FEATURE ARTICLE

Three Blind Spots in Holstein Genomics

Why the 40-Euro Chip Is Powerful - Yet Still Misses Crucial Information

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Genomic breeding values have radically accelerated Holstein breeding. Generational intervals, selection decisions, and bull markets function fundamentally differently today than they did 15 years ago. Nevertheless, the commercial SNP chip has three structural blind spots that no reset or base renewal can eliminate: mitochondrial DNA, copy number variations, and Mendelian sampling variance. Those who ignore them pay the price in the heifer barn, on the embryo market, and in mating strategies - without knowing why. An analysis of the gap between chip limitations, barn reality, and the breeding question of what an index score can actually reflect.

> ***Source note:*** *This article draws on the Bullvine article "The $45 Holstein Genomic Test: Three Blind Spots CDCB's April Reset Missed" by Andrew Hunt (The Bullvine, May 10, 2026) as its primary source, translates his arguments into the German practical context, and expands upon them with additional peer-reviewed studies on mtDNA, copy number variations, and Mendelian sampling variance. The original source is fully cited in the bibliography.*

The "Base-Change" Illusion

Before the criticism comes the acknowledgment: Genomic breeding values have truly transformed Holstein breeding. Since the USDA/AIPL introduced official U.S. genomic evaluations in January 2009 and the CDCB adopted them in 2013, genetic progress has accelerated significantly. Sire-path generation intervals fell from about seven years to less than three. Annual gains in production and health have measurably increased (García-Ruiz et al., 2016). In Germany, the introduction of Single-Step in April 2025 further refined this process methodologically (Misztal et al., 2009; Aguilar et al., 2010). Anyone who disputes this has not been paying close attention over the past 15 years.

With every base update - CDCB's April 2025 reset in the U.S., as well as the regular adjustments at vit Verden - the frame of reference shifts, however. Every breeding value in your inbox suddenly reads differently, even though not a single cow in the barn has changed. The yardstick has shifted - not the animal. For a production farm on the selection list, this is a nuisance. For a breeding operation that sells embryos, markets young bulls, or prices pregnancies based on index scores, it's a different story.

> *The number for which you paid 40 euros per heifer is a model output - not a measurement.*

And it is precisely this model that is constantly being rebuilt at a pace that no one in their own barn can control. The basic adjustments are at least visible - they show up in the evaluation. The three structural blind spots that this article is really about do not.

What the SNP chip sees - and what it doesn't

The commercial 40-euro chip (available in Germany through KuhVision/HerdScan, and in the U.S. through CDCB providers) achieves quite a lot:

- Single-nucleotide polymorphisms (SNPs) across approximately 60,000 to 80,000 markers in the core genome
- Pedigree verification and correction
- Carrier status for known genetic defects and haplotypes (BLAD, CVM, HH1–HH6, HCD, etc.)
- Inbreeding estimation via genomic relatedness
- Estimated heritability values (gZW, gRZG, gTPI) for officially evaluated traits

What it does not include:

- Mitochondrial DNA - the entire maternal genome
- Structural variation - deletions, duplications, copy number variations
- Which specific half of the genome a calf has actually inherited (Mendelian sampling)
- The individual Mendelian sampling variance for polygenic traits
- The actual performance an animal will deliver in the specific environment of a farm

> **Key point:** *What the chip doesn't see may, in the end, cost more than what it does see.*

Blind Spot 1: The Mitochondrial "Mother Lode"

The first blind spot is found in every cow - and is not visible on any commercial genomics report. It is mitochondrial DNA (mtDNA), the maternal genome, which is passed down from the cow to every daughter, granddaughter, and great-granddaughter via the egg. Without recombination, without mixing, without a paternal contribution.

Every commercial genotyping chip scans only nuclear DNA. The mtDNA is left out. The groundbreaking work by Schutz et al. (1994) demonstrated as early as the 1990s that cytoplasmic line effects account for a measurable portion of the production variance in dairy cows. Boettcher, Steverink, Beitz, Freeman, and Van Vleck (1996) confirmed these findings using larger datasets. Subsequent studies showed that mtDNA variants can influence milk yield, fat and protein content, as well as reproductive parameters (Boettcher & Gibson, 1997; Tsai et al., 2020).

The magnitude varies depending on the study, population, and trait. But the finding is consistent: there is a maternal signal that is real, heritable, and economically relevant in practice - and that cannot be structurally captured by the SNP chip.

> *You can replace the nuclear genetics in every generation. You can never get rid of the mtDNA.*

This quickly becomes relevant in practical mating decisions. Two embryo lots, the same bull, identical index scores. One donor comes from a four-times-tested maternal line with classified daughters and stable performance data. The other is a two-year-old genomic sensation with no daughter data. On paper, the two embryos are interchangeable. Genetically, they are not. Every female descendant carries her mother's mtDNA unchanged - across all future generations.

This is precisely where the often-underestimated economic value of deep cow families lies. Breeders who have been flushing in tested maternal lines for decades were not being sentimental. They were co-selecting a trait that the 40-euro test does not capture.

> **Key point:** *Anyone who prices embryos based solely on their index score is effectively giving away the maternal genome for free.*

Blind Spot 2: The CNV Phantom in the Machine

The second blind spot is called copy number variation - and it has triggered one of the best-documented breeding disasters in the modern European dairy cattle market.

SNP chips are designed to detect single-letter changes in DNA. They are not designed to detect when an entire DNA segment is deleted or duplicated during meiosis. When this happens, the flanking markers are often preserved. The chip dutifully reports "marker present." The functional gene it was actually supposed to detect - has disappeared.

In Scandinavian Red cattle populations, researchers identified the actual cause during fine-mapping of a large fertility QTL: not an SNP, but a 660-kilobase deletion on chromosome 12, which led to embryonic lethality when two carriers were mated. The deletion segregated at a frequency of 13 to 23% in Finnish, Swedish, and Danish Red subpopulations (Kadri et al., 2014). It remained undetected for so long because it was linked to markers that increased milk yield - selection simply carried it along.

> *A weakness that the chip fails to detect does not propagate any less - but rather goes undetected for years.*

The same structural mechanism is at work today in the Holstein population. Bickhart et al. (2012, with extensive follow-up studies) identified dozens of CNV regions in the Holstein genome, several of which are associated with production and health traits. Subsequent work by Liu, Bickhart, and Sonstegard (2018) as well as Capomaccio et al. (2020) significantly expanded the catalog. Hayes and Daetwyler (2019) classify structural variation as one of the key gaps in explaining the relationship between genomic models and observed phenotypes.

Added to these are the well-known Holstein haplotypes HH1 through HH6 as well as HCD, which are tracked in the vit-Verden and CDCB routines. They themselves serve as a reminder that structural and haplotype-based effects can pass unnoticed through elite pedigrees for years - until someone specifically looks for them. If a Holstein herd exhibits chronic hoof problems or a drop in fertility that does not align with the health indices, it is reasonable to suspect that part of the discrepancy lies in structural variation - which the chip simply cannot resolve.

> **Key point:** *What appears to be a "mysterious weakness" is often structural variation - methodologically invisible, but biologically real.*

Blind Spot 3: Mendelian Roulette

The third blind spot is what breeders using selection programs have been complaining about for thirty years - and for which they now have a mechanistic explanation. A calf inherits 50% from the sire and 50% from the dam. Which 50% it inherits in each case is random. For monogenic traits such as polled, red factor, kappa-casein, or known genetic defects, this is manageable. For polygenic traits such as udder composite, conformation, or fertility, it is the single largest source of individual prediction error.

Genomic models capture only a fraction of this. Even advanced methods such as single-step GBLUP improve individual Mendelian sampling predictions only incrementally (Mrode, 2014; Habier, Fernando & Dekkers, 2007). The rest is statistical noise that cannot be eliminated through modeling.

> *Two full sisters from the same elite flushing can have very different genetic outcomes. The chip cannot tell you which of the two fared better.*

This is precisely the statistical reality familiar to every breeder who has flushed a top cow twice and witnessed one daughter rise to become a sire's dam, while her full sister never made it past "GP." Daetwyler et al. (2010) formally demonstrated that genomic prediction accuracy for individual Mendelian sampling effects is subject to an asymptotic limit even under optimal conditions. The effect is all the more pronounced the lower the heritability of a trait - and that is precisely where today's economically critical fitness and health traits lie.

In practice, this hits the middle 60% of a heifer cohort the hardest. At the extremes - at the very top and the very bottom - the signals converge. There, the index sum is robust enough to support a reliable decision. In the middle, where most keep-or-slaughter decisions are actually made, the confidence intervals overlap so much that the index rank alone is no longer a valid basis for decision-making.

Re-Ranking: Why the Top Bull in April Isn't the Top Bull in December

Every genomic index is a model output. With each estimation run, the reference population grows, trait weights are reviewed, and the base population rotates according to plan. A breeder who compares an old index value with a current one is comparing it against a moving benchmark.

For young genomic bulls, the re-ranking between the first genomic estimate and daughter testing is extensively documented. Wiggans et al. (2017) showed in a review of the USDA Genomic Selection experience that a measurable proportion of the initial top-quintile bulls fall out of this quintile once daughter data become available. This is not a criticism of the method. It is an inherent characteristic of predicting complex traits from limited information. De Roos, Hayes, and Goddard (2008) formally modeled this effect and demonstrated that the persistence of genomic predictions across generations declines rapidly.

> **Key point:** *If your mating strategy only works if your three top bulls all make it to the daughter test, your strategy has a timing problem - not a genomics problem.*

What does this cost in the heifer barn?

Published rearing costs from Penn State Extension estimate the cost up to first calving in the U.S. at $2,200 to $2,600, with OMAFRA reference budgets in the same range. For Germany, comparable estimates range from 1,800 to 2,200 euros, depending on the region and system (DLG, ongoing; LfL Bavaria, 2023). Both figures are high enough to highlight the economic consequences of poor decisions.

Calculation example: A farm with 200 cows raises 60 heifers annually. If just five heifers per year are retained or culled based on a trait with low on-farm prediction reliability, that amounts to 10,000 euros per year at 2,000 euros per heifer - and 50,000 euros over five years, not including the opportunity costs of the better animals that were not retained.

For a breeding operation that markets embryos, the same math applies on the revenue side: The error rate isn't evident in the barn, but at the auction - as a bull calf that wasn't accepted or as an embryo lot that sold below value. Different columns, same logic.

> *Five wrong decisions per year on a trait that your chip in your herd cannot reliably predict add up to a five-figure loss over five years - usually silently and unnoticed.*

Three Approaches to the Blind Spot Problem

Breeders currently deal with this situation in three ways. Each works in certain situations and fails in others.

Path 1: Trust the Composite - follow the index ranking.

Fast, simple, defensible. Works acceptably at the extremes of a heifer cohort - at the very top and the very bottom. Fails in the middle 60%, where the confidence intervals overlap. Best suited for: large operations that prioritize speed over precision. The cost:

misallocation of capital at the individual animal level - more frequently than the rank suggests.

Path 2: Combine genomics, phenotype, and maternal line.

Use genomics as a filter for the bottom quartile. For the middle of the cohort, go to the barn, classify the animals, review performance data, and assess the maternal line. Slower, more judgment-intensive - but it captures precisely the mtDNA and maternal line signals that the chip is structurally unable to detect. Best suited for: pedigree-conscious operations, embryo transfer (ET) programs, and marketers who receive premium prices for verifiable maternal line depth.

Path 3: CORREL Audit - Your Own Validation.

A one-time 30-day effort: Compare genomics values from the last two years with the actual classification and 305-day values for the same animals. Calculate a correlation for each trait. The results show, trait by trait, which indices you can trust in your herd - and which you cannot.

The 30-Day Audit: Path 3 in Detail

The greatest leverage for every breeder lies in data that is already available: MLP reports, classification data, and original genomic profiles. From these three sources, a farm-specific validation overview can be created in just a few hours.

Procedure:

- Export genomics reports from the last two years at the trait level (not just index totals)
- Combine first-lactation classification scores and 305-day production records for the same animals
- In a table, create one row per animal and one column per trait for “predicted” and “observed”
- Run the Excel CORREL function across all animals for each trait

The results can be interpreted as a practical guideline for your operation - not as a formal statistical cutoff:

- CORREL ≥ 0.40: strong signal. The chip reliably predicts this trait in your herd. Give it full weight.
- CORREL 0.25 to 0.40: marginal signal. Use as a tiebreaker; supplement with phenotype and maternal line.
- CORREL ≤ 0.25: weak signal. Practically no better than guessing. Remove from the selection logic; use family data instead.

> *Path 3 does not replace Path 1 or 2. It tells you which path to take for which trait.*

Important: The thresholds are not statistically guaranteed limits - they are operational heuristics. In borderline cases, it’s worth consulting with a breeding advisor or vit Verden. But

the basic principle remains: If you don't compare your own data, you won't know whether the index rank is meaningful in your own herd.

Where Genomics Wins Hands-Down

Criticism of the blind spots must not obscure the areas where genomics has clear, hard-to-beat strengths. There, it should be used consistently, without hedging:

- Pedigree verification - faster, cheaper, and more accurate than any previous pedigree audit (Heaton et al., 2014)
- Carrier status for known genetic defects - BLAD, CVM, DUMPS, BY, Mulefoot, Cholesterol Deficiency
- Haplotype management – HH1 through HH6, JH1, HCD, and similar constellations (VanRaden et al., 2011; Cole et al., 2016)
- Inbreeding control via genomic relatedness - significantly more precise than pedigree-based estimates (Doekes et al., 2018)
- Selection at the extremes of the cohort - safe at the top, safe at the bottom

Caution is warranted when indices are applied to low-heritability traits, individual linear values, or maternally inherited characteristics. Genomics is a tool, not an oracle. Those who use it as a tool win. Those who treat it as an oracle pay the price.

> **Key message:** *Use genomics aggressively where it is superior. Be more cautious where it is structurally limited.*

Seven practical implications for German Holstein breeders

Concrete implications for German breeding practice can be derived from the relationships described above - regardless of farm size:

1. **Do not abandon maternal line depth.**

   Those who breed for shows, auctions, or the embryo market are marketing a value that the index score does not reflect. The mtDNA of deep maternal lines is a real economic factor. Evaluate cow families separately from the index score.

2. **Design your young bull strategy around re-ranking.**

   Plan matings so that they will still be effective even if the order of your top three bulls changes by the next evaluation. Spreading your matings across multiple tested and genomic bulls is better than focusing on a single top sire.

3. **Combine genomic, classification, and MLP data.**

   If you have all three data sources for the same animals, you have the foundation for the 30-day audit. The effort required: one afternoon in Excel. The benefit: trait-specific clarity regarding the reliability of your own predictions.

4. **Consider structural variation when dealing with chronic problems.**

   Persistent hoof problems, fertility declines, or udder health drift that do not align with health indices are candidates for structural or maternal effects. More genotyping won't solve this. Other data - from hoof trimmers, veterinarians, and family records - is needed.

5. **Do not price embryos based solely on index scores.**

   When two embryos have identical index scores, the maternal line depth determines the actual market value. Those who fail to make this distinction sell assets below their value - or buy them too cheaply.

6. **Do not eliminate MLP and classification for cost reasons.**

   Every genomic prediction depends on phenotypic data collected on commercial farms. Anyone who drops out of the data collection cycle will, in the medium term, undermine the very tool they are paying for.

7. **Aggressively deploy genomics where it is superior.**

   Pedigree, genetic defects, haplotypes, and inbreeding control: these are areas where genomics is superior, and it should be used here without hesitation. For low-heritability individual traits, this includes the barn assessment and the maternal line.

Conclusion: The cow family remains

Genomic breeding values are one of the most effective tools cattle breeding has ever had. No one wants to fall behind. But every tool has structural limitations - and no reset, no baseline adjustment, and no single-step procedure can change that. Mitochondrial DNA, copy number variations, and Mendelian sampling variance remain beyond the reach of the commercial chip.

The breeders who will be leading the pack in five years aren't the ones with the densest chip or the most spectacular flushing program. They are the ones who honestly verify which traits their genomic tool actually predicts for their genetics - and which it does not. This knowledge isn't found on the test report. It emerges in an Excel spreadsheet that the farm builds itself, alongside a pedigree it has long curated.

> *The cow family is not a matter of nostalgia. It is the information that the 40-euro chip does not provide.*

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