AN ANALYSIS OF INBREEDING IN THE EUROPEAN BISON¹

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DURING a study of inbreeding in man (SLATIS, REIS, and HOENE 1958) it became evident that the most striking consequences of inbreeding were increases in childhood death and in serious illnesses. It seemed worthwhile to investigate the death rates in the young of other large mammals. This would be of interest because large mammals fall into two sharply divided categories, (1) domesticated forms with a long history of inbreeding and artificial selection, and (2) wild forms none of whose ancestors have been subject to artificial selection.

It is known that some lethal genes exist in domesticated animals and one might predict that those species that have not undergone much inbreeding and artificial selection would have a higher frequency of recessive lethal genes. To gather information about wild species, data were sought from zoological gardens. The only satisfactory data appear to be for the European Bison, a species with no history of artificial selection, although with small population sizes in recent centuries.

MATERIALS AND METHODS

The pedigree books

The European Bison (occasionally known by its German name, *Wisent*), *Bison bonasus* (L.), had dwindled to two small populations by the middle of the nineteenth century. One subspecies, *Bison bonasus caucasius* Grevé, was limited to a region in the northwestern Caucasus Mountains, where it died out about 1930. The other subspecies, *Bison bonasus bonasus* (L.), was maintained in the forest of Bialowieza, Poland, until poachers extinguished the herd in 1922. The Caucasian subspecies was always completely wild, rarely even being observed. The herd at Bialowieza was carefully protected but was never subject to artificial selection. Within historical times, neither subspecies has been numerous.

The International Society for the Protection of the European Bison was established after the slaughter of the herd at Bialowieza and the near extinction of its largest daughter herd. Records were collected on the birth, death, and ancestry of the few living animals. These data were published in 1932 in the *Berichte der Internationalen Gesellschaft zur Erhaltung des Wisents*, and subsequent data have been published under this title (1932–1937) and more recently under the title of *Pedigree Book of the European Bison* (1949–1958). This paper will analyse the information contained in these volumes through the issue of 1958, which includes all data through 1954. The records give internal evidence of being carefully

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kept. Each animal, including stillbirths, is identified by a name and a number. For the purposes of this paper, only numbers will be quoted. These recording methods were adopted around 1924, and 553 animals born after 1 January, 1925 (animals with numbers above 140), have been used for analysis. Two other animals were omitted because of a lack of satisfactory information.

All living purebred European Bison are the descendants of a basic group of 17 animals whose parents are not accurately known. One of these (#100) was a bull born in the Caucasus Mountains in 1907 and brought to Germany as a calf. Its descendants represent the only Caucasian influence on the modern species, all of the other genes being derived from the Bialowieza herd. One female (#2) was born in the Berlin Zoo about 1883 of unknown parentage (presumably tracing back to Bialowieza). Seven animals (#85, 86, 89, 95, 96, 122, and 123) were taken from the herd at Bialowieza between 1903 and 1913. A daughter colony of the Bialowieza herd at Pszczyna, Poland (Pless, Germany, prior to 1919), was the birthplace of the other eight animals (#1, 7, 16, 32, 33, 42, 45, and 46), all born between 1881 and 1918. At least some of these Pszczyna animals (if not all) were more closely related to each other than to animals picked randomly from the Bialowieza herd. It is probable that most of the Pszczyna animals were inbred.

Although the Bison go back to 17 animals, their genetic constitution represents recombinations of only 12 diploid sets of genes. Animals #1 and 2 had an offspring, #3, which mated with #7 to produce #15, and these animals are represented in the modern species only through the descendants of #15. Similarly, one pair of Pszczyna animals (#32 and 33) and one pair of Bialowieza animals (#85 and 86) are represented through single offspring (#35 and 87, respectively). One other pair of Bialowieza animals (#122 and 123) is represented only through an inbred descendant (#147). Thus, the 213 living European Bison can present a maximum of 24 alleles at any locus, except as mutations may have increased this. The two indexes of inbreeding that will be used do not distinguish between pedigrees that go back as far as they can be traced and those that stop at the last ancestor to which two or more separate lines can be traced. Therefore, it is of no consequence whether the calculations are made with respect to the genes carried by animals #15, 35, and 87, as was actually the case, or their ancestors, #1, 2, 7, 32, 33, 85, and 86. However, because #147 was inbred, it is necessary to trace descent from #122 and 123. Thus, the species has been treated as derived from a "foundation herd" of only 13 animals (see Table 1), all of which were alive during the latter part of 1918 and most of 1919.

These 13 genomes are unevenly represented in the living animals. Table 1 indicates the proportion of the genes of the 213 Bison living on 31 December, 1954, which traces back to each of the foundation animals. Six animals come within 25 percent of the mean, 0.077. However, #46 contributed only about a tenth of this mean, and the combined contribution of #122 and 123 is equally small (five eighths of this represents the genes of #122 and three eighths, those of #123). Two other animals are just below half of the mean value.

Animals #42 and 45, survivors of the predations of poachers on the herd at

TABLE 1

The genetic contribution of the foundation herd animals to the living species

Animal	Proportion
Born at Bialowieza	
(# 85 + 86) 87*	.072
# 89	.096
# 95	.038
# 96	.063
#122 + 123 (147)*	.009
Born at Pszczyna	
(# 1 + 2† = 7) 15*	.082
# 16	.091
(# 32 + 33) 35*	.027
# 42	.188
# 45	.264
# 46	.009
Born in the Caucasus Mountains	
#100	.061

* The preceding animals are represented only through this descendant (see text). The 13 animals not in parenthesis formed the foundation herd.

† Born in the Berlin Zoo.

Pszczyna, founded a remarkably inbred herd by themselves in 1922. Their genes now represent 0.452 of the genetic constitution of the species (this value was about 0.50 when 17 animals, representing the major herd descended purely from this pair, were wiped out by foot-and-mouth disease in December, 1953). Twenty-nine living and 75 dead animals have no foundation herd ancestors other than #42 and 45, while only 52 living animals do not trace back to this pair through any ancestral line. No individual traces back to all of the foundation herd animals and only a few represent all but two of the 13, though many represent all but three. The Caucasian animal, though contributing only six percent of the genes of the species, is an ancestor of 70 percent of the living individuals, having almost as many descendants as #42 and 45. All of the other foundation herd animals are less frequently ancestors of the living animals, down to #122 and 123, who have only 18 living descendants (8.5 percent of the species).

Measures of inbreeding

The standard coefficient of inbreeding, F (WRIGHT 1921), has been calculated for each animal. Similarly, for each animal a value has been calculated which is being designated as ll . This is the *likelihood of genetic death* if each ancestor in the foundation herd possessed a single recessive lethal gene, l , and if each of these lethals was at a different locus. For these purposes, a "lethal" gene will be understood to mean a gene which kills late enough in embryology to be recorded as a stillbirth, or which kills liveborn animals prior to the onset of reproductive maturity (about the second birthday, in Bison). Penetrance and recessivity are assumed to be complete. If a set of records were to include information on fertility, the frequency of genes causing sterility could also be computed.

Although ll is not a direct measure of the quantity of inbreeding in the ancestry of an animal, it may "measure as directly as possible the effects to be expected" from the observed inbreeding (WRIGHT 1922). On this definition, it seems reasonable to refer to ll as an inbreeding coefficient.

The characteristics of ll

F and ll are both inbreeding coefficients, but they are only slightly related. When one is zero, the other must also be zero. If a given ancestor is assumed to have been Ll and leads through a separate and uncomplicated line of descent to each of the parents of a particular animal, the value of ll will be half of the F value due to this same ancestor, since F measures homozygosity for L and for l , but ll measures homozygosity for the latter only. However, if the lines of descent are not separate or if one or more of the intermediate ancestors is also inbred with respect to the given ancestor, then the value of ll will be less than half that of F. Several examples of the calculation of ll will aid in understanding this.

Figure 1 shows the known ancestry of #220. Animal #173 has an F value of

FIGURE 1.—The pedigree of animal #220.

FIGURE 2.—The pedigree of animal #828.

0.25 and a ll value of 0.125. Animal #220 has an F value of 0.375 and a ll value of 0.1429. The calculation of the ll value was performed in the following manner. We assume that animal #42 is heterozygous for a lethal at one locus and that #45 is heterozygous for some other lethal. Since #220 can be homozygous only for the lethal in #45, the lethal carried by #42 will not be considered. We know that the probability that #173 would have been homozygous for any lethal carried by #45 is $1/8$. Since #173 was a viable animal, it was not homozygous for the lethal, and so had $4/7$ probability of being heterozygous and $3/7$ probability of being homozygous normal. Thus, #220 had $1/4$ of $4/7$ probability of being a homozygote, and her ll value is $1/7$, or 0.1429.

The fact that there is not a direct relationship between F and ll values may be seen from Figure 2, which indicates these values for the pedigree of #828, a highly inbred descendant of #42 and 45. It will be instructive to observe the calculation of the ll values of #256 (and also #546, her full brother), for which $F = 0.375$, $ll = 0.1339$. Her sire, #195, had $1/8$ probability of being homozygous for a lethal carried by #45. As a viable animal, he would carry such a lethal $4/7$ of the time. Only in half of these instances would her dam, #49, have been carrying the lethal also, and #256 would be ll $4/7$ times $1/2$ times $1/4$, or 0.0714 . We must add to this the probability of the lethal carried by #42 being homozygous in #256, which is $1/8$ of the $1/2$ chance that #49 is ll , which is $1/16$, or 0.0625 . Therefore, the total ll value for #256 is 0.1339 . Similarly, the ll values for #547, 701, and 828 are compounds of the ll values for lethals carried by both #42 and 45. It is clear in this pedigree that an increase in F can be accompanied by a decrease in ll and that the changes are not closely correlated.

For small values of F , there is a definite correlation with ll in these data. However, for the 199 animals with F equal to or greater than 0.2500 (this point being chosen arbitrarily as the lowest F value for which any observed ll value exceeded 0.10), there is actually a significant negative correlation between F and ll , r being -0.205 .

It will be observed that as a measure of inbreeding, ll is defined as the *sum* of the ll values for each ancestor taken separately. Thus, in the example given above, the ll values were calculated separately for descent from #42 and from #45 and these two values were added together. In practice this is by far the simplest thing to do but it is not quite accurate. The true value of ll in the above example is 0.1295 , rather than 0.1339 , as can be computed by designating #42 as $AaBB$ and #45 as $AABb$ and assuming either homozygous recessive to be lethal. In the example of Figure 1, if #45 had himself been the offspring of two animals of the foundation herd, then the ll value of #220 as calculated in this paper would still be 0.1429 , but the true ll value would be 0.1450 . The difference between the summed ll value and the true ll value is dependent upon the occurrence within the pedigree of ancestors who might have been inbred for lethals from a foundation herd animal. As can be seen from these two examples, the true value may be either greater or less than the summed value. The calculation of the true ll value often is excessively complex. For this reason, the technique of summing the separate ll values has been adopted. The discrepancy that results is never very large. The following paragraph pertains only to the summed ll values, rather than to the true values.

The maximum value of ll will occur after repeated backcrossing to a heterozygous ancestor. In this situation, ll approaches 0.1464 asymptotically (F approaches 0.5). The beginning of such a pedigree is shown in Figure 1. As the intensity of inbreeding increases in inbreeding other than backcrossing, F approaching 1.0 , lethal genes become infrequent. The ll value will be only a small fraction of the F value, as can be observed in Figure 2. The maximum value of ll in a nonbackcrossed organism will occur in the mating of sibs whose parents

are of unrelated, outbred stocks. In this instance, $ll = 0.125$ ($F = 0.25$). Larger ll values occur in organisms in which self-fertilization is possible, but special cases will not be discussed here.

As has been noted, the ll value assumes that the genes are completely penetrant with respect to lethality. If one were to calculate the mortality produced in a pedigree such as that shown in Figure 1 by a gene which killed half of the individuals homozygous for it, the ll value for #173 would be 0.0625, or half of the value under complete penetrance, but the value for #220 would be 0.0833, or more than half of the value under complete penetrance. This example demonstrates the general rule that for pedigrees in which there are no inbred ancestors, incompletely penetrant genes produce mortality equal to the ll value multiplied by the penetrance. (This is true for F as well as for ll and this might be the reason for the usefulness of the theory of lethal equivalents in the uncomplicated human pedigrees studied by MORTON, CROW, and MULLER (1956). In that paper, F was the inbreeding coefficient ostensibly employed, but it was modified to be identical with ll .) The example given above also demonstrates that if at least one inbred ancestor occurs in a pedigree, the mortality produced by a semilethal gene will be greater than the ll value multiplied by the penetrance of the gene. The precise effect of this will depend on the complexity of the pedigrees and the distribution of semilethal genes in the population.

Thus, semilethal genes will have a serious effect on the ll calculations. In relation to ll values they will contribute to lethality in a variable manner, rather than in a manner directly related to their penetrance. This will tend to raise the estimate of the number of lethal equivalents carried by the average foundation herd ancestor. There is no simple way of correcting for this effect. However, the difference between F and ll will be shown in the discussion to have a bearing on this problem.

Treatment of the data

Each animal has been classified according to its life span. There are three categories, (1) perinatal death, which includes stillbirths and deaths within one month of birth, (2) juvenile death, which includes all deaths before the second birthday other than those in category (1), and (3) survival past the second birthday.

The records list 119 animals that were not inbred (F and ll are zero). Of these, 15 died in the perinatal period. However, two animals probably died because of foot-and-mouth disease. These two deaths were essentially nonselective in nature, since it appears that this disease usually kills all of the animals in a stricken herd. Other foot-and-mouth deaths plus many deaths associated with the end of World War II have also been classified as nonselective. Statistically, it seems appropriate to treat the young animals that clearly suffered nonselective deaths as if they had not died at all. Within any period prior to maturity, their life experience would approximate that of half as many animals surviving the period (i.e., two calves dying of clearly nonselective causes within the perinatal period would roughly

represent the same experience of life as one calf that survived this period). Thus, the outbreds are treated as if there had been 13 deaths (all deaths not classified as nonselective) among 119 minus half of two, or 118 animals, for a rate of 0.1102 perinatal deaths. There were 104 animals that survived to one month of age and of these, eight might have died of selective factors and four clearly died of the afore-mentioned nonselective causes. In addition, one outbred animal is less than two years of age. The four nonselective deaths and the animal which has not yet completed the age period are treated identically, half being classified as having survived the period, so that there are eight deaths among 104 less 5/2 animals (8/101.5), or 0.0788 juvenile deaths. Each class of inbred animals has been treated in the same manner. The frequencies of animals surviving the perinatal and juvenile periods have been shown in Table 2 according to their degree of inbreeding.

RESULTS

For reasons to be noted in detail in the discussion, it is important to discover the relationship between degree of inbreeding and premature death. For this purpose, it was decided to calculate the change in death rate as the degree of inbreeding changes, which is the regression of death rate on degree of inbreeding. This regression has been calculated for (1) the total population as classified by F , (2) the total population as classified by ll , (3) the total population as classified by ll except for the omission for each animal of any contribution to ll due to descent from #100 (the Caucasian bull), and (4) only that contribution to ll due to descent from #100. Because F measures homozygosity for normal alleles as well as for lethals, it is necessary to double the value of the regression of death rate on inbreeding as calculated for F to make these results comparable with the calculations based on ll . The values cited in Table 3 have been corrected in this manner.

These regression coefficients have been calculated separately for perinatal and for juvenile death. In each of these categories the calculations have been performed in two manners. The first of these is a least squares estimate made with the data of Table 2. A standard error of this estimate may be obtained as

$$\frac{\sigma_y \sqrt{1-r_{xy}^2}}{\sigma_x \sqrt{N-2}}.$$

The value of σ_y can be assumed to be $\gamma(1-\gamma)$, which is the standard

deviation of a proportion. Since the correlation between x and y , r_{xy} , is assumed to be small (but is not assumed to be zero), the second term in the numerator can be disregarded as not being very different from one. The terms in the denominator are easily obtained from the data. The standard errors indicate that a significant relationship between death rate and degree of inbreeding exists for all categories except (1) perinatal death as measured by F and (2) juvenile death as measured by ll when excluding the contribution due to descent from #100.

A second estimate of the regression is based on a pooling of all the data for inbred individuals at their mean value. The regression is calculated from the change in the frequency of death over the difference between outbreeding (F and ll as defined for the category equal zero) and the mean level of inbreeding.

TABLE 2
Relationship between degree of inbreeding and frequency of early death

Mean F value	0.0	.025	.075	.125	.175	.225	.275	.325	.375	.425	.475	.525	.575		
Perinatal death															
Frequency	.1102	.0000	.1467	.1649	.1000	.0426	.1458	.2162	.0962	.1667	.0952	.1111	.0000		
n	118	3	75	48.5	60	47	48	37	52	30	21	9	3		
Juvenile death															
Frequency	.0788	.5000	.1789	.1316	.1373	.0563	.2000	.1091	.0909	.2174	.0645	.1667	.4000		
n	101.5	2	61.5	38	51	35.5	35	27.5	44	23	15.5	6	2.5		
Mean ll value	0.0	.015	.025	.035	.045	.055	.065	.075	.085	.095	.105	.115	.125	.135	.145
Perinatal death															
Frequency	.1102	.1053	.1071	.1212	.1739	.0222	.0909	.1190	.1852	.0500	.2308	.1667	.1818	.0000	.0000
n	118	19	28	33	80.5	45	55	42	27	20	39	24	11	9	1
Juvenile death															
Frequency	.0788	.2222	.1818	.0741	.1440	.1316	.1176	.2154	.2000	.0571	.1111	.2500	.0000	.0000	.0000
n	101.5	13.5	22	27	62.5	38	42.5	32.5	20	17.5	27	20	9	9	1

TABLE 3
The regression coefficient of death rate on degree of inbreeding

Index of inbreeding	Population	Perinatal death	Outbred vs. inbred	Juvenile death	Outbred vs. inbred
		Least squares $\pm$ S.E.		Least squares $\pm$ S.E.	
F*	Total	.024 $\pm$.060	.137	.140 $\pm$.068	.505
<i>ll</i>	Total	.320 $\pm$.120	.252	.283 $\pm$.135	.930
<i>ll</i>	Exclusive of descent from #100	.266 $\pm$.120	.344	.155 $\pm$.135	1.052
<i>ll</i>	Descent only from #100	2.526 $\pm$.722	— .628	4.992 $\pm$.834	13.723

* All values in this row have been doubled.

In addition to these two methods, an estimate of each regression has been obtained by use of the method of maximum likelihood, as employed by MORTON, CROW, and MULLER (1956). This method gives a somewhat more exact answer than is given by the usual method of least squares. However, the data are transformed in a manner which tends to make the regression coefficients as determined by the method of maximum likelihood greater than by the method of least squares. Because of the ease of calculating a standard error with the least squares method, the maximum likelihood results have not been used and are not given.

With the exception of one pair of twins, the Bison have produced only single-born offspring in captivity. The measure of female fertility was to assume that one birth is possible for each completed year of life after the third. Only a few animals that have been observed have lived long enough to pass out of the fertile period, and on the whole, the females have been kept in situations where yearly breeding was always possible. The outbred females averaged 0.4638 births per year in this fertile period and the inbred females averaged 0.4623 births. Obviously there is no evidence for a loss of fertility among inbred females. Similarly, there appears to be no evidence for a dependence of early mortality on the inbreeding of the dam. In fact, perinatal deaths had a frequency of 0.150 among the offspring of outbred females but only 0.098 among those of inbred females. Juvenile deaths dropped from 0.130 among the offspring of outbred females to 0.123 among those of inbred females.

DISCUSSION

Most geneticists believe that of the deleterious recessive mutations arising in a population, some are approximately neutral in heterozygous condition. Other genes that have a deleterious recessive effect exist in populations because they are favorable in heterozygotes. However, there are at present two conflicting schools of thought concerning the usual effects of heterozygosity and homozygosity. One group believes that the vigor of crossbred individuals is due largely to the covering of harmful recessive genes (dominance theory). The other group believes that at many loci the possession of two different alleles is advantageous over being homozygous for the best available allele (overdominance theory). CROW (1952) has discussed the evidence for each of these points of view.

Where inbreeding is complex, there is not a strong correlation between F and II . These measures of inbreeding may aid in distinguishing the two possible causes of the effects of heterozygosity. F is a measure of the increase in homozygosity after the initiation of inbreeding. Although the use of F is not related to overdominance theory, if it is correct that loss of heterozygosity will be related to loss of vigor and therefore, to early death (overdominance theory), then F should be a relatively direct index of early death. On the other hand, II is a measure only of the homozygosity of genes that are being heavily selected against. Therefore, II will be a relatively direct index of the early death that is due to the action of recessive lethal genes (dominance theory).

In addition to the above, difficulties introduced by the existence of semilethal

genes are differentiated by F and ll . Strong semilethals would behave much like completely penetrant lethals, and so would agree in their effects more with the ll value than with the F value. Semilethal genes of low penetrance would, essentially, be responsible for a lowering of viability of animals with a high F (and a relatively low ll) value. The existence of genes of this type in moderate numbers would be a major mechanism contributing to the apparent success of F as a predictive index of viability.

In Table 3, one may observe that there is a positive relationship between inbreeding and early death in the European Bison. It is reasonable to assume that this relationship is real, being another instance of the deleterious effects of inbreeding. If one of the indexes of inbreeding is more closely correlated with early death than is the other, it will suggest that the chief mechanism behind the inbreeding-death relationship is through the theory that is represented by the appropriate index.

The regression coefficients in Table 3 have been estimated in two ways. One method has been to compare the average increase in frequency of death with the change from outbreeding to the average amount of inbreeding, and the other method has been to calculate the regression of frequency of death on degree of inbreeding. A good index of inbreeding should order the animals in such a manner that these two methods would give very similar values. The only close approximation between these two methods is for perinatal deaths when ordered for ll value. The F estimates for perinatal death are relatively distant from each other. There is only a slightly better agreement between the ll estimates for juvenile death than between the F estimates.

These observations indicate that ll has, in one instance, described the relationship between inbreeding and death in a better manner than has F . Both ll and F have failed to describe the inbreeding-death relationship for juvenile deaths, largely because of the low death rate among the most highly inbred animals. As will be noted in the next few paragraphs, the most highly inbred animals are largely of a single strain which might by chance have had a very small number of recessive lethal genes. If, as seems possible from these pedigrees, highly inbred strains can be developed without appreciable loss of vitality, then it will become clear that dominance is the major mechanism of hybrid vigor. A similarly simple explanation of the low mortality of an inbred strain is not reasonable on the theory of overdominance.

The least squares fit of the ll data indicates that the average number of recessive genes causing perinatal death is of the order of 0.3 per foundation herd animal and a similar average number of such genes causes juvenile death. However, the uneven contributions of the various foundation herd animals to these calculations makes it advisable to break down the data according to the individual animals, where possible. The most interesting animal in this respect is #100, the Caucasian bull. He, representing a separate subspecies, might possibly have differed from the other animals to a great extent. Two separate tests have been performed in comparing this animal with the others. Firstly, the regression of frequency of perinatal and juvenile deaths on ll value has been recalculated

disregarding the contribution of #100. There has been a decrease in the slope of both lines, and particularly in the slope of the line for juvenile death. These results have been checked by a calculation of the relationship between death and that part of the ll value contributed by #100. The perinatal deaths show a slope of 2.5 and the juvenile deaths show a slope of 5.0. These values can not be considered as more than suggestive. However, they indicate that the Caucasian Bison almost certainly was heterozygous for genes that were lethal to homozygotes in both the perinatal and juvenile stages, and that the latter were more numerous.

Some of the animals born prior to 1925, and therefore not included in this analysis, independently confirm the presence of lethal factors in #100. The only offspring of #100 by one of his daughters died in the juvenile period, and of the four instances in which an offspring of #100 was mated with one of its own progeny, one died in the perinatal or juvenile period.

The small regression coefficients for the total ll values after elimination of the contribution of #100 make it difficult to observe any evidence for lethal genes among the other animals. Animals #42 and 45 have been studied for the apparent number of recessive genes that they carried. About half of the total ll value of the species is due to this pair. The regression of frequency of death on ll value are slightly positive: 0.105 ± 0.119 for perinatal death and 0.165 ± 0.131 for juvenile death. These small regression coefficients are not significantly different from zero. (In making these calculations for #42 and 45, only truly out-bred animals were listed for $ll = 0.00$. In the last two rows of Table 3, all animals that are not inbred by the definition of the category are listed as having $ll = 0.00$. The method of analysis is different because inbreeding for #100 is positively correlated with early death and negatively correlated with inbreeding for #42 and 45.)

The regression of death rate on inbreeding will not be cited for any of the other animals. The ll values for descent from other foundation herd Bison are strongly correlated with ll values for descent from #100. However, the regression of death rate on ll values for the combination of all animals exclusive of #100 is not much different from that of #42 and 45 alone. This would indicate that the remaining animals collectively have only a minor relationship between death rate and inbreeding coefficient.

Reference to Table 1 will demonstrate that about 70 percent of the non-Caucasian genes of the living Bison are derived from the herd at Pszczyna. This herd was founded in 1865 by the importation of a bull and three cows from Bialowieza. One can assume that during the next few years the inbreeding was intensive. In 1893, another three bulls and two cows were imported from Bialowieza. The only other addition to the Pszczyna herd was the acquisition in 1900 of a bull that is thought to have been both the son and grandson of #1, who was himself a product of this herd.

The inbreeding in the Pszczyna herd may have done much to eliminate the lethal genes carried by the Bison of Bialowieza. The Pszczyna herd numbered about 70 animals during the 1890's. The animals added at that time would not

have changed the composition of so large a herd drastically, though they would have increased the average number of lethal genes per animal above the level to which it had fallen through inbreeding. It would seem that #42 and 45 (born about 1904 and 1917, respectively) may have been free of lethal genes. In addition, Pszczyna-born animals #1 and 16 are each recorded as having had three viable offspring by one of their own offspring, indicating that neither of them was carrying a large number of recessive lethal genes.

Unfortunately, satisfactory records do not exist for an evaluation of the average number of recessive genes carried by the animals listed in Table 1 as born in the Bialowieza herd. Most of their inbred descendants are also heavily inbred for genes from animal #100. Records exist of 11 highly inbred descendants of the Bialowieza animals (F at least 0.25, I at least 0.10), all born by 1926, and of these, six died in the perinatal period. Thus, the number of lethal genes carried by individuals taken from the herd at Bialowieza was not great enough to preclude the survival of some highly inbred descendants. These results are consistent with an estimate that the average Bialowieza animal was carrying of the order of half a dozen fully penetrant recessive lethal genes or lethal gene equivalents.

It is clear that the process of mutation must be such that new mutants are being formed in each species with great regularity. The completely recessive mutants that are unfavorable in homozygous condition will be selected against in a manner which keeps their frequency roughly constant. In a small population, such as the ancestral populations of these Bison, there undoubtedly were many potentially mutant loci which were not represented by mutant alleles at any given time. However, for all such loci taken collectively, the Bison undoubtedly carried sufficient mutants to give the average animal a number of recessives. The data for the Caucasian animal estimate (with little accuracy) that he carried of the order of seven recessive lethal genes. The Bialowieza animals probably had fewer than this number. The Pszczyna animals obviously had very few recessive lethal genes. A remarkably high intensity of inbreeding in the first years of the Pszczyna herd might explain its later lack of lethals. Furthermore, because the number of recessive genes per animal varies randomly, there is the possibility that the original bull sent to Pszczyna fortuitously had a small number of lethal genes. This would help explain the success that this herd had in starting with small numbers while other small herds did not thrive and survive.

It is of interest that the average number of recessive lethal genes carried by a wild Bison in the forests of Poland or the Caucasus Mountains appears to have been about six and this number is similar to the number of recessive genes found to cause death or serious illness in man (SLATIS, REIS, and HOENE 1958).

SUMMARY

The European Bison provide data which can be utilized for a study of the effects of inbreeding. At the end of 1954 the species contained 213 living animals but it can contain at most only 24 alleles at any locus, exclusive of very recent

mutations. A small proportion of the genetic ancestry of the species traces to a subspecies which lived in the Caucasus Mountains, a larger proportion traces directly to a large herd in Poland, and most of the genes appear to have passed through an inbred subline of the Polish herd.

A new index of inbreeding, ll , has been devised. The effects of inbreeding as measured by this index were compared with the effects as measured by the standard index of inbreeding, F . The relationship between the degree of inbreeding and the frequency of perinatal and juvenile death was found to be slightly greater for ll than for F . The two indexes of inbreeding treat the data differently in that ll measures the effect of homozygosity for recessive lethal genes while F measures the loss of heterozygosity. It appears from this analysis that the deleterious effects of inbreeding may be due more to the presence of recessive lethal genes in wild, outbred ancestors than to the need of the individual to maintain a high level of heterozygosity; that is to say, that dominance rather than overdominance may be the main mechanism of hybrid vigor in these mammals.

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