Ann. Zool. Fennici 34: 167–175
Helsinki 18 September 1997
ISSN 0003-455X
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Developmental stability and the harbour seal epizootic in 1988

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Received 10 October 1996, accepted 16 December 1996

Developmental instability of harbour seals (*Phoca vitulina*) that died during the epizootic in 1988 in the Kattegat was compared with that of the population prior to the epizootic to investigate whether animals that died were the developmentally less stable individuals in the population. As an assessment of developmental instability the fractal dimension of the paired *maxillo palatinae* suture was calculated and the degree of fluctuating asymmetry in teeth, foramina and the paired suture was estimated in a total of 240 skulls. Moreover, prevalences of three pathological changes of the skull were compared. No difference was found in developmental instability or prevalence of skull lesions between animals that died in the epizootic and estimates from the population prior to the epizootic. The lack of difference could be in accordance with the suggestion that organochlorines had an influence on mortality during the epizootic. Levels of organochlorines have decreased in the last decade before the epizootic, and thereby could have equalized differences in levels of developmental stability.

1. Introduction

Minor developmental upsets, for example random differences in an enzyme concentration, are ubiquitous and they are magnified by the complex nature of growth processes and, unless controlled, the result is a disturbance of pre-determined growth. Examples include minor random departures from symmetry in bilaterally paired characters, which are produced by the same gene complexes and undergo the same patterns of development in the same environment. Different buffer mechanisms exist to correct such perturbations. Environmental or genetic stress may increase the frequency of random minor departures from pre-determined growth both indirectly by disturbing the buffer mechanisms, but also directly by increasing the frequency of developmental upsets (Emlen *et al.* 1993). Developmental stability is the ability of an individual to buffer its development against disturbance and to resist stress of environmental and genetic origin (Palmer 1994, Møller & Swaddle 1997). For example, developmental stability was reduced in laboratory rats exposed to prenatal or postnatal stress in the form of noise, cold or heat (Siegel & Doyle 1975, Siegel & Mooney 1987, Siegel *et al.* 1992).

Estimates of developmental instability are considered a more sensitive approach when evaluating the condition of natural populations (Clarke

1995) than more commonly used methods, such as estimates of fecundity. A range of studies (reviewed in Møller & Swaddle 1997) have shown an association between developmental instability and different fitness components such as growth rate, fecundity and survival. This has led to the suggestion of using developmental stability for assessing the quality of the environment as perceived by free-living organisms (Clarke 1995, Møller 1996). Moreover a positive association between developmental stability and general health has been reported in a wide range of organisms (Møller 1996, A. P. Møller & R. Thornhill unpubl.), and developmentally stable individuals are believed to be more fit than less stable individuals (Møller 1996).

Fluctuating asymmetry (FA) is the most commonly used index of developmental instability (Palmer & Strobeck 1986). The small random departures from symmetry in bilateral characters are referred to as asymmetry, and asymmetry in a group of individuals (for example a population) is referred to as FA. The magnitude of FA reflects the ability of individuals in a population to buffer their development. It is used as a tool to evaluate the condition of individuals in natural populations (Jones 1989). For example magnitudes of FA increased in the fin rays of grunion *(Leuresthes tenuis*) when exposed to increasing levels of DDT (Valentine & Soulé 1973).

When diseases hit populations of animals it is generally expected that the less fit individuals are mostly affected. In 1988, a *Morbillivirus* called *phocine distemper* virus caused an epizootic among harbour seal (*Phoca vitulina*) in northern Europe. Other more well known morbilliviruses are those causing measles and rinderpest (Blixenkrone-Møller 1993). Many seals died from complications caused by infections existing prior to the epizootic. I expected that individuals that died during the epizootic had reduced developmental stability compared with the survivors. The high mortality of 60% in the Kattegat population could be merely an effect of *phocine distemper* virus (PDV) (the primary cause of the disease) being introduced into a population without specific immunity to the infectious agent (Heide-Jørgensen *et al.* 1992). However, the less fit individuals were probably more vulnerable to the complications of the virus infection because of higher levels of infections prior to the epizootic. Many individuals died from complications of the virus rather than the virus itself (Baker & Ross 1992).

This paper presents a study of three predictions. First, individuals killed by *phocine distemper* virus infection in 1988 were developmentally more unstable than survivors. A direct comparison between individuals that died and survivors would have been the ideal, but this was impossible due to sparse museum material of survivors. Therefore, the animals that died in the epizootic were compared with the population prior to the epizootic. Few immature compared with mature individuals and many more males than females died in Kattegat than expected from other studies of diseases in harbour seals (Härkönen & Heide-Jørgensen 1990). Therefore, second, males were expected to be developmentally less stable than females, and, third, mature individuals in the population prior to the epizootic to be developmental less stable than immature individuals.

To investigate the three predictions I used four different estimates of developmental instability in seals. First, developmental instability was estimated from FA in teeth dimensions. Second, foramina in the skull, which are small openings in the skull for nerves and blood vessels, were used as another estimate of developmental instability. Pathological changes due to skull lesions, which occur in seals, are in some cases difficult to distinguish from the morphological changes that result from disturbance in development. Therefore, I only used a restricted number of easily recognizable paired characters to estimate developmental stability. Third, it is possible to calculate an empirical fractal dimension for a skull suture because these demonstrate scales of self-similarity. Self-similarity means that the parent pattern is repeated on different scales (Long 1985). I used the fractal dimension of skull bone sutures as an index of developmental instability. This has been recommended by Alados *et al.* (1996), who used this method to assess developmental instability in inbred populations of two species of gazelles, and moreover diseases have been associated with loss of fractal dimension (Honda *et al.* 1992, Escos *et al.* 1995). Fourth, I used the difference in fractal dimension between the right and the left *maxillo palatinae* suture as an estimate of developmental instability.

The prevalence of two skull lesions has been studied in seals from Kattegat (Mortensen *et al.* 1992). The first resembles parodontitis in humans, while the second is alveolar exostosis. I investigated the prevalence of these two pathological changes in addition to the prevalence of enlargement of foramen mentalia. This was used as a measure of the condition of the animals.

2. Materials and methods

The skulls of harbour seals (*Phoca vitulina vitulina*) from the Zoological Museum, Copenhagen (CM), and the Swedish Museum of Natural History, Stockholm (SM) were used. Table 1 presents the material. All the skulls originated from nature reserves in Kattegat which holds a single population (Härkönen & Heide-Jørgensen 1990). A total of 240 skulls (in 17 cases only the mandibles) from two periods were examined: (i) 1955–87: Animals from the population prior to the epizootic that were born in a period with high levels of pollution. 63 individuals were shot for systematic collection for CM, while 52 individuals drowned in fishermans gears, and (ii) 1988: Animals collected during the epizootic, most individuals were (based on manner of death) expected to have died from PDV (M. Olsson pers. comm.). Subadults have been born in a period of decreasing levels of pollution (Olsson & Reutergårdh 1986). Information on age and sex was obtained from index cards at CM and from P. Mortensen (SM). Morphological characteristics of mandibles and teeth, as described in Allen (1902) and Doutt (1942) were used to estimate age, when data were unavailable. On the basis of these data and estimates specimens were classified into three age groups: (i) cubs: 0–0.5 year, (ii) subadults: 0.5–5 years and (iii) adults: more than 5 years.

2.1. Developmental stability measures

Teeth, foramina and sutures were used to assess developmental stability. A digital caliper was used to measure the maximum mesiodistal lengths (transition between the crown and the root) of the 2nd and the 3rd molar in the right and left side of upper and lower jaws to the nearest 0.01 mm (illustration given in Schandorff (1997)). The magnitude of FA was estimated from the difference in length between each bilateral pair of teeth. To estimate measurement error the measurements were replicated on two subsequent days. A two-way ANOVA (sides × individuals) was conducted on data on teeth to test for the significance of FA relative to measurement error (following Palmer & Strobeck 1986). The test asks whether the difference between sides vary more among individuals than would be expected, given the size of the measurement error. The interaction MS containing information about FA (and anti-asymmetry) was tested against error MS (reflecting measurement error) showing that FA (anti-asymmetry) was significantly larger than measurement error in all cases ($0.024 < (\text{interaction MS}) < 0.111$, $0.001 < (\text{error MS}) < 0.002$, $26 < df < 69$, $P < 0.001$). No measurements were attempted on broken or worn teeth.

The difference in number between sides of six bilateral pairs of foramina was used to estimate the magnitude of FA (illustration given in Schandorff (1997)). The numbers were counted macroscopically. Each pair of foramina was counted twice on 46 skulls on two subsequent days to estimate measurement error (%ME). According to Palmer (1994), there is no appropriate way to test for the significance of FA relative to counting error in meristic traits. The measurement errors were calculated as the sum of differences (in FA) between first and second count relative to the sum of FA. *F. posterior palatinae* %ME = 7; *F. hypoglosi* %ME = 0; *F. dentale* %ME = 0; *F. basiorbitalis acessorius* %ME = 11; *F. dorsal condylar* %ME = 14; *F. palatinae minor* %ME = 29. *Foramina palatinae minor* was excluded because of the high %ME.

The pattern of the left and right *maxillo palatinae* sutures was drawn on standard size (28 × 20 cm) paper from slides taken at the same focal distance (30 cm). The patterns were copied to double size before measurements. The walking rulers method (Emlen *et al.* 1993) was used to calculate the empirical fractal dimension of the sutures. Lengths ($L(y)$) of the suture pattern were measured with dividers adjusted to 8 cm, 4 cm, 1 cm and 0.5 cm. The divider was used to measure the length of the line in a series of steps, each step following the curvature of the line as precisely as possible. The length of the line was recorded as the number of steps multiplied by the adjusted yardstick. The length of the suture pattern increased when length of the yardstick (y) used for measurements decreased. The fractal dimension (D) was calculated as:

$$L(y) = ky^{(1-D)} \quad (1),$$

by regression of log ($L(y)$) on log$y$ over four values of y. A similar closure of the sutures was ensured by using only adult specimens. The whole session was replicated for 20 sutures on two subsequent days to estimate percentage of measurement error, which was 0.038% (original measurements).

Table 1. Sex and age distribution of harbour seal skulls from the Kattegat.

Period	Adults			Subadults			Cubs		Total
	Male	Female	Sex unknown	Male	Female	Sex unknown	Male	Female	
1955–87	14	11	–	36	36	–	8	10	115
1988	31	37	13	12	19	11	1	–	124

The other method, which was based on fractal dimensions in the *maxillo palatinae* sutures, was an assesment of developmental stability from the difference between the right and left fractal dimension in the same skull. Using FA data from seven individuals the measurement error was estimated to be 17.9% in a one-way ANOVA with individuals as the fixed factor, and where n is the number of repeated measurements per individual (Merilä & Björklund 1995).

2.2. Pathological changes of the skull

The pathological changes were diagnosed after macroscopical examination of each skull. Type 1 was exostosis (apposition of bone) localized to the part of the lower jaw supporting the teeth (*processus alveolaris*), especially in the area of the molars. A detailed description and illustration is given in Schandorff (1997). Type 2 was a pathological change with external characteristics of parodontitis, which means more or less severe loss of alveolar bone tissue around the teeth and in other parts of the jaws (illustration in Schandorff (1997)). Often teeth were missing. Following Mortensen *et al.* (1992), loss of alveolar bone tissue around a minimum of one tooth to a degree where bony support was lost, was used as a criterion. Type 3 was a pathological change characterized by smooth-edged enlargements of foramen mentalia. An enlargement more than four times the size of the other dental foramina was used as a criterion.

2.3. Tests for FA

The guidelines for FA analysis described by Palmer (1994) were followed.

2.3.1. Test for departures from normality and for size dependence

For data on teeth the two-way ANOVA (sides × individuals) used to test for significance of FA relative to measurement error was also used to test for directional asymmetry. For data on foramina and fractal FA a mean left minus right character value equalling zero was tested in a one sample t-test (small samples were tested in Kolmogorov-Smirnov tests (Sokal & Rohlf 1981)). Other types of departures from normality were tested in two one-sample t-tests carried out on skewness and kurtosis, respectively. To test for size dependence in teeth a regression analysis of the magnitude of asymmetry and the average tooth size was conducted. Dependence of fractal FA on fractal dimension and absolute age, respectively, was tested in a regression analysis.

2.3.2. Test for difference between age classes, sexes and periods

It is recommended to use multible indices of FA whenever it is possible, because different indices are sensitive to different factors (departures from normality, outliers). I used three different indices of FA: FA4, FA5 and FA10 (see below) of Palmer and Strobeck (1986) to estimate levels of FA in teeth. To estimate the level of fractal FA and level of FA in foramina only FA4 was used.

$$\text{FA4: } \sigma^2 (R - L) \quad (1)$$

$$\text{FA5: } \Sigma (R - L)^2/N \quad (2)$$

$$\text{FA10: } \sigma^2 (\text{MS}_{sj} - \text{MS}_m)/M \quad (3)$$

(Note: R and L are right and left side measurement, respectively, FA10 is an index ruling out measurement error (MS_m), σ^2 = variance, MS_{sj} = mean square (side × genotype) from two way ANOVA, $M(m)$ = number of replicate measurements, s = number of sides, j = number of individuals.)

Differences between age classes, sexes or periods were compared with an F-test (Fowler & Cohen 1990). Moreover a two-way ANOVA (samples × traits) of the variation in $(R - L)$ for multiple traits per individual on data on teeth was used to compare differences between periods (Palmer 1993). To test for a statistically significant difference among periods and sexes for fractal dimensions a Mann-Whitney U-test (Fowler & Cohen 1990) was used. To test for differences in prevalence of pathological changes between different periods a G-test (2 × 2 contingency table) with Williams correction was used (Fowler & Cohen 1990). A sequential Bonferroni test was applied (Rice 1989) when more than 5 related tests were conducted to avoid significant results arising as a consequence of a large number of tests.

3. Results

A detailed presentation of descriptive statistics is given in the appendix in Schandorff (1997).

The two-way ANOVA (sides × individuals) showed that there was no significant directional asymmetry in data on teeth ($0.745 <$ (side MS) < 1.169, $0 < F < 4.375$, $0.32 < P < 1$).

In some cases departures from normality in teeth measurements and foramina counts occurred. However these variables were included since it was found that the departure was due to one or two extreme values. The regression analyses of teeth size versus magnitude of FA showed that the magnitude of FA in teeth was independent of overall teeth size in all cases (regression analyses conducted on each trait in each period: $-0.074 < r < 0.086$, df = 14–68, $0.16 < P < 0.94$). Furthermore, in the regression analysis of fractal FA versus fractal dimension no significant correlation was found (regression analysis: $r = 0.027$, $df = 50$, $P = 0.82$). Some results have been pooled

for sex and/or age, after it was assured that the polling did not affect the results of the statistics.

3.1. Differences in developmental instability between 1955–87 and 1988

Animals that died in 1988 were not developmentally less stable than those of the 1955–87 sample (Table 2). The ANOVA were non-significant for teeth data, and level of FA do not show any consistent tendencies of being higher in one period than another. This is also seen in the few statistical significant *F*-tests, that yield opposite results. Also no significant difference in mean fractal dimension was found (mean (*S.E.*): 1959–87: 1.055 (0.002), $N = 46$; 1988: 1.052 (0.004), $N = 46$). A significant difference was nevertheless seen in fractal FA, indicating that individuals from 1988 were more developmentally stable than those in the 1955–87 sample (1959–87: 0.0008; 1988: 0.0002, $F = 4.00$, $df = 38$, $P < 0.01$).

3.2. Sex differences in developmental instability

A difference in developmental instability between sexes was not seen in 1955–87 or 1988 (Table 3). No consistent tendency for either sex to show higher levels of FA (as reflected in variances and *F*-tests) is seen. Furthermore, no difference was seen when comparing fractal dimensions (mean (*S.E.*): male: 1.053 (0.003), $N = 15$; female: 1.053 (0.002), $N = 13$). However, a significantly higher

Table 2. Magnitude of asymmetry of skull characters of harbour seals in 1955–87 and 1988. Values are variances. Test statistics are *F*-tests.

Index – trait	1955–87	*N*	1988	*N*	*F*	*P*
FA5 – lower 2. molar	0.112	28	0.060	33	1.87	< 0.05
FA5 – lower 3. molar	0.025	33	0.025	41	1.00	*n.s.*
FA5 – upper 2. molar	0.039	29	0.076	45	1.95	< 0.05
FA5 – upper 3. molar	0.040	28	0.028	39	1.43	*n.s.*
FA10 – lower 2. molar	0.050	28	0.030	33	1.67	< 0.05
FA10 – lower 3. molar	0.012	33	0.012	41	1.00	*n.s.*
FA10 – upper 2. molar	0.020	29	0.038	45	1.90	< 0.05
FA10 – upper 3. molar	0.020	28	0.015	39	1.33	*n.s.*
ANOVA (all molars)	SS	*d.f.*	Variance	*F*	*P*	
Year	13 395	1	13 395	1.21	*n.s.*	
Interaction	7 869	2	3 934.5	0.35	*n.s.*	
Within	151 800	209	11 054			
Index – trait	1955–87	*N*	1988	*N*	*F*	*P*
Adults						
FA4 – *F. posterior palatine*	0.625	16	0.577	78	1.08	*n.s.*
FA4 – *F. hypoglosi*	0.667	18	0.536	69	1.24	*n.s.*
FA4 – *F. dentale*	2.059	17	2.300	77	1.12	*n.s.*
FA4 – *F. basiorbitalis acs.*	1.294	17	0.520	77	2.49	< 0.02
FA4 – *F. dorsal condylar*	0.615	11	0.886	70	1.44	*n.s.*
Subadults						
FA4 – *F. posterior palatine*	0.409	44	0.438	32	1.07	*n.s.*
FA4 – *F. hypoglosi*	0.125	40	0.432	37	3.46	< 0.001
FA4 – *F. dentale*	2.785	65	3.000	37	1.08	*n.s.*
FA4 – *F. basiorbitalis acs.*	0.558	43	0.469	32	1.19	*n.s*
FA4 – *F. dorsal condylar*	0.615	39	0.757	37	1.23	*n.s.*

FA4 is not presented for teeth because the results are identical with those of FA5.

level of fractal *FA* was found in females than in males (male: 0.00021; female: 0.00058, $F = 2.76$ $df = 26$, $P < 0.05$).

3.3. Differences in developmental instability among age groups

For teeth no test showed significant differences among age groups in 1955–87 (Table 4). In 1988 one out of four tests showed a significant difference. In foramina, adults tended to have higher levels of FA than subadults in 1955–87 and 1988. This tendency were only statistically significant in two traits in 1955–87. Moreover cubs often showed no level of FA (data not shown). These tendencies were the reason for separating age groups when examining differences between sexes and periods, respectively. Fractal FA was not significantly related to age ($r = 0.00$, $df = 40$, $P = 0.42$).

3.4. Pathological changes

There was no statistical difference in prevalence of the different pathological changes except for alveolar exostosis among subadults, where the 1988 sample showed a higher prevalence of exostosis than the 1955–87 sample (Table 5).

4. Discussion

All four methods showed that harbour seals that died during the epizootic were not the developmentally less stable than individuals from before the epizootic. A comparison between survivors and non-survivors in 1988 would give the best basis when investigating whether the developmentally less stable individuals died in 1988. However, no sample of survivors exists and instead I compared the 1988 sample with the sample from 1955–87. The 1955–87 sample was comprised both of individuals that potentially could have died in the epizootic as well as potential survivors, so an overlap between the 1988 and the 1955–87 samples potentially exists. This overlap may make a direct test of a difference in developmental stability between the periods difficult. Males showed higher mortality than females in the epizootic and mature individuals higher mortality than immatures. I expected the difference to be reflected in developmental stability, but that was not the case.

Table 3. Sex differences in asymmetry of skull characters of harbour seals. Values are variances. Test statistics are *F*-tests.

	1955–87						1988					
Index – trait	Male	*N*	Female	*N*	*F*	*P*	Male	*N*	Female	*N*	*F*	*P*
FA5 – Lower 2. molar	0.158	16	0.102	16	1.55	*n.s.*	0.058	15	0.071	15	1.2	*n.s.*
FA5 – Lower 3. molar	0.028	33	0.021	41	1.33	*n.s.*	0.044	14	0.013	19	3.4	< 0.01
FA5 – Upper 2. molar	0.031	23	0.055	11	1.77	*n.s.*	0.062	17	0.022	19	2.8	< 0.02
FA5 – Upper 3. molar	0.039	22	0.055	23	1.41	*n.s.*	0.023	15	0.024	18	1	*n.s.*
Adults												
FA5 – *F. posterior palatine*	0.727	11	0.400	5	1.82	*n.s.*	0.724	29	0.556	36	1.30	*n.s.*
FA5 – *F. hypoglosi*	0.727	11	0.571	7	1.27	*n.s.*	0.615	26	0.516	31	1.2	*n.s.*
FA5 – *F. dentale*	2.400	10	1.571	7	1.53	*n.s.*	1.690	29	3.028	36	1.8	*n.s.*
FA5 – *F. basiorbitalis acs.*	1.583	12	0.600	5	2.64	*n.s.*	0.448	29	0.629	35	1.40	*n.s.*
FA5 – *F. dorsal condylar*	0.444	7	1.000	4	2.25	*n.s.*	0.731	26	1.031	32	1.4	*n.s.*
Subadults												
FA5 – *F. posterior palatine*	0.440	25	0.368	19	1.20	*n.s.*	0.667	9	0.357	14	1.9	*n.s.*
FA5 – *F. hypoglosi*	0.090	23	0.176	17	1.96	*n.s.*	0.900	10	0.222	18	4.1	< 0.001
FA5 – *F. dentale*	1.971	34	3.670	31	1.86	< 0.05	2.250	12	2.882	17	1.3	*n.s.*
FA5 – *F. basiorbitalis acs.*	0.708	24	0.368	19	1.92	*n.s.*	0.444	9	0.500	14	1.1	*n.s.*
FA5 – *F. dorsal condylar*	0.409	22	0.882	17	2.16	< 0.05	0.600	10	0.889	18	1.5	*n.s.*

FA4 is not presented for teeth because the results are identical with those of FA5.

A possible explanation for the lack of a relationship between mortality and developmental instability, as estimated in the present study, is that the mortality differences between the sex and age classes were due to behaviour-mediated effects. Immature individuals are not reproductively active and they may not spend as much time at the breeding sites as mature individuals. Therefore, they may be exposed to a lower risk of infection. According to Heide-Jørgensen *et al.* (1992), viral diseases among seals resemble epidemics of venereal diseases in humans. This view is consistent with the higher mortality among mature individuals, but does not explain the higher mortality of males in a population where approximately all mature females reproduce (Härkönen & Heide-Jørgensen 1990). Therefore, they should encounter the same risk of infection as males. Behaviourally mediated effects cannot explain the three to six times lower mortality in the waters of East Scotland estimated to 10–20%. I suggest that a possibly higher resistance was a result of a better immune function in seals in the less polluted waters of East Scotland (Hall *et al.* 1992). Harbour seals fed with fish from polluted waters showed impaired immunological functions that

Table 4. Age difference in asymmetry of skull characters of harbour seals. Values are variances. Test statistics are *F*-tests.

Index – trait	Adults	N	Subadults	*N*	*F*	*P*
1955–87						
FA5 – lower 2. molar	0.142	8	0.100	25	1.42	*n.s.*
FA5 – lower 3. molar	0.018	8	0.026	17	1.44	*n.s.*
FA5 – upper 2. molar	0.020	9	0.032	54	1.60	*n.s.*
FA5 – upper 3. molar	0.061	7	0.031	17	1.97	*n.s.*
FA4 – *F. posterior palatinae*	0.625	16	0.409	44	1.53	*n.s.*
FA4 – *F. hypoglosi*	0.667	18	0.125	40	5.34	< 0.001
FA4 – *F. dentale*	2.059	17	2.790	65	1.36	*n.s.*
FA4 – *F. basiorbitalis acessorius*	1.294	17	0.558	43	2.32	< 0.02
FA4 – *F. dorsal condylar*	0.615	13	0.615	39	1.00	*n.s.*
1988						
FA5 – lower 2. molar	0.095	11	0.042	22	2.26	< 0.05
FA5 – lower 3. molar	0.021	14	0.024	27	1.14	*n.s.*
FA5 – upper 2. molar	0.063	20	0.087	25	1.38	*n.s.*
FA5 – upper 3. molar	0.016	14	0.037	25	2.31	*n.s.*
FA4 – *F. posterior palatinae*	0.577	78	0.438	32	1.31	*n.s.*
FA4 – *F. hypoglosi*	0.536	69	0.432	37	1.24	*n.s.*
FA4 – *F. dentale*	2.300	77	3.00	37	1.30	*n.s.*
FA4 – *F. basiorbitalis acessorius*	0.520	77	0.469	32	1.11	*n.s.*
FA4 – *F. dorsal condylar*	0.886	70	0.757	37	1.17	*n.s.*

Table 5. Prevalence of pathological changes in harbour seals in 1955–87 and 1988. Numbers are number of individuals. Test statistics are *G*-tests.

	Period	Adults				Subadults			
		No lesion	Lesion	Total	*P*	No lesion	Lesion	Total	*P*
Parodontitis	1955–87	12	12	24	69	1	70		
	1988	39	38	77	*n.s.*	37	2	39	*n.s.*
Alveolar exostosis	1955–87	1	21	22	54	17	71		
	1988	5	75	80	*n.s.*	15	23	38	< 0.01
Enlargement of *f. mentalia*	1955–87	12	4	16	44	27	71		
	1988	49	30	79	*n.s.*	26	12	38	*n.s.*

made them more susceptible to viral infections (Swart *et al.* 1994).

A higher prevalence of skull lesions in the Kattegat population of harbour seals have been related to organochlorines (Mortensen *et al.* 1992, Schandorff 1997). Skull lesions indicate an impairment of immune functions (Bergman *et al.* 1992). A higher prevalence of severe skull lesions was also observed in Baltic grey seals (Bergman *et al.* 1986, 1992), which are exposed to higher levels of organochlorines than harbour seals in Kattegat (Haraguchi *et al.* 1992). Older individuals had a higher frequency of skull lesions in harbour seals (Mortensen *et al.* 1992), but not in Baltic grey seals (Bergman *et al.* 1992). This may be explained by higher levels of organochlorines encountered by grey seals early in life (Haraguchi *et al.* 1992), or an inability to cope with organochlorines.

The increasing prevalence of skull lesions with age seen in harbour seals could be an effect of organochlorines, which are known to accumulate in seals with age and more in males than in females because the females detoxicate into their cubs (Helle *et al.* 1977, Hall *et al.* 1992). This detoxication happens primarily through nursing, where the organochlorines are transferred from mother to cub in the lipids of milk. This fact is in accordance with a higher prevalence of skull lesions in males than females (Mortensen *et al.* 1992, Schandorff 1997). These age and sex differences in the prevalence of skull lesions parallel patterns of mortality in 1988, where particularly adults and males died in high proportions.

The results of this study may be explained by organochlorines being important for mortality in 1988, and at the same time it is possible that individuals that died during the epizootic actually were the developmentally less stable individuals. We may expect a difference in developmental stability between subadults in the 1955–87 and the 1988 sample, if animals with high levels of organochlorines died. A difference was not found perhaps because the subadults from 1988 consisted of animals born in a period of decreasing levels of pollution (Olsson & Reutergårdh 1986). Although the subadults that died in the epizootic had high levels of organochlorines compared with the survivors, the levels may not be higher than average levels in 1955–87. This explanation would be in accordance with the results of fractal FA showing reduced developmental stability in 1955–87 compared with the 1988 sample. Individuals in the 1988 sample were primarily born after 1978 (the period of decreasing levels of pollution) resulting in a high developmental stability in these individuals in 1988.

Prevalence of alveolar exostosis, parodontitis and enlargement of foramen mentalia did not differ between the two periods, except for alveolar exostosis, where a larger prevalence was found in subadults in the 1988 sample. This lesion is expressed more in older animals (Mortensen *et al.* 1992, Schandorff 1997), and since the 1988 sample had a higher proportion of older individuals than the 1955–87 sample, this could explain the higher prevalence. From the apparent positive relationship between skull lesions and higher levels of organochlorines in the marine environment (Mortensen 1992), a higher prevalence of skull lesions should be expected in the 1988 sample compared with the 1955–87 sample, if organochlorines were important for mortality in the epizootic. If this were the case, animals that died would have high levels of organochlorines and therefore a high prevalence of skull lesions. However, this was not seen.

A comparison of individuals that died during the epizootic with the population before the epizootic did not show a difference in developmental stability or in prevalence of pathological changes. The lack of difference may have been caused by biased sampling or a lack of a relationship between developmental instability and susceptibility to the disease.

Acknowlegdements. I thank Prof. Anders Pape Møller for constructive criticism on drafts of the manuscript. The Swedish Museum of Natural History, Stockholm, and Zoological Museum, Copenhagen, provided free access to their collections of skulls.

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