

Dear Committee Members- Dr. Bulla's article is well written and describes many of the reasons that clones are not phenotypically identical with the original and why clonemates will not be identical with each other. The only slight quibble I have is that the consensus seems to be that telomeres reset their length in cloned embryos, although this is hard to prove conclusively. The research showing that Dolly's telomeres were shorter than normal is not convincing to me- also, while Dolly died of a respiratory infection, so did the other sheep in that barn that were not clones.

None of this invalidates the main points of Dr. Bulla, that clones will differ from each other phenotypically, and sometimes have abnormalities; many of the mechanisms causing dissimilarity are similar to causes of differences between identical twins. One other point is that most of problems that Dr. Bulla discusses correct themselves in the next generation, that is the clones may have issues, but the information available suggests that the offspring of clones do not have such problems; for example, the population of sperm of a clone of a stallion will be essentially identical to the population of sperm from the original both genetically and phenotypically, and will produce similar offspring.

A consequences of the phenotypic differences among clones, often obvious when looking at clonemates, is that clones of race winners usually will not win races (phenotypic dissimilarity).. On the other hand, clones of sires whose offspring win races, will also sire race winners at similar frequency (genetic similarity).

I attach a paper with additional details that I wrote some years ago; the paper covers most of the issues that Dr. Bulla mentioned, and comes to similar conclusions. George Seidel

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