P2), fat:lactose ratio (F:L1, F:L2), protein:lactose ratio (P:L1, P:L2)).

	h^2	SE
Fat-%	0.203	0.009
Prot-%	0.337	0.011
Lact%	0.322	0.010
UREA	0.095	0.006
F:P1	0.155	0.008
F:L1	0.193	0.009
E:L1	0.287	0.010
F:P2	0.139	0.007
F:L2	0.225	0.009
P:L2	0.384	0.010

Genetic correlations between content traits and the metabolic disorders FEV, KET and CUL are shown in Table 4. The highest genetic correlations were found between Protein-% and Fat-% and KET (-0.28 and -0.21, respectively) and UREA and FEV (-0.21). Calculated ratios for the first test-day however revealed higher genetic correlations between F:P1 and KET and CUL, respectively (0.38, Table 5). In general, the genetic correlations between FEV, KET and CUL and second test-day ratios were lower than those of the first test-day. An explanation could be that most of the diagnoses of these traits occur right at the start of the lactation. Based on phenotypic data only, Manzenreiter *et al.* (2013) questioned the sole use of the fat:protein ratio with a threshold of 1.5 in Fleckvieh as an indicator for ketosis. Only 58% of the cows with a ketosis diagnosis had a fat-protein-ratio smaller than or equal to 1.5. For management purposes using phenotypic data, the authors suggested to adapt the thresholds depending on breed and also to further evaluate the fat:lactose ratio as an additional information.

Table 4: Genetic correlations and their standard errors (in brackets) between metabolic disorders (milk fever (FEV), ketosis (KET) and culled for metabolic disorder (CUL)) and milk contents at the first test-day (fat content (Fat-%), protein content (Prot-%), lactose content (LACT-%) and urea content (UREA)).

	Fat-%	Prot-%	Lact-%	UREA
FEV	0.08 (0.05)	-0.12 (0.05)	0.05 (0.05)	-0.21 (0.06)
KET	0.21 (0.09)	-0.28 (0.08)	-0.15 (0.09)	-0.11 (0.11)
CUL	0.26 (0.10)	-0.16 (0.07)	0.01 (0.07)	-0.13 (0.09)

Table 5: Genetic correlations and their standard errors (in brackets) between metabolic disorders (milk fever (FEV), ketosis (KET) and culled for metabolic disorder (CUL)) and ratios calculated for the first and second test-day (fat:protein ratio (F:P1, F:P2), fat:lactose ratio (F:L1, F:L2), protein:lactose ratio (P:L1, P:L2)).

	F:P1	F:L1	E:L1	F:P2	F:L2	E:L2
FEV	0.18 (0.05)	-0.07 (0.05)	-0.13 (0.05)	0.01 (0.06)	-0.11 (0.05)	-0.12 (0.05)
KET	0.38 (0.10)	-0.25 (0.10)	-0.21 (0.09)	0.25 (0.09)	0.15 (0.10)	-0.12 (0.09)
CUL	0.38 (0.09)	-0.26 (0.09)	-0.18 (0.10)	0.16 (0.08)	0.04 (0.05)	-0.09 (0.08)

Results suggest that in case of an introduction of a metabolic index in Fleckvieh, milk fever, ketosis and culling due to a metabolic disorder should be considered. For both acidosis and displaced abomasum, very low frequencies and heritabilities were found, thus those traits should not be included in a future index. Possible indicator traits are the fat:protein ratio and fat:lactose ratio of the first, possibly also of the second test-day. As the feet and legs complex may also be affected in course of negative energy balance, ratios might also serve as indicators for leg and claw traits. Another possibility would thus be the combination of both, metabolic and feet and legs disorders into one index.

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