Selection for bull fertility: a review

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ABSTRACT: Fertility is a critically important factor in cattle production because it directly relates to the ability to produce the offspring necessary to offset costs in production systems. Female fertility has received much attention and has been enhanced through assisted reproductive technologies, as well as genetic selection; however, improving bull fertility has been largely ignored. Improvements in bull reproductive performance are necessary to optimize the efficiency of cattle production. Selection and management to improve bull fertility not only have the potential to increase conception rates but also have the capacity to improve other economically relevant production traits. Bull fertility has reportedly been genetically correlated with traits such as average daily gain, heifer pregnancy, and calving interval. Published studies show that bull fertility traits are low to moderately heritable, indicating that improvements in bull fertility can be realized through selection. Although female fertility has continued to progress according to increasing conception rates, the reported correlation between male and female fertility is low, indicating that male fertility cannot be improved by selection for female fertility. Correlations between several bull fertility traits, such as concentration, number of spermatozoa, motility, and number of spermatozoa abnormalities, vary among studies. Using male fertility traits in selection indices would provide producers with more advanced selection tools. The objective of this review was to discuss current beef bull fertility measurements and to discuss the future of genetic evaluation of beef bull fertility and potential genetic improvement strategies.

Key words: Bull, cattle, conception rate, male fertility, semen

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INTRODUCTION

Fertility is generally defined as the ability to conceive offspring (Utt, 2016). Fertility traits have a large impact on production and, therefore, are of great economic value in the livestock industry (Abdollahi-Arpanahi et al., 2017). Reproductive efficiency impacts beef producers' profitability and often determines whether beef producers reach their production goals, regardless of whether it is explicitly included in the breeding objective (Harris, 1970). Reproduction is a very complex trait that involves events including gametogenesis, fertilization, uterine attachment, embryogenesis, and fetal development (Abdollahi-Arpanahi et al., 2017). Reproduction, and more definitively, conception, is influenced by several environmental factors including nutrition, temperature, and overall animal health. The female's conception rate can be impacted by these common factors, as well as age and season of the year (Senger, 2012). Optimizing environmental conditions of

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the female and using assisted reproductive technologies such as estrous synchronization, artificial insemination (AI), and embryo transfer can improve conception rate, but beef producers still only experience an average single service AI conception probability of 60% (USDA, 2010). Conception rate is also contingent on the bull and the quality of the semen; therefore, improvements to the conception rate of the U.S. beef cattle herd can be further enhanced by obtaining accurate measures of bull fertility (Berry et al., 2014).

A significant percentage of reproductive failure is attributable to bull subfertility due to low semen quantity, poor semen quality, and/or health status (DeJarnette et al., 2004); therefore, only evaluating and selecting for female fertility traits have tremendous consequences. Bull fertility has significant production impacts (Braundmeier and Miller, 2001). Bull fertility is currently phenotypically evaluated with breeding soundness examinations (BSEs) and at AI centers, but measures of sire fertility are not currently included in genetic evaluation in the beef industry. Incorporation of phenotypes from BSEs or AI centers would allow national genetic evaluation of these traits, but no system currently exists to transfer these phenotypes to breed associations. Bull fertility traits are generally low to moderately heritable, so genetic selection could be utilized to improve bull fertility. In addition, genomic technologies should enhance genetic prediction, as evidenced by genomic studies utilizing dairy bulls. Incorporating both traditional genetic evaluation approaches combined with genomic technologies has the opportunity to provide beef producers with genetic selection tools for male fertility.

IMPORTANCE OF MALE FERTILITY

Only focusing on female reproductive capabilities limits the potential for increased phenotypic performance. Braundmeier and Miller (2001) recognized using subfertile or infertile semen has consequences, not only to the AI companies distributing it, but also to the producers using it. Discovering how to improve male fertility traits not only has the potential to increase the conception rate but also has the capacity to improve other traits. For example, research shows that selection for increased scrotal circumference (SC) size decreased the calving interval (Meyer et al., 1991), improved daughter pregnancy rates (Toelle and Robison, 1985; Van Melis et al., 2010), and increased average daily gain (Raidan et al., 2016). Although it is arguable whether or not SC is an indication of female fertility due to some conflicting results in the literature (Evans et al., 1999; Martínez-Velázquez et al., 2003), it is currently the only male fertility-related phenotype collected and utilized for genetic evaluation. There is a need for additional male fertility traits to be collected and evaluate the relationship with economically relevant traits.

The economic impacts of bull fertility have tremendous influence in the AI industry. The National Association of Animal Breeders (NAAB, 2018) reported that nearly 3.7 million units of beef bull semen were custom collected and 4 million units were distributed throughout the United States in 2018. Furthermore, the United States exported 3.9 million units of beef bull semen in 2018 totaling over $13 million. In the last study performed by the National Animal Health Monitoring System, it was reported that only 7.62% of beef cattle producers had adopted AI technology. Along with time and labor, the cost was cited as one of the main reasons for not utilizing the reproductive technology (USDA, 2009). Utilizing selection to improve semen quality has the potential ability to increase the quality and, therefore, quantity of semen produced by genetically superior sires. Not only will an increased amount of semen from genetically superior sires be more widely available but could also be available at a more reasonable price which may entice more producers to implement AI and cause a more rapid rate of genetic change in the beef industry. More importantly, improved semen quality would cause an increased conception rate, which would result in a decreased cost per pregnancy for the producer.

GENETIC EVALUATION OF BULL FERTILITY

Phenotypic semen quality traits are often the starting point for evaluating fertility and should be considered when purchasing a bull to ensure he is capable of producing. However, it has been documented that bull fertility is influenced by genetic factors (Huang et al., 2011; Corbet et al., 2013), so bull fertility could be improved through selection within the context of breeding programs. Semen quality is recognized as a major determinant of bull fertility and estimates of heritability have been published. Measurements obtained from BSEs have been used to determine genetic parameters and heritability of SC, spermatozoa motility (MOT), semen abnormalities (ABNO), and common defects of the penis (Smith, 1989; Christmas et al., 2001).

Semen production traits are relatively easy to measure, and there are often multiple records available per bull if they have been collected at an AI center. Selection for semen production and quality traits is an attractive prospect because heritability estimates for semen production and quality traits are relatively similar across studies, and there are favorable genetic relationships between semen quality traits.

Many different terminologies are used across the industry to describe semen production and quality traits. Table 1 summarizes the different measurements utilized to quantify bull fertility. Due to the nature of collecting bull fertility phenotypes, the measures are generally subjective and recorded by different laboratory technicians. This introduces the possibility of bias or difficulty in combining records across multiple studs and technicians. This is potentially problematic for the purposes of genetic evaluation, though fitting appropriate contemporary groups or fixed effects should alleviate some of these issues. The biological process of the development of spermatozoa is affected by many environmental factors. Factors such as age (Taylor et al., 1985; Karoui et al., 2011), nutrition (Coulter et al., 1997; Brito et al., 2006), temperature (Hall, 1989; Barth and Waldner, 2002), semen collector (Amann and Almquist, 1976; Chenoweth, 1983), and collection interval (Fuerst-Waltl et al., 2006) can all impact spermatozoa production and should be accounted for when evaluating these traits. Furthermore, recent advances in technology promise to provide more objective semen quantity and quality measures. For example, computer-assisted semen analysis provides objective measures of semen production and quality traits (Druet et al., 2009; Corbet et al.,

Table 1. Description of various bull traits used to define bull fertility

Trait	Abbreviation	Description	Units
Production traits			
Concentration	CONC	Relative amount of spermatozoa per ejaculate, measured by a colorimeter	millions/mL
Number of spermatozoa	NSP	Calculated by multiplying spermatozoa concentration and semen volume; expressed in millions	millions
Volume	VOL	Total amount of the ejaculate, measured by weight	mL
Quality measures			
Mass activity	MASS	Score from 0–5 with 0 being no activity and 5 being rapid, distinct swirls/rapid swirling, slower swirling, generalized oscillation, and sporadic oscillation, similar measurement to motility score	score
Motility	MOT	Movement and swimming of sperm	%
Motility score	MOTSC	Score which scale varies depending on study.	score
Progressive motility	PROG	Swimming in a mostly straight line or large circles, measured by computer-assisted semen analysis	%
Morphology			
Percent normal spermatozoa	%NORM	Percentage of morphologically normal spermatozoa	%
Percentage of living spermatozoa	%LIV	Percent of living spermatozoa with an intact cytoplasmic membrane after thawing	%
Abnormalities			
Percentage of spermatozoa with an abnormal cytoplasmic droplet	DROP	Percentage of spermatozoa with a cytoplasmic droplet defect	%
Percentage of spermatozoa with an abnormal head	HEAD	Percentage of spermatozoa with a defect to the head of the spermatozoa	%
Percentage of spermatozoa with an abnormal tail	TAIL	Percentage of spermatozoa with a defect to the tail of the spermatozoa	%
Primary abnormalities	PRIM	Generally, defects of the head of the spermatozoa	%
Secondary abnormalities	SEC	Slight defects of the tails	%
Total abnormalities	ABNO	Percentage of abnormalities in the total ejaculate sample	%
Scrotal circumference			
Scrotal circumference	SC	Circumference measured at the widest point of the scrotum with both testes fully distended	cm

2013). The dairy industry has realized benefits from some of these technology advancements utilized in the evaluation of fertility traits. Measurements of mitochondrial activity and acrosome reaction have been utilized to evaluate phenotypic differences in semen quality (Graham et al., 1990; Bucher et al., 2019). These more objective evaluations of semen quality provide an opportunity to eliminate some of the issues surrounding use of more subjective data, provided that the industry is willing to share the data with breed associations for inclusion into these evaluations. Though some benefits have been realized from these technologies in the dairy industry, there are few published studies that utilize these technologies for evaluation of beef bulls.

Genetic Parameters

Tables 2 and 3 outline the reported heritabilities published within the scientific literature for semen production and quality traits, respectively. All reported studies used a best linear unbiased prediction (BLUP) univariate or multivariate animal model, with the exception of Smith et al. (1989) which used a least squares procedure to analyze the data with a multivariate sire model. Each reported study had varying fixed effects incorporated in the model. Many of the studies included the fixed effects of bull age, season, and year. Additional fixed effects include AI center, ejaculate number, day of the week, and semen collector. Relevant discussion pertaining to the differing fixed effects will be further discussed with the pertinent studies.

Scrotal Circumference

SC is often used to predict the number of sperm produced and is measured using circular tape around the widest part of the testicles (Whittier and Bailey, 2009). Several different studies with various ages and breeds of bulls have shown SC to have a moderate-to-high heritability(Table 2). Interestingly, in a study of tropical composite bulls, SC heritability was estimated at 6, 12, 18, and 24 months of age and reported estimates were nearly the same (Corbet et al., 2013). Thus, age does not seem to result in changes in the heritability estimate of SC.

Semen Production Traits

Volume. Semen volume (VOL) is the total amount of the ejaculate, which is expressed in milliliters. A majority of the reported studies estimated a moderate heritability for semen volume as summarized in Table 2. In a study of AI ram fertility, David et al. (2006) estimated the heritability of VOL to be close to moderately heritable, which mirrored that of the heritabilities found in the bull fertility studies. Kaps et al. (2000) and Kealey et al. (2006) reported low heritability estimates for VOL in yearling beef bulls. Kaps et al. (2000) and Kealey et al. (2006) obtained VOL measurements from BSEs taken on Simmental and Hereford bulls, respectively. Breed and age could have caused the difference from the higher reported heritabilities. All other reported estimates were obtained from beef and dairy bulls at an AI center, which would have a large number of more mature bulls.

Concentration. Concentration (CONC) is the amount of spermatozoa in the ejaculate, expressed as millions per milliliter and is measured with a colorimeter (Suchocki and Syzda, 2015; Table 2). Concentration has been reported as a moderately heritable trait, with one outlier estimate. Mathevon et al. (1998) estimated CONC to have a high heritability, which could be attributable to the small population size (n = 198).

Number of spermatozoa. Number of spermatozoa (NSP) is a function of both CONC and VOL, and reported estimates are shown in Table 2. It is expressed in millions of spermatozoa per ejaculate and is calculated by multiplying CONC and VOL. The number of spermatozoa is generally moderately heritable, which is logical because VOL was estimated to be moderately heritable and CONC was generally low to moderately heritable. Variability in the estimates could also be attributed to the varying fixed effects utilized in the different studies.

Semen Quality Measures

Motility. Motility is a trait which refers to the ability of the spermatozoa to move progressively forward (Senger, 2012). Motility, also called progressive motility (PROG) by studies that utilized a computer-assisted semen analyzer, is a term often used to characterize whether spermatozoa are swimming in mostly straight lines or large circles. Motility and progressive motility are scored as a percentage of the ejaculate. To obtain the percentage, a sample of the ejaculate is inspected under a microscope. Spermatozoa are counted and classified as motile or not motile until 100 spermatozoa are counted. Due to the subjectivity of obtaining the measurement, variation can occur in the measurements between different observers. Heritability estimates for motility and progressive motility

Table 2. Heritabilities for commonly measured semen production traits

Traits	*n*	Estimate	Standard error	Breed	Source
Scrotal circumference, 6 months	2,424	0.41	0.08	Tropical Composite	Corbet et al. (2013)
Scrotal circumference, 12 months	717	0.36	0.06	Angus	Knights et al. (1984)
	4,233	0.53	0.06	Hereford	Bourdon and Brinks (1986)
	549	0.40	0.09	Hereford, Angus, & Red Angus	Smith et al. (1989)
	1,282	0.56	*	Angus	Christmas et al. (2001)
	1,281	0.46	0.08	Angus	Garmyn et al. (2011)
	2,424	0.46	0.09	Tropical Composite	Corbet et al. (2013)
Scrotal circumference, 18 months	—	0.40	0.02	Nellore	Silva et al. (2011)
	2,424	0.43	0.09	Tropical Composite	Corbet et al. (2013)
Scrotal circumference, 24 months	2,424	0.44	0.09	Tropical Composite	Corbet et al. (2013)
Volume	2,351	0.18	*	Holstein	Taylor et al. (1985)
	2,387	0.65	0.09	Normande	Ducrocq and Humblot (1995)
	198	0.24	*	Holstein	Mathevon et al. (1998)
	955	0.04	0.54	Simmental	Kaps et al. (2000)
	841	0.09	0.08	Line 1 Hereford	Kealey et al. (2006)
	974	0.22	0.03	Lacaune Rams	David et al. (2006)
	301	0.18	0.02	Beef/ Dairy Cross	Gredler et al. (2007)
	515	0.22	0.05	Holstein	Druet et al. (2009)
	1,212	0.26	0.06	Holstein	Suchocki and Syzda (2015
	787	0.20	0.04	Beef & Dairy	Berry et al. (2019)
Sperm concentration	717	0.13	0.06	Angus	Knights et al. (1984)
	2,387	0.39	0.10	Normande	Ducrocq and Humblot (1995)
	198	0.52	*	Holstein	Mathevon et al. (1998)
	955	0.26	0.01	Simmental	Kaps et al. (2000)
	841	0.16	0.08	Line 1 Hereford	Kealey et al. (2006)
	974	0.24	0.04	Lacaune Rams	David et al. (2006)
	301	0.14	0.04	Beef/Dairy Cross	Gredler et al. (2007)
	1,212	0.34	0.07	Holstein	Suchocki and Syzda, 2015
	787	0.20	0.07	Beef & Dairy	Berry et al. (2019)
Number of spermatozoa	717	0.24	0.05	Angus	Knights et al. (1984)
	2,351	0.03	*	Holstein	Taylor et al. (1985)
	198	0.38	*	Holstein	Mathevon et al. (1998)
	974	0.18	0.03	Lacaune Rams	David et al. (2006)
	301	0.22	0.02	Beef/Dairy Cross	Gredler et al. (2007)
	1,212	0.27	0.06	Holstein	Suchocki and Syzda (2015)
	787	0.38	0.05	Beef & Dairy	Berry et al. (2019)

*No standard error reported; *n* = number of bulls.

differed greatly between different studies and are summarized in Table 3. Some studies reported very low heritability estimates while others reported very high-to-moderate heritability estimates. In addition to the subjectivity of the measurement, the studies utilized different breeds, different data collection, and varying fixed effects.

Motility score. Motility score (MOTSC) or mass activity (MASS) is a subjective score based on a scale which varies depending on the individual standards of the collection protocol. A higher MOTSC is more desirable. Ducrocq and Humblot (1995) based MOTSC from 0 to 4; Gredler et al. (2007) did not provide the scoring system used; and Suchocki and Syzda (2015) based MOTSC from 1 to 3. Table 3 summarizes motility score heritability estimates. Gredler et al. (2007) reported a much lower heritability than Ducrocq and Humblot (1995) and Suchocki and Syzda (2015).

Percentage of living spermatozoa after freezing. Few studies have estimated the heritability of percentage of living spermatozoa after freezing (%LIV; Table 3). Data collected from an AI center would allow for the estimation of %LIV. Percentage of living spermatozoa after freezing has been estimated to have a low-to-moderate heritability, with relatively small standard deviations (Kealey et al., 2006; Druet et al., 2009; Berry et al., 2019). The low-to-moderate correlation estimate is surprising considering the potential impact of the freezing

process on spermatozoa and the small population size (n = 841, 515, and 787, respectively).

Percent normal spermatozoa. Gross percentages of normal spermatozoa (%NORM) measures the percentage of spermatozoa with an acceptable morphology and is recorded as a part of a BSE. The heritability estimates for this trait are extremely variable as reported in Table 3. Yilmaz et al. (2004), Kealey et al. (2006), and Corbet et al. (2013) reported moderate-to-high heritability estimates,

Table 3. Heritabilities for commonly measured semen quality traits

Traits	n	Estimate	Standard Error	Breed	Source
Spermatozoa motility	717	0.13	0.06	Angus	Knights et al. (1984)
	549	0.08	0.07	Hereford, Angus, & Red Angus	Smith et al. (1989)
	2,387	0.30	0.09	Normande	Ducrocq and Humblot (1995)
	198	0.31	*	Holstein	Mathevon et al. (1998)
	1,282	0.07	*	Angus	Christmas et al. (2001)
	974	0.18	0.03	Lacaune Rams	David et al. (2006)
	841	0.22	0.09	Line 1 Hereford	Kealey et al. (2006)
	515	0.43	0.08	Holstein	Druet et al. (2009)
	1,281	0.05	0.03	Angus	Garmyn et al. (2011)
	2,424	0.15	0.05	Tropical Composite	Corbet et al. (2013)
	1,212	0.31	0.06	Holstein	Suchocki and Syzda, 2015
	787	0.37	0.03	Beef & Dairy	Berry et al. (2019)
Progressive motility	515	0.13	0.13	Holstein	Druet et al. (2009)
	2,424	0.15	0.05	Tropical Composite	Corbet et al. (2013)
Motility score	2,387	0.23	0.08	Normande	Ducrocq and Humblot (1995)
	301	0.04	0.01	Beef/ Dairy Cross	Gredler et al. (2007)
	1,212	0.26	0.05	Holstein	Suchocki and Syzda, 2015
Mass activity	2,424	0.13	0.05	Tropical Composite	Corbet et al. (2013)
Percentage of living sperm-atozoa after thawing	841	0.22	0.09	Line 1 Hereford	Kealey et al. (2006)
	515	0.21	0.08	Holstein	Druet et al. (2009)
	787	0.25	0.08	Beef & Dairy	Berry et al. (2019)
Percent normal spermatozoa	549	0.07	0.06	Hereford, Angus & Red Angus	Smith et al. (1989)
	765	0.47	0.07	Angus	Yilmaz et al. (2004)
	841	0.35	0.10	Line 1 Hereford	Kealey et al. (2006)
	301	0.10	0.03	Beef/Dairy Cross	Gredler et al. (2007)
	2,424	0.41	0.10	Tropical Composite	Corbet et al. (2013)
Abnormal spermatozoa per-centage/ Total abnormalities	2,387	0.19	0.07	Normande	Ducrocq and Humblot (1995)
	1,282	0.29	*	Angus	Christmas et al. (2001)
	515	0.25	0.10	Holstein	Druet et al. (2009)
	—	0.15	0.01	Nellore	Silva et al. (2011)
	1,281	0.25	0.07	Angus	Garmyn et al. (2011)
Primary abnormalities	549	0.31	0.09	Hereford, Angus, & Red Angus	Smith et al. (1989)
	1,282	0.35	*	Angus	Christmas et al. (2001)
	841	0.30	0.10	Line 1 Hereford	Kealey et al. (2006)
	1,281	0.27	0.07	Angus	Garmyn et al. (2011)
Secondary abnormalities	549	0.02	0.05	Hereford, Angus, & Red Angus	Smith et al. (1989)
	1,282	0.26	*	Angus	Christmas et al. (2001)
	841	0.33	0.09	Line 1 Hereford	Kealey et al. (2006)
	1,281	0.23	0.08	Angus	Garmyn et al. (2011)
Percentage of spermatozoa with abnormal head	515	0.35	0.12	Holstein	Druet et al. (2009)
Percentage of spermatozoa with abnormal tail	515	0.19	0.12	Holstein	Druet et al. (2009)
Percentage of spermatozoa with abnormal cytoplasmic droplet	515	0.19	0.08	Holstein	Druet et al. (2009)

*No standard error reported; n = number of bulls.

whereas Smith et al. (1989) and Gredler et al. (2007) reported low heritability estimates. Variation in results are unlikely due to age or breed composition, as all are yearling beef bulls, with the exception of Corbet et al. (2013) which utilized tropical composite bulls. Differences could be caused by population size; Yilmaz et al. (2004), Corbet et al. (2013), and Kealey et al. (2006) had population sizes ranging from 765 to 2424; and Smith et al. (1989) had a population size of 549.

Abnormalities. Abnormalities or percent abnormalities are a measure of the spermatozoa in an ejaculate that are aberrant or have undesirable characteristics. Abnormalities generally have estimates that were low to moderately heritable and are summarized in Table 3. Abnormalities can be further divided into primary (PRIM) or secondary (SEC), and estimates are additionally low-to-moderate heritability estimates, with the exception of a few outliers with high standard errors (Table 3). Other studies classify abnormalities more descriptively as percentage of spermatozoa with an abnormal head (HEAD), tail (TAIL), and/or cytoplasmic droplet (DROP).

GENETIC AND PHENOTYPIC CORRELATIONS

Scrotal Circumference with Semen Quality Measures

Smith et al. (1989) was one of the first to report a genetic correlation estimate between SC and MOT. The low, unfavorable estimate with a high standard error could be attributed to the low estimates of genetic variation in the population of 549 bulls, of which 450 were Herefords. Contradicting Smith et al. (1989), Christmas (2001), and Corbet et al. (2013) found a moderate, favorable genetic correlation between SC and MOT. Corbet et al. (2013) estimated the genetic correlation between MASS and SC to be moderate and favorable. Corbet et al. (2013) was the only study that reported MASS, which is a motility measure. The estimate reported MASS was highly correlated with SC, indicating a larger SC would result in increased MASS. Table 4 presents the reported correlations.

Contradictory genetic relationships have been reported between SC and %NORM as shown in Table 4. Smith et al. (1989) reported a negative genetic correlation, which is unfavorable; nonetheless, Corbet et al. (2013) reported a positive, favorable estimate. A contributing factor to the different correlations could be the fact that Smith et al. (1989) utilized yearling beef bulls, and Corbet et al. (2013) provided estimates on 18-month-old *Bos indicus* cattle.

Smith et al. (1989) reported genetic correlations between SC and PRIM and SEC. The genetic correlation between SC and SEC is outside of the parameter space, which could have been caused by the genetic evaluation model utilized (least squares). The least squares procedure only uses the relationships among siblings to calculate heritability estimates. In contrast, BLUP utilizes more pedigree information to make predictions. Considering the Smith et al. (1989) heritability estimate for SEC was not statistically different than zero, the lack of genetic diversity in the population could also explain the reason for the anomalous genetic correlation between SC and SEC. Christmas et al. (2001) reported negative, favorable genetic correlations between SC and abnormal spermatozoa traits, which translates to higher SC corresponding to fewer abnormalities. Garmyn et al. (2011) also reported negative, favorable genetic correlations between abnormalities and SC, although the correlation estimates were lower than Christmas et al. (2001). Genetic and phenotypic correlations between SC and semen quality traits for all reported studies are typically in the same direction, but phenotypic correlations are generally much lower. Refer to Table 4 to compare genetic and phenotypic correlations.

Semen Production Traits

Table 4 also includes genetic and phenotypic correlations between semen production traits. Additionally, Table 4 includes the corresponding standard errors. Reported genetic correlations between VOL and CONC are generally negative and unfavorable because as VOL increases, CONC decreases. On the contrary, Berry et al. (2019) estimated that the genetic correlation between VOL and CONC was positive and moderate; however, the standard error was large ± 0.20 and the phenotypic correlation was estimated to be extremely low.

Reported genetic correlations between VOL and NSP are all high, positive, and favorable, which is to be expected as total NSP is impacted by the VOL. The genetic correlations do vary in strength, but all are generally high estimates. The genetic correlation between CONC and NSP yielded similar results, as CONC is the additional factor that goes into calculating total NSP. As summarized in Table 4, Druet et al. (2009), Taylor et al. (1985), Gredler et al. (2007), Berry et al. (2019), and David et al. (2006)

Table 4. Phenotypic (r_P) and genetic (r_G) correlations between semen production and quality traits

n	r_P	r_G	Breed	Source
Volume and concentration				
2,351	−0.07	−0.19	Holstein	Taylor et al. (1985)
2,387	−0.15	−0.42 ± 0.17	Normande	Ducrocq and Humblot (1995)
974	0.04	−0.24	Lacaune Rams	David et al. (2006)
301	−0.17	0.06 ± 0.13	Beef/Dairy Cross	Gredler et al. (2007)
515	0.02	−0.55 ± 0.18	Holstein	Druet et al. (2009)
	0.10	0.40 ± 0.20	Beef & Dairy	Berry et al. (2019)
Volume and number of spermatozoa				
2,351	0.55	0.45	Holstein	Taylor et al. (1985)
974	0.86	0.84	Lacaune Rams	David et al. (2006)
301	0.70	0.83 ± 0.13	Beef/ Dairy Cross	Gredler et al. (2007)
515	0.61	0.47 ± 0.18	Holstein	Druet et al. (2009)
787	0.63	0.66 ± 0.16	Beef & Dairy	Berry et al. (2019)
Volume and motility				
841	—	−0.38	Line 1 Hereford	Kealey et al. (2006)
515	−0.03	−0.20 ± 0.19	Holstein	Druet et al. (2009)
787	0.05	0.07 ± 0.06	Beef & Dairy	Berry et al. (2019)
974	−0.02	−0.04	Lacaune Rams	David et al. (2006)
Volume and motility score				
2,387	—	−0.03 ± 0.21	Normade	Ducrocq and Humblot (1995)
301	−0.12	0.21 ± 0.17	Beef/Dairy Cross	Gredler et al. (2007)
515	0.01	−0.17 ± 0.19	Holstein	Druet et al. (2009)
Volume and progressive motility				
515	−0.05	−0.53 ± 0.45	Holstein	Druet et al. (2009)
Volume and percentage of living spermatozoa				
2,387	−0.05	−0.16 ± 0.21	Normande	Ducrocq and Humblot (1995)
841	—	−0.09	Line 1 Hereford	Kealey et al. (2006)
515	−0.10	−0.47 ± 0.26	Holstein	Druet et al. (2009)
787	0.01	0.32 ± 0.22	Beef & Dairy	Berry et al. (2019)
Volume and abnormality				
2,387	−0.13	−0.26 ± 0.24	Normande	Ducrocq and Humblot (1995)
515	0.0	0.23 ± 0.26	Holstein	Druet et al. (2009)
Volume and percentage of normal spermatozoa				
301	−0.13	0.31 ± 0.15	Beef/Dairy Cross	Gredler et al. (2007)
841	—	0.32	Line 1 Hereford	Kealey et al. (2006)
Volume and percentage of spermatozoa with an abnormal head				
515	0.02	−0.12 ± 0.27	Holstein	Druet et al. (2009)
Volume and percentage of spermatozoa with an abnormal tail				
515	0.0	0.43 ± 0.35	Holstein	Druet et al. (2009)
Volume and percentage of spermatozoa with an abnormal cytoplasmic droplet				
515	0.01	0.32 ± 0.28	Holstein	Druet et al. (2009)
Volume and primary abnormalities				
841	—	0.33	Line 1 Hereford	Kealey et al. (2006)
Volume and secondary abnormalities				
841	—	−0.34	Line 1 Hereford	Kealey et al. (2006)
Concentration and number of spermatozoa				
717	−0.85	−1.03	Angus	Knights et al. (1984)
2,351	0.67	0.72	Holstein	Taylor et al. (1985)
974	0.51	0.30	Lacaune Rams	David et al. (2006)
301	0.52	0.60 ± 0.17	Beef/Dairy Cross	Gredler et al. (2007)
515	0.71	0.46 ± 0.18	Holstein	Druet et al. (2009)
787	0.68	0.42 ± 0.21	Beef & Dairy	Berry et al. (2019)
Concentration and motility				
717	1.00	1.01	Angus	Knights et al. (1984)
974	0.18	0.04	Lacaune Rams	David et al. (2006)
841	—	0.81	Line 1 Hereford	Kealey et al. (2006)

Table 4. Continued

n	r_P	r_G	Breed	Source
515	0.01	0.12 ± 0.20	Holstein	Druet et al. 2009
787	0.20	0.29 ± 0.04	Beef & Dairy	Berry et al. 2019
n	r_P	r_G	Breed	Source
Concentration and motility score				
2,387	—	0.67 ± 0.11	Normande	Ducrocq and Humblot (1995)
301	0.23	0.48 ± 0.17	Beef/Dairy Cross	Gredler et al. (2007)
515	0.02	−0.01 ± 0.20	Holstein	Druet et al. (2009)
Concentration and progressive motility				
515	0.06	0.35 ± 0.36	Holstein	Druet et al. (2009)
Concentration and percentage of living spermatozoa				
717	−0.81	0.01	Angus	Knights et al. (1984)
2,387	0.47	0.54 ± 0.15	Normande	Ducrocq and Humblot (1995)
515	0.04	0.29 ± 0.26	Holstein	Druet et al. (2009)
787	0.15	0.37 ± 0.23	Beef & Dairy	Berry et al. (2019)
Concentration and abnormal spermatozoa				
2,387	−0.07	−0.27 ± 0.24	Normande	Ducrocq and Humblot (1995)
515	−0.07	−0.34 ± 0.24	Holstein	Druet et al. (2009)
Concentration and percentage of normal spermatozoa				
841	—	0.36	Line 1 Hereford	Kealey et al. (2006)
301	0.27	0.41 ± 0.17	Beef/Dairy Cross	Gredler et al. (2007)
Concentration and percentage of spermatozoa with an abnormal head				
515	−0.02	−0.23 ± 0.24	Holstein	Druet et al. (2009)
Concentration and percentage of spermatozoa with an abnormal tail				
515	−0.06	−0.33 ± 0.30	Holstein	Druet et al. (2009)
Concentration and percentage of spermatozoa with an abnormal cytoplasmic droplet				
515	−0.08	−0.09 ± 0.28	Holstein	Druet et al. (2009)
Concentration and primary abnormalities				
841	—	0.36	Line 1 Hereford	Kealey et al. (2006)
Concentration and secondary abnormalities				
841	—	−0.58	Line 1 Hereford	Kealey et al. (2006)
Number of spermatozoa and motility				
974	0.06	−0.01	Lacaune Rams	David et al. (2006)
515	−0.03	−0.12 ± 0.25	Holstein	Druet et al. (2009)
787	0.13	0.36 ± 0.08	Beef & Dairy	Berry et al. (2019)
Number of spermatozoa and motility score				
717	—	−1.04	Angus	Knights et al. (1984)
301	0.06	0.50 ± 0.13	Beef/Dairy Cross	Gredler et al. (2007)
515	0.01	−0.24 ± 0.29	Holstein	Druet et al. (2009)
Number of spermatozoa and progressive motility				
515	0.02	−0.22 ± 0.67	Holstein	Druet et al. (2009)
Number of spermatozoa and percentage of living spermatozoa				
515	0.03	−0.18 ± 0.38	Holstein	Druet et al. (2009)
Number of spermatozoa and abnormal spermatozoa				
515	−0.05	−0.09 ± 0.38	Holstein	Druet et al. (2009)
Number of spermatozoa and percentage of normal spermatozoa				
301	0.07	0.54 ± 0.11	Beef/Dairy Cross	Gredler et al. (2007)
Number of spermatozoa and percentage of spermatozoa with an abnormal head				
515	−0.02	−0.38 ± 0.36	Holstein	Druet et al. (2009)
Number of spermatozoa and percentage of spermatozoa with an abnormal tail				
515	−0.03	0.14 ± 0.54	Holstein	Druet et al. (2009)
Number of spermatozoa and percentage of spermatozoa with an abnormal cytoplasmic droplet				
515	−0.05	0.33 ± 0.43	Holstein	Druet et al. (2009)
Motility and motility score				
515	0.66	0.88 ± 0.06	Holstein	Druet et al. (2009)
Motility and progressive motility				
515	0.48	0.74 ± 0.30	Holstein	Druet et al. (2009)

Table 4. Continued

n	r_P	r_G	Breed	Source
Motility and mass activity				
2,424	0.84	0.98 ± 0.01	Tropical Composite	Corbet et al. (2013)
Motility and percentage of living spermatozoa				
515	0.40	0.58 ± 0.15	Holstein	Druet et al. (2009)
787	0.54	0.89 ± 0.04	Beef & Dairy	Berry et al. (2019)
n	r_P	r_G	Breed	Source
Motility and abnormal spermatozoa				
515	−0.20	−0.55 ± 0.19	Holstein	Druet et al. (2009)
Motility and percentage of normal spermatozoa				
549	0.38	0.43 ± 0.64	Hereford, Angus, & Red Angus	Smith et al. (1989)
841	—	0.51	Line 1 Hereford	Kealey et al. (2006)
2,424	0.56	0.77 ± 0.09	Tropical Composite	Corbet et al. (2013)
Motility and percentage of spermatozoa with an abnormal head				
515	0.17	−0.56 ± 0.18	Holstein	Druet et al. (2009)
Motility and percentage of spermatozoa with an abnormal tail				
515	−0.11	−0.24 ± 0.24	Holstein	Druet et al. (2009)
Motility and percentage of spermatozoa with an abnormal cytoplasmic droplet				
515	−0.07	0.13 ± 0.23	Holstein	Druet et al. (2009)
Motility and primary abnormalities				
549	−0.31	−0.36 ± 0.55	Hereford, Angus, & Red Angus	Smith et al. (1989)
841	—	0.57	Line 1 Hereford	Kealey et al. (2006)
Motility and secondary abnormalities				
549	−0.22	0.71 ± 0.89	Hereford, Angus, & Red Angus	Smith et al. (1989)
841	—	−0.54	Line 1 Hereford	Kealey et al. (2006)
Motility and scrotal circumference				
549	0.13	−0.04 ± 0.40	Hereford, Angus, & Red Angus	Smith et al. (1989)
1,282	—	0.56	Angus	Christmas et al. (2001)
1,281	0.11	0.36 ± 0.29	Angus	Garmyn et al. (2011)
2,424	0.42	0.56 ± 0.10	Tropical Composite	Corbet et al. (2013)
Motility score and progressive motility				
515	0.40	0.71 ± 0.27	Holstein	Druet et al. (2009)
Motility score and percentage of living spermatozoa				
2,387	0.73	0.83 ± 0.09	Normande	Ducrocq and Humblot (1995)
515	0.31	0.61 ± 0.15	Holstein	Druet et al. (2009)
Motility score and total abnormalities				
2,387	−0.34	−0.68 ± 0.16	Normande	Ducrocq and Humblot (1995)
515	−0.16	−0.40 ± 0.18	Holstein	Druet et al. (2009)
Motility score and percentage of normal spermatozoa				
301	0.55	0.90 ± 0.05	Beef/ Dairy Cross	Gredler et al. (2007)
Motility score and percentage of spermatozoa with an abnormal head				
515	−0.15	−0.54 ± 0.17	Holstein	Druet et al. (2009)
Motility score and percentage of spermatozoa with an abnormal tail				
515	−0.05	−0.02 ± 0.23	Holstein	Druet et al. (2009)
Motility score and percentage of spermatozoa with an abnormal cytoplasmic droplet				
515	−0.14	−0.07 ± 0.21	Holstein	Druet et al. (2009)
Progressive motility and percentage of living spermatozoa				
515	0.70	0.89 ± 0.17	Holstein	Druet et al. (2009)
Progress motility and total abnormalities				
515	−0.24	−0.38 ± 0.38	Holstein	Druet et al. (2009)
Progressive motility and percentage of spermatozoa with an abnormal head				
515	−0.17	−0.39 ± 0.41	Holstein	Druet et al. (2009)
Progressive motility and percentage of spermatozoa with an abnormal tail				
515	−0.17	−0.15 ± 0.57	Holstein	Druet et al. (2009)
Progressive motility and percentage of spermatozoa with an abnormal cytoplasmic droplet				
515	−0.13	−0.08 ± 0.44	Holstein	Druet et al. (2009)

Table 4. Continued

n	r_P	r_G	Breed	Source
Mass activity and percentage of normal spermatozoa				
2,424	0.52	0.87 ± 0.08	Tropical Composite	Corbet et al. (2013)
Mass activity and scrotal circumference				
2,424	0.43	0.60 ± 0.10	Tropical Composite	Corbet et al. (2013)
Percentage of living spermatozoa and total abnormalities				
2,387	−0.37	0.43 ± 0.22	Normande	Ducrocq and Humblot (1995)
515	−0.20	0.23 ± 0.29	Holstein	Druet et al. (2009)
Percentage of living spermatozoa and percentage of normal spermatozoa				
841	—	0.32	Line 1 Hereford	Kealey et al. 2006
n	r_P	r_G	Breed	Source
Percentage of living spermatozoa and percentage of spermatozoa with an abnormal head				
515	−0.15	−0.44 ± 0.28	Holstein	Druet et al. (2009)
Percentage of living spermatozoa and percentage of spermatozoa with an abnormal tail				
515	−0.12	0.10 ± 0.39	Holstein	Druet et al. (2009)
Percentage of living spermatozoa and percentage of spermatozoa with an abnormal cytoplasmic droplet				
515	−0.11	0.05 ± 0.30	Holstein	Druet et al. (2009)
Percentage of living spermatozoa and primary abnormalities				
841	—	0.33	Line 1 Hereford	Kealey et al. (2006)
Percentage of living spermatozoa and secondary abnormalities				
841	—	−0.43	Line 1 Hereford	Kealey et al. (2006)
Percentage of living spermatozoa and scrotal circumference				
—	—	−0.24	Nellore	Silva et al. (2013)
Total abnormalities and percentage of spermatozoa with an abnormal head				
515	0.61	0.71 ± 0.18	Holstein	Druet et al. (2009)
Total abnormalities and percentage of spermatozoa with an abnormal tail				
515	0.77	0.78 ± 0.16	Holstein	Druet et al. (2009)
Percentage of spermatozoa with an abnormal cytoplasmic droplet				
515	0.36	0.15 ± 0.34	Holstein	Druet et al. 2009
Total abnormalities and scrotal circumference				
1,282	—	−0.32	Angus	Christmas et al. (2001)
1,281	-0.11	−0.23 ± 0.18	Angus	Garmyn et al. (2011)
Percentage of normal spermatozoa and primary abnormalities				
549	−0.57	−0.85 ± 0.63	Hereford, Angus, & Red Angus	Smith et al. (1989)
Percentage of normal spermatozoa and secondary abnormalities				
549	−0.80	0.16 ± 1.54	Hereford, Angus, & Red Angus	Smith et al. (1989)
Percentage of normal spermatozoa and scrotal circumference				
549	−0.17	0.36 ± 0.34	Hereford, Angus, & Red Angus	Smith et al. (1989)
2,424	0.31	0.55 ± 0.13	Tropical Composite	Corbet et al. (2013)
Percentage of spermatozoa with an abnormal head and percentage of spermatozoa with an abnormal tail				
515	0.02	0.13 ± 0.39	Holstein	Druet et al. (2009)
Percentage of spermatozoa with an abnormal head and percentage of spermatozoa with an abnormal cytoplasmic droplet				
515	0.12	−0.43 ± 0.30	Holstein	Druet et al. (2009)
Percentage of spermatozoa with an abnormal tail and percentage of spermatozoa with an abnormal cytoplasmic droplet				
515	0.15	0.06 ± 0.37		Druet et al. (2009)
Primary abnormalities and secondary abnormalities				
549	0.17	0.14 ± 0.64	Hereford, Angus, & Red Angus	Smith et al. (1989)
841	—	−0.87	Line 1 Hereford	Kealey et al. (2006)
Primary abnormalities and scrotal circumference				
549	−0.09	0.14 ± 0.22	Hereford, Angus, & Red Angus	Smith et al. (1989)
1,282	—	−0.25	Angus	Christmas et al. (2001)
1,281	−0.10	−0.19 ± 0.17	Angus	Garmyn et al. (2011)
Secondary abnormalities and scrotal circumference				
549	−0.10	1.22 ± 1.57	Hereford, Angus, & Red Angus	Smith et al. (1989)
1,282	—	−0.40	Angus	Christmas et al. (2001)
1,281	−0.05	−0.11 ± 0.19	Angus	Garmyn et al. (2011)

n = number of bulls

reported positive correlations, which are desirable because as CONC increases, so does the NSP in an ejaculate.

Semen Quality Measures

Relatively few studies have quantified the interrelationship of varying MOT measures (Table 4). Druet et al. (2009) reported a strong, positive relationship, as would be expected, between MOT and MOTSC. Druet et al. (2009) measured the percentage of progressive spermatozoa (%PROG) and reported a strong, positive estimate for its genetic correlation with MOT; however, the standard error was ±0.30. A phenotypic correlation of moderate, positive correlation estimate was reported by Druet et al. (2009), indicating that there is an environmental effect that reduces the phenotypic correlations. A strong, favorable correlation is expected, as the PROG is dependent on the MOT of the ejaculate. Motility is also strongly, favorably genetically correlated with %LIV (0.58; Druet et al. 2009). Furthermore, MOTSC is favorably correlated with %PROG and %LIV with strong, positive relationships (Druet et al. 2009). Corbet et al. (2013) estimated the genetic correlation between PROG and MASS and found a high, favorable correlation. In this population, these correlations are high enough to suggest that PROG and MASS are the same traits.

Moderate, favorable genetic correlations were reported between MOT and %NORM (Smith et al., 1989; Kealey et al., 2006). In a group of tropical composite bulls, Corbet et al. (2013) reported a strong, favorable genetic correlation between PROG and %NORM.

Genetic relationships between MOT and traits related to the percentage of abnormal spermatozoa were negative, indicating that abnormalities were associated with a low MOT (Druet et al. 2009). The results were comparable with the negative, favorable genetic correlation estimates between MOTSC and abnormal spermatozoa traits reported by Ducrocq and Humblot (1995) and Druet et al. (2009), which are summarized in Table 4. Smith et al. (1989) reported similar negative genetic correlations between MOT and PRIM and SEC. On the other hand, Kealey et al. (2006) reported a positive, unfavorable genetic correlation between MOT and PRIM. No standard errors were provided. Total abnormalities were negatively and favorably correlated to %PROG; however, the standard error was ±0.38 (Druet et al. 2009). Furthermore, Druet et al. (2009) reported percentages of spermatozoa with abnormal HEAD, TAIL, or DROP that reflected the same negative, favorable genetic correlation with %PROG and %LIV, but these too had large standard errors. Druet et al. (2009) concluded that as the %PROG increased, so did the percentage of living spermatozoa after thawing based on a strong, favorable genetic and phenotypic correlation between those traits.

Corbet et al. (2013) indicated that as the MASS increased, so did the %NORM with a strong, favorable genetic correlation estimate. The %NORM was found to be strongly, favorably genetically correlated to PRIM (Smith et al., 1989). However, Smith et al. (1989) reported %NORM was unfavorably genetically correlated with the percentage of SEC, but the reported standard error is outside of the parameter space, likely due to the least squares methodology utilized. Table 4 summarizes the genetic and phenotypic correlations.

As expected, Druet et al. (2009) reported strong, favorable genetic correlations between ABNO and HEAD and TAIL. Unexpectedly, an unfavorable, negative genetic correlation with DROP was reported; however, the standard error was large. Percentage of spermatozoa with an abnormal head was lowly, positively genetically correlated with TAIL, but negatively genetically correlated with DROP. Percentage of spermatozoa with an abnormal tail is very lowly genetically correlated with DROP. It is difficult to speculate whether or not the correlations could be considered favorable or not, but certainly, some correlation could be advantageous for selection against abnormalities. The phenotypic correlation between ABNO and DROP was substantially different from the genetic correlation, which means there was a large environmental component that affected the cytoplasmic droplets. In addition, Druet et al. (2009) reported no phenotypic correlation between HEAD and TAIL (0.02).

Production Traits with Semen Quality Measures

Kealey et al. (2006) and Druet et al. (2009) reported negative, unfavorable genetic correlation between VOL and MOT in a population of Line 1 Herefords and dairy cattle, respectively. Gredler et al. (2007) reported a moderate genetic correlation with a large standard error between VOL and MOTSC; however, the phenotypic correlation was low and negative. Volume was unfavorably genetically correlated to %PROG and %LIV (Druet et al. 2009). Ducrocq and Humblot (1995) also reported a negative, unfavorable genetic correlation between VOL and %LIV. Volume was unfavorably genetically correlated with ABNO (Druet et al. 2009). On the other hand, Ducrocq and Humblot (1995)

reported a negative, moderate genetic correlation between VOL and ABNO. The varying correlation estimates are reported in Table 4.

Druet et al. (2009) found a low, favorable genetic correlation between CONC and MOT. Kealey et al. (2006) also found a positive, favorable genetic correlation between CONC and MOT, but it was much stronger. Furthermore, Knights et al. (1984) estimated the genetic correlation between the two traits; however, they did not report standard errors. Furthermore, as CONC increased, so did the %PROG. Druet et al. (2009) reported a favorable genetic correlation, but the genetic correlation was not different from zero. Ducrocq and Humblot (1995) estimated the genetic correlation between CONC and %LIV to be moderate and positive with a similar phenotypic correlation (Table 4). Druet et al. (2009) also reported a favorable genetic correlation between CONC and %LIV, but the standard error was high. More recently, Berry et al. (2019) reported a moderate, positive genetic correlation, but the standard error was also large for this estimate (Table 4). Concentration was favorably correlated to ABNO with a negative, moderate correlation estimate (Druet et al. 2009). Furthermore, Kealey et al. (2006) reported genetic correlations between CONC and PRIM to be positive and moderate. This unfavorable genetic correlation contradicts the favorable genetic correlation that Kealey et al. (2006) reported between CONC and SEC. Without reported standard errors, it is difficult to evaluate the estimate's accuracy.

Varying estimates have been published for genetic correlations between NSP and MOT and are reported in Table 4. Druet et al. (2009) found a negative genetic correlation, whereas Berry et al. (2019) reported a positive, favorable genetic correlation between NSP and MOT. Furthermore, Druet et al. (2009) reported that the genetic correlation between NSP and MOTSC was also unfavorable, but this estimate had a large standard error (Table 4). On the other hand, Gredler et al. (2007) estimated the correlation between NSP and MOTSC to be favorable. Gredler et al. (2007) also estimated a favorable genetic correlation between NSP and %NORM. Druet et al. (2009) reported a negative genetic correlation between NSP and HEAD, although the estimate had a large standard error, indicating that the estimate was not statistically different from zero. On the other hand, NSP had a favorable genetic correlation with TAIL or DROP (Druet et al., 2009). The genetic correlation estimates indicate as NSP increases, HEAD decreases, but TAIL and DROP increase.

BULL FERTILITY INDEX

The sire conception rate (SCR) evaluation encompasses inbreeding of the bull, inbreeding of the embryo from the mating, age of the bull, AI center where the bull was collected at, year of collection, and the effect of the bull itself. The SCR is a selection index which provides genetic prediction for AI dairy bulls. It has been realized that SCR does not account for bulls with poor quality semen attributes as bulls with inferior semen quality are removed from the population because of the strict semen freezing requirements established by AI centers. While there are concerns pertaining to the variation in SCR phenotypes, there is more than 10% difference in conception rate between high and low fertility bulls measured by SCR (Penagaricano et al., 2012; Rezende et al., 2018).

Determining which bull fertility traits to measure when making selection decisions has proven to be difficult, and it has been suggested that the only way to truly measure bull fertility is by directly measuring conception rate (Penagaricano et al., 2012). Bull fertility indices have the opportunity to provide beef producers with the ability to make selection decisions on traits which are difficult to measure or not observed until later in life, such as conception rate. Additionally, utilizing genomic prediction has the potential to provide more predictive power for estimating genetic merit for bull fertility. Many of the advancements in utilizing genomic prediction pertaining to measuring bull fertility have been led by the dairy industry (Han et al., 2016; Rezende et al., 2018; Nani et al., 2019). While ample beef bull fertility phenotypes do exist from both bull studs and individual producers who use BSEs, the lack of data flow between the phenotype holders and evaluation providers prevents the ability to provide such an index to beef producers using traditional evaluation techniques, let alone genomic prediction.

GENOMIC PREDICTIONS

The genomic prediction of polygenic traits, such as fertility, has revolutionized agriculture and human medicine. Although genomic prediction for improved cow fertility has received a lot of attention, beef bull fertility has largely been ignored (Abdollahi-Arpanahi et al., 2017). Utilizing genomic prediction, researchers can utilize information on the bull's entire genome to assist in predicting the fertility of the bull. Incorporation of genome sequence and other biological information with genomic predictions has the potential to

Table 5. Genes associated with male fertility traits

Associated sperm trait	Gene identified	Chromosome	Breed	Source
Percentage of living spermatozoa	*SPATA16*	1	Fleckvieh	Ferenčaković et al. (2017
	GPX5	1	Fleckvieh	Ferenčaković et al. (2017
	NYD-SP5	1	Fleckvieh	Ferenčaković et al. (2017
	PIAS1	1	Fleckvieh	Ferenčaković et al. (2017
	FEM1B	1	Fleckvieh	Ferenčaković et al. (2017
	SPESP1	1	Fleckvieh	Ferenčaković et al. (2017
	NOX5	1	Fleckvieh	Ferenčaković et al. (2017
	FEM1B	10	Fleckvieh	Ferenčaković et al. (2017
	SPESP1	10	Fleckvieh	Ferenčaković et al. (2017
	NOX5	10	Fleckvieh	Ferenčaković et al. (2017
	TSPYL5	14	Fleckvieh	Ferenčaković et al. (2017
Total number of spermatozoa	*RPL10L*	10	Fleckvieh	Ferenčaković et al. (2017
	SLC25A31	17	Fleckvieh	Ferenčaković et al. (2017
	CDH18	20	Fleckvieh	Ferenčaković et al. (2017
	KCNU1	27	Fleckvieh	Ferenčaković et al. (2017
Sperm motility	*COX7A2L*	11	Jersey	Rezende et al. (2018)
	ZMYND10	22	Jersey	Rezende et al. (2018)
	SLC25A20	22	Jersey	Rezende et al. (2018)
	DNAH3	25	Jersey	Rezende et al. (2018)
	PARP11	5	Holstein	Han and Penagaricano (2016)
	AKAP3	5	Holstein	Han and Penagaricano (2016)
	CNNM4	11	Jersey	Han and Penagaricano (2016)
Acrosome reaction/Fertilization	*PKDREJ*	5	Jersey	Rezende et al. (2018)
	STX2	17	Jersey	Rezende et al. (2018)
	TBC1D20	13	Holstein	Nicolini et al. (2018)
	CCT6A	25	Holstein	Han and Penagaricano (2016)
	CACNA1H	25	Holstein	Penagaricano et al. (2012)
	ITGB5	1	Holstein	Feugang et al. (2009)
	FER1L5	11	Jersey	Rezende et al. (2018)
Spermatogenesis	*EPB41L2*	9	Jersey	Rezende et al. (2018)
	IP6K1	22	Jersey	Rezende et al. (2018)
	ADAM28	8	Holstein	Nicolini et al. (2018)
	PIWIL3	17	Holstein	Nicolini et al. (2018)
	TMEM119	17	Holstein	Nicolini et al. (2018)
	TDRD9	21	Holstein	Han and Penagaricano (2016)
	CKB	21	Holstein	Han and Penagaricano (2016)
	MGRN1	25	Holstein	Han and Penagaricano (2016)
	SEPT12	25	Holstein	Han and Penagaricano (2016)
	DYNC1I2	2	Holstein	Penagaricano et al. (2012)
	LOC784935	5	Holstein	Penagaricano et al. (2012)
	ZNF541	18	Holstein	Penagaricano et al. (2012)
	LOC617302	25	Holstein	Penagaricano et al. (2012)
Testis development	*PDGFD*	15	Jersey	Rezende et al. (2018)
	DNAJA1	8	Holstein	Nicolini et al. (2018)
Gametogenesis	*ROGDI*	25	Holstein	Penagaricano et al. (2012)
	SPO11	13	Holstein	Nicolini et al. (2018)
	RAD21L1	13	Holstein	Nicolini et al. (2018)
	BCL2L1	13	Holstein	Nicolini et al. (2018)
Sire conception rate	*PROP1*	7	Holstein	Lan et al. (2013)
	FGF2	17	Holstein	Khatib et al. (2010)
	STAT5A	19	Holstein	Khatib et al. (2010)
	RIMS1	9	Holstein	Han and Penagaricano (2016)
	CTCFL	13	Holstein	Han and Penagaricano (2016)
	SPO11	13	Holstein	Han and Penagaricano (2016)

Table 5. Continued

Associated sperm trait	Gene identified	Chromosome	Breed	Source
	CADM1	15	Holstein	Han and Penagaricano (2016)
	IGF1R	21	Holstein	Han and Penagaricano (2016)
	BRF1	21	Holstein	Han and Penagaricano (2016)
	KAT8	25	Holstein	Han and Penagaricano (2016)
	ITGAM	25	Holstein	Han and Penagaricano (2016)
	TYW1	25	Holstein	Han and Penagaricano (2016)
	LOC521021	25	Holstein	Penagaricano et al. (2012)
	COL1A2-AS1	4	Holstein	Feugang et al. (2009)
	RIMS1	9	Holstein	Feugang et al. (2009)
	SFNX1	10	Holstein	Feugang et al. (2009)

make crucial connections between biology and performance that may save producers money as well as improve the accuracy of genomic prediction in the future (Taylor et al., 2018).

Genome-wide association studies have been successful in identifying genomic regions and individual variants associated with numerous complex traits, and some weighted genomic prediction models can incorporate this information by placing more emphasis on these markers in the prediction. The incorporation of these data into predictive models could positively affect both model predictive ability and model robustness (Abdollahi-Arpanahi et al., 2017). While few genomic studies have been performed on beef bulls, analyses including dairy bulls have identified several SNPs within or near genes associated with male fertility traits. Table 5 outlines various genes which have been associated with male fertility traits.

A few genes pertaining to spermatogenesis have been identified. The gene *PIWIL3* has been identified as associated with spermatozoa development in Holstein cattle (Nicolini et al., 2018). This gene encodes a member of the PIWI family, which is a group of proteins that are essential for spermatogenesis and post-transcriptional events leading to translation (Paronetto and Sette, 2010). Another gene found on chromosome 17 that is associated with spermatogenesis is *TMEM119* (Nicolini et al., 2018). *TMEM119* encodes a transmembrane protein actively involved in osteoblast differentiation and is expressed in spermatocytes and spermatids in developing testis (Mizuhashi et al., 2015).

Several genes associated with spermatozoa motility were identified by Ferenčaković et al. (2017), when studying inbreeding depression in Austrian Fleckvieh bulls. *SLC25A31* is associated with mediating energy generation and consumption in the distal flagellum, therefore, influencing motility. *CDH18* is a member of the cadherin superfamily which is responsible for mediating calcium-dependent cell-to-cell adhesion and has been found to be associated with spermatozoa motility (Pacheco et al., 2011). *KCNU1* is a gene found on chromosome 27 which is known to encode a testis-specific potassium channel. This channel is involved with maintaining normal sperm morphology and motility (Schreiber et al., 1998). Rezende et al. (2018) identified an SNP within *COX7A2L* on chromosome 11. This gene encodes cytochrome c oxidase which is responsible for promoting respiratory supercomplex assembly and regulates energy generation. It has been speculated that this process is involved in sperm motility. Rezende et al. (2018) additionally identified other SNPs within or near genes *ZMYND10, SLC25A20,* and *DNAH3* that were associated with sperm motility traits. *ZMYND10* is a gene exclusively expressed in the testis which is involved in cilia integrity and has been implicated in sperm dysmotility, and thus infertility (Moore et al., 2013). *SCL25A20* is a gene involved in ATP production and cell energy metabolism (Asghari et al., 2017). *DNAH3* encodes a member of the dynein family and has been proven to cause abnormalities pertaining to the sperm flagella and as a result negatively impact sperm motility (Ben Khelifa et al., 2014).

Many genes associated with the percentage of living sperm are noted in the literature. A gene on chromosome X called *SPATA16* was found to cause infertility in humans (Dam et al., 2007). The infertility results from spermatogenesis defects which cause malformation of the spermatozoa. Ferenčaković et al. (2017) found an SNP associated with this gene in Austrian Fleckvieh cattle that was associated with formation of the spermatozoa. Additionally, Ferenčaković et al. (2017) found an SNP on chromosome 1 associated with the *GPX5* gene. This gene encodes epididymis secretory sperm-binding protein Li 75p, which is a

protein with predicted involvement in protecting the spermatozoa membrane from lipid peroxidation during oxidative stress, and potentially preventing a premature acrosome reaction (Hall et al., 1998). Ferenčaković et al. (2017) also identified *NYD-SP5* and *PIAS1* on chromosome 1 to be associated with the percentage of living spermatozoa. Yin et al. (2005) found *NYD-SP5*, which encodes testis development protein and plays a regulatory role in spermatogenesis. Vernocchi et al. (2014) noted that *PIAS1* is associated with androgen receptor-mediated initiation and the maintenance of spermatogenesis.

Additional genes have been found to be involved in various male fertility processes. For example, *SPO11* and *RAD21L1* were found to be associated with gametogenesis (Nicolini et al., 2018). *SPO11* is a topoisomerase-like protein responsible for the formation of DNA double-strand breaks that occur during meiotic recombination (Keeney et al., 1997). *RAD21L1* encodes a protein that is involved in multiple aspects of meiosis (Choi et al., 2008). Genes associated with acrosome reaction and the fertilization process were identified in a study with Jersey bulls (Rezende et al., 2018). These genes are *PKDREJ, STX2,* and *FER1L5*. *PKDREJ* encodes a sperm surface receptor which mediates the sperm–egg interaction (Hamm et al., 2007). *STX2* encodes a protein family that controls membrane fusion during the acrosome reaction (Hutt et al., 2005). *FER1L5* encodes proteins which are important during fusion events involved in spermatogenesis (Washington and Ward, 2006).

Several genomic regions have been associated with sire conception using genome wide association studies (GWAS). The genes and their chromosome locations are outlined in Table 5. Sire conception rate is currently only predicted in the dairy industry. The function of the genes associated with sire conception rate ranges from encoding proteins involved in sperm maturation (*PARP11*) and sperm motility genome wide association studies (*AKAP3*) to encoding proteins which play a role in testis development (*IGF1R*) and the fertilization process (*CCT6A*; Han and Penagaricano, 2016).

Beyond standard genomic prediction, there is an additional desire to better understand the underlying biology of male fertility. While utilizing this technology to identify genes in the cattle industry is rare, a few examples include utilizing dual targeted β-defensin and exome sequencing to identify bull fertility genes in the dairy industry (Whiston et al., 2017; Lyons et al., 2018). Genes identified have been found to be involved with spermatogenesis (Whiston et al., 2017) and sperm binding (Lyons et al., 2018).

Bull fertility has the possibility to be significantly impacted by genomic selection. SNP genotyping continues to gain popularity in the beef industry (Taylor et al., 2018). While identifying SNPs that are located within or near certain genes pertaining to male fertility traits helps to better understand the expression of certain traits, this information is not essential for genomic selection. However, providing producers with genomic selection tools could cause a more rapid improvement in fertility traits, but in order to make genomic selection for beef bull fertility a reality, more beef bull fertility trait phenotypes are a necessity (Taylor et al., 2018).

CONCLUSIONS

Bull fertility is an economically relevant trait that merits additional research, particularly on beef bulls. Within the U.S. beef cattle herd, producers utilize a large number of sires because of the lack of adoption of reproductive technologies such as AI and embryo transfer. To improve the efficiency of the beef cattle herd, improvements in bull fertility must be made, and change could be further accelerated by utilizing genetic selection tools. If beef producers could make selection decisions for improved fertility, it would save time and resources, improve production efficiency, and increase profitability (Rogers et al., 2012). Heritability estimates of semen production traits, which include VOL, CONC, and NSP, are generally low to moderate. Semen quality measures, for the purpose of this review, are measures related to motility and morphology. Reported genetic correlations provide useful candidates for indirect selection to improve fertility traits. Correlations between semen traits are generally moderate and favorable, indicating that selection for one trait could benefit another.

Genomic selection is another useful tool which can increase genetic prediction capabilities and improve selection. Genomic prediction has an even greater impact on the prediction of polygenic traits, such as fertility, because of the ability to incorporate biological information into genetic prediction. While the dairy industry has been a leader in identifying genomic regions associated with male fertility traits, the beef industry has identified relatively few SNPs related to beef bull fertility.

Studying bull fertility in the beef industry could increase beef production, improve the efficiency of the livestock industry, and provide benefits to male fertility traits in other species. Limited published

research and a need for improvement make additional beef bull fertility research a necessity.

LITERATURE CITED

Abdollahi-Arpanahi, R., G. Morota, and F. Penagaricano. 2017. Predicting bull fertility using genomic data and biological information. J. Dairy Sci. 100:9656–9666. doi:10.3168/jds.2017-13288

Amann, R. P., and J. O. Almquist. 1976. Bull management to maximize sperm output. In: Proceedings of the Sixth Technical Conference on Artificial Insemination and Reproduction. Milwaukie, USA, 20-21 February 1976. Pp. 1–10.

Asghari, A., S. A. Marashi, and N. Ansari-Pour. 2017. A sperm-specific proteome-scale metabolic network model identifies non-glycolytic genes for energy deficiency in asthenozoospermia. Syst. Biol. Reprod. Med. 63:100–112. doi:10.1080/19396368.2016.1263367

Barth, A. D., and C. L. Waldner. 2002. Factors affecting breeding soundness classification of beef bulls examined at the Western College of Veterinary Medicine. Can Vet J. 43:274–284.

Ben Khelifa, M., C. Coutton, R. Zouari, T. Karaouzène, J. Rendu, M. Bidart, S. Yassine, V. Pierre, J. Delaroche, S. Hennebicq, et al. 2014. Mutations in DNAH1, which encodes an inner arm heavy chain dynein, lead to male infertility from multiple morphological abnormalities of the sperm flagella. Am. J. Hum. Genet. 94:95–104. doi:10.1016/j.ajhg.2013.11.017

Berry, D. P., B. Eivers, G. Dunne, and S. McParland. 2019. Genetics of bull semen characteristics in a multi-breed cattle population. Theriogenology 123:202–208. doi:10.1016/j.theriogenology.2018.10.006

Berry, D. P., E. Wall, and J. E. Pryce. 2014. Genetics and genomics of reproductive performance in dairy and beef cattle. Animal. 8(Suppl. 1):105–121. doi:10.1017/S1751731114000743

Bourdon, R. M., and J. S. Brinks. 1986. Scrotal circumference in yearling Hereford bulls: adjustment factors, heritabilities and genetic, environmental and phenotypic relationships with growth traits. J. Anim. Sci. 62:958–967. doi:10.2527/jas1986.624958x

Braundmeier, A. G., and D. J. Miller. 2001. The search is on: finding accurate molecular markers of male fertility. J. Dairy Sci. 84:1915–1925. doi:10.3168/jds.s0022-0302(01)74633-4

Brito, L. C., A.D. Barth, N.C. Rawlings, R.E. Wilde, D.H. Crews Jr., P.S. Mire, J.P. Kastelic. 2006. Effect of improved nutrition during calfhood on serum metabolic hormones, gonadotropins, and testosterone concentrations, and on testicular development in bulls. Domestic Anim. Endocrinology. 33:460–469. doi:10.1016/j.domaniend.2006.09.004

Bucher, K., E. Malama, M. Siuda, F. Janett, and H. Bollwein. 2019. Multicolor flow cytometric analysis of cryopreserved bovine sperm: a tool for the evaluation of bull fertility. J. Dairy Sci. 102:11652–11669. doi:10.3168/jds.2019-16572

Chenoweth, P.J. 1983. Sexual behavior of the bull: a review. J. of Dairy Sci. 66:173–179. doi:10.3168/jds.S0022-0302(83)81770-6

Choi, E., C. Han, I. Park, B. Lee, S. Jin, H. Choi, D. H. Kim, Z. Y. Park, E. M. Eddy, and C. Cho. 2008. A novel germ cell-specific protein, SHIP1, forms a complex with chromatin remodeling activity during spermatogenesis. J. Biol. Chem. 283:35283–35294. doi:10.1074/jbc.M805590200

Christmas R. A., D. W. Moser, M. F. Spire, J. M. Sargeant, and S. K. Tucker. 2001. Genetic relationships among breeding soundness traits in yearling bulls. In: Cattlemen's day. Manhattan (KS); Kansas Agricultural Experiment Station Research Reports. p. 1–3. doi:10.4148/ 2378–5977.1712

Corbet, N. J., B. M. Burns, D. J. Johnston, M. L. Wolcott, D. H. Corbet, B. K. Venus, Y. Li, M. R. McGowan, and R. G. Holroyd. 2013. Male traits and herd reproductive capability in tropical beef cattle. 2. Genetic parameters of bull traits. Anim. Prod. Sci. 53:101–113. doi:10.1071/AN12163

Coulter, G.H., R. B. Cook, and J.P. Kastelic. 1997. Effects of dietary energy on scrotal surface temperature, seminal quality, and sperm production in young beef bulls. J. Anim. Sci. 75:1048–1052. doi:10.2527/1997.7541048x

Dam, A. H., I. Koscinski, J. A. Kremer, C. Moutou, A. S. Jaeger, A. R. Oudakker, H. Tournaye, N. Charlet, C. Lagier-Tourenne, H. van Bokhoven, et al. 2007. Homozygous mutation in SPATA16 is associated with male infertility in human globozoospermia. Am. J. Hum. Genet. 81:813–820. doi:10.1086/521314

David I., L. Bodin, G. Lagriffoul, E. Madfedi, C. Robert-Granie. 2006. Genetic parameters of ram semen traits using an animal model accounting for serial correlation. Proceedings of the Eighth World Congress on Genetics Applied to Livestock Production. Belo Horizonte (Brazil), 13–18 August 2006.

DeJarnette, J. M., C. E. Marshal, R. W. Lenz, D. R. Monke, W. H. Ayars, and C. G. Sattler. 2004. Sustaining the fertility of artificially inseminated dairy cattle: the role of the artificial insemination industry. J. Dairy Sci. 87(Suppl. E):93–104. doi:10.3168/jds.S0022-0302(04)70065-X

Druet, T., S. Fritz, E. Sellem, B. Basso, O. Gérard, L. Salas-Cortes, P. Humblot, X. Druart, and A. Eggen. 2009. Estimation of genetic parameters and genome scan for 15 semen characteristics traits of Holstein bulls. J. Anim. Breed. Genet. 126:269–277. doi:10.1111/j.1439-0388.2008.00788.x

Ducrocq, V., and P. Humblot. 1995. Genetic characteristics and evolution of semen production of young Normande bulls. Livest. Prod. Sci. 41:1–10. doi:10.1016/0301-6226(94)00029-7

Evans, J. L., B. L. Golden, R. M. Bourdon, and K. L. Long. 1999. Additive genetic relationships between heifer pregnancy and scrotal circumference in Hereford cattle. J. Anim. Sci. 77:2621–2628. doi:10.2527/1999.77102621x

Ferenčaković, M., J. Sölkner, M. Kapš, and I. Curik. 2017. Genome-wide mapping and estimation of inbreeding depression of semen quality traits in a cattle population. J. Dairy Sci. 100:4721–4730. doi:10.3168/jds.2016–12164

Feugang, J. M., A. Kaya, G. P. Page, L. Chen, T. Mehta, K. Hirani, L. Nazareth, E. Topper, R. Gibbs, and E. Memili. 2009. Two-stage genome-wide association study identifies integrin beta 5 as having potential role in bull fertility. BMC Genom. 10:176. doi:10.1186/1471-2164-10-176

Garmyn, A. J., D. W. Moser, R. A. Christmas, and J. M. Bormann. 2011. Estimate of genetic parameters and effects of cytoplasmic line on scrotal circumference and semen quality traits in Angus bulls. J. Anim. Sci. 89:693–698. doi:10.2527/jas.2010–3534

Graham, J. K., E. Kunze, and R. H. Hammerstedt. 1990. Analysis of sperm cell viability, acrosomal integrity,

and mitochondrial function using flow cytometry. Biol. Reprod. 43:55–64. doi:10.1095/biolreprod43.1.55

Gredler, B., C. Fuerst, B. Fuerst-Waltl, H. Schwarzenbacher, and J. Sölkner. 2007. Genetic parameters for semen production traits in Austrian dual-purpose Simmental bulls. Reprod. Domest. Anim. 42:326–328. doi:10.1111/j.1439-0531.2006.00778.x

Hall, L., K. Williams, A. C. Perry, J. Frayne, and J. A. Jury. 1998. The majority of human glutathione peroxidase type 5 (GPX5) transcripts are incorrectly spliced: implications for the role of GPX5 in the male reproductive tract. Biochem. J. 333 (Pt 1):5–9. doi:10.1042/bj3330005

Hall, S .J. G. 1989. Chillingham cattle: social and maintenance behavior in an ungulate that breeds all year around. Anim. Behavior. 38:215–225. doi:10.1016/S0003-3472(89)80084-3

Hamm, D., B. S. Mautz, M. F. Wolfner, C. F. Aquadro, and W. J. Swanson. 2007. Evidence of amino acid diversity-enhancing selection within humans and among primates at the candidate sperm-receptor gene PKDREJ. Am. J. Hum. Genet. 81:44–52. doi:10.1086/518695

Han, Y., and F. Penagaricano. 2016. Unravelling the genomic architecture of bull fertility in Holstein cattle. BMC Genet. 17:143. doi:10.1186/s12863-016-0454-6

Harris, D. L. 1970. Breeding for efficiency in livestock production: defining the economic objectives. J. Anim. Sci. 30:860–865. doi:10.2527/jas1970.306860x

Huang, J. Z. Ju, Q. Li, Q. Hou, C. Wang, J. Li, R. Li, L. Wang, T. Sun, S. Hang, et al. 2011. Solexa sequencing of novel and differentially expressed microRNAs in testicular and ovarian tissues in Holstein cattle. Int. J. Biol. Sci. 7:1016–1026. doi:10.7150/ijbs.7.1016

Hutt, D. M., J. M. Baltz, and J. K. Ngsee. 2005. Synaptotagmin VI and VIII and syntaxin 2 are essential for the mouse sperm acrosome reaction. J. Biol. Chem. 280:20197–20203. doi:10.1074/jbc.M412920200

Kaps, M., M. Posavi, N. Stipic, and B. Mikulic. 2000. Genetic evaluation of semen and growth traits of young simmental bulls in performance test. Agric. Conspec. Sci. 65:15–20.

Kealey, C. G., M. D. MacNeil, M. W. Tess, T. W. Geary, and R. A. Bellows. 2006. Genetic parameter estimates for scrotal circumference and semen characteristics of line 1 Hereford bulls. J. Anim. Sci. 84:283–290. doi:10.2527/2006.842283x

Keeney, S., C. N. Giroux, and N. Kleckner. 1997. Meiosis-specific DNA double-strand breaks are catalyzed by Spo11, a member of a widely conserved protein family. Cell 88:375–384. doi:10.1016/s0092-8674(00)81876-0

Khatib, H., R. L. Monson, W. Huang, R. Khatib, V. Schutzkus, H. Khateeb, and J. J. Parrish. 2010. Short communication: validation of in vitro fertility genes in a Holstein bull population. J. Dairy Sci. 93:2244–2249. doi:10.3168/jds.2009-2805

Knights, S. A., R. L. Baker, D. Gianola, and J. B. Gibb. 1984. Estimates of heritabilities and of genetic and phenotypic correlations among growth and reproductive traits in yearling Angus bulls. J. Anim. Sci. 58:887–893. doi:10.2527/jas1984.584887x

Lan, X. Y., F. Penagaricano, L. DeJung, K. A. Weigel, and H. Khatib. 2013. Short communication: a missense mutation in the PROP1 (prophet of Pit 1) gene affects male fertility and milk production traits in the US Holstein population. J. Dairy Sci. 96:1255–1257. doi:10.3168/jds.2012-6019

Lyons, A., F. Narciandi, E. Donnellan, J. Romero-Aguirregomezcorta, C. O. Farrelly, P. Lonergan, K. G. Meade, and S. Fair. 2018. Recombinant β-defensin 126 promotes bull sperm binding to bovine oviductal epithelia. Reprod. Fertil. Dev. 30:1472–1481. doi:10.1071/RD17415

Martínez-Velázquez, G., K. E. Gregory, G. L. Bennett, and L. D. Van Vleck. 2003. Genetic relationships between scrotal circumference and female reproductive traits. J. Anim. Sci. 81:395–401. doi:10.2527/2003.812395x

Mathevon, M., M. M. Buhr, and J. C. Dekkers. 1998. Environmental, management, and genetic factors affecting semen production in Holstein bulls. J. Dairy Sci. 81:3321–3330. doi:10.3168/jds.S0022-0302(98)75898-9

Meyer, K., K. Hammond, M. J. Mackinnon, and P. F. Parnell. 1991. Estimates of covariances between reproduction and growth in Australian beef cattle. J. Anim. Sci. 69:3533–3543. doi:10.2527/1991.6993533x

Mizuhashi, K., T. Chaya, T. Kanamoto, Y. Omori, and T. Furukawa. 2015. Obif, a transmembrane protein, is required for bone mineralization and spermatogenesis in mice. Plos One 10:e0133704. doi:10.1371/journal.pone.0133704

Moore, D. J., A. Onoufriadis, A. Shoemark, M. A. Simpson, P. I. zur Lage, S. C. de Castro, L. Bartoloni, G. Gallone, S. Petridi, W. J. Woollard, et al. 2013. Mutations in ZMYND10, a gene essential for proper axonemal assembly of inner and outer dynein arms in humans and flies, cause primary ciliary dyskinesia. Am. J. Hum. Genet. 93:346–356. doi:10.1016/j.ajhg.2013.07.009

NAAB. 2018. Semen production. National Association of Animal Breeders. https://www.naab-css.org/semen-sales [accessed February 6, 2019].

Nani, J. P., F. M. Rezende, and F. Penagaricano. 2019. Predicting male fertility in dairy cattle using markers with large effect and functional annotation data. BMC Genom. 20:258. doi:10.1186/s12864-019-5644-y

Nicolini, P., R. Amorín, Y. Han, and F. Penagaricano. 2018. Whole-genome scan reveals significant non-additive effects for sire conception rate in Holstein cattle. BMC Genet. 19:14. doi:10.1186/s12863-018-0600-4

Pacheco, S. E., E. A. Houseman, B. C. Christensen, C. J. Marsit, K. T. Kelsey, M. Sigman, and K. Boekelheide. 2011. Integrative DNA methylation and gene expression analyses identify DNA packaging and epigenetic regulatory genes associated with low motility sperm. Plos One 6:e20280. doi:10.1371/journal.pone.0020280

Paronetto, M. P., and C. Sette. 2010. Role of RNA-binding proteins in mammalian spermatogenesis. Int. J. Androl. 33:2–12. doi:10.1111/j.1365-2605.2009.00959.x

Penagaricano, F., K. A. Weigel, and H. Khatib. 2012. Genome-wide association study identifies candidate markers for bull fertility in Holstein dairy cattle. Anim. Genet. 43(Suppl. 1):65–71. doi:10.1111/j.1365-2052.2012.02350.x

Raidan, F. S. S., J. S. A. Tineo, M. M. de Moreaes, T. C. Escarce, A. E. M. de Araujo, M. M. de Caralho Gomes, H. T. Venture, F. L. B. Toral. 2016. Associations among growth, scrotal circumference, and visual score of beef cattle in performance tests of pasture or in feedlots. R. Bras. Zootec. 46:309–316. doi:10.1590/s1806-92902017000400006

Rezende, F. M., G. O. Dietsch, and F. Penagaricano. 2018. Genetic dissection of bull fertility in US Jersey dairy cattle. Anim. Genet. 49:393–402. doi:10.1111/age.12710

Rogers, J. C., S. L. Bird, J. E. Larson, N. Dilorenzo, C. R. Dahken, A. Dicostanzo, and G. C. Lamb. 2012. An economic evaluation of estrous synchronization and timed artificial insemination in suckled beef cows. J. Anim. Sci. 90:4055–4062. doi:10.2527/jas.2011–4836

Schreiber, M., A. Wei, A. Yuan, J. Gaut, M. Saito, and L. Salkoff. 1998. Slo3, a novel pH-sensitive K+ channel from mammalian spermatocytes. J. Biol. Chem. 273:3509–3516. doi:10.1074/jbc.273.6.3509

Senger, P. L. 2012. Pathways to pregnancy and parturition, 3rd ed. Redmond (OR): Current Conceptions, Inc.

Silva, M. R., V. B. Pedrosa, J. C. B. Silva, J. P. Eler, J. D. Guimarães, and L. G Albuquerque. 2011. Testicular traits as selection criteria for young Nellore bulls. J. Anim. Sci. 89:2061–2067. doi:10.2527/jas2012-6067

Smith, B. A., J. S. Brinks, and G. V. Richardson. 1989. Estimation of genetic parameters among breeding soundness examination components and growth traits in yearling bulls. J. Anim. Sci. 67:2892–2896. doi:10.2527/jas1989.67112892x

Suchocki, T., and J. Szyda. 2015. Genome-wide association study for semen production traits in Holstein-Friesian bulls. J. Dairy Sci. 98:5774–5780. doi:10.3168/jds.2014–8951

Taylor, J. F., B. Bean, C. E. Marshall, and J. J. Sullivan. 1985. Genetic and environmental components of semen production traits of artificial insemination Holstein bulls. J. Dairy Sci. 68:270.3–2733. doi:10.3168/jds.S0022-0302(85)81155-3

Taylor, J. F., R. D. Schnabel, and P. Stuovsky. 2018. Review: genomics of bull fertility. Animal 12(Suppl. 1):172–183. doi:10.1017/S1751731118000599

Toelle, V. D., and O. W. Robison. 1985. Estimates of genetic correlations between testicular measurements and female reproductive traits in cattle. J. Anim. Sci. 60:89–100. doi:10.2527/jas1985.60189x

USDA. 2009. Part II: reference of beef cow-calf management practices in the United States. United State Department of Agriculture National Animal Health Systems Monitoring System Beef Cow-Calf studies. Ft. Collins (CO). https://www.aphis.usda.gov/animal_health/nahms/beefcowcalf/downloads/beef0708/Beef0708_dr_PartII_1.pdf [accessed February 6, 2019].

USDA. 2010. Part IV: reference of beef cow-calf management practices in the United States. United State Department of Agriculture National Animal Health Systems Monitoring System Beef Cow-Calf studies. Ft. Collins (CO). https://www.aphis.usda.gov/animal_health/nahms/beefcowcalf/downloads/beef0708/Beef0708_dr_PartIV_1.pdf [accessed July 31, 2019].

Utt, M. D. 2016. Prediction of bull fertility. Anim. Reprod. Sci. 169:37–44. doi:10.1016/j.anireprosci.2015.12.011

Van Melis, M. H., J. P. Eler, G. J. M. Rose, J. B. S Ferraz, L. G. G. Figueredo, E. C. Mattos, H. N. Oliveira. 2010. Additive genetic relationships between scrotal circumferences, heifer pregnancy, and stayability in Nellore cattle. J. Anim. Sci. 88:3809–3813. doi:10.2527/jas.2009–2127

Vernocchi, V., M. G. Morselli, S. Varesi, S. Nonnis, E. Maffioli, A. Negri, G. Tedeschi, and G. C. Luvoni. 2014. Sperm ubiquitination in epididymal feline semen. Theriogenology 82:636–642. doi:10.1016/j.theriogenology.2014.06.002

Washington, N. L., and S. Ward. 2006. FER-1 regulates $Ca2^{+}$-mediated membrane fusion during *C. elegans* spermatogenesis. J. Cell Sci. 119(Pt 12):2552–2562. doi:10.1242/jcs.02980.

Whiston, R., E. K. Finlay, M. S. McCabe, P. Cormican, P. Flynn, A. Cromie, P. J. Hansen, A. Lyons, S. Fair, P. Lonergan, et al. 2017. A dual targeted β-defensin and exome sequencing approach to identify, validate and functionally characterise genes associated with bull fertility. Sci. Rep. 7:12287. doi:10.1038/s41598-017-12498-x

Whittier, W. D., and T. Bailey. 2009. Predicting bull fertility. Blacksburg (VA): Virginia Cooperative Extension. p. 1–3.

Yilmaz, A., M. E. Davis, and R. C. Simmen. 2004. Estimation of (co)variance components for reproductive traits in Angus beef cattle divergently selected for blood serum IGF-I concentration. J. Anim. Sci. 82:2285–2292. doi:10.2527/2004.8282285x.s

Yin, L. L., J. M. Li, Z. M. Zhou, and J. H. Sha. 2005. Identification of a novel testis-specific gene and its potential roles in testis development/spermatogenesis. Asian J. Androl. 7:127–137. doi:10.1111/j.1745-7262.2005.00041.x