About IQ-genes, GWAS, and a mathematical model for intelligence

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It is (almost) universally agreed that IQ is a highly heritable phenotypic trait largely caused by many genes, that is, polygenic, similar in this respect to height. The latest genome-wide association study (GWAS) on IQ genes found in total 52 IQ related genes [1]. They were estimated to explain some 5% of differences in IQ. The heritable fraction of IQ is usually placed between 60% and 80%. The higher bound comes from twin studies measured in adulthood. The lower bound could be 40% since in the childhood heritability is weaker than the home environment. This observation may mean that the environment has larger effect in the childhood, which sounds reasonable, but it may also mean that many IQ-genes have effect only in the adulthood and are sleeping in the childhood. This latter hypothesis is actually better. It must be so. Humans are less intelligent as children than as adults, though the genome does not change. It must be so that many IQ-genes become active after early childhood.

If 52 genes correspond to 5%, then, assuming that all IQ genes have the same explanatory strength, we need some 624 to 832 IQ genes in order to explain 60% to 80% of IQ by genes. There are several natural questions, such as the following:

1) If there are some 620 to 840 IQ genes, why researchers have only found 52 so far?
2) If there are some 620-840 IQ genes, why we do not see populations, where the average intelligence is, say, 20 standard deviations higher than in some other population?.
3) Why GWA studies on IQ suggest that there is only a small contribution from the proposed IQ genes? Is it mathematically even possible to have only a small contribution from the found IQ genes accepting that 60-80% of IQ is genetic according to other IQ research?

This article answers these questions and discusses some other specific issues.

Genome-wide association studies for a phenotype trait, such as IQ, find a set of SNPs (single nucleotide polymorphisms) that correlate with the phenotypic trait. A large set of potential SNPs is selected and the study needs a large population of individuals who have both the genome and the phenotypic trait measured. The study counts how often a certain SNP allele is associated with the phenotypic trait and how large effect it has on the phenotype. A small set of most effective SNPs is selected by imposing a selected threshold, and with this set one can calculate a polygenic score (PGS or PS) for any individual. Averaging over all individuals in a population allows comparing populations.

In GWAS of IQ, about half of the identified SNPs have been inside a gene and able to code proteins. What about those SNPs, which cannot code proteins? They are an artificial correlation created by the GWAS method. SNPs that actually can have effect must be inside a gene and able to code proteins. Much of our genome is in fact old junk of evolution, which does not any more code proteins. The fact, that the GWAS method produces artificial correlations, show that the results are not necessarily reliable. Without knowing that the SNPs are in non-coding regions, we would identify them as IQ-genes since there is the correlation. Correlation is not enough to prove causation. There must be IQ-genes, this everybody agrees, but they need not be those that genome-wide association studies have so far identified. Yet, the expectations are high that the newly found IQ-genes are real ones. We will accept this assumption in this article, if only for the purposes of discussion.

Let us construct a very crude first model for drawing some basic mathematical conclusions of the method. We will make several simplifying assumptions. Let us assume that the different SNPs are independent random variables which have two possible values: *H* giving a higher IQ and *L* giving a lower IQ. These are the alleles of the corresponding IQ gene and everyone in the population must have one of these alleles. There could be more than one SNP in a gene, but we will assume there is one IQ SNP in each IQ-gene, just to make the model simpler.

At the moment we will not consider the fact that a human has two copies of each gene: one from each parent, and can have one *H* and another *L* allele. We will add this essential part to the model a bit later. Now we assume that every individual has only one gene and it can have only two alleles. We will also assume that for every SNP the effect, or value, of the *H* allele to the IQ of an individual is the same *v*, and the *L*-allele has no effect on the IQ. We will also assume that for every SNP the penetration of the *H*-allele in the population is equal and has the value *p*.

With these simple assumptions we define the polygenic score of an individual *i* as

$$PGS_i = \sum_{j=1}^{n} \beta_j x_{i,j}$$

where $\beta_j = vp$ is the value times the penetration of the *H*-allele of SNP_j. Here $x_{i,j} = 1$ if the individual *i* has the *H*-allele of SNP_j, and $x_{i,j} = 0$ if the individual *i* has the *L*-allele.

Le us select $n=841=29^2$ as the number of IQ SNPs. It is close to the higher bound of 80% heritability for IQ supported by the twin studies, and it is a square for convenience. Under these simplifying and not quite realistic assumptions the probability that an individual has more than *m H*-alleles is quite well approximated by the normal distribution, assuming that *n* is large enough. The number *n*=841 is sufficient, thus with a fairly good precision:

$$\Pr[\#H \geq m] = \int_t^{\infty} e^{-\frac{1}{2}u^2}\, du \text{ where } t = \frac{m - np}{\sqrt{np(1-p)}}.$$

This is of course just one form of the central limit theorem: enough Bernoulli trials tend to the normal distribution. The value *t*=1 gives the probability of individuals being below one standard deviation (SD), and *t*=*k* below *k* SDs. Thus, 84% of the population has *t* at most 1 and the number *m* of *H*-alleles differs from *np* by maximum $\sqrt{np(1-p)}$, so *np* is the average and $\sqrt{np(1-p)}$ is the standard deviation. The standard deviation in IQ is usually taken as 15 and if 80% of IQ is genetic, the genetically inherited IQ has the standard deviation 12. This 12 must then be the result of the 841 IQ genes. The effect of having $\sqrt{np(1-p)}$ more *H*-alleles of the IQ-genes than the average value *np* gives the individual the genetic IQ of 112. (This figure is modified by the 20% environmental IQ.) The effect of one IQ gene, i.e., one *H*-allele of one SNP, on the intelligence is therefore $12/\sqrt{np(1-p)}$. As $\sqrt{p(1-p)} \leq 0.5$ and *n*=841, the effect is at least $12/(29*0.5) = 0.83$. We come to the third question posed in the beginning: the effect of one IQ-gene is at least 0.83 IQ points, it does not seem so small. Why is it claimed that the effect is small?

Our model has many simplifications, but they are not the reason for this result. The situation is that if some trait is determined by *n* independent random variables, like the SNPs, then the variation of the sum of these variables is normally distributed under rather light conditions and it varies on the scale of $\sqrt{n}$. If the total effect of the sum of these random variables on some trait is quite large, like a standard deviation of 12 IQ, and we want each random variable to have a very small effect, like for instance 0.1 IQ points, then to have one SD over the average requires the sum of 120 random variables, which is on the scale of $\sqrt{n}$.

In out model $\sqrt{n}$ would have to be at least 12/(0.1*0.5)=240. Then *n* must be on the range of 57.000. Just to mention, a human only has about 20.000 protein coding genes.

Clearly, it is not mathematically possible to have a very small IQ effect for one *H*-allele in this sense. What seems to be meant by a very small effect is something else. In genome-wide association studies one is measuring the phenotypic trait, let it be for instance by forming pairs of individuals where the first has IQ one SD higher. Then one is looking at the SNPs and calculating in how many of these pairs the individual with higher IQ has more *H*-alleles of the selected set of SNPs. If the study has selected 52 *H*-alleles, each in its own gene, and there are in total 841 IQ-related *H*-alleles, and all have the same effect on IQ, then the result is that the 52 *H*-alleles explain 52/841 part, 6%, of the genetically heritable IQ differences. As we have assumed IQ is 80% heritable, these 52 *H*-alleles explain 4.8% of IQ differences. Just like the GWAS article announcing having found 52 IQ-genes [1] stated, the genes explain 5% of the IQ variation. From that follows that each *H*-allele explains 4.8/52=0.09% of IQ differences, so basically 0.1% as in the example above. Thinking in this way each IQ-gene has a very small effect, but at the same time this means that in case two individuals differ by these particular 52 *H*-alleles, the one, which has 52 *H*-alleles more, has at least 52*0.83=43 points higher IQ. Seen in this alternative way, the effect of a single *H*-allele is large. The third question in the beginning has been answered. It was just of the way to understand the world small in this context.

Let us next think about the second question. Why we do not have populations with IQ ten or twenty times higher than somewhere else? Why the differences are not larger than 1 or 2 SDs? The same question can be posed concerning height: height is a highly heritable polygenic trait and human populations differ by average height, thus, why do they not differ more than they do? Giants and dwarfs usually have a disorder of the growth hormone. They are not typical to any populations. The smallest are the Pygmies and the tallest are the Dutch, the difference is not that large. Is there some mathematical problem with large differences?

Let us see with the IQ case. Let there be two populations, one where the frequency of *H*-alleles of IQ genes is p_1 and another where it is p_2, p_s being larger than p_1 and the average np_2 for the second population being *t* standard deviations larger than the average np_1 for the first population:

$$np_2 = np_1 + t\sqrt{np_1(1-p_1)}\,.$$

This means that the second population has 12*t* points higher average IQ. In order to use this formula for the second population, we have made the assumption that it also has normally distributed values for the number of IQ genes in an individual. Especially this means that the second population is not formed by selecting individuals from the first population in a way that does not produce normally distributed values.

Let us try with the value p_1=0.5. Then $np_2 = 420.5 + 14.5t$. There are 841 IQ genes, thus the number *t* cannot be larger than 29, but why indeed we could not find a population where *t* would be close to 29? The average IQ of such a population would be 29*12=348 IQ points above the average of the first population. Clearly, no such super smart population can be found anywhere on the Earth, yet we are able to make a selection of individuals with a high *H*-allele count, or by IQ-tests, and eventually get to such huge differences in the average IQ, that is, assuming that p_1 is around 0.5 or smaller. It is intuitively clear that such an experiment will not succeed. Had such methods been successful, some racist dictator would have created a super race already. The highest to this direction are the 4 million Chinese in Singapore and the 6 million Jews in the USA. The IQ of neither population goes more than one SD over the IQ 100 of whites. We should put some mechanism to our model, which stops increasing IQ too much by selection. This can be made by assuming that p_1 is very close to one already. It is in fact a quite natural solution: why should high IQ genes not have been

selected by evolution? If they are useful, they should have nearly complete penetration in a population.

How much can we increase the average IQ by selection? Clearly, at least 15 points is possible, there are the Chinese in Singapore and the Jews in the States. It would seem possible to select individuals with IQ 5 SD over 100, why not? Then, if IQ is 80% heritable, according to the breeder´s equation, see [5], the descendants have IQ 4 SD above the original average. This is where the regression to the mean stops, or at least JayMan says so [5].

We get a population with the average genetic IQ of 148 and can add some 10 points from environmental factors. A smart population of average IQ 158, it is roughly as far as we can get with selection. Going higher than this will first face the problem of finding women with so high IQ, but going up still, we cannot find enough men either.

Realistically, we surely can put an upper bound for t as 6. This upper bound can be enforced in our model by requiring that if t=6, then p_2=1. Setting these requirements allows us to solve p_1:

$$841 = 841\,p_1 + \sqrt{841\,p_1(1-p_1)} \quad \text{i.e.,} \quad p_1^2 - 1.958951\,p + 0.958951 = 0\,.$$

We get p_1=0.9589524. This is about 0.96 as this simple model does not need many decimals.

We see that this simple model gives us some insight. The reason we cannot by selection raise the average IQ very much is that most individuals already have the *H*-alleles of the IQ genes. Why, indeed, should they not? What is the genetic advantage of not being intelligent? We maybe can see the genetic advantage of not growing much larger or staying much smaller than humans now are. Larger size necessitates finding more food and small size animals are prey for larger carnivores. There is a balance. With the brain energy consumption there probably also is a balance. This means, the brain size has some optimal range. There apparently is a positive correlation between the brain size and IQ, but the more cleaver people are not especially known to be consuming more food than the less cleaver. Very possibly there is no special disadvantage of having *H*-alleles of IQ genes in our simple model. As a result, penetration *H*-alleles, that the population genome contains, grows close to one and if it is not close to one, there are specific reasons, such as that the allele has been obtained from another population rather recently and the penetration is still growing. This is, in our simple model, to be precise. Not in the real world.

At this point we must revise our simple model. It is too simplified and cannot describe the recessive advantage, which limits the penetration to a number below one. In reality an individual inherits two copies of each gene, one from each parent. Assuming that each IQ-gene has only one SNP and each SNP has *H* and *L* alleles, we have three cases for the genome of an individual: LL, LH and HH. Let $x_{i,j}$ get the values 0, 1, 2, where 0 corresponds to LL, 1 to LH and 2 to HH. We will also drop the requirement that each H-allele has equal effect v on intelligence and that each H-allele has the same penetration p. For each SNP_j we allow different numbers v_j and p_j.

These extensions to the model do not change that much: as long as n is large and effect of one *H*-alleles is sufficiently small, the distribution still tends to normal. With these changes the model is rather realistic. We can study a situation where the LH case gives an IQ advantage, while the HH case has a negative effect on reproduction. This is the recessive advantage situation. In the recessive advantage the penetration of a gene cannot reach 100% since that would imply that all are homozygous (HH) and have the disadvantage. There is a simple equation that theoretically should give the penetration. If the fertility of individuals having two *L*-alleles (LL individuals) 100*s percent and for HH individuals 100*r percent of the fertility of LH individuals, $0 \le s, r \le 1$, then the population size in the next generation is

$s(1-p)+p-p^2+rp^2$. The parts in the sum are the descendants of LL, LH and HH respectively. The population size is maximized by $p=(1/2)(1-s)/(1-r)$. Theoretically this should be the penetration, because a subpopulation with this penetration grows fastest and replaces other subpopulations. The practice is more complicated.

IQ-genes with recessive advantage have been proposed in [3] for explaining the high intelligence of Ashkenazi Jews. No later research has verified that the genes they proposed have any genetic recessive advantage, but let us note that the recessive advantage does not need to be genetic. The Romanov royal lineage suffered from hemophilia, but there was a strong social recessive advantage: after all, healthy carriers of the disease were royals. Likewise, belonging to a privileged (for instance, ethnic) group can be a sufficiently strong social recessive advantage that it can keep the disease in the genome.

We notice that the *L*-allele may be seen as a harmful gene. In a population where most individuals have the *H*-allele of most IQ-genes, such harmful *L*-alleles are rare. They are the reason for very low IQ, while the reason for very high IQ is having more of the quite common *H*-alleles. We can say that very high IQ is caused by the same mechanism that causes normal IQ, while very low IQ is caused by a different mechanism: possessing one or more rare IQ-lowering genes. Such an observation has been made in [2] based on empirical data.

Some populations may have different numbers of *H*-alleles. It may be that Europeans have more *H*-alleles than Africans, and that Europeans also have new harmful *L*-alleles, which Africans do not have. If so, then IQ 70 in Europe can be an indication of a harmful IQ-lowering gene and the effect can be a serious handicap in mental tasks, while IQ 70 in Africa can indicate that the individual has few *H*-alleles, but it does not imply that he has harmful alleles. The effect on mental abilities can be much milder. Their situation may be similar to that of children. Children have the IQ-increasing genes in their genome, but they are not active yet. They do not have rare IQ-lowering genes. This is like having fewer *H*-alleles. It may be quite fitting to compare such adults to children rather than mentally handicapped.

Let us finally address the first question in the beginning using the insight from our simple model. That the researchers cannot find the IQ genes means that the IQ-related SNPs do not come up in the association study as explanatory factors for the IQ differences. The study has set a threshold, the P-value. The 52 IQ-genes pass the threshold, the others do not. Let us assume that almost all in the population have the *H*-alleles of the IQ-genes because that is the most natural explanation why selection cannot increase average IQ too much. Let us also accept the average value p_1=0.96, which was calculated. Then the average number of *H*-alleles in an individual is 807. The very highest IQ individuals in a population have only 34 *H*-alleles of IQ-genes more than what the average person has. The standard deviation is $\sqrt{np_1(1-p_1)}$ is 5.68 and it is difficult to find individuals higher than 5 SD, which means 28 genes. By checking another population with a different history, we may find another 20 or so IQ genes. The achievement of finding not less than 52 IQ genes from a large study of 74.000 individuals fits well to these considerations, but why did they not find more?

Individuals, who differ from the average by SD, do not have the same 5.68 different IQ-genes. They usually have different IQ-genes, and if all IQ-genes had as strong effect on IQ, the study would have found all 841 IQ-related SNPs. It must be so that the majority of the IQ-related SNPs have still smaller effect and there are much more than 841 IQ-genes. In order to make a simple model, let the 52 genes have three times bigger effect on IQ than the other IQ-genes, thus each of the 52 can be counted as three IQ-genes and together they represent virtual 156 genes. We can take a round number $2500=50^2$ as the number of IQ-genes, of which 156 are the part of the 52 IQ-genes and 2344 are the weaker genes that were not found yet. Actually there are 52+2344=2396 IQ-genes in our revised model, but they behave as

2500 genes. The standard deviation $\sqrt{np_1(1-p_1)}$ is then 9.8 genes, but most of the IQ-genes are not found as they fall below the threshold.

All three questions in the beginning have been answered at least in some way.

Let us look at some specific issues, as promised. Earlier we mentioned that the second population must have normally distributed values for the number of IQ-genes in an individual and this restricts the selection procedure. Let us now look at the effect of a selection procedure, which does not fill this requirement. We form the second population by selecting from the first population those who have higher than average IQ. As such a selection cannot measure the heritable IQ, it must use the phenotypic IQ, which has the standard deviation 15 and which includes the environmental factors. The average IQ value of the selected population is obtained as

$$\int_{-\infty}^{+\infty} f_1(z)z\,dz = \int_{0}^{+\infty} f_1(z)z\,dz$$

where $f_1(z) = 2\frac{1}{\sigma\sqrt{2\pi}}e^{-\frac{1}{2\sigma^2}z^2}$ is the normal distribution density multiplied by 2 in order to normalize the total probability to one. The integral is easily calculated

$$\frac{1}{\sigma}\sqrt{\frac{2}{\pi}}\int_0^\infty e^{-\frac{1}{2\sigma^2}z^2} z\,dz = \frac{1}{\sigma}\sqrt{\frac{2}{\pi}}\left(-\sigma^2\right)\int_0^\infty e^{-\frac{1}{2\sigma^2}z^2}\left(-\frac{1}{\sigma^2}z\right)dz = -\sigma\sqrt{\frac{2}{\pi}}(0-1) = \sigma\sqrt{\frac{2}{\pi}}$$

The average is:

$$\int_{-\infty}^{+\infty} f_1(z)z\,dz = \sigma\sqrt{\frac{2}{\pi}} = 0.798\sigma = 11.97 \quad \text{if } \sigma = 15\,.$$

The variance for this distribution is also easy to calculate:

$$\frac{1}{\sigma}\sqrt{\frac{2}{\pi}}\int_0^\infty e^{-\frac{1}{2\sigma^2}z^2}\left(z-\sigma\sqrt{\frac{2}{\pi}}\right)^2 dz = 2\frac{1}{\sigma}\sqrt{\frac{1}{2\pi}}\int_0^\infty e^{-\frac{1}{2\sigma^2}z^2} z^2\,dz - \sigma\frac{1}{\sigma}\frac{1}{2\pi}\int_0^\infty e^{-\frac{1}{2\sigma^2}z^2} 2z\,dz$$

$$+\sigma^2\frac{2}{\pi}\frac{1}{\sigma}\sqrt{\frac{2}{\pi}}\int_0^\infty e^{-\frac{1}{2\sigma^2}z^2} dz$$

$$= \sigma^2 - \frac{4}{\pi}\sigma^2 + \sigma^2\frac{2}{\pi} = \sigma^2\left(1-\frac{2}{\pi}\right).$$

The standard deviation is thus reduced from 15 to 9, but this reduction is not noticed if the variance is measured from the part of the distribution, which is above 100, and the other part of the distribution is missing. Instead, what is noticed is that in the second population there are twice as many individuals, proportional to the population size, who exceed any high IQ level, such as 150, as in Terman´s study of gifted children started in 1921. The average IQ of 111.97 will regress in the next generation to 109.6 assuming that IQ is 80% heritable and that the population inbreeds.

It should be apparent that these figures closely resemble what is known of the IQ of American Ashkenazi Jews. The average IQ is the US Ashkenazi has been estimated to 112-115 or even higher, but according to Richard Lynn, 107.5-110 are better justified figures. The suggestion, that the high IQ of American Ashkenazi Jews is a result of selection, is supported by the following facts.

Firstly, the Ashkenazi Jews do not have the same IQ everywhere, unlike whites, who practically everywhere seem to have about 100, excluding the Irish and some other outliners. In Israel Ashkenazi Jews have IQ of 103.5 and they have 103.5 also in the Balkans. The high value of 110 is obtained in the USA and GB. In the Balkans, Sephardic Jews have the same average IQ as Ashkenazi Jews. Apparently European Jews of both main types have IQ over

100. The Mizraim Jews from Muslim countries have IQ of 90 and the Ethiopian Jews still lower, but these populations also differ genetically from European Jews. Selection or environmental factors are the most natural ways to explain the range of IQs for European Jews in different countries. Secondly, in Terman`s study of gifted children in the IQ tests made 1919-21 the Jewish subpopulation had twice as many children exceeding 150 than what was expected from the subpopulation size, compared to the white majority. If was not about ten times as many, what would have been expected if the Ashkenazi Jews had had the average IQ of 110 and a normal distribution with SD=15. Thirdly, there apparently was a selection of US Ashkenazi Jews by IQ tests from 1921 to 1939, as I explained in another post [4].

What will happen to the IQ f such a selected population in a longer time? It must finally tend to the normal distribution. If the average has been raised over the normal average, it may seem that the standard deviation must become a bit smaller, as the individuals of the base population already have very high penetration of IQ-increasing genes. But this may not be what happens. The likely result is that the distribution of IQ is for a long time un-symmetric, there are less very intelligent, and this lack of symmetry pushes the average lower. It may just be so that JayMan in [5] is incorrect stating that the regression to the mean stops to the first generation of the offspring. That conclusion is justified by one model, if the model is more refined, it may give another prediction.

We can conclude this section by noticing that selection probably explains the IQ difference of the USA Jews to the white non-Jews. Selection also explains Chinese in Singapore and many other high IQ ethnic minorities in the USA, like the USA Hindu minority with average IQ of 105 selected from India with the average IQ 84, or the USA African minority, which also has very high IQ. The effect of selection is rather well understood and is not a question of controversy. We can ignore these cases.

The controversial issue of IQ differences between races is related to IQ differences found by studying populations which have evolved to their own directions without much interbreeding for some 50.000 years. These populations have had different environmental pressures and they have developed different genes. The main question has been how these people can manage in the modern society. Can they manage in school as well as other populations, can they find jobs and can they avoid social problems_

The three main races, Sub-Saharan Africans, Caucasians, and Mongoloids, are not homogeneous and they are mixed. They are not proxies to what humans were 50.00 years ago. It is much better to focus first on certain very old hunter-gatherer societies, notably aboriginal Australians, San and Pygmies. These very old hunter-gatherer societies have always had a small number of individuals. There cannot have been as many IQ-improving mutations as in bigger societies – the rate of mutations depends on the population – and the evolution pressure for higher IQ has been low since the environment has stayed similar for a long time. We may expect that these populations have had much fewer IQ increasing mutations for some 50.000 years and they may even give the baseline of what was the intelligence of humans when they left Africa.

According to Richard Lynn, these populations have the lowest IQ levels of all human populations: aborigines` average IQ is 64 and San (Bushmen) and Pygmies have still slightly lower IQ. Pygmies have small bodies and consequently also smaller brains. San, like Khoi (Hottentots), have some ancient features, like steatopygia. The brains of aboriginal Australians are smaller than those of Caucasians but they have a larger visual-spatial cortex. In tests aborigines can remember better than whites what was in a room and this skill helps them to find their way in the wilderness. The 52 IQ-related genes, which were identified from European people in [1] do not include aborigine genes, which increase visual cortex and improve visual memory. There are population specific IQ-genes, though the advantages they give may not help much in a modern society. What is known from Paleolithic European

hunter-gatherers is that they did not have small brains. They had a larger brain than modern humans and the brain has been decreasing for the past 30.000 years. It is plausible that the brain was smaller when these people left Africa, since the aborigines have smaller brains. In Europe they faced a more challenging climate. In East-Asia the environmental demands of cold weather were even higher, which can be seen e.g. in mongoloid eye shape. Lynn`s theory, that colder climate has been the main reason for increases in IQ, fits quite well all that is known.

The decrease of the brain size in the last 30.000 years may be an effect of taming. There is a similar decrease in brain size in all tamed animals compared to their wild ancestors. If this is the case, the large brains of Paleolithic European hunter-gatherers were needed for surviving in the wilderness with low-level technology. We may assume that these people had superior visual memory. They may have had eidetic memory, since some people have this capability and such a memory does not combine well with better reasoning. There may have been a shift from superior visual memory to superior visual reasoning and planning. The difference in IQ between children and adults may be largely in planning capabilities.

In Lynn`s theory the reason why one population has higher intelligence than another is not mainly the selection of IQ-genes that both populations have. The main reason is that one population has new IQ-increasing mutations, which some populations do not have. Thus, the higher IQ of Caucasians and East Asians is explained by cold weather, which caused genetic changes in the form of new mutations. This seems to be supported by Piffer`s IQ GWAS studies [6]. Piffer calculate polygenic scores from a small number of IQ-related SNPs. Sub-Saharan Africans had PGS about 300, while Northern Europeans had about 500. If the small number of SNPs can be considered a random sample of IQ-genes, then this result agrees with Lynn`s theory. However, they may not be an unbiased sample. The SNPs have been selected from European population and they may downplay African specific IQ-genes. But assuming that Piffer`s PGS is a reasonably good sample, we can approximate the mutation rate for IQ.

Some 50.000 years ago IQ was 60. We know that Piffer`s PGS value 300 for Africans corresponds to the African IQ of 70 and Piffer`s PGS value 500 for the British corresponds to the standardized IQ of 100. It means that the IQ of aborigines, 60, corresponds to Piffer`s PGS of about 250, so in 50.000 years PGS grew 250 points. That means doubling of intelligence in 50.000 years. Assuming that there are some 2500 IQ-related SNPs, there would have been 1250 IQ-related SNP mutations. The mutation rate depends on the number of people. We must estimate the size of the European population for the last 50.000 years. This is of course very uncertain, but:

50.000 - 15.000 ybp, doubling time 35.000 years.
15.000 – 8.000 ybp, doubling time 3.500 years.
8.000 – 700 ybp, doubling time 1.825 years
700 – 100 ybp, doubling time 600 years.
100 – present, doubling time 50 years.

The present population is some 400 million. In 1900 it was 100 million. In 1300 it was 50 million. We estimate four doublings from 8000 ybp to 700 ybp. Thus in 8000 ybp there were about 3 million. With two doublings from 15.000 ybp to 8.000 ybp, there were 0.75 million in 15.000 and 0.375 million in 50.000 ybp. Calculating a generation as 25 years, in 35.000 million there are 1400 generations, about 0.5 million each. That is 700 million humans. From 15.000 to 8.000 ybp there were 280 generations, each some 2 million. That gives 560 million humans. From 8.000 to 700 ybp there are 292 generations, each some 25 million. That is 7.3 billion. From 700 to 100 ybp there are 24 generations, each some 70 million. That is 1.7 billion. Finally from 100 ybp to present there are 4 generations, each some 200 million. That is 800 million. Together the sum is 11 billion humans in Europe. There are estimates that in

all times there have lived some 100 billion humans. The estimate for Europe may be reasonable.

Assuming that there have been 11 billion people and 1250 IQ-related mutations, we have the mutation rate as 113.6 $*10^{-9}$. We can compare this mutation rate to the proposal in [3] that the Ashkenazi Jews have obtained in 900 years several mutations which increase IQ. The size of the Ashkenazi Jewish population over the 900 years should be estimated. It is reasonable to assume that this population grew four times larger in 100 years.

1150	296
1250	592
1350	2.368
1450	9.472
1550	37.890
1650	151.563
1750	606.250
1850	2.425.000
1950	9.700.000

The sum of the figures is 12.933.431. There are 4 generations in each 100 years and the average size of the generation from the year x to the year x+100 is something like twice the population in the year x. Thus, the total number of European Ashkenazi Jews was roughly 26 million. The estimated number of IQ-related mutations in Ashkenazi Jews would be about 3. At the same 900 years there lived some 350 million Europeans, who had some 40 IQ-related mutations. This example shows why the high intelligence of American Ashkenazi Jews and similar groups is easier explained as a result of fairly recent selection than by long term mutations. Naturally it is possible that IQ-related mutations would have been lost in Europeans and become common in Jews, but there is little reason for such an assumption. Apparently Europeans have been collecting IQ-related mutations for a long time, and very probably many of them after 8000 ybp.

Finally, let us return to the question why the IQ differences between human populations stay in a rather narrow range? Even the hunter-gatherer societies have IQ 60, with education probably 70, that is 2 SD under the British standard. There may be a reason for it. Humans may already be almost as cleaver they will ever be. There may be little to gain from any IQ-related mutation. It may also be that there is considerable recessive advantage of having genes, which lower IQ. This advantage is not genetic in the strict sense, but it may be so that some families have genes for beauty, nice character etc. which get selected, and at the same time some harmful genes get selected. It is just possible that genetic IQ cannot be much raised and all human populations are rather close to this bound.

Who knows?

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