Information provision and network externalities: the impact of genomic testing on the dairy industry

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Link to the latest version:

`https://victorefunes.github.io/files/job-market-paper.pdf`

Abstract

We use differences-in-differences with a matched control group method to estimate the long-term impacts of genomic selection for dairy cattle genetics in the American market. Genomic selection is an application of big data that uses the entire genome of an animal to test for the presence of a set of traits. Unlike pre-existing technologies that require several years of data from a bull's daughters, an animal can be tested as soon as it is born, allowing breeders to identify the "best" animals much faster. Using a data set of all bulls marketed in the US from 2000 to 2020, we find that genomic selection significantly increased genetic gains for all measured traits, particularly milk production, protein, and fat yields, but also increased levels of inbreeding depression, a reduction in the performance of animals whose parents have a high degree of relatedness, as a consequence of genetics companies breeding more animals from established lines to respond to an increased demand towards such lines. Our estimation shows that the increased inbreeding rate of American bulls caused a loss of between 3.6 to 6.7 billion dollars to the entire industry from 2011 to 2019. Solving this externality will require either a mechanism to internalize the harmful effects, such as paying a much higher price for more inbred sires, or a collective action mechanism to select which lines will be bred in the next generation.

1 Introduction

Technological innovations, defined as the set of rules or instructions to combine inputs to produce outputs (Romer, 1990), play a significant role in economic analysis as a factor that drives economic growth. This article investigates the externalities of a new technology that boosts genetic productivity in the dairy industry: genomic selection for bulls. This new technology increased the productivity of the dairy industry as soon as it was introduced; however, since dairy genetics has a public good aspect related to the existence of a common pool of genes that animal breeders draw from, genomic selection has a negative aspect that leads to billions of dollars of losses to the dairy industry.

Animal breeders and genetics companies can identify which bulls (sires) have the highest productivity and transmit it to their offspring, thus increasing the productivity of the next generation of animals. But it also had a negative consequence: increased inbreeding rates due to breeders mating more closely related animals to undercut each other and ensure a higher market share. Inbreeding is a negative externality inflicted by breeders on themselves by pursuing short-term gains in genetic performance at the cost of reducing the diversity of the gene pool.

However, genomic selection was expected to help breeders “discover" new potentially high-performing sires independent of their ancestry. Still, something entirely different happened: genetic companies genotyped almost all of their bulls and released them into the market in 2010, thus expanding the number of sires more than threefold. Due to a much more extensive choice set, the relative value of a line (family) of animals increased as it reduces the quality uncertainty of a new bull whose performance is still unknown. We can think of this as “brand loyalty” towards famous bull families, and the ultimate consequence of this process is an increasing inbreeding rate of all bulls from 2010 onwards.

Inbreeding leads to a higher prevalence of deleterious genetic conditions that inflict a very high cost on the dairy industry. This is not the first time that intense selective breeding has pushed the dairy industry to a bad outcome, Adams et al. (2016), for example, shows that a mutation in a single chromosome that could be traced to a very famous sire called “Pawnee Farm Arlinda Chief” led to more than 500,000 spontaneous abortions and nearly 420 million dollars in losses because of a deleterious mutation in a gene that was passed to his offspring. Genomic selection could have helped breeders find more productive genetic resources outside their gene pool. However, it had the opposite effect since the incentive mechanism that breeders face makes

them compete to supply the most productive bulls, and the best way to breed high-productivity bulls is to pull them from existing famous lines.

Is it the case that breeders are mating more heavily on famous lines? If so, we should see higher inbreeding rates in animals from those well-established families than in other less famous lines. Genomic selection has the opposite effect; it helps breeders identify a highly productive bull when the animal is born so it can be marketed as soon as it reaches sexual maturity (one year old versus four to five years with traditional selection.) Genomic selection increased the genetic gains per year due to shortened generation intervals but also led to increased competition in the animal genetics industry; firms that supply bull genetics are under higher pressure to offer more "efficient" bulls. The most common way to breed a highly productive sire is through linebreeding, which consists of mating two related animals that are descendants of a famous sire. A consequence of the pervasive use of line breeding, the effective population size[1] of bulls is at a historic low ranging from 43 to 66 animals. Now all animals are, on average, more related to each other due to the pervasive use of linebreeding (Cole, 2019).

Inbreeding depression is a negative externality that genetics companies inflict upon each other because of breeding more closely related bulls. According to the Holstein Association of the United States, each additional percent point of inbreeding rate leads to a $23 revenue loss per cow. We estimate that increased inbreeding has led to around one billion dollars of losses for the industry annually since 2012. The most important policy implication of this article is that we can use one or several solutions from the economics "toolbox" to fix the issue, such as Pigovian taxes on breeders who supply highly inbred bulls or governmental regulations to keep inbreeding levels below a certain threshold.

This article fills a critical gap in the literature; there are no other papers that analyze inbreeding as a **network externality** (Liebowitz and Margolis, 1994), (Katz and Shapiro, 1985), where companies enjoy the benefits (faster genetic gains) and suffer the costs (decreased fertility caused by the increased frequency of deleterious mutations) from changes in the size of their associated network. In this case, the pedigree (relatedness) is the force that binds all the nodes in the network, where two animals are part of the same network (line) if they descend from the same ancestor. Similarly, all articles that analyze the market for cattle genetics, such as Kerr (1984), Melton et al. (1994), Richards and Jeffrey (1996) or Schroeder et al. (1992) study cattle as a closed system and their objective was to determine what are the implicit prices of each genetic

[1]The number of individuals that an idealized population would need to have for inbreeding rates to be the same as in the actual population.

trait using hedonic methods. These early papers then focus on the decisions taken by individual breeders or dairy farmers without paying attention to their broader impact.

The paper is structured as follows: Section 2 presents the relevant background details and a detailed causal mechanism to explain why introducing genomic selection led to higher inbreeding rates in subsequent generations of dairy bulls. Section 3 explains the data sources and shows how some descriptive statistics can help us delineate the relationship between our observed variables. Section 4 outlines the identification strategy and methodological framework used to identify the causal effect of genomic selection on inbreeding rates. Finally, in section 6, we explain the results and discuss their implications for the future of the American dairy industry.

2 Conceptual Framework

Selective breeding has been practiced since prehistoric times; however, the discovery of inheritance mechanisms and genetics in the 19^{th} and 20^{th} centuries allowed breeders to identify the link between desired traits in animals and their inheritance mechanisms (Lush, 1937). From an economic standpoint, an animal breeder supplies a product (animal genetics) with an inseparable bundle of traits embodied in a single animal. To produce such an animal, the breeder combines a set of inputs that can be split into two groups: a series of inputs exogenously supplied by the producer (labor, feed, capital, veterinary services) and a series of inputs embodied in the animal (traits.) Those inputs are combined on a production function subject to the prices of exogenous inputs (observed) and genetic inputs, whose prices are implicit but can be estimated based on the price paid for a bull's genetics using the hedonic approach (Schroeder et al., 1992, Sy et al., 1997).

Melton et al. (1994, 1979) add an additional element: breeders are paid based on the (positive) deviation of the trait levels of their bulls relative to the population average. Breeders compete to maximize profits; to do so, they will attempt to release bulls into the market with the highest possible trait values. Dairy farmers demand genetics to improve their productivity in the next period by choosing the bulls with the best characteristics. However, the traits that constitute an animal are marketed as a "bundle" of traits embedded in each animal. Dairy farmers are only interested in the productivity of their cows because their profits depend on it; they are not directly interested in how inbred their cows are; therefore, inbreeding rates cannot be priced directly as an additional cost to dairy farmers.

Inbreeding is the mating of individuals more closely related to each other than the average for the population(Bourdon, 2000, p. 337). Inbreeding has two consequences: first, it increases the likelihood that an animal's offspring will perform similarly to its ancestors; for example, if a particularly tall bull is mated with a related cow, its offspring will likely be taller than average because the genes that carry such trait are likely to be present in both parents. Second, it favors the expression of deleterious recessive alleles; since an animal has two copies of a recessive gene from each parent, it is more likely to express any such traits than an outbred animal.

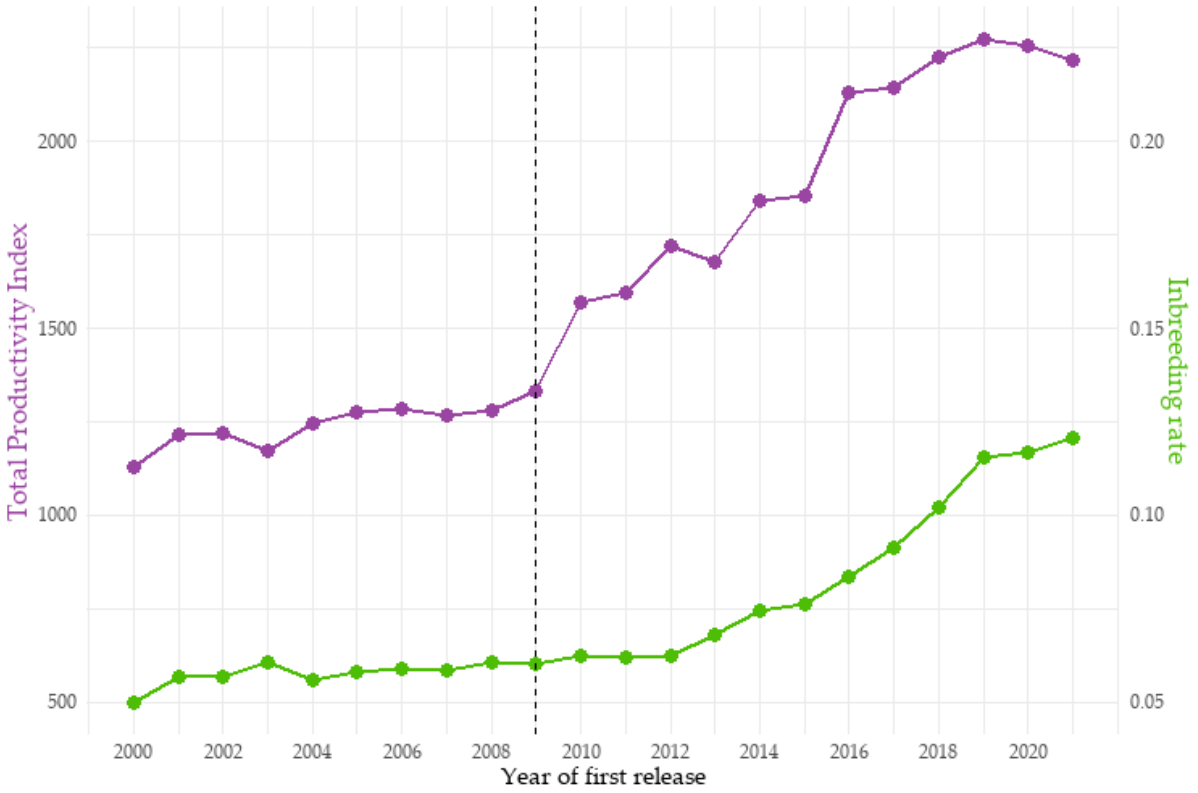

Figure 1: Average Total Performance Index and Inbreeding rates of Holstein bulls
Source: NAAB

Figure 1 shows the close link between the **Total Performance Index** (TPI), a weighted average of a series of genetic traits, and the average inbreeding rate. Genomic selection led to an immediate jump in the productivity index but to a lagged response in inbreeding rates because of the gestation length and time to reach maturity that originate the cattle cycle (Rosen et al., 1994), breeding decisions in year t, leading to a birth of a new calf in $t+1$, that will be released into the market in year $t+2$.

The increment in the rate of change in hereditary traits caused by genomic selection can be decomposed into a set of components: heritability, selection intensity, and generation interval (Kerr, 1984). Equation 1 is called the "breeder's equation":

$$\text{genetic improvement} = \frac{\text{heritability} \times \text{selection intensity} \times \text{trait variance}}{\text{generation interval}} \quad (1)$$

Heritability is a population parameter specific to each trait that measures the strength of the relationship between the genetic traits of parents and their offspring, and so is the standard deviation of the observed trait values. **Selection intensity** is the differential in average trait values between selected animals and the general population at a given time. Selection intensity measures how “choosy" breeders are in deciding which individuals are selected; more intensity of selection means that only the best animals (according to some criterion) are selected to have offspring. If animals were randomly selected, those magnitudes would not differ. The generation interval is the time required to replace one generation with the next: the shorter the interval, the faster the genetic change. Genetic improvement is driven by two forces: selection intensity and generation interval. An increase in selectivity or a decrease in the generation interval increases genetic gains per generation.

Before 2009, it took one to five years for a bull to father a large enough number of daughters to test them for the presence of genetic traits such as milk, protein, or fat yields. With genomic testing, an animal can be tested as soon as it is born for the presence of specific traits in its genome. Consequently, the generation interval, the average age of parents when offspring are born (Wiggans and Carrillo, 2022), has decreased from around five and a half years to less than two years, close to the biological minimum of one year.

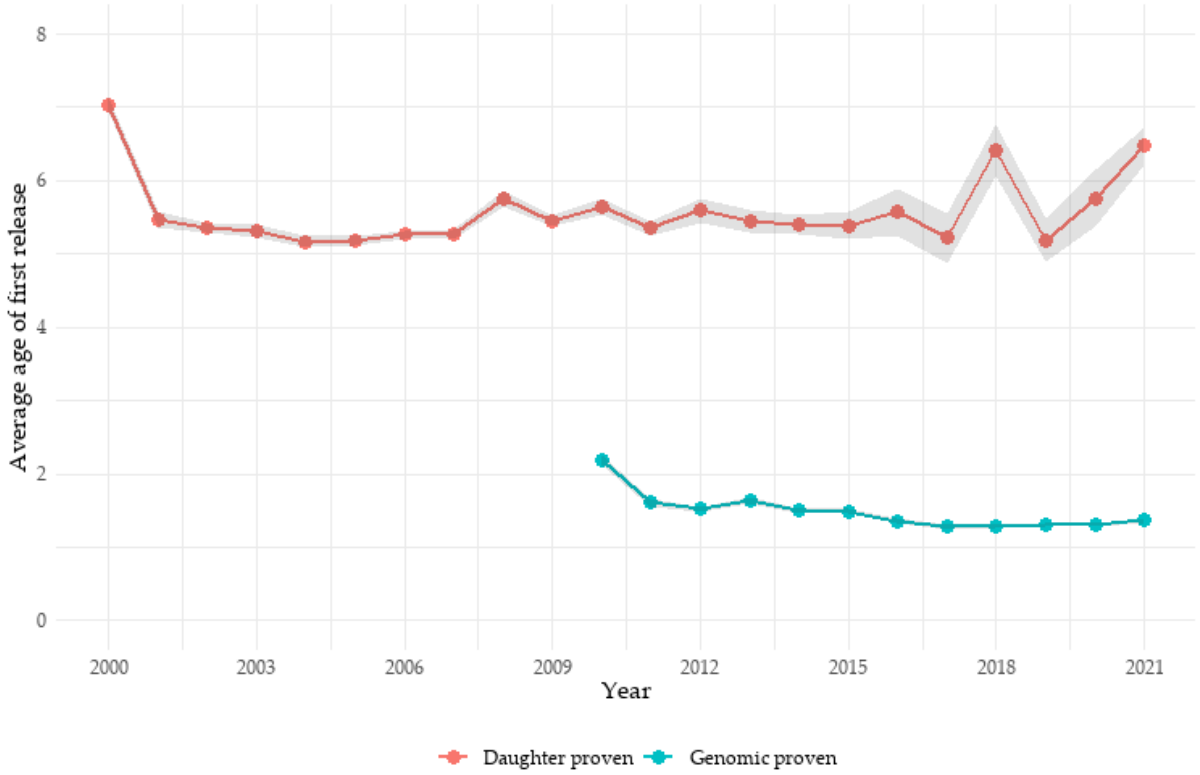

Figure 2: Average age at first proof for Holstein bulls
Source: NAAB

The most important consequence of a shorter generation interval is an increase in genetic progress. Figure 2 also shows that the number of daughter-proven bulls has steadily decreased, and the wider confidence intervals for the average age reflect that. A key element of our analysis is the existence of what we call “supersires", high-prestige bulls who became the founders of lines (families). The more genetics companies breed the same lines, the more newborn bulls will be related to a specific ancestor. We define a bull as a supersire in terms of the size of its offspring; since we have records of all male descendants from each sire that have been released on the dairy genetic market, we can then identify which bulls have had the largest offspring; more precisely, a bull born between 1997 and 2007 is a supersire if the size of his offspring is on the 95^{th} percentile of the offspring distribution.

We test the hypothesis that genomic selection caused higher inbreeding rates by constructing a counterfactual line of bulls that possessed similar traits but were less popular at the introduction of genomics. We identify their descendants and respective inbreeding rates; they increased disproportionately more for the descendants of supersires. A likely explanation for this process is that genetics companies did not invest in the most productive bulls but instead in the most famous lines in an attempt to gain market share. The more companies breed into those lines,

the higher the inbreeding rate of the next generation of bulls. This leads to a deterioration of the expected traits of future generations of bulls and, thus, to significant losses for the dairy industry in terms of reduced productivity.

3 Data Description

Improved genetics via the selection of bulls (sires) and cows (dams) is a crucial driver for productivity improvements in the dairy industry. Milk production in the United States has tripled in the last forty years, and over half of this increase is solely due to genetics (`https://uscdcb.com/impact/`). The United States is also the world's largest exporter of bovine genetics, with a share of 46.4% of the total value of exports in 2019. Every improvement in genetics in the United States will be quickly transmitted to the rest of the world dairy industry.

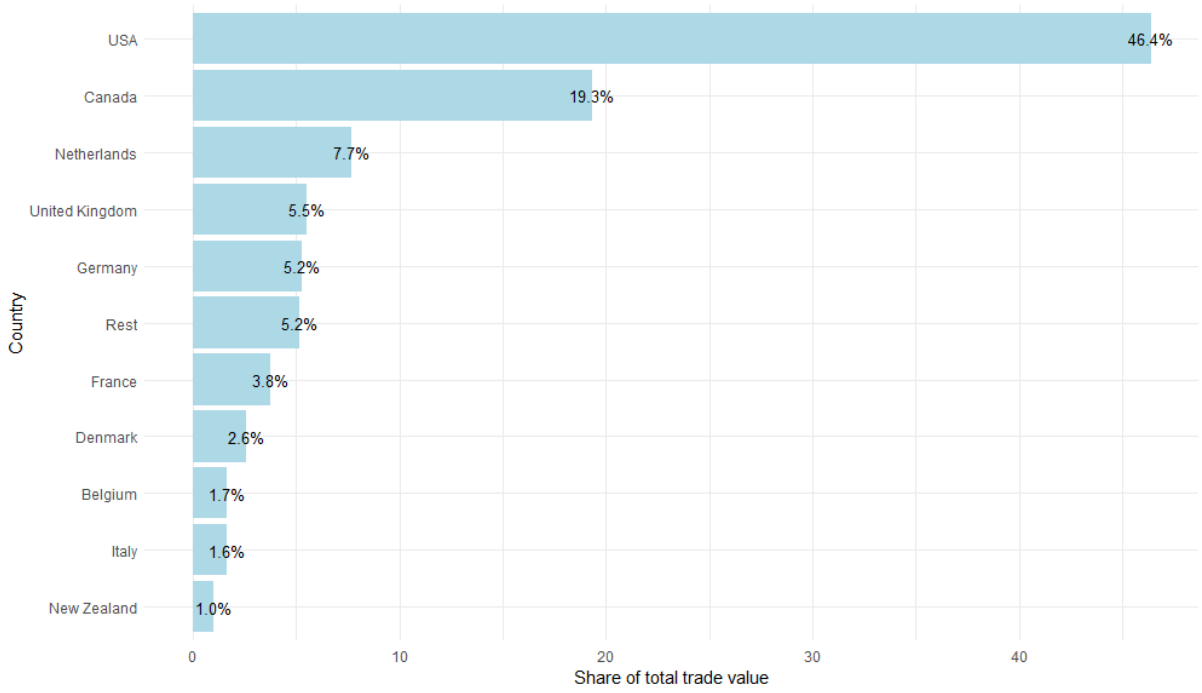

Figure 3: Share of trade Value of Bovine Semen exports by country (2019)
Source: UN Comtrade database

Traditional selection techniques relied on information from pedigrees and observed trait values to assess an animal's fitness. Genomic selection uses data from an animal's genome to accurately predict breeding values, particularly in cases where traits are expressed only in females. In such case, it was necessary to measure the traits of a large number of daughters from the same bull; for that to happen, these cows must have reached the age of sexual maturity

(one year); hence, the entire process could take three to four years until enough information was gathered.

Instead, genomic selection uses the bull's DNA to detect gene combinations significantly different from the expected frequency if they were randomly associated. This process detects the presence of certain genetic traits that are highly correlated with specific gene combinations. It can be undertaken as soon as the calf is born, so by the time the animal has reached sexual maturity (one year old), it can be marketed immediately.

Figure 5 shows that genomic selection increased the number of animals released into the market since there is intense competition for supplying the most productive bulls; the relative value of a line is now higher than ever. Since dairy farmers have a more extensive choice set of bulls to buy genetics from, lines act similar to "brands," as they reduce the quality uncertainty from a product. Finally, this "loyalty" to established lines leads to breeders supplying more bulls from such families than from lesser-known lines that have similar or better traits.

Brand loyalty arises in this context due to the nature of animal genetics, dairy farmers demand a particular set of genetic traits related to the productivity and health of their cows, but they cannot be sold separately; they have to be acquired as a "bundle" at a single price (Ladd and Martin, 1976, Melton et al., 1994) that is a linear function of the entire set of genetic traits, which makes a comparison between any two or more animals extremely difficult. This implies that there are very high search costs to farmers when deciding which animal to buy genetics from, Stigler (1961) and Farley (1964) argue that it is reasonable for an economic agent to seek further information on a single purchase as long as the expected reduction in price per unit as a result of additional search times the quantity purchased is equal to the marginal cost from searching for lower prices (or higher qualities.)

Figure 4: Total number of new bulls released in the market **Source**: NAAB

Genomic selection increased the number of new bulls released into the market, as shown in Figure 4. Consequently, search costs for dairy farmers increased as well, thus promoting greater brand loyalty for "superstar" lines, that is, bulls descended from a notorious ancestor (e.g., bulls that fathered many daughters). The ultimate consequence of this process is an increase in inbreeding for the entire population of dairy bulls; the more related bulls are, the more likely their offspring is to preserve their genetic traits. Higher inbreeding levels lead to a higher likelihood of observing **inbreeding depression**, a decrease in the performance of inbred animals due to lower fertility, higher incidence of rare diseases, and reduced longevity.

The National Dairy Herd Improvement Program (NDHIP) collects, manages, and analyzes data from American dairy cattle (Hutchins and Hueth, 2023). The information it collects assesses the industry's productivity and estimates the paternal contribution to these traits. The raw data collected from farms is used to estimate a statistical model to predict the genetic contribution of an animal as a sire (or dam) after controlling for environmental variables such as herd, age, pedigree, and season effects. The predicted values for each trait from this model are commonly called the **predicted transmitting ability** (PTA) of a trait.

Animal breeders and genetics companies compete to offer an animal with the most significant possible improvement over the cohort's average. As a consequence, firms will try to produce

bulls with the highest productivity traits; similarly, this will lead to higher rates of inbreeding since the best way to ensure high productivity is through linebreeding, that is, by building a pedigree that has a relatively stable degree of relationship relative to a supersire. We call “large" companies those with the most significant average number of bulls across the entire 2000-2020 period in the North American genetics market. Similarly, a firm is called “preexisting” if it participated in the bull genetics markets before 2009; otherwise, they are considered “new."

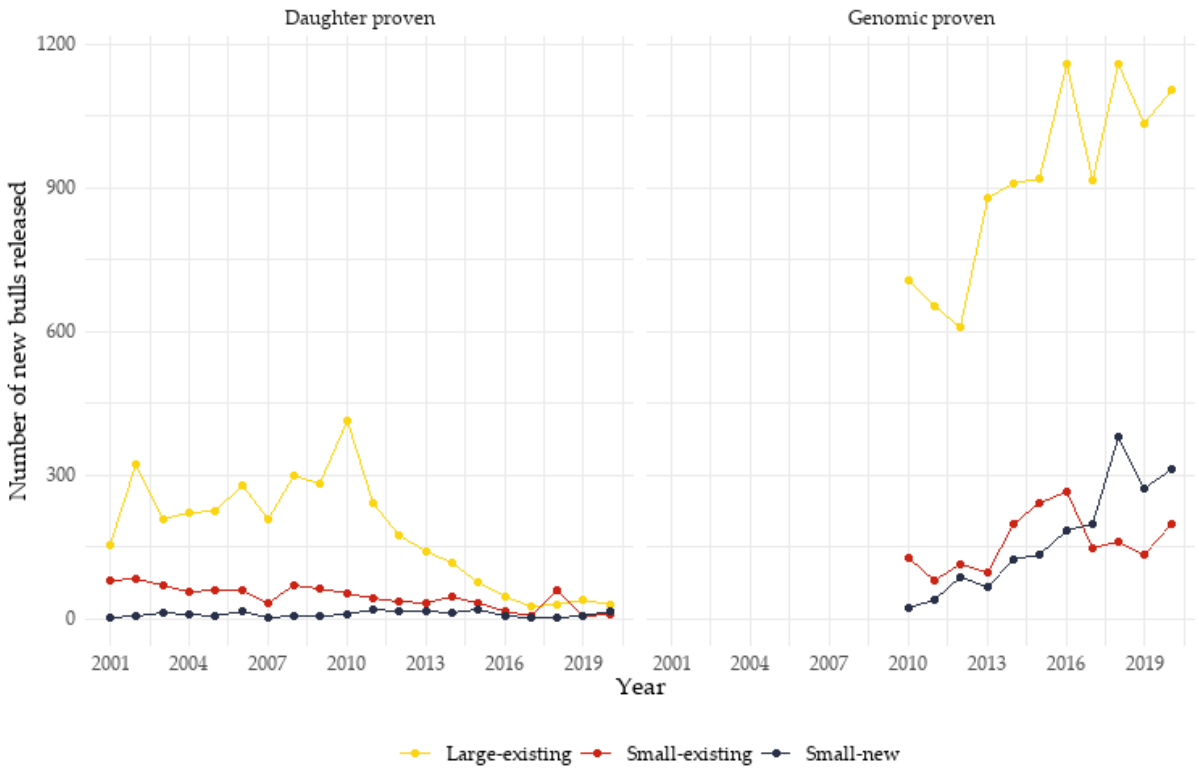

Figure 5: Number of genotyped bulls by firm size
Source: NAAB

Genomic testing allows genetic companies to detect suitable bulls earlier; thus, they can release those animals into the market as soon as they reach sexual maturity. Figure 5 shows the total number of new bulls released in the market by year and type of firm.

In the cattle genetics industry, genomic testing has been adopted remarkably fast. Figure 6 shows how fast genomic testing was adopted in our sample. The high starting costs for genotyping help explain this since large companies were the ones that could initially afford it. Still, more companies have genotyped their calves since genomic selection was released. Larger firms genotyped a more significant percentage of their newborn calves in 2010, and the share has grown steadily, while smaller firms followed them at a slower rate.

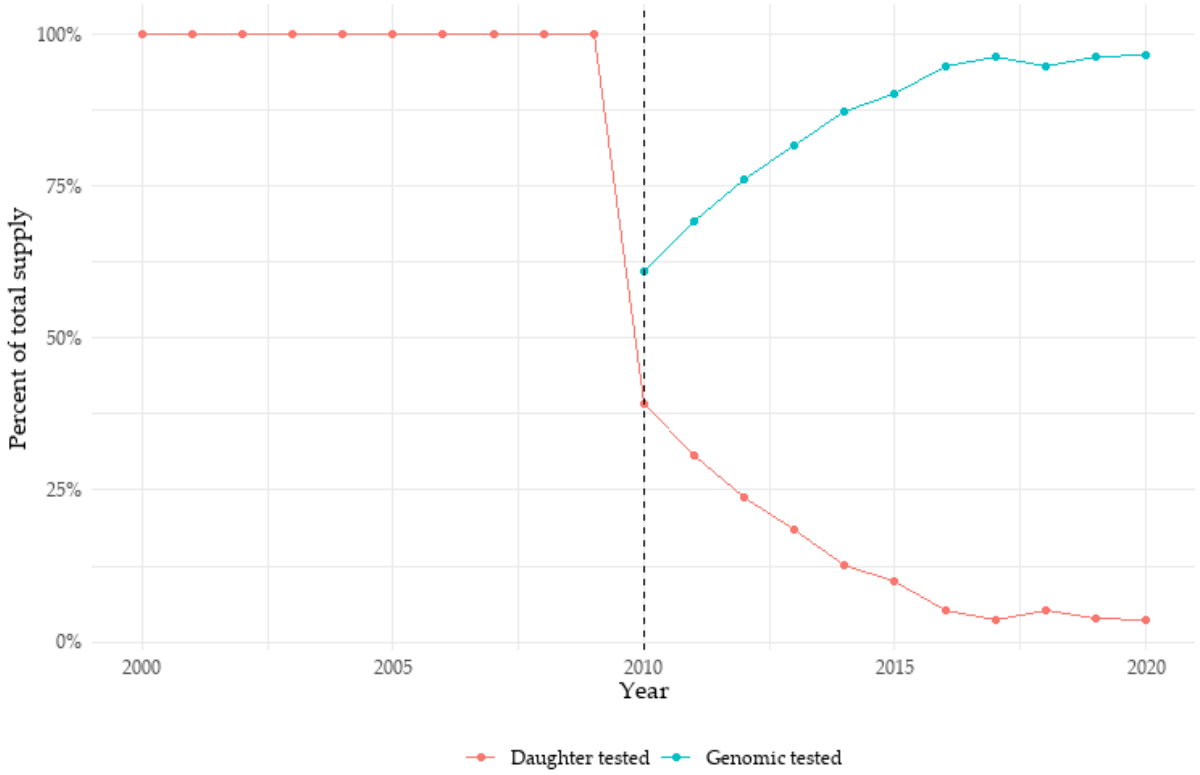

Figure 6: Share of each status of bull by year

Starting in 2010, large firms started genotyping their bulls, and consequently, the number of new bulls increased threefold in that year, when all bulls aged 1 to 4 were released simultaneously. The staggered adoption of genomic selection has further consolidated the market and led to more competition for superstar lines. However, the more inbred selected animals are, by definition, also increases the inbreeding of an average animal in any cohort, thus making inbreeding depression more likely within a herd. Figure 5 shows the total number of new bulls released in the market by year and type of firm. We call “large" companies with the most significant share of the North American genetics market. Similarly, a firm is called “preexisting" if it participated in the market of bull genetics before 2009; otherwise, they are considered “new."

4 Empirical Framework

We want to test the hypothesis that the introduction of genomic selection led to higher inbreeding rates because of increased demand for well-known pedigrees fueled by an increment in the productivity of dairy cattle. We want to know the causal effects of introducing this technology; ideally, we need a treatment group that was affected by genomic selection and a treatment group that was not.

We must identify families whose founders had similar trait levels as superstars but had less “momentum"; that is, a smaller offspring. This is a comparable group because breeders could have invested in them but did not because they were not demanded as highly as superstars. We can track these lines over time. If their inbreeding rates did not differ substantially from those of superstars, we could argue that genomic selection allowed breeders to identify new, less-inbred bulls independent of their ancestry.

Figure 6 shows that the pace of adoption was very high from the beginning; this makes it unlikely to use non-genomic proven bulls as a counterfactual, mainly because we don’t know why breeders choose not to genotype certain bulls after 2009. Similarly, we have very few foreign-born bulls to use as a control group; we also don’t know the selection criteria to import genetics from those animals. Both alternative control groups suffer from an additional issue: they will have a significantly smaller offspring, thus leaving us with little statistical power to detect any relevant treatment effect. To overcome these issues, we use propensity score matching (Smith and Todd, 2005) to define a control group that resembles the treatment in all observable characteristics (except for our outcome: inbreeding rates.)

We define a **line** as the set of direct (male) descendants from a particular bull, which, in turn, can be described in terms of his number of male descendants; a bull is a superstar if his number of sons is in the 95th percentile of the distribution of offspring. Breeders are under high competitive pressure to supply bulls with the highest PTAs. However, the best way to do so is by linebreeding a family of bulls so that the relative distance to an ancestor remains constant across generations. Steyn et al. (2022) and Steyn et al. (2023) show that younger sires capture a more significant share of present-day population genetic variation; hence, genotypes of older sires can be discarded from (genomic) evaluations. As generations begin to accumulate, the contribution of a particular sire decreases (on average) exponentially, so we can disregard the impact of distant ancestors.

In the sample, we include all animals whose sires were born between 1998 and 2004, and we call those sires the “founders" of each line. Our definition depends critically on when the founders of such lines were born; if we choose an earlier date, we cannot find a statistically significant effect due to the high relatedness of all bulls. The reason behind this is that bulls are advertised only with their sire and grandsire; any ancestry beyond that is not taken into account by dairy farmers. If we choose a later date, both treatment and control groups will have little difference in inbreeding rates because there is not enough time for the control group lines to develop. Figure 9 in the appendix summarizes the sample selection mechanism; the treatment

and control groups are comprised of the descendants of two groups: supersires (treatment) and matched sires (control).

After identifying the sires, we can recursively build their pedigrees by matching bulls with their sires until we find the latest animal in the sample. We want to measure the causal effect of introducing genomic testing in 2009 on the average inbreeding in extant lines; the first problem we must tackle is the nature of a valid comparison group. The introduction of genomic testing caused animal breeders to supply more animals from well-known lines, that is, descended from superstar sires; our control group must be the lines descended from other non-superstar sires.

First, we need to match the founders of such lines with other sires that did not father as many sons but had comparable genetic traits. Second, we find all descendants from both groups and compare the evolution of their inbreeding rates through time. The idea behind this procedure is that when those bulls were in the market, any reasonably similar sire could have been chosen but was not, and thus, can be used to construct a counterfactual line.

For every family, we must construct a counterfactual family with new bulls being born every year, each with an inbreeding rate that we can average across cohorts. Then, the **treatment group** are all lines of animals comprised of direct descendants of superstar sires. Similarly, the **control group** includes descendants from comparable animals matched to superstar bulls by their genetic trait levels.

Our next objective is to identify the counterfactual lines by matching superstar sires with alternative lines. To do so, we use a propensity score matching estimator, let $p(X) = \Pr(T_j = 1|\mathbf{X})$ where T is a dummy equal to 1 if sire j's offspring is on the 95^{th} percentile of the distribution and X_j be a vector comprised of the following:

1. Predicted Transmitting Ability for production traits (milk, fat, and protein), health traits (mastitis, metritis, productive life), and reproductive traits (daughter pregnancy rate, effective calving, gestation length).

2. Dummies for the absence of certain recessive genetic disorders (Holstein Foundation, 2021) such as *brachyspina*, cholesterol deficiency, and mule foot.

3. Dummies for the presence of genes associated with specific qualitative traits (polled, red coat color, complex vertebral malformation).

4. Dummies for the presence of haplotypes (specific sequences of DNA at different locations in the chromosome) that affect fertility (Holstein Foundation, 2018) when present in

homozygous form (likely to be expressed when two carrier animals are mated).

We then estimate the propensity score (Cameron and Trivedi, 2005, p. 873), the probability of being a superstar given the level of PTAs and the presence of the entire set of variables listed above. The validity of our method relies on a series of assumptions (Heckman et al., 1997):

Assumption 1 (Stable Unit Treatment Value Assumption): $(F^{treatment}, F^{control}) \perp\!\!\!\perp T/\mathbf{X}$.

The average inbreeding rates of any two lines do not vary with the treatment assigned to other lines. For each unit, there are no different forms of each treatment level (Imbens and Rubin, 2015), conditional on a set of covariates. This assumption implies that conditional on $\mathbf{X}$, the distribution of inbreeding rates for both groups will be the same for all founders, but it must differ for their offspring. Figure 10, in the Appendix, shows the extent to which the inbreeding rates distribution of our matched sample resembles that of the treatment group. Unlike unmatched sires, their inbreeding distribution has a similar mean and variance conditional on the set of genetic traits.

Assumption 2: $0 < \Pr\left(T_j = 1 \middle| \mathbf{X}\right) < 1$ for all $\mathbf{X}$

This assumption implies that we can define a propensity score for every value in the treatment group. Assumptions 1 and 2 together are the "strong ignorability" assumption in Rosenbaum and Rubin (1983), and they are necessary in conjunction to identify causal parameters. The set of matching variables above exhausts the entire set of possible selection criteria for breeders; no unobserved variable can influence mating decisions. Figure 11 shows the degree of overlapping between superstars and alternative sires; since there is a considerable difference in the size of the groups, superstars make up only a tiny fraction of bulls; however, the overlapping is good in the range $0.05 - 0.50$, but there exist a few superstars that had little close matches in the sample.

A central part of our argument is that treatment and control groups should have similar inbreeding levels before genomic selection. To test that assumption, in Table 1, we calculate the difference in averages of treatment and control groups for inbreeding rates and a few selected traits. There is no statistically significant difference for most traits between treatment and control groups, so our matching method ensures that the difference between both groups is due to the "prestige" of superstar lines because the bulls in the treatment group had a larger offspring than those in the control group. Each year these bulls will have a new set of descendants being born, each with its inbreeding rate and genetic traits; if our hypothesis holds, then the inbreeding rates of the descendants of supersires should be significantly higher than those of non-superstar descendants.

We then estimate the differential impact of genomic selection (our treatment) on the two types of lines, those from supersires and those from alternative sires. Equation **??** includes leads and lags of the treatment to check to which degree a pre-treatment balance exists in both groups. This is not a test for parallel trends on our differences-in-differences specification (Cunningham, 2021), but it helps us ascertain how comparable both groups are. Table 1 shows that there is no significant pre-treatment difference in both groups and that coefficients from 2010-2011 are not statistically significant either, which coincides with the values from Figure 1 where there is a significant increment in inbreeding rates starting in 2012.

We observed the average inbreeding rates of the treatment and matched control groups before and after introducing genomic selection. If we divide the 2005-2017 period into two groups, pre-intervention (2005-2009) and post-intervention (2010-2017), denoted as 1 and 2 and let T_j denotes whether a bull from line j belongs to treatment ($T_j = 1$) or control ($T_j = 0$) groups, then:

$$F_{jt} = T_j F_{jt}^{treat} + (1 - T_j) F_{jt}^{control}$$

Where $j = 1, \ldots, J$ are the lines, each comprised of n_j bulls such that $i = 1, \ldots, n_j$. Our causal estimand of interest is the Average Treatment on the Treated (ATT) in the post-intervention period:

$$ATT_{post} = E\left[F_{post}^{treat} - F_{post}^{control} \middle| T_j = 1\right]$$

This quantity measures the average causal effect on the treated lines in the post-treatment period (Roth et al., 2023). To correctly identify causal effects, a series of assumptions must hold (Sun and Abraham, 2021):

Assumption 3 (Parallel trends): $E\left[F_{post}^{control} - F_{pre}^{control} \middle| T_j = 1\right] = E\left[F_{post}^{control} - F_{pre}^{control} \middle| T_j = 0\right]$

We assume that the average inbreeding rates for both groups would have evolved in parallel without introducing genomic selection. This assumption is not testable because we cannot observe untreated inbreeding rates after 2009, but it constrains our model specification to a Two-Way Fixed Effects specification we call Model (1):

$$F_{ikt} = treat_i + (treat_i * post_t)\beta + post_t + \alpha_k + \varepsilon_{ijt} \qquad \text{(Model (1))}$$

Where F_{ikt} is the inbreeding rate of bull i born from a line that belongs to either the treatment

or control group born in year t, supplied by firm k, β_t is a year of birth fixed effect, and α_j is a firm fixed effect.

Assumption 4 (No anticipatory effects): $F_{pre}^{control} = F_{pre}^{treat}$

This assumption ensures that both groups were comparable before the treatment; inbreeding rates of superstar lines did not increase before the introduction of genomic selection; we use matching methods to ensure that this condition holds.

Under Assumptions 3 and 4, we have that:

$$ATT_{post} = E\left[F_{post}^{treat} - F_{post}^{control}\middle| T_j = 1\right] - E\left[F_{pre}^{treat} - F_{pre}^{control}\middle| T_j = 0\right] \tag{2}$$

The ATT is identified as the difference-in-differences of observed outcomes in the pre and post-intervention periods. All elements in this equation are observed and, therefore, estimable. Suppose superstars had superior traits to our matched control group. In that case, this condition is violated, and we would not be able to parse out the causal effect of inbreeding from the effect of the bull's genetic superiority. Assumptions 3 and 4 ensure that the OLS estimates $\hat{\beta}$ from Equation **??** are consistent and asymptotically valid (Roth et al., 2023).

Assumption 5: Let $W_j = (F_{j,pre}, F_{j,post}, T_j)'$ be a vector of outcomes and treatment status for unit i. Then, we observe a sample of N independent and identically distributed random draws $W_i \sim F_W(w)$ for a distribution F satisfying parallel trends.

Under Assumptions 3, 4, and 5: $\sqrt{N}(\hat{\beta} - ATT_{post}) \xrightarrow{D} N(0, \sigma^2)$, then we can consistently estimate the variance σ^2 clustering at the line level (Bertrand et al., 2004) asymptotically ($N \to \infty$ and T fixed).

We can also decompose the variations in treatment time since every year, a certain number of bulls will be born from lines in both groups, each with its inbreeding rate that can be averaged across treatment and control groups. Equation **??** shows the complete specification with leads and lags of the treated group. This equation decomposes the estimated ATT relative to the treatment average by birth year. Sun and Abraham (2021) call this specification "Dynamic specification." However, there is no staggered treatment since genomic selection affects all lines in the same year, but inbreeding rates evolve independently after the treatment.

This assumption states that the treatment status of an individual from any given line is not affected by the treatment status of others. In our setting, the treatment (genomic selection) affects all individuals, so there is no selection into treatment. To measure the effects of genomic selection on inbreeding, we estimate the differences-in-differences model:

$$F_{ikt} = treat_i + treat_i * \sum_{\substack{t=2005 \\ t \neq 2009}}^{2017} \beta_t \mathbf{I}(yob = t) + \alpha_t + \alpha_k + \varepsilon_{ijt} \quad \text{(Model (2))}$$

$$F_{ikt} = treat_i + treat_i * \sum_{\substack{t=2005 \\ t \neq 2009}}^{2017} \beta_t \mathbf{I}(yob = t) + \alpha_t + \alpha_k + \mathbf{X}_{it}\gamma + \varepsilon_{ijt} \quad \text{(Model (3))}$$

$$F_{ikt} = treat_i + treat_i * \sum_{\substack{t=2005 \\ t \neq 2009}}^{2017} \beta_t \mathbf{I}(yob = t) + \alpha_t + \alpha_k + \mathbf{X}_{it}\gamma + (\mathbf{X}_{it} * post_t)\,\rho + \varepsilon_{ijt} \quad \text{(Model (4))}$$

Model (2) is a specification that does not contain any covariates (PTAs), and it decomposes the ATT on inbreeding rates by year of birth cohort. Model (3) incorporates PTAs ($\mathbf{X}_{it}$) and firm-level fixed effects, and Model (4) includes interactions of PTAs and the post-treatment dummy. We include the last specifications to account for trait-level trends. We also know that the treatment affects both inbreeding rates and PTAs, but we do not have a structural model for their impact (Lewbel, 2019), so we include them in a non-linear fashion as both levels and interactions to reduce the uncertainty about the functional form of our controls.

5 Results

Figure 7 shows treatment and control groups' average inbreeding rates and confidence intervals across years. Genomic selection did not increase inbreeding rates immediately; both curves began to diverge substantially after 2011. Inbreeding rates, in turn, started growing two years later, and both curves diverged significantly from 2012 onwards.

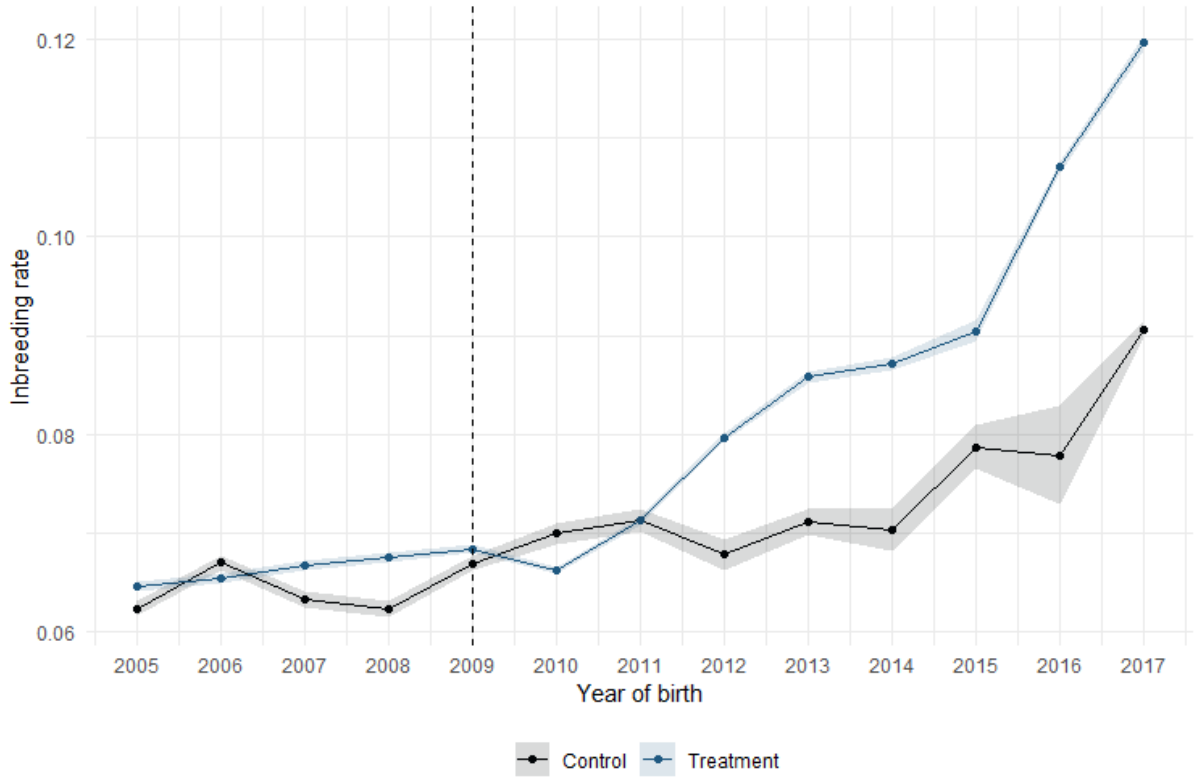

Figure 7: Average inbreeding rates in treatment and control groups

This plot closely matches the evolution of the population average inbreeding rates plotted in Figure 1. This behavior is consistent with our hypothesis that breeders prefer well-known over new lines. Our findings coincide with results reported by Guinan et al. (2023), who also look at the time trends of productivity and reproduction traits in US dairy cattle. The authors also document a decrease in the generation interval and an increase in inbreeding rates of Holstein and Jersey cattle but do not consider the latter an issue because the expected inbreeding rates of future offspring adjust genetic trait values.

Table 2 shows the results of three models; model (1) is the analog to Figure 7, including only the interactions between treatment and year of birth. Model (2) splits the treatment effect by year of birth averages, Model (3) includes the complete set of genetic traits and firm level fixed effects, and Model (4) is the model with traits and a post-treatment dummy. In all specifications, standard errors are clustered at the line level to better deal with the inconsistency generated by the autocorrelation of inbreeding rates between different generations (Bertrand et al., 2004).

Model (1) is the simple differences-in-differences estimation where $post_t$ is a dummy equal to 1 if the bull was born after 2009. The results show that bulls descending from supersires increased their inbreeding rates by 1.35 percent more than those who descend from less popular bulls, on average. Since the impact of genomic testing on inbreeding rates only became noticeable

after 2011, the coefficient underestimates the ATT of genomic selection. Model (2) shows that the treatment-year of birth interactions are positive and significant from 2012 onwards, with the 2017 effect being the highest.

Models (3) and (4) in Table 2 show that there is a positive and significant incremental variation for the treated versus control groups in years 2012-2017; however, the addition of controls in either levels or interactions attenuates the coefficients for 2014 and 2015 to the point of reducing the significance of interactions between treatment and year of birth. Still, in all instances, the coefficients are positive. Regression models show a positive and increasing inbreeding rate in the treatment group comprised of all descendants from prestige sires relative to the control group of descendants of comparable non-superstar sires.

The last period in the sample is 2017 instead of 2020 because we cannot make inferences after 2018 due to a reduction in the number of animals in the control sample; the number of comparable descendants is too small for us to consider them a valid comparison group. This drastic reduction is a consequence of lower fertility rates in the control group; since those sires fathered fewer sons, their sons will beget even fewer grandsons, and after a few years, this group collapses.

Table 2 also includes the results for the F-test for joint significance of all pre-treatment differences in trends for the interaction of treatment and year of birth. The p-values show no significance in two of the three specifications and a 5% significance for the pre-treatment coefficients of Model (4). This is not a test for parallel trends since the assumption relies on unobservable counterfactuals, letting $\hat{\beta}_k$ be the coefficient for any of the post-treatment periods, then:

$$E\left[\beta_k\right] = ATT_k + \left(E\left[F_{ik}^{control} - F_{i0}^{control}\middle| T_i = 1\right] - E\left[F_{ik}^{control} - F_{i0}^{control}\middle| T_i = 0\right]\right)$$

The parallel trends assumption implies that the term in parentheses is equal to zero, then the coefficients correctly capture the ATT for any post-treatment period $k = t - 2009 > 0$. On the other hand, its pre-treatment analog is identified as:

$$E\left[\beta_{(-k)}\right] = E\left[F_{i(-k)}^{control} - F_{i0}^{control}\middle| T_i = 1\right] - E\left[F_{i(-k)}^{control} - F_{i0}^{control}\middle| T_i = 0\right]$$

We have just shown that these coefficients are not significantly different from zero for all pre-treatment periods, individually and jointly. It is usually considered evidence in favor of the parallel trends assumption. However, this is not necessarily the case since this test has

low power against a linear or quadratic pre-trend alternative (Roth, 2022). In our case, we can dismiss this concern; since our matching method ensures the fulfillment of the condition for the founders, the entire effect must be due to their sons and grandsons.

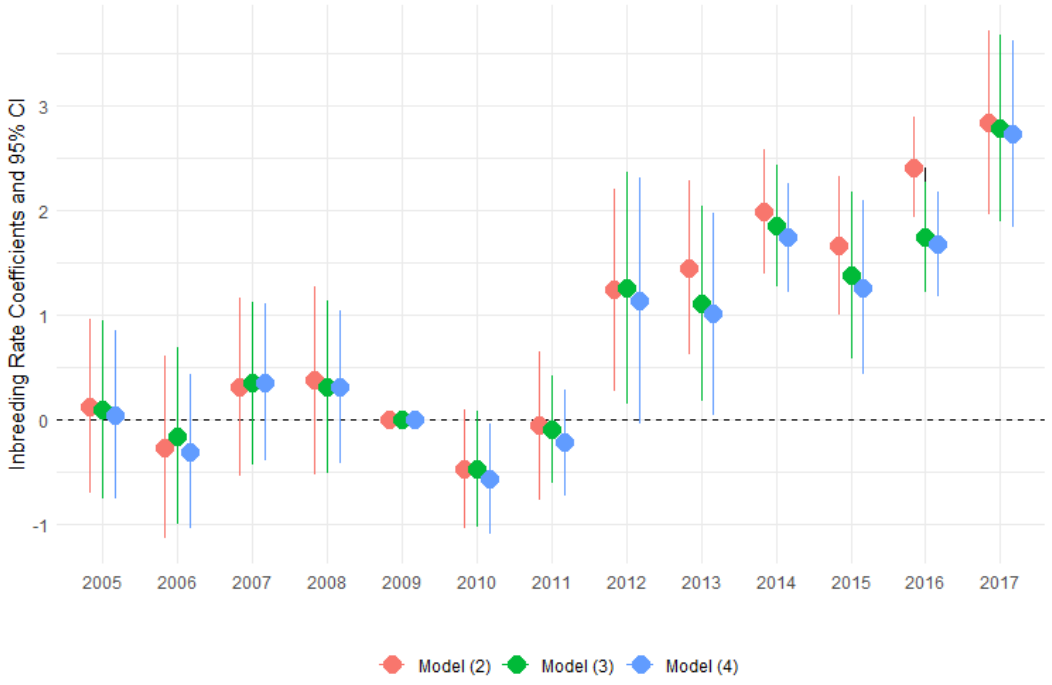

Figure 8: Coefficient plots for different specifications

Figure 8 shows the coefficients from the interaction of year fixed effects and treatment status to assess the impact of inbreeding per year correctly; every year after 2012, the inbreeding rate of the treatment group is between 1 and 2.8 percent points higher than the control group. The coefficient values decrease slightly after controlling for their genetic traits but remain statistically significant. The figure also shows that all pre-treatment coefficients are not significant; we interpret this as evidence in favor of the reasonability of the research design. Figure 8 uses the *sup-t* uniform confidence intervals for the pre-treatment periods of Montiel-Olea and Plagborg-Møller (2019) as suggested by Freyaldenhoven et al. (2019) instead of the standard pointwise confidence. These confidence intervals contain the actual path of the coefficients 95% of the time and can better help understand which kinds of pre-trends are consistent with the data.

So far, we have discussed our results regarding inbreeding rates only, but we can attach a monetary value to increased inbreeding rates. The American Holstein Breeders Association estimates that every additional inbreeding percent over the average is worth 23 US dollars per animal. The total cost depends on what we consider the average inbreeding; we can use the control group rates as a counterfactual and calculate the cost from increased inbreeding

of the treatment group, multiplied by the total stock of Holstein cattle; in that instance, we estimate a total cost from increased inbreeding of 3.6 billion dollars for the period 2011-2019. Alternatively, by industry standards, the maximum recommended inbreeding rate is 6.25%[2], so we can calculate how much is the cost from inbreeding levels greater than such value, we estimate the total cost for the same period as 6.7 billion dollars for the entire dairy industry.

6 Discussion

This article investigates the consequences of genomic selection on the American dairy industry. On the one hand, this new technology significantly improved productivity, but on the other hand, it increased the average inbreeding rate of all bulls in the market. Such increment in productivity was fueled by two effects: a decreased generation interval motivated by a new technology that accurately estimates a bull's genetic traits as soon as it is born and more intense competition between animal breeders to produce high-performing bulls. Faster access to information allowed breeders to select better bulls faster, and consequently, genetic gains per generation are now much higher than they used to be in the past.

The most popular method to breed highly productive bulls is by breeding lines that descend from a famous ancestor; we call these animals supersires and show that breeding on these well-known lines is the most critical driver of a greater degree of inbreeding among bulls. As the average inbreeding rate increases, every new generation of animals experiences an increased risk of experiencing inbreeding depression, which in turn leads to lower fertility and a higher prevalence of genetic diseases. Inbreeding depression is a cost that breeders impose on others, a negative externality that is a function of the number of breeders that release closely related bulls into the market. Since pedigrees transmit the externality, it is a network externality that affects all breeders and dairy farmers.

It is impossible to directly test the impact of genomic selection on inbreeding rates because there is no natural control group and no lines unaffected by genomic selection. We used a propensity score matching method to find equivalent families based on the founder's genetic traits but had fewer offspring; breeders could have invested in them but did not. We then track those families across years and observe the average inbreeding rates of their offspring. We show that bulls from the treatment group (those who descend from famous ancestors) have

[2] https://www.agproud.com/articles/36810-the-impact-of-genomics-on-rapid-increase-of-inbreeding-of-holsteins

consistently higher inbreeding rates than those from less-known ancestors from 2012 to 2017. Our analysis shows that the cost of this increased inbreeding amounts to around 500 million dollars.

Inbreeding rates can be reduced significantly by importing bulls from other countries that are not (or less) related to supersires. Another possible solution is to adjust the values of genetic traits by inbreeding; the Holstein Association of the United States adjusts the values of PTAs according to inbreeding levels to penalize breeders that select bulls with high inbreeding rates to account for the higher reproduction costs of such animals. The value of this penalty is a crucial element in reducing inbreeding by incentivizing dairy farmers and animal breeders to breed lines that are less related on average by, for example, importing bulls from abroad or by crossbreeding Holsteins with other high-yield milk breeds.

7 Appendix

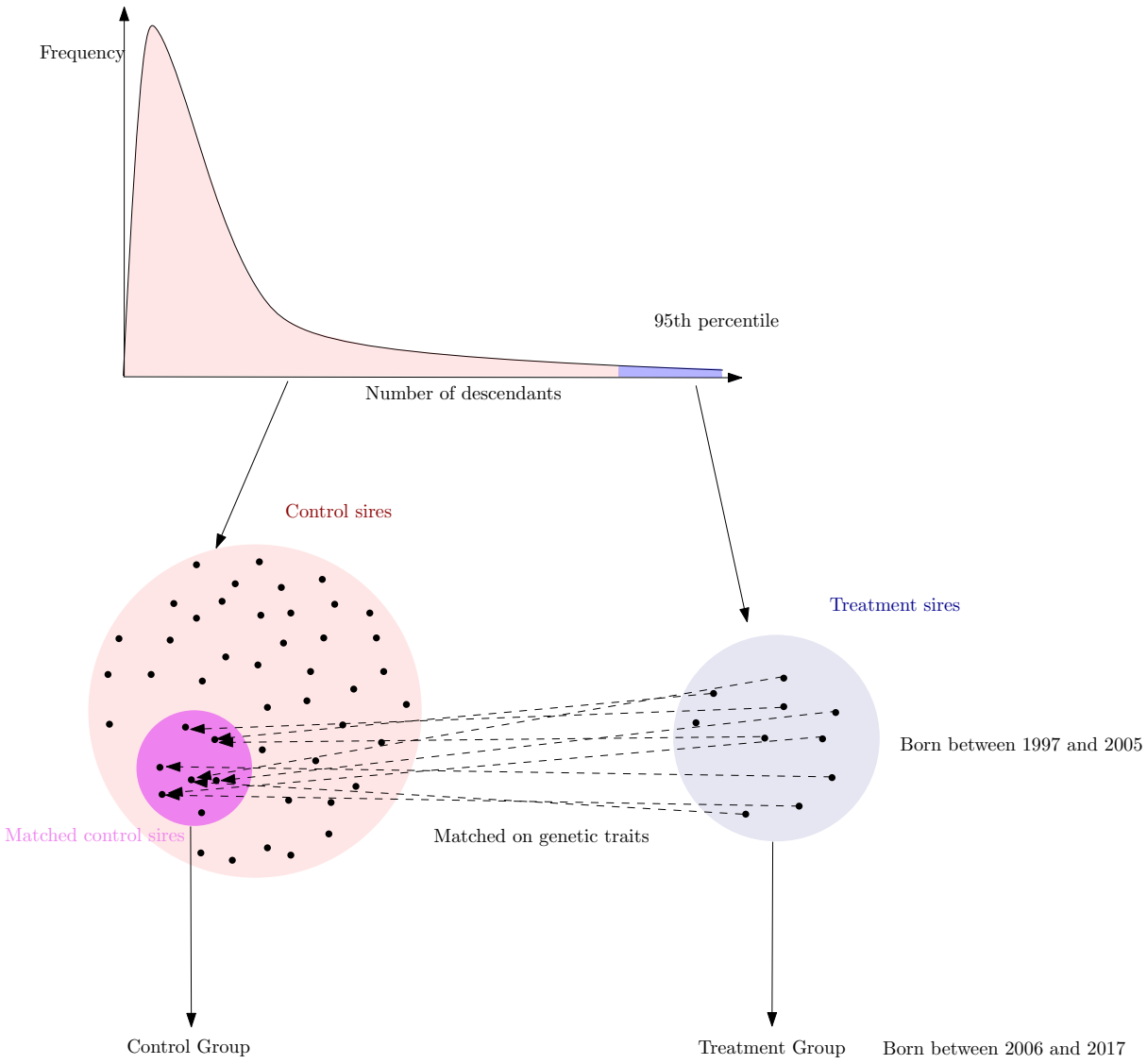

Figure 9: Sample selection mechanism

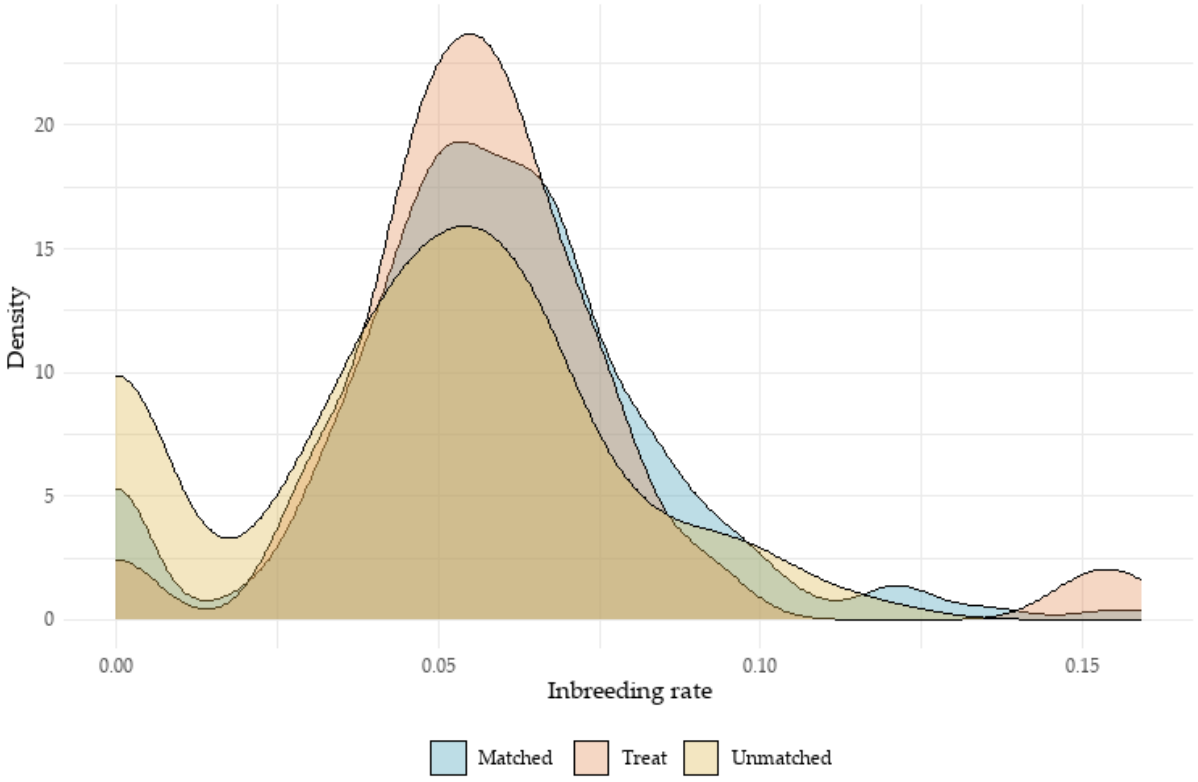

Figure 10: Distribution of Inbreeding rates across pre-treatment groups

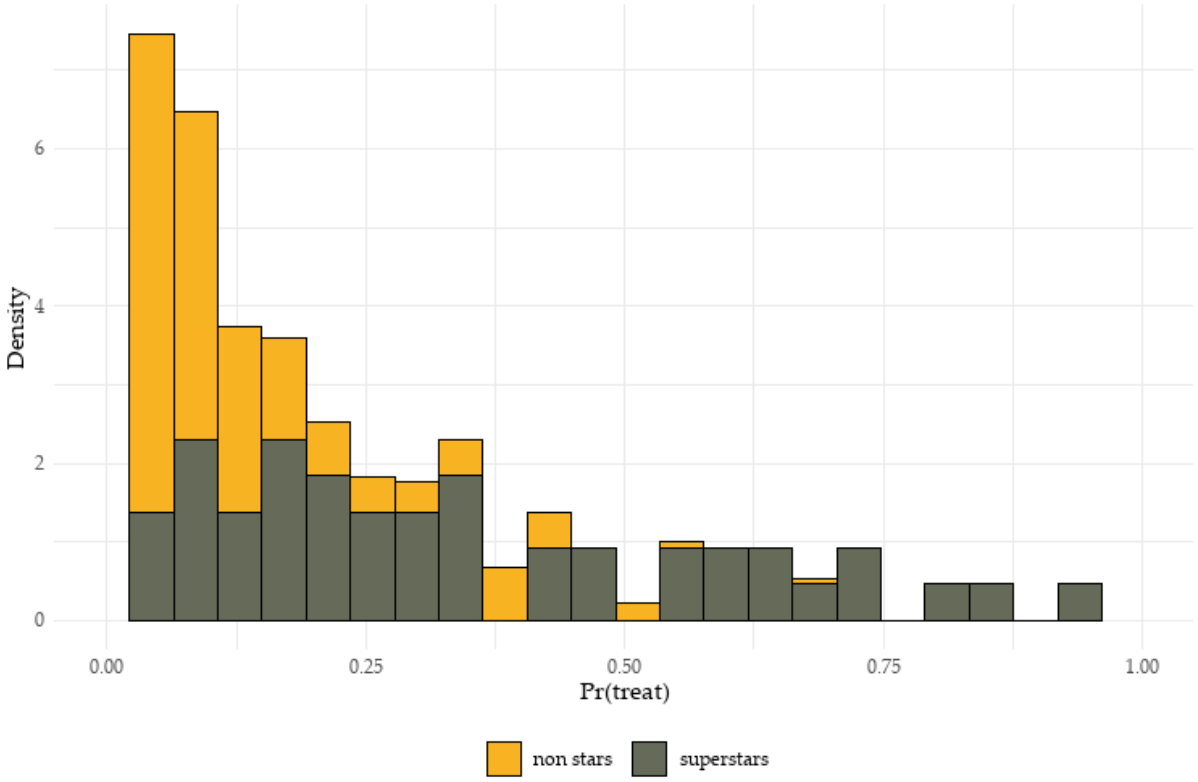

Figure 11: Propensity score by class of bull

Table 1: Balance table for outcome and matched variables

Variable	Control mean (n = 224)	Treatment mean (n = 43)	t.statistic	p.value	df
Inbreeding rate	2.968	2.904	0.426	0.672	58.147
Milk	0.059	0.059	-0.036	0.971	59.956
Fat	-0.122	-0.170	0.346	0.730	76.548
Protein	-0.851	-1.009	0.416	0.679	61.059
Somatic cell score	-0.151	-0.037	-0.675	0.502	66.764
Productive life	-0.619	-0.677	0.173	0.864	59.829
Daughter preg. rate	-1.224	-0.489	-1.336	0.186	70.101
Heifer conc. rate	-11.953	-5.553	-1.807	0.075*	71.841
Cow conc. rate	-0.420	-0.209	-1.278	0.206	65.480
Livability	-0.393	-0.177	-1.816	0.074*	70.905
Type	-0.827	-0.551	-1.721	0.090*	69.510
Gest. length	0.342	-0.055	1.719	0.090*	64.087
Heifer liv.	-0.569	-0.579	0.032	0.975	58.933
Eff. calving	0.101	0.232	-1.549	0.127	58.339
Mastitis	-0.888	-0.764	-0.288	0.774	58.621
Metritis	-0.840	-0.891	0.202	0.840	62.094
Strength	-0.589	-0.581	-0.047	0.963	57.734
Rear legs rear view	-191.265	-24.936	-1.581	0.119	67.713
Rear legs side view	-0.027	-0.013	-0.495	0.623	60.964
Foot leg score	-1.348	-1.123	-0.666	0.508	59.099
Teat rear place	-8.284	-3.255	-1.859	0.067	68.421
Rump angle	-0.360	-0.136	-1.447	0.153	64.334
Thurl width	-0.010	0.050	-0.342	0.734	63.537
Foot angle	-0.767	-0.417	-2.044	0.045**	68.106
Fore udder	-0.743	-0.432	-1.735	0.087*	66.269
Rear udder height	0.142	0.193	-0.254	0.800	60.330
Rear udder width	3.044	2.995	0.690	0.493	52.692
Udder cleft	-0.386	-0.349	-0.235	0.815	77.188
Udder depth	-0.206	-0.267	0.409	0.684	67.108
Teat fron place	-0.343	-0.195	-1.006	0.318	66.497
Teat length	0.197	0.159	0.195	0.846	59.002
Milk fever	-0.312	-0.140	-0.948	0.347	62.811
Stature	-0.255	-0.233	-0.138	0.890	72.664
Dairy form	-0.573	-0.403	-1.442	0.153	73.087
Body depth	-0.401	-0.193	-1.127	0.264	61.777
Sire calv. ease	-0.738	-0.589	-0.989	0.326	73.000
Daughter calv. ease	2.427	2.287	1.188	0.239	63.963

Notes: ***Significant at the 1 percent level.
**Significant at the 5 percent level.
*Significant at the 10 percent level.

Table 2: Difference-in-Differences estimates from Equation **??**

	Inbreeding rate (%)			
	No covariates	No covariates	Traits	Traits and interactions
	(1)	(2)	(3)	(4)
treat	0.152 (0.129)	0.084 (0.230)	0.018 (0.218)	0.090 (0.177)
$treat \times post\_2009$	1.346*** (0.366)			
$treat \times \mathbf{I}(yob = 2005)$		0.129 (0.298)	0.095 (0.295)	0.048 (0.277)
$treat \times \mathbf{I}(yob = 2006)$		−0.262 (0.313)	−0.158 (0.292)	−0.306 (0.254)
$treat \times \mathbf{I}(yob = 2007)$		0.312 (0.306)	0.350 (0.268)	0.358 (0.259)
$treat \times \mathbf{I}(yob = 2008)$		0.375 (0.324)	0.311 (0.285)	0.313 (0.251)
$treat \times \mathbf{I}(yob = 2010)$		−0.467 (0.290)	−0.472* (0.285)	−0.567** (0.270)
$treat \times \mathbf{I}(yob = 2011)$		−0.057 (0.363)	−0.090 (0.262)	−0.220 (0.260)
$treat \times \mathbf{I}(yob = 2012)$		1.239** (0.492)	1.255** (0.564)	1.139* (0.601)
$treat \times \mathbf{I}(yob = 2013)$		1.453*** (0.425)	1.107** (0.477)	1.010** (0.491)
$treat \times \mathbf{I}(yob = 2014)$		1.990*** (0.304)	1.851*** (0.298)	1.739*** (0.267)
$treat \times \mathbf{I}(yob = 2015)$		1.660*** (0.339)	1.383*** (0.407)	1.259*** (0.424)
$treat \times \mathbf{I}(yob = 2016)$		2.410*** (0.246)	1.745*** (0.272)	1.677*** (0.252)
$treat \times \mathbf{I}(yob = 2017)$		2.840*** (0.448)	2.784*** (0.454)	2.735*** (0.454)
p-value for nonzero pre-effect		0.289	0.3	0.041**
Observations	14,480	14,480	14,480	14,480
R^2	0.160	0.440	0.468	0.474
Adjusted R^2	0.160	0.439	0.465	0.470
Residual Std. Error	2.265 (df = 14476)	1.851 (df = 14454)	1.807 (df = 14415)	1.798 (df = 14393)

Notes: ***Significant at the 1 percent level.

**Significant at the 5 percent level.

*Significant at the 10 percent level.

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