Musings

From: "JSeltzer"
To: <cangen@cody.library.manhattan.edu>
Date: Tue, 24 Feb 2004 14:04:07 -0500
Subject: [Cangen] Re: Evolution caught in the act

Quoting from Susan's post: "Bardwell likens the new pathway for disulfide bond formation to engineering. 'People often speak of Computer Assisted Design (CAD), where you try things out on a computer screen before you manufacture them. We put the bacteria we were working on under a strong genetic selection, like what can happen in evolution, and the bacteria came up with a completely new answer to the problem of how to form disulfide bonds. I think we can now talk about Genetic Assisted Design (GAD)."

I had the opportunity this month to attend lectures by two scientists with quite disparate takes on evolution and speciation.

Michael Behe, a professor of biochemistry at Lehigh University, spoke on "Darwin's Black Box" and what he calls the Irreducible Complexity of a number of biological mechanisms, e.g., the cascade of events that control the clotting of blood. The absence of a single link in the chain and the blood clotting mechanism fails resulting either in bleeding to death or turning the organism's entire blood supply into one big clot. Behe asserts that neo-Darwinism cannot explain the simultaneous evolution of both fibrinogen and plasminogen (the balance of which makes the system work). Like a mousetrap which (according to Behe) requires all its separate components to catch a mouse, so also the blood-clotting chain needs all its separate pieces. The mechanism is Irreducible Complex and mutations that led to fibrinogen had to occur simultaneously with other mutations that led to plasminogen -- a "statistically impossible" simultaneity. Hence, Intelligent Design -- hence, an Intelligent Designer ... an attempt using molecular biology to bring William Paley's Intelligent Design Thesis into the 21st century.

I suggested to Behe that simply because a biological structure appears to be Irreducibly Complex (and which neo-Darwinism cannot, according to Behe, explain) does not, by the process of eliminating a single hypothesis, leave the only answer of Intelligent Design. Indeed, there are other alternative hypotheses that must also be considered. I was surprised to find that Behe was unfamiliar with the Morphogenic Field hypothesis of Rupert Sheldrake. I was also surprised that Behe had not made any attempt to elucidate the information pathway(s) that any explication of the Intelligent Design thesis reasonably demands. When I asked if intelligence (information content as the alternative to the randomness assumed in neo-Darwinian evolution) was an intrinsic property of matter, perhaps infused at the time of the Big Bang, he could offer no response. IMO, a collision with an apparently inexplicable puzzle should not halt further inquiry. A god of the gaps is no god at all.

The other lecture was given by Niles Eldridge (the collaborator with Stephen Jay Gould on the punctuated equilibrium theory of evolution). Eldridge emphasized the evidence from paleontology that reveals long periods of relatively slow changes in most species followed by short periods with new species emerging and old species disappearing -- a long period of stasis followed by a brief period of intense speciation driven by abrupt environmental changes. This lecture, unlike Behe's, was well supported by a considerable amount of evidence.

Yet, I for one am still struck by the remarkable degree of plasticity inherent in the phylogenetic process. In the mere blink of an eye (on a geological time scale), large numbers of new species have emerged when driven by a major environmental shift -- simply because they must. As reported in the referenced study, in the much shorter span of time that it takes for a bacterial colony to either adapt or starve, a novel technique for generating disulfide bonds emerges from the latency of the bacterial genome -- simply because it must.

Is there something else going on here?

Jim Seltzer
Willowind Dalmatians

Date: Wed, 25 Feb 2004 16:22:41 -0500
From: "JSeltzer"
Subject: Re: sex linked traits/meiosis

Jane wrote: "I am trying to respond to something on Mastiff list about the x factor theory...it seems that it is being applied generally to all traits - not just sex linked. As if, "if in doubt, assume sex linked." The question was to do with deciding to breed back to a grandparent - which grandparent would be the better choice - paternal grandsire or the maternal grandsire? I can't get my head around this.
Please help. Am I wrong when I think that the x factor is WAY too narrow an approach to examining the likelihood's of which grandparent was the most influential on his granddaughter as a whole?"

"...So back to my original question - if the original cell contains a gene on a chromosome (other than on the x or the y) and this gene is a recessive for a negative characteristic - shouldn't the gametes that aquifer the x or the y have an equal chance of also acquiring this recessive gene?"

IMO, focussing on sex-linked attributes is too narrow a view. Consider the following categories:

http://www.genome.iastate.edu/edu/genetics/sexlim.html
"Sex traits can be categorized into three types of inheritance:
sex-limited, sex-linked, and sex-influenced.

"Sex-limited traits are traits that are visible only within one sex. For instance, barred coloring in chickens normally is visible only in the roosters.

"Sex-linked traits would be considered traits like sickle cell anemia and color blindness. They are said to be linked because more males (XY) develop these traits than females (XX). This is because the females have a second X gene to counteract the recessive trait. Thus, the trait is
more likely to be visible in the male.

"Sex-influenced traits are autosomal traits that are influenced by sex. If a male has one recessive allele, he will show that trait, but it will take two recessive for the female to show that same trait. One such gene is baldness."

Observe that sex-limited and sex-influenced traits are controlled by autosomal genes (these can derive from either parent), but the expression of these genes depends on the sex of the individual.

See, also,
http://webpages.marshall.edu/~adkinsda/B111OutlinesChromInhSxLink.html

Also, be aware that the expression a genetic trait can be controlled by one parent or the other by using genomic imprinting. Methylation of a cytosine nucleotide in the parental gamete can silence the expression of a gene. There is some controversy on the importance of this mechanism, but several known defects are traceable to its effects.

"Genomic imprinting can be loosely defined as the gamete-of-origin dependent modification of phenotype. That is, the phenotype elicited from a locus is differentially modified by the sex of the parent contributing that particular allele."

Do a GOOGLE search for imprinting or take a look at:
http://www.genome.org/cgi/content/full/8/9/881

Jim Seltzer, Ph.D.
Willowind Dalmatians

From: "JSeltzer"
Subject: Re: population needed for breeding?

Anna wrote: "Now for the question part of the email.... What is considered the needed population of breeding dogs, given that I'm sure out COI is skyhigh?"

This same question came up last year on this list. I answered as follows:

~~~~start repost~~~~~~~

As I understand your question you are asking how large a population must be to avoid the negative effects of random gene loss and loss of heterozygosity (LOH).

By way of backgound, you should know that there is a natural and unavoidable increase in the mean inbreeding for any finite breeding population, the smaller the population, the faster the increase. Various mate selection methods might cause a more rapid LOH, but random mating (Hardy-Weinberg assumptions) will give the minimum increase in COI per generation:

COI increase = 1/ (2N)

where N is the population size. Take away the random mating requirement and the formula becomes:

COI increase = 1/(2Ne)

where Ne is the "effective population size"

If there are Nm breeding males and Nf breeding females in the population, then

Ne = 4 Nm Nf / (Nm + Nf)

So what is the minimum Ne to sustain a "healthy" population? Professor Per-Erik Sundgren (Swedish University of Agricultural Sciences) proposes a 5-generation rule. The COI increase in 5 generations should be held to a maximum of 2.5% (0.5% per generation). You might find others with their own rules, but Sundgren's is as good as any.
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0.5% = 1/ (2Ne) or

Ne = 100

Observe that Ne is NOT the number of AKC-registered dogs. It is the effective population size and applies only to active breeders. So if there is only a small number of breeding males compared to the females, the Ne will be small (limited mainly by the number of males).

Mathematicians will recognize the quantity

2 Nm Nf / (Nm + Nf) as the harmonic mean of the numbers of breeding males and females.

~~~~~end repost~~~~~~~

Anna: "So, it has come up again- two (or more?) breeders are stating that we have enough dogs in the US to only use Black Russian Terriers that are cleared in hips and elbows. A total of nineteen dogs is enough? Out of those nineteen, two dogs are unregistered, five are over eight years old (two are over ten), effectively reducing the breeding population to fifteen or less."

Using Ne = 15, we obtain:

COI increase = 1/30 = 3.3% per generation. N.B. this is the inevitable generational increase in inbreeding (random mating assumptions) based on this population size. If the breed is already inbred, then this increase will further add to that value.

This is well above Per-Erik Sundgren's healthy population rule.

I suggest you try to find ways to enlarge the population base before becoming overly restrictive in your selection criteria.

Regards,
Jim Seltzer, Ph.D.
Willowind Dalmatians

2) the "type" of wild animals is usually more consistent than that of domestic ones. The reasons for that center around the phenomenon of "genetic buffering" which results from and is maintained by natural selection. There has recently been considerable discussion on this list about the reasons for the breakdown of genetic buffering among domestic animals, a breakdown which leads to greater phenotypic variability and then to attempts by breeders to standardize "type" by various breeding strategies, most of which involve various degrees of inbreeding and result in a loss of genetic diversity within the population. We discussed the apparent paradox that wild animals in general have a greater genetic diversity, but a more uniform phenotype, than domestic ones, and the implications of that paradox for breeding policies. All of that can be found, I believe, in the archives of this list, and reading some of it might prove informative ... <G>

3) the laws of genetics are the same for all animals, wild or domestic.

John
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Dr. John Burchard
Tepe Gawra Salukis