How can we estimate inheritance?

Bachelor thesis
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Abstract

Inheritance is central in evolutionary studies. In this review I explain how inheritance can be estimated. Inheritance in the field is estimated using the fact that children inherit traits from their parents. Regressing offspring traits on parental traits gives an estimate of inheritance called heritability. I explain how heritabilities are calculated, discuss the biases that heritability estimates imply and discuss ways to resolve these problems. The biases I will talk about are: misidentified parents, the fact that heritability is variance dependent, maternal effects and shared environments of parents and offspring. Also, I will discuss that heritability estimates depend on the environment and heritability estimates are affected by selection. Finally, two alternative methods to estimate inheritance are discussed, crossing experiments and genomics. At last, I will give my vision on whether we should still use heritability estimates to estimate inheritance.

Source front page image: http://www.capitalotc.com/darwins-finches-adapted-their-beak-shapes-to-food-sources-study-shows/28964/
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Introduction

To understand biology, we need to understand evolution. An important part of evolution is natural selection. We already have learned a lot about selection in lab situations, but since the environment in labs is constant, natural selection might work differently in natural ecosystems, where the environment changes constantly. Because of this, it is important to do research on evolution in wild populations of organisms. To do so, first of all one needs to prove that there is natural selection working on the trait that is studied by proving the requirements of natural selection. The requirements for natural selection are (i) variation in the trait, (ii) that the trait is heritable and (iii) that some variants survive and reproduce at higher rates than others *(Freeman & Herron, 2014)*. Without variation in a trait, everyone is the same and there is nothing to select on. If a population is monitored properly, variation in traits is often measurable. Examples of such traits are clutch size, birth and adult weight and lifetime reproductive success. Also variation in the success of traits is often measurable, if the survival and reproduction of individuals is known. The harder part of proving natural selection is proving requirement (ii) that a trait is heritable. Many traits and the variation in these traits are determined by a lot of genes and their interactions, so it is hard to find which genes are exactly responsible for the trait and its variation and thus to prove inheritance in that way. A common approach to estimate inheritance is to estimate the so called heritability, based on comparing parents and offspring. In this essay I aim to explain how heritability is measured, what the pitfalls are and how we can deal with them. In the discussion I will give my view on the question if we still should use this way of estimating inheritance.

The definition of heritabilities

Heritability is a measure to estimate if a trait is inherited (I used the word heritability before without aiming at the parameter, but from now on, it will be the parameter I am aiming on whenever I use it). There are two kinds of heritability, the broad-sense heritability (H^2) and the narrow-sense heritability (h^2). The variation in a trait has different sources. The variation in a phenotype (Var(P)) is explained by the variation in the genotype (Var (G)) and the variation in environment (Var(E)) *(Hartl & Clark, 2007)*.

$$Var(P) = Var(G) + Var(E)$$

Broad sense heritability

The definition of the broad-sense heritability is the part of the phenotypic variation that is caused by the variation in genotype *(Hartl & Clark, 2007)*:

$$H^2 = Var(G) / Var(P)$$

This genotypic variation consists of additive genetic variation, dominance effects, epistatic effects and maternal and paternal effects. Additive genetic variation is the variation caused by the average effects of the alleles. This is the part we actually want to prove that it exists, because it proves there are genes responsible for the trait.

Dominance effects are effects that have to do with the fact that some alleles are dominant and others are recessive. This can have an influence on how a trait is expressed in the phenotype, but can sometimes be hard to estimate if a trait is determined by a lot of genes.

Epistatic effects are effects caused by interactions between genes. Some alleles in one gene can influence the transcription of alleles on different genes for example.

Maternal and paternal effects are effects caused by the fact that a specific organism is the mother or the father of an organism, but is not caused by the genes. An example is the cytoplasm in an egg that is made by the mother, but will have effects on the zygote.

Narrow-sense heritability

To make sure there are genes causing the trait and there is really additive genetic variation, Var(A), one can estimate the narrow-sense heritability *(Hartl & Clark, 2007)*:

$h^2 = Var(A)/Var(P)$

Another definition of the narrow-sense heritability is the breeder's equation *(Hartl & Clark, 2007)*:

$h^2 = R/S$

Wherein S is the difference between the population mean and the mean of the selected parents or the selection differential. R is the difference between the new population mean (after selection) and the old population mean or the response to selection, see figure 1.

Since we do not know which parents are favoured in the wild, we cannot literally use the breeder's equation. Also the definition of the narrow-sense heritability is hard to use, since it is almost impossible to make a good estimate of the additive genetic variation (this is the part we want to know, we don't know it yet).

To still calculate the narrow-sense heritability, most researches use a parent-offspring regression. If a trait is equal in males and females, the slope of the regression of the average of the offspring on the average of the parents is equal to the narrow-sense heritability. When parents have a certain value of a trait and the offspring have a value near the value of the parents, this can be a good indicator that inheritance plays a role in the expression of the trait.

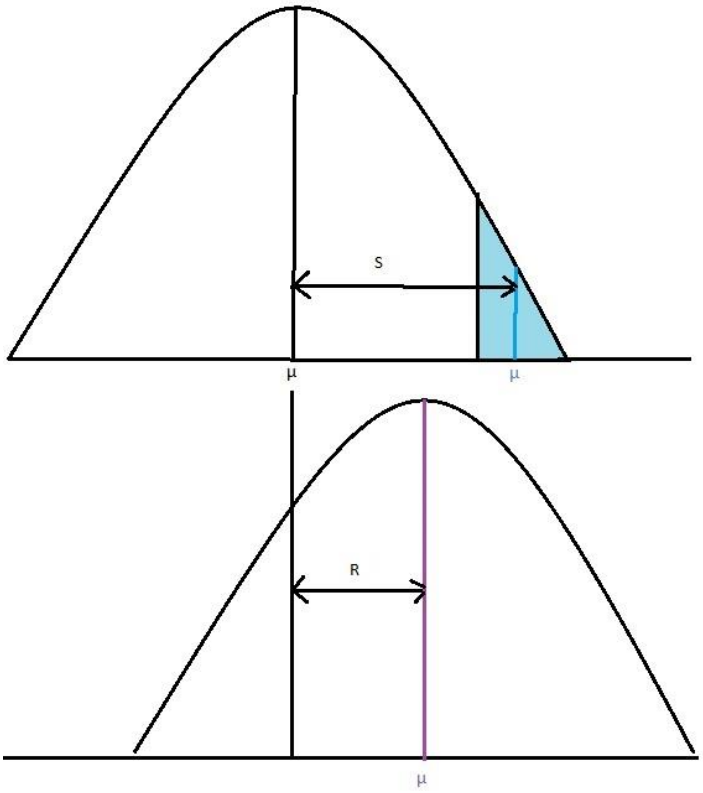

Figure 1: The breeder's equation visualized. $h^2=R/S$. S is the difference between the mean of the total population and the mean of the selected parents. R is the difference between the mean of the original population and the new population mean (after selection).

A good example of this way of a h^2 estimation is given by *Boag (1983*). In his work the narrow-sense heritability of bill depth was estimated in the population of Darwin's Ground Finches (*Geospiza fortis*) on Isla Daphne Major, Galapagos, see figure 2.

Resolving biases in the estimates of narrow-sense heritabilities

Even though estimating the narrow-sense heritability via parent offspring regressions is simple and very easy to use, it is not perfect. There are a lot of biases and problems with this way of estimating h^2. In the rest of this paper I will try to explain what these biases and problems are, what we can do about it and what the alternatives are.

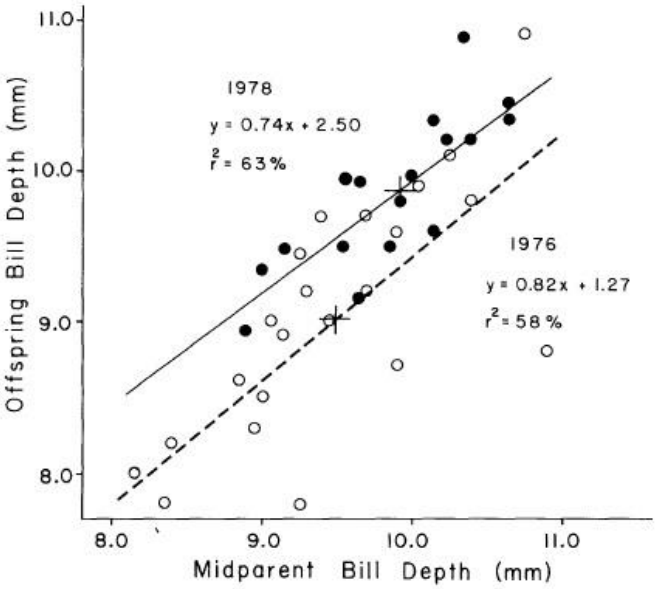

Figure 2: The parent-offspring regression done by Boag (1983). The open circles are of 1976 and the solid circles are of 1978. The slopes of the lines are nearly identical, even though the years differ. The slope of the line is h^2.

Misidentified parents

Estimating paternity just by observations is in most species not quite reliable. A lot of mammal and bird species act monogamous, but actually still mate with more than one partner. From genetic analysis we know that extra pair paternity and conspecific brood parasitism exists. In the first case the breeding female is the true mother, but the helping father is not the real father. In the second case both breeding parents are not the true parents, since a different female, from the same species, laid an egg in the nest. It is important to control for this, because if parents and their non-related offspring have different values of a trait, the slope of the parent-offspring regression will become less steep and the estimate of the narrow-sense heritability will become lower and maybe even non-significant from zero *(Freeman & Herron, 2014)*.

The use of Microsatellites

Currently, a couple of microsatellites (repetitive parts of the DNA used for identification) are being analysed and when one satellite differs between the parent and the offspring, the offspring is being classified as non-related. *Lemons et al. (2015)* advocate for a less conservative method, by using a difference of two microsatellites. This will decrease the amount of type I errors (falsely declared cases of extra pair paternity and conspecific brood parasitism). As a result the type II errors (non-related offspring declared as related offspring) will increase. The study simulated data (with all factors known) and they tried to find out how many young where non-related to their breeding parents. With one microsatellite difference, they found that too many young were declared non-related. However, with a difference of two microsatellites, they came quite close to the real number of non-related offspring in the simulated data (for numbers see article).

Another result of their research was that it is important to choose enough microsatellites. When there will be more microsatellites, there will be more mismatches between parent and offspring, so it is important to choose the right number of allowed mismatches with the number of microsatellites. This probably will differ between species and populations, since every species has different percentages of extra pair paternities and conspecific brood parasitism. So it is important to think about when planning a study.

Keller et al. (2001) did work on extra pair paternity in Darwin's Ground Finches (*Geospiza fortis*). They first sampled the parents and offspring and checked with eight loci if there were differences. When there was a difference between the offspring and the father, they genotyped both again to exclude the possibility of genotyping error. All the extra pair offspring mismatched their breeding

fathers at two or more loci, except for one. However this one was still considered an extra pair young, since mutation was unlikely, because it happens much less often than extra pair paternity.

The frequency of extra pair young in this population was 19.7% and 35.5% of the families had at least one extra pair young. They also found that there were no cases of young that were not related to their mothers, so there were no (measured) cases of conspecific brood parasitism.

Kellers work shows that a low number of microsatellites can still be very useful, if the right microsatellites are being chosen (their combined exclusion probability was 99.96%) and that re-genotyping can increase the power of the experiment tremendously.

It remains remarkable that there were no cases of intraspecific brood parasitism. Since the power of the microsatellites was quite high, we can assume that there were no mistakes in the analysis and that the pedigree of this population was reliable, which means that an estimate of the narrow-sense heritability probably also will be more reliable than in some other populations.

Heritability is variance dependent

Quite a big bias in the use of the narrow-sense heritability is that it is dependent on the variance of the trait. When a trait is very important for an individual (a life-history trait) almost all the variation that arises will have an effect on the survival of the individual. If this effect is positive, its fitness will increase and the mutation will spread quickly through the population. If the mutation has a negative effect on its survival, the fitness of the individual will decline and it will die before it can reproduce or at least have a much lower fitness. This mutation will fade out soon. This means that there will be almost no variation left in the life-history trait in the population. Since there are no genetic differences in this trait between individuals, but the environmental variation will stay, the proportion of additive genetic variance will decrease and the slope of a parent-offspring regression will be low as well, so the estimate of the narrow-sense heritability will be low too. Although these traits are very important for fitness and have genes controlling it, they still have a low estimate of heritability. *Kruuk et al. (2000)* made a graph that shows heritability estimates of a trait on the y-axis and the correlation of the trait with total fitness on the x-axis, see figure 3. It might not be perfect, but one can see a pattern in it.

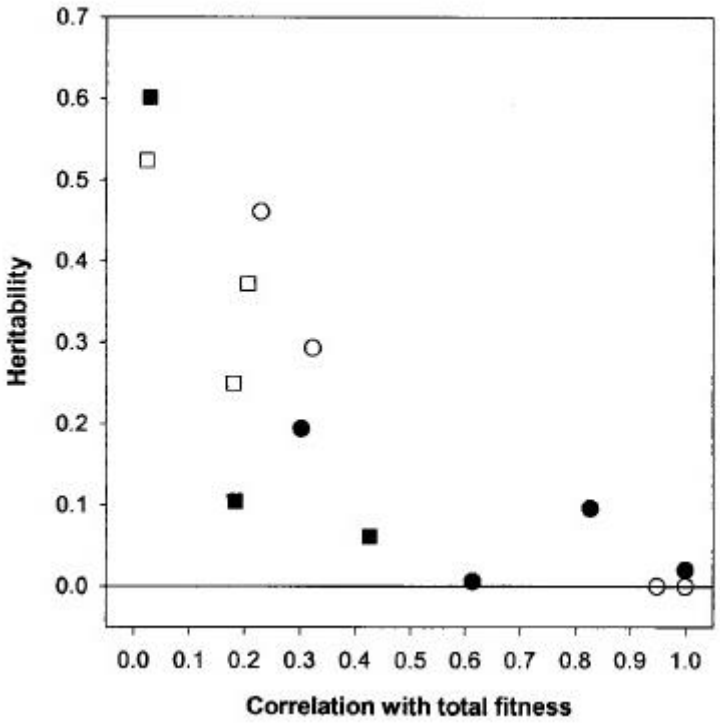

Figure 3: Heritability estimates plotted against each trait's correlation with total fitness. Open circles are female life-history traits, open squares are female morphometric traits, closed circles are male life-history traits and closed squares are male morphometric traits. From Kruuk et al. (2000).

Two meanings of a low heritability

When one wants to know if a trait is heritable and the data show a low heritability, it is impossible to prove that the trait is heritable and important for life-history or that it is not heritable and determined by other causes than the DNA. Of course it makes sense that some traits are life-history traits, without proving it (like lifetime reproductive success) or are not heritable, but for traits that are not as obvious as lifetime reproductive success it can be complex to prove what is true. To really prove that a trait can be inherited, it should be proven that there are genes responsible for its expression. As said in the introduction, this is really complex, since most traits are determined by a lot of genes and their interaction. Other things than variation in a trait are not visible and measurable in the phenotype, so there is no simple alternative for the narrow-sense heritability.

The coefficient of additive genetic variance

One way to deal with the low heritability of life-history traits, is to use the coefficient of additive genetic variance (CVa) instead of the narrow-sense heritability. The CVa scales the component of additive genetic variance by the trait mean instead of by the total variance *(Kruuk, 2000).* This means that it is independent on the magnitude of the other variances.

Houle (1992) found that the CVa values of life-history traits are much higher than that of morphological traits (which are less linked to fitness). If both the narrow-sense heritability and the CVa are estimated, this could be a solution to prove a trait is inherited through genes instead of not being heritable, when h^2 is low.

Maternal effects

Maternal effects are the effects of the mother on the traits of offspring in other ways than via the DNA. Examples of this are the effect of the mother on the immune system of the offspring through breast feeding and the amount of yolk an egg contains dependent on the environment of the egg. These maternal effects are short term, however scientists begin to see that there could also be maternal effects that influence the offspring long term of even permanently.

Evidence from the lab

Breeuwer & Werren (1995) found that the effect of the cytoplasm is much more important than expected. They crossed *Nasonia Vitripennis* with *Nasonia Giraulti* (which is in natural situations impossible due to micro-organisms) and created hybrid females (no males, because both species are haplodiploid). They used one species (X) as

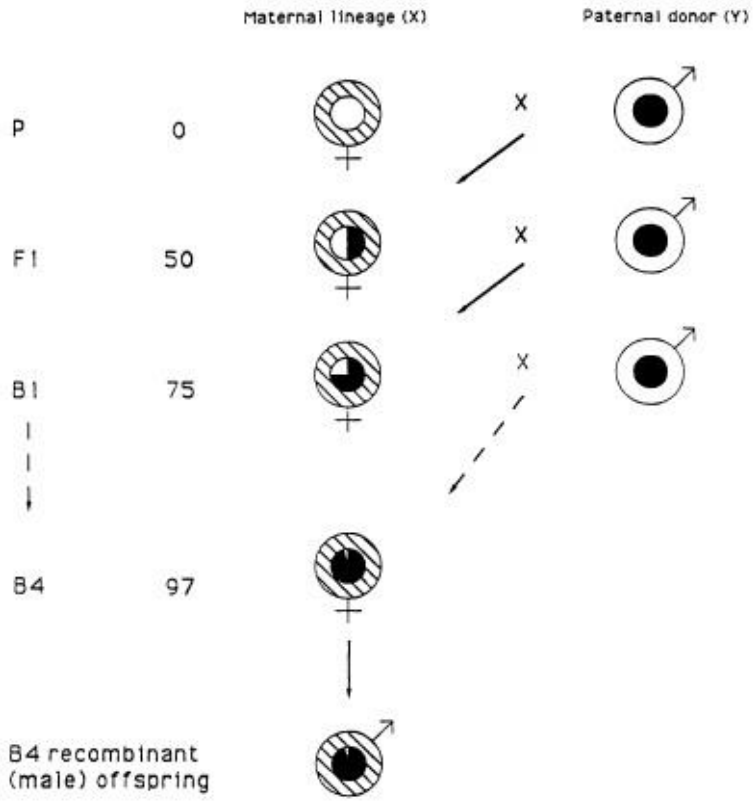

Figure 4: Species X (maternal line) is crossed with species Y (paternal line). The hybrids are crossed back with the paternal line until a hybrid with complete Y nuclear DNA and X cytoplasm exists. From Breeuwer, J.A.J. & Werren, J.H. (1995).

female for the first cross and kept crossing its female hybrid offspring with the other line (Y). In one line X is *N. Vitripennis* and Y is *N. giraulti* and in the other line vice versa (see figure 4).

In this way, they created hybrids with the genes of the Y species and the cytoplasm of the X species, after 16 backcrosses. They found that F2 males were not viable. There genomes were a combination of *N. Vitripennis* and *N. Giraulti*, due to recombination. Because males are haploid, the interactions of the genome with the cytoplasm sometimes showed incompatibilities and these were not restored by a second copy on an extra chromosome, such as in females. The females that had just genes of Y and cytoplasm of X were mated with Y and they showed a 50% mortality rate in their male offspring. This shows that maternal effects are really important and can change the fitness of their offspring tremendously.

Evidence from the field

Mother- vs father-offspring regressions

It is also possible to do research on maternal effects in the field. *Keller (2001)* compared estimates of the narrow-sense heritability of midparent-offspring regressions, mother-offspring regressions and father-offspring regressions. He found significantly higher h^2 estimates for bill size in the mother-offspring regression than in the others. He also made maternal grandmother-offspring regressions and paternal grandmother-offspring regressions and found that h^2 estimate of the maternal grandmother-offspring regression of PC-1 was significantly higher than the estimate of the paternal grandmother-offspring regression. PC-1 was a special measure of the body size (for explanation see article).

Kruuk et al. (2000) also found significant maternal effects on total fitness and on adult breeding success in females (their daughters). *Kirkpatrick & Lande (1989)* and *Wolf et al. (1998)* showed that maternal effects can have a big impact on selection and even can lead to counterintuitive responses to selection. Researchers increasingly recognize the importance of understanding maternal effects to understand natural selection and evolution.

A good, direct way to estimate maternal effects in general, is to compare the mother-offspring regression with the father-offspring regression. However, a lot of traits are different for both sexes (often females have a different length and other morphological traits). One should actually compare the 'female size' the male carries in its genes with the 'female size' the mother carries in her genes, but again this is impossible without knowing the genes behind the trait. Also, some traits are only expressed in one sex, like clutch size, average egg volume and average laying date. A good way to solve these problems is to make grandmother-offspring or grandmother-daughter regressions. However, since there is a generation in between, it is more prone to errors, so a good sample size is important.

Comparing grand-offspring of daughters and sons

Kruuk (2004) suggested a way to estimate genetic maternal effects, so the effect the genes of the grandparents have on the maternal effects of their offspring (the parents). She divided the grand-offspring of a certain male into offspring of his daughters and offspring of his sons. His daughters will be influenced by his maternal effect genes and his genes for the trait (for example growth), but his sons only will be influenced by his growth genes and not by his maternal effect genes. If the grand-offspring of the daughters are heavier than the grand-offspring of the sons, the male has genes for high maternal performance.

The use of animal models

Another way to correct somewhat for maternal effects, is to use the animal model instead of just a parent-offspring regression *(Kruuk, 2004; Wilson et al.,* 2009). The animal model does not only use the information who the parents are (one generation deep), but uses the whole pedigree. If traits continuously just inherit from mother to offspring, this indicates that the genes are on the mitochondrial DNA or that it is not dependent on genes, but for example on the cytoplasm. X-linked genes might have a similar pattern, but these genes should have a bigger impact on males than on females and should be expressed more often in males than in females, while maternal effects are not necessarily favoured or more expressed in one sex.

Correction for Shared environments of parents and offspring

Since birds and mammals show caring behaviour over their eggs and young, they automatically live in the same environment as their young. When parents and young are adapted to the same environment by phenotypical plasticity, they resemble each other more than expected just by their genes. This can cause an inflation of the narrow-sense heritability estimate. Especially the fact that parents and offspring live in the same micro-environment (for example their own nest box or territory within an ecosystem) can make it favourable for them to adapt differently than other parents and offspring, since they live in a different micro-environment. The variation in the population is larger in this way and parents and offspring resemble each other more than the rest of the population, just by living in the same place, which increases the estimate of the additive genetic variation.

For example, when parents are large, they have an advantage in fighting for territories and they will probably have a large and good territory. The chicks they produce have abundant food and also will grow large. However, it is uncertain if the cause of this are the genes of the large parents or the fact that the chicks had a lot of food *(Freeman & Herron, 2014)*.

Cross fostering offspring

To check for this one could perform cross-fostering experiments. A cross-fostering experiment is an experiment in which the young are being placed in a different nest, with different parents. It is best to do this with the eggs (in birds) to make the time the parents and offspring live in the same environment as short as possible. Because the environments of the parents and offspring are different now, the resemblance between them has to be due to their genes (and maybe some early maternal effects) and so there is evidence that the trait is heritable. In mammals cross-fostering experiments are harder, since young stick to their mothers, so there is not really time to change the young. Also, often mothers recognise their offspring and vice versa, so if the young are cross-fostered in the same population, they may run back to their mother and the mother probably will not raise the cross-fostered offspring or allow it to drink milk.

The only chance of success with a cross-fostering experiment with mammals is if the cross-fostering is between populations, since it is impossible for the offspring to return to its own mother. However, the chance that the new mother will adopt the cross-fostered offspring is smaller, since the offspring is from a different population and therefore it will be genetically more distinct than offspring from within the population.

Animal Models

Again, an animal model could help to filter out some of the environmental effects, because it uses the whole pedigree. Since the environment fluctuates over time, great-grandparents of offspring probably lived in a different environment than its parents and itself. If traits of the offspring still resemble those of the great-grandparents, these traits are probably heritable and not dependent on the environment. With only the use of an animal model, it is impossible to conclude that there are no environmental influences anymore, but it can help us to make our heritability estimate better.

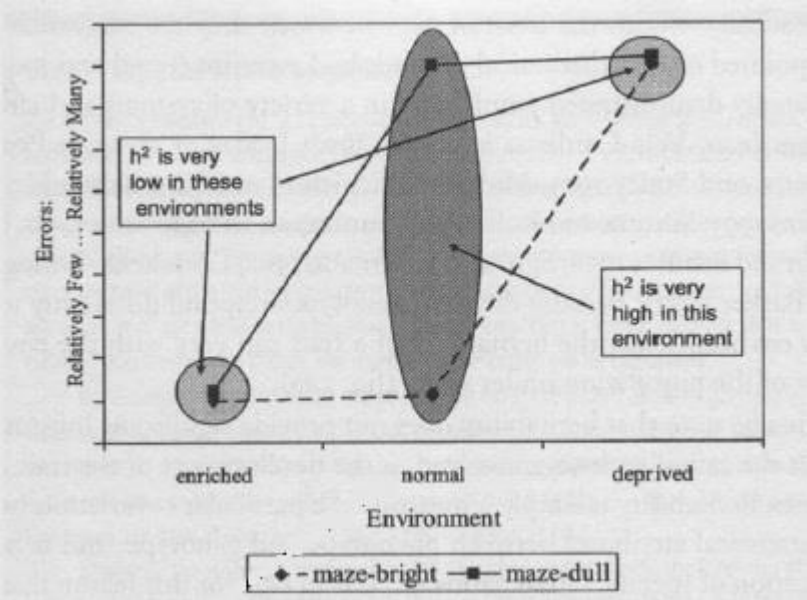

Figure 5: The maze running ability of rats from the McGill bright and dull strains, raised in three different environments (enriched, normal and deprived).

Figure found in the college slides of L.P.W.G.M. Van De Zande for the course Ecological & Evolutionary Genomics, Biology Bsc, University of Groningen

h^2 estimates depend on environment

In different environments, there will be different estimates of the narrow-sense heritability. This is best explained by the research of *Cooper & Zubek (1958)* who did a lab experiment on the maze running ability in rats, with the McGill bright and dull strains. There were 3 groups of rats. They were raised either in an enriched, normal or deprived environment. After 65 days the rats had to do a maze running ability test and their errors were scored. The rats in the enriched environment, both the bright and the dull rats, had a relatively low error rate. In the normal environment however, the bright rats had a low error rate, but the dull rats made many errors. In the deprived environment, both de bright and dull rats had a high error rate, see figure 5. In other words, in the enriched and deprived environment, both kinds of rats showed a similar expression of the trait, so there is not many variation in the trait. This will give a low h^2 estimate. In the normal environment, the bright rats showed a few errors, but the dull rats showed many errors. The variation in this environment is therefor much bigger than in the other environments and so the h^2 estimate will be much higher.

In other words, in some environments there is just somehow more variation than in others, which makes it easier to detect the additive genetic variance. When there is low variance, it is harder to prove that an individual inherited the trait from its parents, instead of gaining it from the environment, since almost the whole population has the trait. *Charmantier & Garant (2005)* showed with a meta-analysis that when the environment is

favourable, the h^2 estimate is higher compared to unfavourable conditions. However, they also showed that there is still a lot unclear in how this environment dependence works. For example, in one avian study they found that the body mass differed in different growth conditions, but not the tarsus length.

It is a problem when heritability estimates depend on the environment. In that case one could find different heritability estimates in different environments, maybe even some that are not statistically different from zero. However, the trait could still be inherited through genes, only this population does not show a lot of variance. This means that almost all heritability estimates should be done on different populations (with different environments) and at different times (also with different environments) to control for the effect the environment has on the estimates. Only when a trait is proven to be heritable in multiple environments, it is justified to claim that a trait is inherited through genes. This makes quantitative genetics much more costly and also makes studies to single populations less useful. Again, an animal model estimate of h^2 is more robust, since the environment changes somewhat in time, but there are still more estimates of different populations needed to really prove that a trait is inherited.

h^2 estimates are affected by selection

Also important is that natural selection itself can have an influence on the h^2 estimate. If natural selection favours a certain value of a trait, the trait will show less variance, because this certain value will spread through the population. If there is less variance, the h^2 estimate will become lower. So if a certain trait is in general not important for a species, but it is for a certain population, the h^2 estimate of the trait will be much lower in this population than in the rest of the species. This can lead to wrong conclusions if only the specific population is studied. However, if more research is done on the species and it is known that there is a big difference between populations, this can be valuable information. It could learn us a lot about natural selection and why traits are important in certain environments.

Discussion

This essay shows that there are clearly biases to heritability in its use to estimate inheritance and that there is no simple solution to solve all of them at once. It is expensive and time consuming to control for all the biases that are solvable and then there are still some that we cannot control for, so the question is whether we should use heritability estimates at all and if we should use it, what it really tells us.

Equilibrium assumptions

The problem of the low heritabilites is solvable with CVa estimates. However, the explanation of why life-history traits have a low heritability is questionable. It assumes a population is in equilibrium and the most beneficial trait mutations are fixed. However, for wild populations it is highly likely that they are not in equilibrium, since the environment fluctuates all the time and the most beneficial phenotype changes with this environment, so the most beneficial genotype also changes all the time. Also, the evidence of *Houle (1992)* that life-history traits have a higher CVa contradicts with the fact that mutations are fixed. The high CVa values are probably due to the fact that life-history traits are dependent on more genes and more complex interactions than morphological traits and therefore have a higher chance on mutations and genetic additive variance. *Price & Schluter (1991)* declare that low heritability estimates of life-history traits are not due to a low additive genetic variance, but due to a high residual variance. They state that variation in metric traits underlies the variation in life-history

traits. For example, the survival of Darwin's ground finches is dependent on its body size *(Boag & Grant, 1981).* The life-history traits not only have a large amount of variation in their underlying metric traits, but there is also a lot variation that only influences life-history traits and not the underlying metric traits. Examples of this are predation chance, the density of potential mates and the chance on (fatal) accidents. Because their residual variance is so much higher than that of metric traits, life-history traits automatically have a lower heritability estimate, independent whether the population is in equilibrium or not. However, there still needs to be a lot of research done to prove if this explanation is true.

Alternatives

Clearly, h^2 is not perfect, but are there better alternatives to still keep on doing research on evolution and natural selection? The two main alternatives are crossing experiments and genomics.

Crossing experiments

To totally understand how selection works, it is easy to think about crossing experiments. By doing crossing experiments, we could learn a lot about maternal and environmental effects. Benefits are that there are no misidentified parents to control for and that it is clear which environmental factor influences selection in a specific way. However, it would not be the same as in wild populations, since the environment is constant in labs or only one thing is changed to see what its influence is. In the wild different aspects of the environment covary. If the temperature rises, this is not the only thing that changes. The whole water cycle will change, by more photosynthetic activity of plants, more evaporation of surface water and so on. These chain reactions in the wild are never completely imitated in labs, so it is impossible to get a complete overview of what for instance a temperature rise will do to a population. One cannot really say much about 'natural' selection in lab situations, since it will always differ from the way it works in nature.

It could, however, help us to understand more about the reliability of heritability estimates. If we know a trait is heritable by crossing experiments and after that we would make a parent-offspring regression or an animal model, this could help us to understand the way selection influences heritability estimates. Lab experiments could help us to understand a lot about all the biases and what to do about it, just like *Cooper & Zubek (1958)* and *Breeuwer & Werren (1995)* did, so it is important to also keep on doing lab work.

It also could help us to understand the assumptions behind our experiments. If there is no genetic variance in life-history traits, since the population is in equilibrium, there is no variation to select on, so a crossing experiment would not lead to new phenotypes or less or more adapted individuals. However when *Price & Schluter (1991)* are right, there should be variation to select on and so the experiment should lead to less or more adapted individuals.

Genomics

Genomics is the study of the whole genome instead of looking at every gene separately. The benefit of this is that one can measure all the genes that are involved in the expression of a trait and it also can learn us something about the interactions between the genes. For example, we want to know if there is a genetic background for body weight. First of all, DNA samples of a bird with a high body weight and a low body weight have to be collected. Of these samples, micro arrays are made. The genes that are expressed differently in the birds possibly have an effect on their difference in body weight. These genes are now called 'candidate genes'. Then there are

more DNA samples needed of birds with all kinds of different body weight and these need to be sequenced. Every candidate gene will be tested for a correlation with body weight. If these correlations are sufficiently different from zero, it can be concluded that there is a genetic background for the trait. A big advantage of this, is that there are no pedigrees needed to conclude something. This makes the work much less intensive than estimates of heritabilities.

Mueller et al. (2011) did such a research on blackcaps *(Sylvia atricapilla)* to find out the genetic background of migratory behaviour. They tested 17 candidate genes for a correlation with the trait. Only one candidate gene, ADCYAP1, showed a correlation. They also reported that this gene only explained about 3 percent of the variation. This is the biggest disadvantage of genomics: There are so many genes that have a role in the expression of a trait, that it is hard to find all of them and if one is found, it will only explain a little bit of the trait. The question is if this is enough to conclude that a trait is heritable.

Also, it still costs a lot of money to sequence DNA and even with up to date computers it is still hard to store all the collected data. After that, all the data still needs to be analysed, which is expensive in time and man power. When these problems with the technique will be solved and maybe analysis can be automatic, genomics can get an important role in evolutionary research. Tests should be improved in their sensitivity (ideally without making the error rates higher) to find more genes that are involved in the expression of a trait, so they can explain a higher percentage of the variation and help to conclude whether a trait is inherited and natural selection can take place.

Conclusion

Personally, I think that we should keep using heritability estimates for research on wild populations. However, I think it is important as well to not just calculate h^2, but also include CVa values in evolutionary studies. The combination of both tells us a lot more about the trait and the selection working on it, than the values apart. Just the CVa tells us more about the traits than just h^2, so we should definitely start using it, but there is already so much data on heritabilities that it would be a waste of data if we stopped using h^2 at all (think of comparing h^2 values of different populations, or within populations on different times). The combination can help us to figure out which traits are life-history traits for example. A low heritability and a high CVa suggest a trait is a life-history trait. However, a low heritability and a low CVa suggest that the trait is not inherited through genes. Also, it is important to keep all the biases in mind and not ignore them and to try to make these errors as small as possible. Genomics is a good tool for in the future, but now a days I think it is still too imperfect to use it a lot in wild studies. Crossing experiments in the lab are very important for our understanding, but cannot replace studies on wild populations.

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