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Developmental stability and skull lesions in the harbour seal (*Phoca vitulina*) in the 19th and 20th Centuries

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Developmental stability in the Kattegat population of harbour seal (*Phoca vitulina*) was assessed to determine whether reduced developmental stability occurred prior to the epizootic in 1988 and possibly predicted the high mortality. As a measure of developmental stability 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 361 skulls. Moreover prevalence of three pathological changes of the skull was investigated. Reduced developmental stability and higher prevalence of pathological changes were associated with a dramatic increase in pollution after World War II. Reduced developmental stability occurred prior to the epizootic, although young seals born after levels of pollution started to decrease tended to be developmentally more stable. No relationship was found between pathological changes and developmental stability.

1. Introduction

The epizootic in 1988 in which more than 18 000 harbour seals (*Phoca vitulina*) died throughout N Europe was initially discovered in the Kattegat. Here 3 909 dead harbour seals were reported (Dietz *et al.* 1989) and mortality for the population was later estimated to 60% (Härkönen & Heide-Jørgensen 1990). The primary cause of the epizootic was a highly contagious virus, *phocine distemper* virus (PDV) (Osterhaus & Vedder 1988). One manifestation of the disease was immunosuppression, which brought along different complications that were often the cause of death (Baker & Ross 1992).

Carter *et al.* (1992) suggested that virus infections of the genus *Morbillivirus*, to which PDV belongs, probably are more widespread in seals than previously thought, although not always resulting in a high mortality. Speculation arose as to why the epizootic in 1988 resulted in a high mortality. It could be an effect of an introduction of the virus into a highly susceptible population without prior infection with PDV (Have *et al.* 1991). Also, or alternatively, a general poor state of health might have existed in the population prior to 1988 and made the individuals more vulnerable to the infection. Anthropogenic factors and infections may have had synergistic effects resulting in a poor state of health (Baker & Ross 1992,

Carter *et al.* 1992, Hall *et al.* 1992). Olsson *et al.* (1992) suggested that immunosuppression existed prior to the virus infection.

Pollution, especially with organochlorines such as PCB and DDT, which are accumulated in marine mammals, has been considered a major threat to their state of health (Olsson *et al.* 1992). A dramatic increase in pollution happened after World War II, and levels of organochlorines did not decrease until the late seventies (Olsson & Reutergårdh 1986). Studies indicate a negative effect of organochlorines on many aspects of health in seals: (i) reproductive failure in harbour, grey (*Halichoerus grypus*) and ringed seal (*Pusa hispida*) (Helle *et al.* 1977, Reinjders 1980), (ii) a disease complex with hormonal imbalance in grey seal (Bergman & Olsson 1986, Bergman *et al.* 1986), and (iii) different skeletal deformities in harbour and grey seal (Mortensen *et al.* 1992). Hormonal imbalance and skeletal deformities are indicative of a deficiency of the immune system (Bergman *et al.* 1992). Impairment of immune function was seen in harbour seals fed fish containing organochlorines at levels similar to those encountered by free-ranging seals (Swart *et al.* 1994). This finding supports that a general poor health existed prior to the epizootic. Observation of many sick seals (Søndergård *et al.* 1976, Bøgebjerg *et al.* 1991) and a high prevalence of skull lesions (Mortensen *et al.* 1992) and parasites (Clausen 1977) were recorded in the Kattegat population.

Pollution and infections are considered to be environmental stress factors. Stress of environmental and genetic origin affects developmental processes as reflected in reduced developmental stability (Møller & Swaddle 1997). Developmental stability reflects the ability of an individual to buffer its development against disturbance. Stress makes the individual less able to produce a predetermined phenotype (Palmer 1994, Møller & Swaddle 1997). Developmental stability is generally high in individuals of natural populations not subject to considerable genetic or environmental stress (Palmer & Strobeck 1986, Jones 1987, Møller & Swaddle 1997). In natural populations of grunion (*Leuresthes tenuis*) exposed to organochlorines, developmental stability was reduced (Valentine & Soulé 1973), and in a population of grey seal in the Baltic Sea, high levels of organochlorines were correlated with reduced developmental stability (Zakharov *et al.* 1986). 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.).

Fluctuating asymmetry (FA) is the most commonly used index of developmental instability (Palmer & Strobeck 1986). Asymmetry is small random departures from symmetry in bilateral characters, while asymmetry in a group of individuals (for example a population) is called FA. The magnitude of FA reflects the ability of the individuals in a population to buffer their development (Møller & Swaddle 1997).

I assessed developmental instability to monitor the general condition of the Kattegat harbour seal population. Developmental stability was assessed by examining the prevalence of morphological changes caused by disturbed growth patterns such as deviation from symmetry in bilateral characters. Skeletal deformities were used as another estimate of the general condition of the Kattegat population. Four different measures of developmental stability were used. First, FA in teeth dimensions was used to estimate the degree of developmental instability. Second, FA in foramina in the skull was used as another estimate of developmental instability. Foramina are small openings in the skull for nerves and blood vessels. Morphological changes in foramina resulting from disturbance in development are in many cases difficult or impossible to distinguish from pathological changes due to skull lesions in seals. Therefore, only a restricted numbers of easily recognizable paired characters were used to estimate the magnitude of FA. Third, sutures have been shown to demonstrate scales of self-similarity (where the parent pattern is repeated again and again on a smaller scale), and this makes it possible to calculate an empirical fractal dimension for a suture (Long 1985). The fractal dimension of skull bone sutures was, as suggested by Emlen *et al.* (1993), used as an index of developmental instability in the present study. Diseases have been associated with loss of fractal dimension (Honda *et al.* 1992, Escós *et al.* 1995), and Alados *et al.* (1996) recommended this method for measuring developmental instability after showing reduced dimensionality in sagital sutures in inbred popula-

tions of two species of gazelles. Moreover, Sumarsono *et al.* (1996) have recently shown severe changes of sutures associated with abnormal growth patterns in mice with Down's syndrome. Fourth, the *maxillo palatinae* suture is paired, which allowed me to investigate the difference in fractal dimension between the right and the left suture and use this as an estimate of developmental instability.

Mortensen *et al.* (1992) studied the prevalence of two skull lesions in seals from Kattegat. One resembles parodontitis in humans, while the other is alveolar exostosis. Prevalence in the 19th Century of these two pathological changes in addition to a third, enlargement of foramen mentalia, was compared with the prevalence after the major increase in pollution. Moreover, the relationship between prevalence of the three pathological changes and the measures of developmental instability was investigated.

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. All the skulls came from Kattegat which holds a single population (Härkönen & Heide-Jørgensen 1990). A presentation of the material is given in Table 1. A total of 361 skulls (in 40 cases only the mandibles) from three periods were examined:

1889–90: Seals collected in the first year of bounty hunting in Denmark and constituting a pre-pollution sample. Although this is an old sample it was the most homogeneous sample prior to 1940.

1955–87: A sample of animals from the population that was affected by PDV. Most individuals were born in a period with high levels of pollution. Cause of death: 63 individuals shot for systematic collection for CM and 52 individuals drowned in fishermans gear.

1988: Animals collected during the epizootic. Almost all individuals were expected to have died from PDV. Subadults were born in a period of decreasing levels of pollution (Olsson & Reutergårdh 1986).

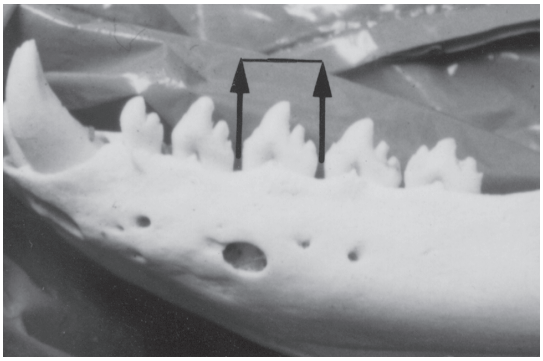

Fig. 1 Maximum mesiodistal length of a molar.

Animals collected in 1955–88 all came from sanctuaries or preserved areas. Information on age and sex were obtained from index cards at CM and from P. Mortensen (SM). When age data were unavailable, morphological characteristics of mandibles and teeth described in Allen (1902) and Doutt (1942) were used for the age estimation. All 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. Measures of developmental instability

Developmental stability was estimated by using characters of the skull: teeth, foramina and sutures. The maximum mesiodistal lengths (transition between the crown and the root) (Fig. 1) of the 2nd and the 3rd molar in right and left side of upper and lower jaws were measured with a digital caliper to the nearest 0.01 mm. The difference in length between each bilateral pair of teeth was used to estimate the magnitude of FA. The measurements were replicated on two subsequent days. A two-way ANOVA (sides × individuals) was conducted on teeth data to test for the significance of FA relative to measurement error (Palmer & Strobeck 1986). The interaction MS containing information about FA (and anti-asymmetry) was tested against error MS (reflecting measurement error) showing that FA was significantly larger than measurement error in all cases ($0.012 <$ (interaction MS) < 0.111, $0.000 <$ (error MS) < 0.002, $15 < df < 69$, $P < 0.001$). No measurements were attempted on broken or worn teeth.

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

Period	Adults			Subadults			Cubs			Total
	Male	Female	Sex unknown	Male	Female	Sex unknown	Male	Female	Sex unknown	
1889–90			17			58			58	122
1955–87	14	11		36	36		8	10		115
1988	31	37	13	12	19	11	1			124

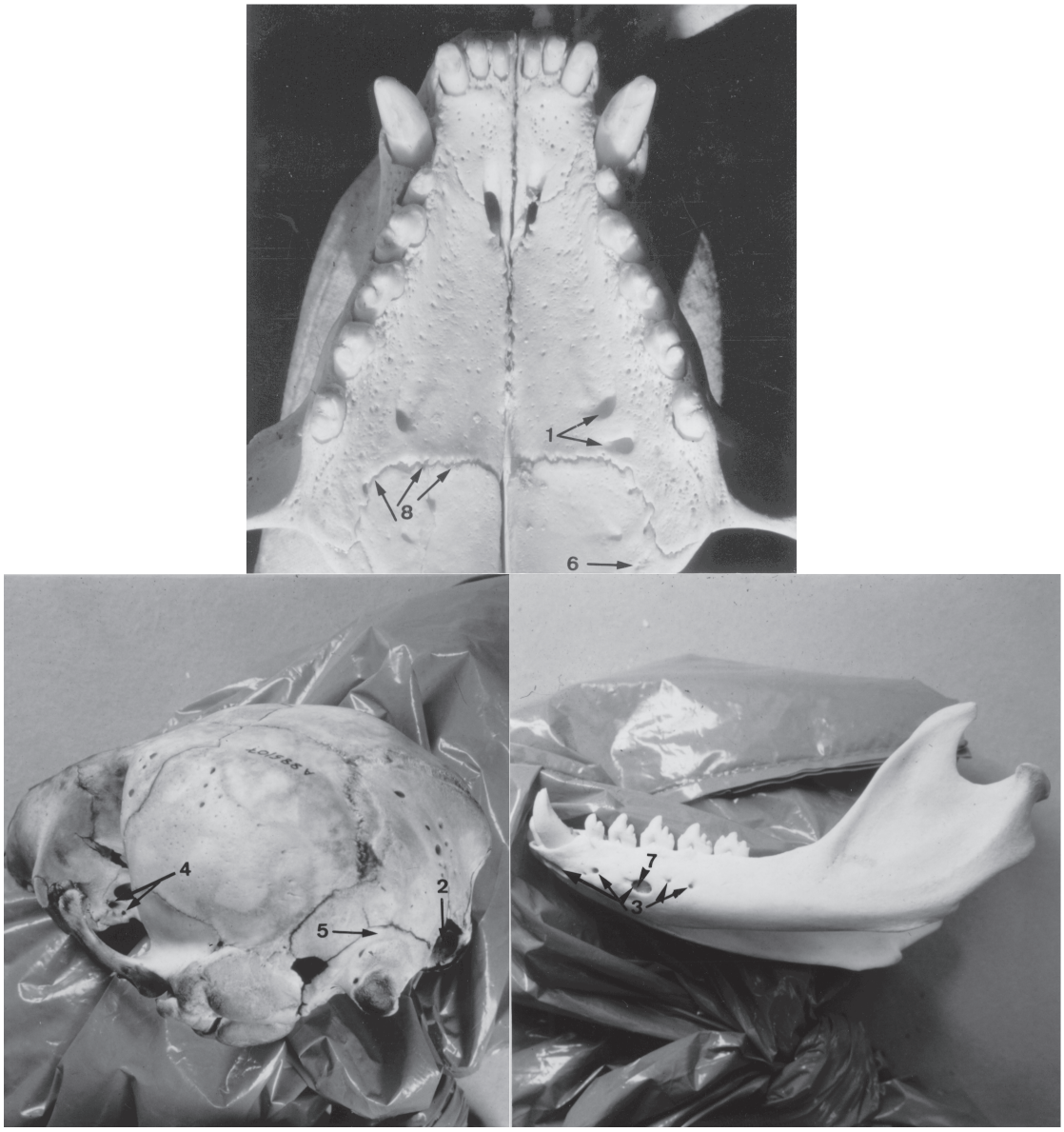

Fig. 2. Morphological characteristics of the skull of harbour seal (*Phoca vitulina*). Top: Ventral aspect of the skull. Bottom right: Left mandible. Bottom left: Posterio-lateral aspect of the skull. — 1: *Foramen posterior palatinae*, — 2: *Foramen hypoglosi*, — 3: *Foramen dentale*, — 4: *Foramen basiorbitalis acessorius*, — 5: *Foramen dorsal condylar* (not in illustration), — 6: *Foramen palatinae minor,* — 7: *Foramen mentalia*, — 8: *Forma sutura maxillopalatinae*. 1–6 used to estimate FA.

Six pairs of foramina were counted macroscopically on the right and left side of the specimen (Fig. 2). The difference in number of foramina on each side was used to estimate the magnitude of FA. Each pair of foramina was counted twice on 46 skulls on two subsequent days to estimate measurement errors (%ME). No appropriate test for the significance of FA relative to counting error in meristic traits have been described (Palmer 1994). Therefore 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.

Slides of the left and right *maxillo palatinae* sutures (Fig. 2) were taken at the same focal distance (30 cm) and projected onto standard size (28 × 20 cm) paper on which

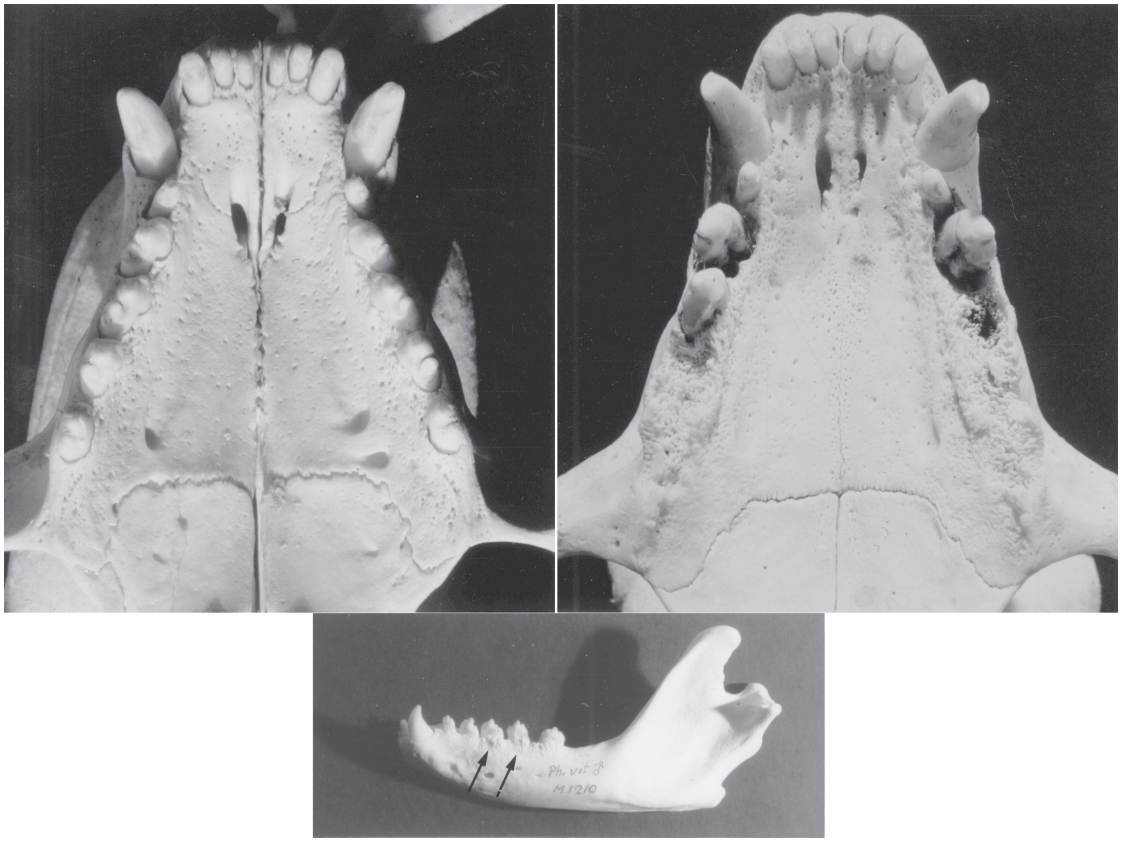

Fig. 3. Skull lesions in skull of harbour seal (*Phoca vitulina*). Top right: External characteristic of parodontitis (note the missing teeths in the regions of the molars). Top left: Normal skull. Bottom: Characteristic of alveolar exostosis.

the pattern of the suture was drawn. Thereafter it was copied to double size before measurements. Dividers adjusted to 8 cm, 4 cm, 1 cm and 0.5 cm were used to measure lengths ($L(y)$) of the suture on the drawings. 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 number of steps is multiplied by the adjusted yardstick to achieve the length of the line. An increase in length is observed when length of the yardstick (x) used for measurements decreases. The fractal dimension (D) is calculated as:

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

by regression of $\log(L(y))$ on $\log y$ over four values of y. For a detailed description of this walking rulers method, see Emlen *et al.* (1993). To ensure a similar closure of the sutures only adult specimens were used. The percentage of measurement error of 0.038% (original measurements) was estimated by replicating the whole session for 20 sutures on two subsequent days.

A second estimate of FA based on fractal dimensions in sutures was calculated as the difference between the right and left fractal dimension in the same skull. A one-way ANOVA with individuals of the fixed factor, where n is the number of repeated measurements per individual, was used to estimate measurement error (Merilä & Björklund 1995). Using data from seven individuals, the measurement error was estimated to be 17.9% (ME% of FA).

Each skull was examined macroscopically to diagnose one or more of three pathological changes. Type 1 was characterized by exostosis (apposition of bone) localized to the part of the lower jaw supporting the teeth (processus alveolaris), especially in the area of the molars (Fig. 3). The exostoses are tapering, almost formed as flames. They grow from the marginal of the bone parallel to the teeth and vertically parallel to the surface of the bone. I based this description of alveolar exostosis on X-ray photographs and on macroscopical examination. X-rays were conducted at the Jaw-Surgical Department, Aalborg Hospital. X-rays showed no signs of intra-osseous exostosis (condensations in the central parts of the alveolar bone) and macroscopical examination was therefore sufficient for verification. Type 2 had external characteristics of parodontitis (Fig. 3): Pathological change with loss (sometimes severe) of alveolar bone tissue around the teeth and in other parts of the jaws. Often

teeth were missing. As criterion, the loss of alveolar bone tissue around a minimum of one tooth to a degree where bony support was lost (Mortensen *et al.* 1992) was used. Type 3 was characterized by smooth-edged enlargements of foramen mentalia (Fig. 2). As criterion for this change, an enlargement more than four times the size of the other dental foramina was used.

2.2. Tests for FA

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

2.2.1. Test for departures from normality and size dependence

The significance of FA relative to directional asymmetry in data on teeth was tested in the two-way ANOVA (sides × individuals), which also tested for measurement error (Palmer 1994). *t*-tests were used to test for a signed mean left minus right character value equaling zero on data on foramina and fractal FA (Kolmogorov-Smirnov tests were applied to small samples (Sokal & Rohlf 1981)). To test for other types of departures from normality one-sample *t*-tests were carried out on skewness and kurtosis, respectively.

Regression of the magnitude of asymmetry against average tooth size were made to test for size dependence. Moreover a regression was used to test for a relation between fractal FA and fractal dimension.

2.2.2. Test for differences between age classes and periods

Four different indices of FA were used on teeth data. Palmer (1994) recommended the use of more than one index, whenever possible, because different indicies are sensitive to different factors (directional asymmetry, outliers). The first three indices, FA4, FA5 and FA10 of Palmer and Strobeck (1986) were used to estimate levels of FA in single traits (a trait is for example one bilateral pair of teeth), and for statistical comparison between different periods an *F*-test was used (Fowler & Cohen 1990).

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

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

$$\text{FA10: } \sigma^2 (MS_{sj} - MS_m)/M \quad (3)$$

(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.)

The fourth index was a two-way ANOVA (samples × traits) (Palmer 1994) used on teeth data to test for differences among periods for multiple traits in the individuals. As an index of FA in foramina and fractal FA only FA4 were used. The same procedure was used to compare developmental instability of animals shot versus animals caught in nets. I used a Mann-Whitney *U*-test (Fowler & Cohen 1990) to test for a significant difference among periods for fractal dimensions. To test for differences in prevalence of pathological changes between sexes, age groups and different periods, a *G*-test (2 × 2 contingency table) with Williams correction was used (Fowler & Cohen 1990). A Mann-Whitney *U*-test was used to test for a relation between pathological changes and fractal dimension and fractal FA, respectively. A relationship between pathological changes and FA in foramina was tested using *F*-tests (after testing for departures from normality). To ensure that possible significant results were not a consequence of a large number of tests, a sequential Bonferroni test was applied when more than 5 related tests were conducted (Rice 1989).

3. Results

A detailed presentation of descriptive statistics is given in the appendices.

The two-way ANOVA (sides × individuals) showed that there was no significant directional asymmetry in data on teeth ($0.745 < (\text{side MS}) < 1.169, 0 < F < 1.75, 0.05 < P < 1$) except for lower second molar in data from 1889–90 ($(\text{side MS}) = 0.112$, $F = 9.33$, $df = 1.6$, $P < 0.05$), which was excluded from further analyses.

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 magnitude of FA in teeth was independent of overall teeth size (regression analyses conducted on each trait in each period: $-0.074 < r < 0.086$, $df = 14–68$, $0.16 < P < 0.94$). Furthermore, a correlation of fractal FA with fractal dimension was not significant (regression analysis: $r = 0.027$, $df = 50$, $P = 0.82$). No difference between animals shot and animals that died in fishing gear was found in levels of FA in foramen or teeth (*F*-tests conducted for each sex, age class and for pooled data: $0.022 < F < 2.95$, $df = 13–42$, $0.05 < P < 0.96$), and for this reason specimens were pooled.

In some tests different age classes and/or sexes have been pooled, after assuring that this did not change the results of the statistics.

3.1. Differences in developmental instability among age groups in 1889–90

For teeth, no test showed significant differences between adults and subadults (Table 2). In foramina, adults tended to have higher levels of FA than subadults, but this difference was only statistically significant in one out of 5 characters.

3.2. Differences in developmental instability between pre- and post-pollution samples

Five out of 20 *F*-tests conducted on foramina data yielded significant differences showing the seals in 1889–90 to be developmentally more stable than those from 1955–87 and 1988 (Table 3). Moreover another 11 *F*-tests showed higher variances (higher level of FA) for the seals from 1955–87 and 1988 compared with seals from 1889–90, although these differences were not statistically significant.

The ANOVA conducted on teeth data did not yield significant differences between the seals from 1889–90 and from 1955–87 and 1988, respectively. In 5 out of 6 *F*-tests, variances (levels of FA) were higher in 1955–87 and 1988 compared with 1889–90, although these differences were only statistically significant in two cases.

Levels of fractal FA were significantly different between 1889–90 and 1955–87 (FA4: 1889–90: 0.00026; 1955–87: 0.00083; $F = 3.19$, $df = 28$, $P < 0.05$), but not between 1889–90 and 1988 (FA4: 1889–90: 0.00026, $N = 9$; 1988: 0.00021, $N = 21$). No differences were seen when fractal dimension was compared (mean (*S.E.*): 1889–90: 1.045 (0.003), $N = 20$; 1955–87: 1.055 (0.002), $N = 46$; 1988: 1.052 (0.004), $N = 46$).

3.3. Pathological changes

The prevalence of the three pathological changes increased significantly since the last century (Fig. 4, Table 4). The prevalence of alveolar exostosis and enlargement of foramen mentalia did not differ significantly between males and females for either adults or subadults, while a pooled sample of adult animals from 1955–88 showed a higher prevalence of paradontitis in males than in females.

3.4. Pathological changes and developmental instability

Individuals from 1955–87 and 1988 with parodontitis did not show increased levels of developmental instability compared with individuals without this lesion (Table 5). Neither was a relation found between the enlargement of foramen mentalia and developmental stability. For lesion type 2 the fractal dimension was larger in individuals with a pathological change, although the difference in the average fractal dimension was not statistically significant (mean (*S.E.*): lesion: 1.054 (0.002), $N = 21$, no lesion: 1.049 (0.001), $N = 20$). Lesion type 3 showed same pattern in 1955–87 (mean (*S.E.*): lesion: 1.054 (0.001), $N = 12$, no lesion: 1.049 (0.003), $N = 28$) and in 1988 (mean (*S.E.*): lesion: 1.052 (0.002), $N = 15$, no lesion: 1.048 (0.003), $N = 24$).

Table 2. Age difference in asymmetry of skull characters of harbour seals in 1889–90. Values are variances. Test statistics are *F*-tests.

Index	Trait	Adults	*N*	Subadults	*N*	*F*	*P*
FA5	Lower 3. molar	0.012	5	0.017	26	1.42	*n.s.*
FA5	Upper 2. molar	0.037	4	0.061	11	1.65	*n.s.*
FA5	Upper 3. molar	0.011	5	0.018	22	1.64	*n.s.*
FA4	*F. posterior palatinae*	0.556	18	0.387	31	1.44	*n.s.*
FA4	*F. hypoglosi*	0.167	6	0.143	14	1.17	*n.s.*
FA4	*F. dentale*	3.071	14	1.930	43	1.59	*n.s.*
FA4	*F. basiorbitalis acessorius*	0.769	13	0.263	18	2.92	< 0.05
FA4	*F. dorsal condylar*	0.290	8	0.500	14	1.72	*n.s.*

4. Discussion

4.1. Developmental instability of harbour seals in the pre- and post-pollution samples

Many findings in the present study indicated a reduced level of developmental stability in the later samples of seals that lived in a more polluted environment. The levels of fractal FA were significantly larger in 1955–87 compared with 1889–90, but similar levels were seen in 1889–90 and 1988. If the use of fractal dimensions is a more sensitive approach than commonly used indices of FA (Alados *et al.* 1996), the similar levels in the two periods may be explained by the 1988 sample being comprised of animals mainly born in 1978–83; a period of decreasing levels of pollution (Olsson & Reutergårdh 1986). No difference was seen in the average fractal dimension between 1889–90, 1955–87 and 1988. The similar estimates among samples were probably due to the low complexity of the suture, which may prevent disturbance of growth resulting in considerable deviations in fractal dimension.

4.2. Pathological changes

The prevalence of alveolar exostosis, parodontitis and enlargement of foramen mentalia increased with increasing levels of pollution. A high prevalence of alveolar exostosis existed even before any significant pollution. This finding changes the current view of the etiology of the lesion. Exostosis

Table 3. Magnitude of asymmetry of skull characters of harbour seal in pre- and post-pollution samples. Values are variances. Test statistics are *F*-tests.

Index	Trait	1889–90	*N*	1955–87	*N*	*F*	*P*	1889–90	*N*	1988	*N*	*F*	*P*
FA5	Lower 3. molar	0.017	33	0.025	69	1.47	*n.s.*	0.017	33	0.025	41	1.47	*n.s.*
FA5	Upper 2. molar	0.053	16	0.039	29	1.36	*n.s.*	0.053	16	0.076	45	1.43	*n.s.*
FA5	Upper 3. molar	0.018	28	0.040	41	2.22	< 0.05	0.018	28	0.028	39	1.56	*n.s.*
FA10	Lower 3. molar	0.008	33	0.012	69	1.50	*n.s.*	0.008	33	0.012	41	1.50	*n.s.*
FA10	Upper 2. molar	0.025	16	0.020	29	1.25	*n.s.*	0.025	16	0.038	45	1.52	*n.s.*
FA10	Upper 3. molar	0.007	28	0.020	41	2.9	< 0.05	0.007	28	0.015	39	2.14	< 0.05

ANOVA (all molars)	SS	*df*	variance	*F*	*P*	SS	*d.f.*	variance	*F*	*P*
Year	13 395	1	13 395	1.21	*n.s.*	27 880	1	27 880	1.89	*n.s.*
Interaction	7 869	2	3 934.5	0.35	*n.s.*	913.6	2	456.8	0.03	*n.s.*
Within	151 800	209	11 054			2 119 464	196	14 718		

Index	Trait	1889–90	*N*	1955–87	*N*	*F*	*P*	1889–90	*N*	1988	*N*	*F*	*P*
Adults													
FA4	*F. posterior palatine*	0.556	18	0.625	16	1.1	*n.s.*	0.556	18	0.577	78	1.04	*n.s.*
FA4	*F. hypoglosi*	0.167	6	0.667	18	4	< 0.05	0.167	6	0.536	69	3.21	< 0.05
FA4	*F. dentale*	3.070	14	2.059	17	1.5	*n.s.*	3.070	14	2.300	77	1.33	*n.s.*
FA4	*F. basiorbitalis acs.*	0.769	13	1.294	17	1.7	*n.s.*	0.769	13	0.520	77	1.48	*n.s.*
FA4	*F. dorsal condylar*	0.290	8	0.615	11	2.1	*n.s.*	0.290	8	0.886	70	3.06	< 0.05
Subadults													
FA4	*F. posterior palatine*	0.387	31	0.409	44	1.1	*n.s.*	0.387	31	0.438	32	1.13	*n.s.*
FA4	*F. hypoglosi*	0.143	14	0.125	40	1.1	*n.s.*	0.143	14	0.432	37	3.02	< 0.05
FA4	*F. dentale*	1 930	43	2.785	65	1.4	*n.s.*	1 930	43	3.000	37	1.60	*n.s.*
FA4	*F. basiorbitalis acs.*	0.263	18	0.558	43	2.1	< 0.05	0.263	18	0.469	32	1.78	*n.s.*
FA4	*F. dorsal condylar*	0.500	14	0.615	39	1.2	*n.s.*	0.500	14	0.757	37	1.51	*n.s.*

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

in the jaws is a normal deviant in humans (Touyz & Tau 1991ab, Eggen & Natvig 1994). The high prevalence of exostoses even in the 19th Century indicates that this is a commonly occurring feature in harbour seals. The prevalence of paradontitis was higher in males and in older individuals, in accordance with the suggested correlation with organochlorines (Bergman *et al.* 1992). Organochlorines accumulate to higher levels in males than in females because females detoxicate themselves into their cubs (Helle *et al.* 1977, Hall *et al.* 1992). Organochlorines are known to be immunosuppressive (Swart *et al.* 1994), and immunsuppression in laboratory rats is often shown to worsen the pathological condition of parodontitis (Klausen 1991).

4.3. Pathological changes and developmental stability

A high prevalence of pathological changes and increased developmental instability have both been associated with organochlorines, and a relationship between pathology and developmental instability seems possible. Other studies have shown a correlation between different diseases and reduced developmental stability (Møller 1996). No relationship between parodontitis and increased developmental instability (measured by fractal dimension and FA in foramina) was found.

For the enlargement of foramen mentalia, no clear-cut relation with reduced developmental stability was seen in either comparison of fractal dimensions or levels of FA in foramina, in accordance with the possibility of the enlargement being a normal genetic phenodeviant. The high prevalence of alveolar exostosis in all samples made an examination of this lesion impossible.

Zakharov and Yablokov (1990) showed a dramatic change in developmental stability in Baltic grey seals during a period of increased pollution. Despite a potential species-specific effect, the less dramatic change in the present study is consistent with the much higher levels of organochlorines in grey seals in the Baltic proper than in harbour seals in the Kattegat (Clausen 1977, Holden 1978, Haraguchi *et al.* 1992, Heide-Jørgensen *et al.* 1992). A direct comparison of this study with the study of Baltic grey seals may be difficult. The

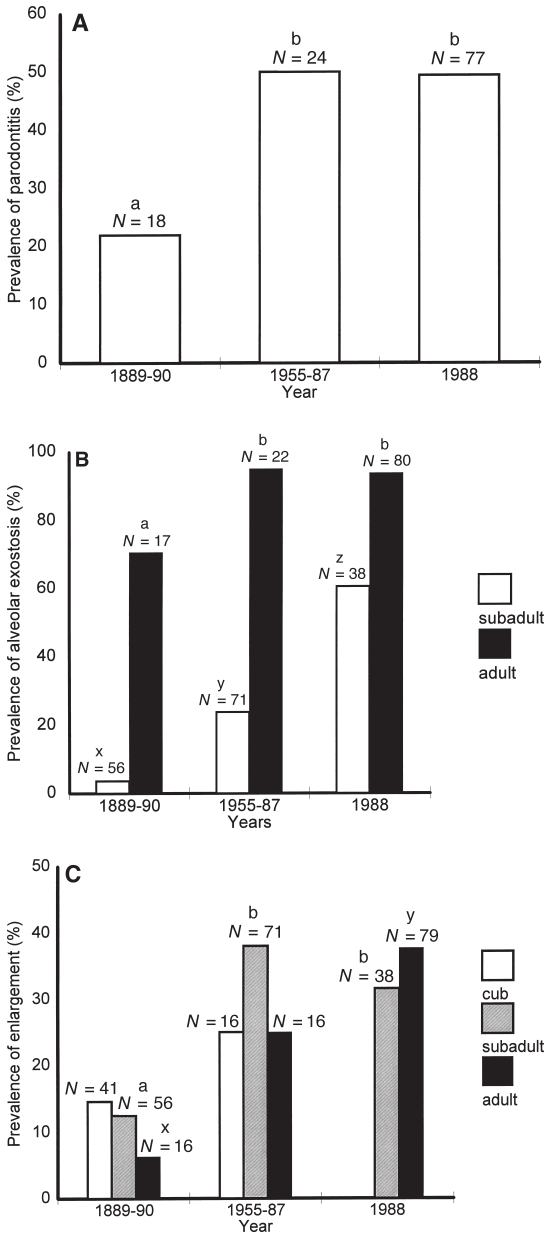

Fig. 4. — A: Prevalence of parodontitis. — B: Prevalence of alveolar exostosis. — C: Prevalence of enlargement of foramen mentalia. — A–C: Prevalences that were significantly different ($P < 0.05$) in a *G*-test are provided with different subscripts. Each age class was treated separately.

high levels of organochlorines in the Baltic increase the prevalence of skull lesions with pathological changes (Bergman *et al.* 1986, 1992), which are very similar to many of the morphological characters used in the study of the grey

seal (e.g. small foramina). This means that some of the morphological changes seen in the very large number of characters (37) in the study of Baltic grey seals actually may have had a pathological origin without being caused by disruption of development.

Alados *et al.* (1996) concluded that the fractal dimension of skull sutures is a useful and sensitive index of developmental stability, but in the present study the low fractal dimension of the maxillo palatinae suture made the fractal dimension by itself inapplicable as an estimate of developmental stability. The fractal dimension is correlated with the degree of suture interlocking and recurvation (Hartwig 1991), which are features that prevent bone separation (Long 1985). The low fractal dimension of the maxillo palatinae suture probably arises because the degree of interlocking and recurvation are of minor importance in the skull of harbour seals, which do not use their skulls directly in male–male interactions.

The difference seen in results of fractal FA and FA in foramina is in accordance with fractal asymmetry being a more sensitive method as suggested by Alados *et al.* (1996) for the sagital suture in two species of gazelles. They found reduced developmental stability using fractal dimensions, while FA in foramina did not show a similar result.

The prevalence of paradontitis was in accordance with the results of Mortensen *et al.* (1992), while this was not so for alveolar exostosis. In my study I included incipient stages of this type of exostosis characterized by reactive bone for-

Table 4. Prevalence of pathological changes in harbour seals in pre- and postpollution samples. Numbers are numbers of individuals. [a] = 1955–87, all ages pooled. [b] = 1988, all ages pooled.

	Adults		Subadults		Cubs	
	No lesion	Lesion	No lesion	Lesion	No lesion	Lesion
Parodontitis						
1889–90	14	4				
1955–87	12	12				
1988	39	38				
Males	16	25				
Females	30	18				
Alveolar exostosis						
1889–90	5	12	54	2	46	0
1955–87	1	21	54	17	15	0
1988	5	75	15	23		
Males	2	45	37	16		
Females	3	41	23	14		
Enlargement of foramen mentalia						
1888–89	15	1	49	7	35	6
1955–87	12	4	44	27	12	4
1988	49	30	26	12		
Females	34	15[a]				
Males	32	20[a]				
Females	36	16[b]				
Males	49	26[b]				

Significance of differences tested with *G*-test.

	Alveolar exostosis		Enlargement of *f. mentalia*		*Parodontitis*
	Adults	Subadults	Adults	Subadults	Adults
1889–90 vs. 1955–87	< 0.05	< 0.01	*n.s.*	< 0.01	< 0.01
1889–90 vs. 1988	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01
1955–87 vs. 1988	*n.s.*	< 0.01	*n.s.*	*n.s.*	*n.s.*
males vs. females	*n.s.*	*n.s.*	*n.s.*	*n.s.*	< 0.05

mations having a rough surface. When including incipient stages, a very high prevalence was found in adults. The prevalence of alveolar exostosis in the two samples from this century was more than twice as high as that observed in a study partly based on the same material (Mortensen *et al.* 1992). In the pre-pollution sample the prevalence was nine times higher than in the study of Mortensen *et al.* (1992), which indicates that the presence of this lesion is not due to pollution. The expression is, however, more profound in the latter part of the present century, and Peter Mortensen (pers. comm.) suggested pollutants or new strains of bacteria to be responsible. This is in accordance with the many small blood vessels in the exostoses, which could indicate inflammation processes caused by bacteria in irritation processes.

The post-pollution samples were characterized by a higher prevalence of pathological changes and reduced developmental stability compared with the pre-pollution sample. This indicates a poor condition prior to the epizootic, which may have had an influence on mortality in 1988. Moreover individuals born in a period of decreasing levels of pollution tended to be developmentally more stable than individuals from the pollution era.

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References

Alados, C. L., Escós, J. & Emlen, J. 1996: Fluctuating asymmetry and fractal dimension of the saggital suture as

Table 5. Developmental stability and pathological changes in harbour seals. Magnitude of asymmetry in animals with or without pathological changes. Values are variances. Test statistics are *G*-tests.

Traits	Lesion	*N*	No lesion	*N*	*P*
Parodontitis 1955–87					
F. posterior palatinae	0.800	10	0.333	6	*n.s.*
F. hypoglosi	0.900	10	0.286	7	*n.s.*
F. dentale	1.900	9	2.250	8	*n.s.*
F. basiorbitalis acs.	1.000	9	0.000	8	< 0.001
F. dorsal condylar	1.555	7	1.000	5	*n.s.*
Parodontitis 1988					
F. posterior palatinae	0.500	40	0.568	37	*n.s.*
F. hypoglosi	0.514	35	0.594	32	*n.s.*
F. dentale	2.167	36	2.842	38	*n.s.*
F. basiorbitalis acs.	0.425	40	0.611	36	*n.s.*
F. dorsal condylar	0.486	35	1.333	33	< 0.01

Traits	Change	*N*	No change	*N*	*P*
Enlargement of *f. mentalia* 1955–87					
F. posterior palatinae	0.457	35	0.556	18	*n.s.*
F. hypoglosi	0.281	32	0.294	17	*n.s.*
F. dentale	2.529	51	2.655	29	*n.s.*
F. basiorbitalis acs.	0.618	34	0.948	19	*n.s.*
F. dorsal condylar	0.667	27	0.722	18	*n.s.*
Enlargement of *f. mentalia* 1988					
F. posterior palatinae	0.522	69	0.550	40	*n.s.*
F. hypoglosi	0.556	63	0.447	38	*n.s.*
F. dentale	2.597	72	2.385	39	*n.s.*
F. basiorbitalis acs.	0.574	68	0.400	40	*n.s.*
F. dorsal condylar	0.984	64	0.658	38	*n.s.*

FA11 test statistics are *F*-tests.

inbreeding depression indicators in Dama and Dorcas gazelle. — Can. J. Zool. 73: 1967–1974.

Allen, J. A. 1902: The hair seals (Family Phocidae) of the North Pacific Ocean and Bering Sea. — Bull. Am. Mus. Nat. Hist. 16: 459–499.

Baker, J. R. & Ross, H. M. 1992: The role of bacteria in phocine distemper. — Sci. Total Environ. 115: 9–14.

Bergman, A. & Olsson, M. 1986: Pathology of Baltic grey seal and ringed seal females with special references to adrenocortical hyperplasia: is environmental pollution the cause of a widely distributed disease syndrome? — Finnish Game Res. 44: 47–62.

Bergman, A., Olsson, M. & Reiland, S. 1986: High frequency of a skeletal deformation in Baltic grey seal skulls. — ICES, C: M 1986/N: 15: 1–7.

Bergman, A., Olsson, M. & Reiland, S. 1992: Skullbone lesions in the Baltic grey seal (Halichoerus grypus). — Ambio 21: 517–519.

Bøgebjerg, E., Tougaard, S., Madsen, J. & Nørgaard, N. 1991: Status of the harbour seal Phoca vitulina populations in the Danish waters 1976–1989, and short-term effects of the epidemic in 1988. — Dan. Rev. of Game Biol. 14: 2–16.

Carter, S. D., Hughes, D. E., Taylor, V. J. & Bell, S. C. 1992: Immune responses in common and grey seals during the seal epizootic. — Sci. Total Environ. 115: 83–91.

Clausen, B. 1977: Diseases and toxochemicals in the common seal in Denmark. Proc. Symp. Cons. Baltic Seals April 26–28. — Finnish Game Res. 37: 38–39.

Dietz, R., Heide-Jørgensen, M. P. & Härkönen, T. 1989: Mass death of habor seals (Phoca vitulina) in Europe. — Ambio 18: 258–264.

Doutt, J. K. 1942: A review of the genus Phoca. — Ann. Carnegie Mus. 29: 61–125.

Eggen, S. & Natvig, B. 1994: Concurrence of torus mandibularis and torus palatinus. —Scand. J. Dent. Res. 102: 60–63.

Emlen, J. M., Freeman, D. C. & Graham, J. H. 1993: Growth dynamics and the origin of fluctuating asymmetry. — Genetica 89: 77–96.

Escós, J. M., Alados, C. L. & Emlen, J. M. 1995: Fractal structures and fractal functions as disease indicators. — Oikos 74: 1–5.

Fowler, J. & Cohen L. 1990: Practical statistics for field biology. — Open University Press, Philadelphia.

Hall, A. J., Law, R. J., Wells, D. E., Harwood, J., Ross, H. M., Kennedy, S., Allchi, C. R., Campbell, L. A. & Pomeroy, P. P. 1992: Organochlorine levels in common seals (Phoca vitulina) which were victims and survivors of the 1988 phovina distemper epizootic. — Sci. Total Environ. 115: 145–162.

Haraguchi, K., Athanasiadou, M., Bergman, Å., Hovander, L. & Jensen, S. 1992: PCB and PCB methyl sulfones in selected groups of seals from Swedish waters. — Ambio 21: 546–549.

Härkönen, T. & Heide-Jørgensen, M. P. 1990: Comparative life histories of East Atlantic and other harbour seal populations. — Ophelia, 32: 211–235.

Hartwig, W.C. 1991: Fractal analysis of the sagittal suture morphology. — J. Morphol. 210: 289–298.

Have, P., Nielsen, J. & Bøtner, A. 1991: The seal death in Danish waters. 2. Virological studies. — Acta vet. Scand. 32: 211–219.

Heide-Jørgensen, M. P., Härkönen, T., Dietz, R. & Thompson, P. M. 1992: Retrospective of the 1988 European seal epizootic. Diseases Aquat. Organisms 13: 37–62.

Helle, E., Olsson, M. & Jensen, S. 1977: PCB levels correlated with pathological changes in seal uteri. — Ambio 5: 261–263.

Holden, A. V. 1978: Pollutants and seals – a review. — Mammal Rev. 8: 53–66.

Honda, E., Domon, M., Sasaki, T., Obayashi, N. & Ida, M. 1992: Fractal dimensions of ductal patterns in the parotid glands of normal subjects and patiens with Sjögren syndrome. — Invest. Radiol. 27: 790–795.

Jones, J. S. 1987: An asymmetrical view of fitness. — Nature 325: 299.

Klausen, B. 1991: Microbiological and immunological aspects of experimental periodontal disease in rats. — Disputats. KBH tandlægehøjskole.

Long, C. A. 1985: Intricate sutures as fractal curves. — J. Morphol. 185: 285–295.

Merilä, J. & Björklund, M. 1995: Fluctuating asymmetry and measurement error. — Syst. Biol. 44: 97–101.

Møller, A. P. 1996: Developmental instability and parasitism: A review. — Oikos. 77: 189–196.

Møller, A. P. & Swaddle, J. P. 1997: Developmental stability and evolutionary biology. — Oxford University Press, Oxford. (In press.)

Mortensen, P., Bergman, A., Bignert, A., Hansen, H. J., Härkönen, T. & Olsson, M. 1992: Prevalence of skull lesions in habor seals (Phoca vitulina) in Swedish and Danish museum collections during the period 1835–1988. — Ambio 21: 520–524.

Olsson, M. & Reutergårdh, L. 1986: DDT and PCB pollution trends in the Swedish aquatic environment. — Ambio 15: 103–109.

Olsson, M., Karlsson, B. & Ahnland, E. 1992: Seals and seal protection: A presentation of a Swedish research project. — Ambio 21: 494–496.

Osterhaus, A. D. M. E. & Vedder, E. J. 1988: Identification of virus causing recent seal deaths. — Nature 335: 20.

Palmer, A. R. & Strobeck, C. 1986: Fluctuating asymmetry: Measurement, analysis, patterns. — Ann. Rev. Ecol. Syst. 17: 391–421.

Palmer, A. R. 1994: Fluctuating asymmetry analyses: A primer. — In: Markow, J. (ed.), Developmental instability: Its origin and evolutionary implications: 335–364. Kluwer, Dordrecht.

Reijnders, P. J. H. 1980: Organochlorine and heavy metal residues in harbour seals from the Wadden Sea and their possible effects on reproduction. — Neth. J. Sea Res. 14: 30–65

Rice, W. R. 1989: Analysing tables of statistical tests. — Evolution 43: 223–225.

Sokal, R. R. & Rohlf, F. J. 1981: Biometry. — W. H. Freeman, San Francisco.

Søndergård, N., Joensen, A. H. & Bøgebjerg, E. 1976: Sælernes forekomst og sæljagten i Danmark. — Danske Vildtundersøgelser 26. 80 pp.

Sumarsono, S. H., Wilson, T. J., Tymms, M. J., Venter, D. J., Corrick, C. M., Kola, R., Lahoud, M. H., Papas, T. S., Seth, A. & Kola, I. 1996: Down's syndrome-like skeletal abnormalities in Ets2 transgenic mice. — Nature 379: 534–537.

Swart, R. L, Ross, P. S., Vedder, L. J., Timmerman, H. H., Heisterkamp, S., Loveren, H. V., Vos, J. G. Reijnders, P. J. H. & Osterhaus, A. D. M. E. 1994: Impairment of immune function in harbor Seals (Phoca vitulina) feeding on fish from polluted waters. — Ambio 23: 155–159.

Touyz, L. Z. G. & Tau, S. 1991a: A classification of palatal osseous alveolar marginal exostoses — POAMES J. Dent. Ass. South Africa 46: 419–421.

Touyz, L. Z. G. & Tau, S. 1991b: Frequency and distribution of palatal osseous alveolar marginal exostoses — POAMES. J. Dent. Ass. South Africa 46: 471–473.

Valentine, D. W. & Soulé, M. 1973: Effetcs of p,p´-DDT on developmental stability of pectoral fin rays in the grunion, Leuresthes tenuis. — Fish. Bull. 71: 921–926.

Zakharov, V. M, Olsson, M. & Yablokov, A.V. 1986: Does environmental pollution affect the development of the Baltic grey seal (Halichoerus grypus). Influence of human activities on the Baltic ecosystem. — Soviet-Swedish Symposium, April 1986: 96–108.

Zakharov, V. M. & Yablokov A. V. 1990: Skull asymmetry in the Baltic grey seal: Effects of environmental pollution. — Ambio 19: 266–269.

Appendices

Appendix 1. Descriptive data of foramina asymmetry. *R* = right side measurement, *L* = left side measurement.

Trait		(*R* + *L*)/2		*R* – *L*					
	N	Mean	*S.E.*	Mean	*S.E.*	Skew	*S.E.*	Kurtosis	*S.E.*
Adults 1889–90									
F. posterior palatinae	18	1.61	0.11	0.22	0.18	0.63	0.54	1.03	1.03
F. hypoglosi	6	1.58	0.20	0.17	0.17	2.45	0.85	6.00	1.74
F. dentale	14	7.68	0.32	0.07	0.47	– 0.21	0.60	1.11	1.15
F. basiorbitalis acs.	13	2.31	0.09	– 0.31	0.24	– 0.24	0.24	– 0.04	1.19
F. dorsal condylar	8	1.50	0.13	– 0.50	0.19	0.00	0.19	– 2.80	1.48
Subadults 1889–90									
F. posterior palatinae	31	1.48	0.08	0.00	0.11	0.84	0.42	2.78	0.82
F. hypoglosi	14	1.14	0.15	0.00	0.10	0.00	0.59	6.50	1.15
F. dentale	43	7.56	0.15	0.35	0.21	– 0.08	0.36	0.53	0.71
F. basiorbitalis acs.	18	2.02	0.10	0.17	0.12	0.32	0.54	0.92	1.03
F. dorsal condylar	14	2.30	0.09	– 0.14	0.19	– 0.52	0.60	0.24	1.15
Cubs 1889–90									
F. posterior palatinae	5	1.20	0.20	0.00	0.00				
F. hypoglosi	8	1.00	0.00	0.00	0.00				
F. dentale	29	7.38	0.19	0.03	0.28	0.81	0.43	2.93	0.85
F. basiorbitalis acs.	20	1.93	0.20	0.20	0.14	1.38	0.51	3.40	0.99
F. dorsal condylar	6	2.41	0.33	– 0.17	0.50	1.21	0.85	– 0.45	1.74
Adults 1955–87									
F. posterior palatinae	16	1.31	0.06	– 0.13	0.20	– 0.32	0.56	– 1.36	1.09
F. hypoglosi	18	1.50	0.12	– 0.22	0.19	– 0.78	0.54	0.24	1.04
F. dentale	17	8.20	0.36	– 0.41	0.35	0.24	0.55	0.99	1.06
F. basiorbitalis acs.	17	2.35	0.13	0.23	0.28	– 0.30	0.55	1.20	1.06
F. dorsal condylar	13	1.69	0.24	– 0.30	0.22	0.54	0.62	1.22	1.19
Subadults 1955–87									
F. posterior palatinae	44	1.30	0.06	0.14	0.10	0.47	0.36	1.04	1.48
F. hypoglosi	40	1.06	0.03	0.03	0.06	0.37	0.37	5.60	7.91
F. dentale	65	7.60	0.15	0.05	0.21	0.29	0.30	0.71	1.21
F. basiorbitalis acs.	43	2.23	0.07	0.73	0.11	0.07	0.36	– 0.32	– 0.45
F. dorsal condylar	39	1.90	0.11	0.77	0.13	– 0.36	0.38	0.08	0.11
Cubs 1955–87									
F. posterior palatinae	12	1.29	0.10	– 0.25	0.19	0.17	0.64	– 0.09	1.23
F. hypoglosi	14	1.00	0.00	0.00					
F. dentale	17	7.18	0.18	– 0.24					
F. basiorbitalis acs.	11	2.23	0.10	0.54	0.16	0.15	0.66	1.86	1.28
F. dorsal condylar	14	1.84	0.23	– 0.07	0.34	0.38	0.60	– 0.49	1.15

(continues …)

Appendix 1. Continues.

Trait		(*R* + *L*)/2		*R* – *L*					
	N	Mean	*S.E.*	Mean	*S.E.*	Skew	*S.E.*	Kurtosis	*S.E.*
Adult females 1955–87									
F. posterior palatinae	5	1.20	0.12	0.00	0.38	0.00	0.91	2.00	2
F. hypoglosi	7	1.57	0.25	0.00	0.32	0.00	0.79	– 1.20	1.59
F. dentale	7	7.64	0.53	– 0.71	0.59	0.25	0.79	– 0.94	1.58
F. basiorbitalis acs.	5	2.30	0.25	– 0.20	0.56	0.51	0.91	– 0.61	2
F. dorsal condylar	4	1.75	0.48	– 0.50	0.33	– 2.00	1.01	4.00	2.61
Adult males 1955–87									
F. posterior palatinae	11	1.36	0.07	– 0.18	0.19	0.89	0.66	– 1.62	1.28
F. hypoglosi	11	1.45	0.13	– 0.36	0.23	0.36	0.66	0.64	1.28
F. dentale	10	8.60	0.46	– 0.20	0.40	0.20	0.69	1.12	1.33
F. basiorbitalis acs.	12	2.38	0.15	– 0.25	0.22	– 0.11	0.64	1.06	1.23
F. dorsal condylar	9	1.67	0.29	0.26	0.33	– 0.04	0.72	– 0.22	1.40
Subadult females 1955–87									
F. posterior palatinae	26	1.27	0.08	0.04	0.14	– 0.05	0.52	0.04	1.01
F. hypoglosi	25	1.42	0.03	0.04	0.10	0.43	0.55	3.96	1.06
F. dentale	40	7.92	0.18	– 0.18	0.35	0.66	0.42	0.85	0.82
F. basiorbitalis acs.	25	2.18	0.10	0.04	0.14	1.52	0.52	5.60	1.01
F. dorsal condylar	25	1.89	0.14	– 0.08	0.23	– 0.60	0.55	– 0.18	1.06
Subadult males 1955–87									
F. posterior palatinae	30	1.24	0.07	0.07	0.13	0.79	0.46	1.92	0.90
F. hypoglosi	29	1.03	0.02	0.00	0.06	0.00	0.48	5.00	0.93
F. dentale	42	7.74	0.18	0.14	0.24	– 0.25	0.40	0.42	0.78
F. basiorbitalis acs.	29	2.20	0.07	0.28	0.16	– 0.60	0.47	– 1.16	0.91
F. dorsal condylar	28	1.91	0.14	– 0.25	0.13	0.14	0.49	– 0.28	0.95
Adults shot 1955–87									
F. posterior palatinae	11	1.36	0.07	– 0.36	0.26	0.84	0.66	– 0.76	1.27
F. hypoglosi	13	1.46	0.15	0.68	0.19	0.20	0.62	– 0.46	1.19
F. dentale	15	8.43	0.35	– 0.46	0.38	0.64	0.58	1.26	1.12
F. basiorbitalis acs.	12	2.46	0.17	– 0.42	0.38	0.07	0.63	0.62	0.51
F. dorsal condylar	9	1.94	0.31	– 0.33	0.29	– 0.65	0.72	0.82	0.59
Adults caught in net 1955–87									
F. posterior palatinae	5	1.20	0.12	0.40	0.28	0.60	0.91	– 3.33	2.00
F. hypoglosi	5	1.60	0.19	– 0.40	0.49	– 0.40	0.91	– 0.17	2.00
F. dentale	2								
F. basiorbitalis acs.	5	2.10	0.10	0.20	0.63	2.23	0.91	5.00	2.00
F. dorsal condylar	4	1.13	0.13	– 0.25	0.79	– 2.00	1.01	4.00	2.61
Subadults shot 1955–87									
F. posterior palatinae	4	1.19	0.08	0.00	0.25	0.00	0.56	1.89	1.09
F. hypoglosi	12	1.08	0.06	0.00	0.12	0.00	0.64	5.50	1.23
F. dentale	40	7.70	0.21	– 0.15	0.27	0.49	0.37	1.26	0.73
F. basiorbitalis acs.	16	2.19	0.09	0.13	0.15	– 0.06	0.56	0.05	1.09
F. dorsal condylar	11	1.77	0.25	– 0.45	0.20	– 0.21	0.66	– 2.44	1.27
Subadults caught in net 1955–87									
F. posterior palatinae	40	1.34	0.06	0.08	0.11	0.38	0.37	0.49	0.73
F. hypoglosi	42	1.60	0.02	0.02	0.04	1.03	0.36	12.00	0.71
F. dentale	42	6.50	0.15	0.12	0.22	0.36	0.36	0.33	0.72
F. basiorbitalis acs.	38	2.10	0.08	0.18	0.12	0.13	0.38	– 0.17	0.75
F. dorsal condylar	42	1.13	0.11	– 0.09	0.15	0.05	0.36	0.18	0.72
Adults 1988									
F. posterior palatinae	78	1.42	0.04	0.01	0.09	0.52	0.27	0.17	0.54
F. hypoglosi	69	1.60	0.07	– 0.01	0.09	0.02	0.29	0.17	0.57
F. dentale	7	7.45	0.15	0.13	0.57	0.00	0.27	– 0.06	0.54
F. basiorbitalis acs.	77	2.18	0.05	– 0.02	0.08	0.04	0.27	0.17	0.54
F. dorsal condylar	70	1.56	0.07	– 0.02	0.11	0.16	0.29	2.09	0.57

(continues …)

Appendix 1. Continues.

Trait		(*R* + *L*)/2		*R* – *L*					
	N	Mean	*S.E.*	Mean	*S.E.*	Skew	*S.E.*	Kurtosis	*S.E.*
Subadults 1988									
F. posterior palatinae	32	1.35	0.10	0.06	0.12	– 0.07	0.41	– 0.61	0.81
F. hypoglosi	37	1.69	0.09	– 0.03	0.11	0.58	0.39	1.11	0.76
F. dentale	37	7.74	0.18	0.51	0.28	0.02	0.39	0.12	0.76
F. basiorbitalis acs.	32	2.30	0.08	0.16	0.12	0.47	0.41	0.83	0.81
F. dorsal condylar	37	1.94	0.16	– 0.11	0.14	– 0.57	0.39	– 0.11	0.76
Adult females 1988									
F. posterior palatinae	36	1.39	0.06	0.06	0.14	0.33	0.39	– 0.06	0.77
F. hypoglosi	31	1.71	0.10	0.00	0.14	0.55	0.42	0.68	0.82
F. dentale	36	7.43	0.19	0.08	0.22	0.06	0.39	– 0.18	0.77
F. basiorbitalis acs.	35	2.20	0.09	0.00	0.11	0.36	0.40	– 0.43	0.78
F. dorsal condylar	32	1.41	0.09	– 0.28	0.15	– 0.23	0.41	1.99	0.81
Adults shot 1955–87									
F. posterior palatinae	29	1.47	0.07	– 0.03	0.14	0.78	0.43	0.33	0.85
F. hypoglosi	26	1.65	0.09	– 0.07	0.14	– 0.09	0.16	0.46	0.89
F. dentale	29	7.62	0.32	0.00	0.32	0.09	0.43	– 0.57	0.43
F. basiorbitalis acs.	29	2.26	0.07	– 0.03	0.15	0.04	0.43	– 0.67	0.85
F. dorsal condylar	26	1.60	0.12	0.19	0.20	1.30	0.46	3.70	0.89
Subadult females 1988									
F. posterior palatinae	14	1.17	0.12	– 0.07	0.16	0.02	0.59	0.30	1.15
F. hypoglosi	18	1.61	0.12	0.00	0.11	0.00	0.53	2.44	1.04
F. dentale	17	7.88	0.30	0.47	0.41	0.31	0.55	– 8.00	1.06
F. basiorbitalis acs.	14	2.25	0.13	0.36	0.19	– 0.43	0.60	– 0.39	1.15
F. dorsal condylar	18	2.00	0.26	– 0.22	0.22	– 0.45	0.54	– 0.39	1.04
Subadult males 1988									
F. posterior palatinae	9	1.50	0.24	0.22	0.27	– 0.50	0.72	– 1.27	1.40
F. hypoglosi	10	1.23	0.22	– 0.40	0.30	2.00	0.69	4.18	1.33
F. dentale	12	7.92	0.27	0.83	0.43	– 0.41	0.64	– 0.29	1.23
F. basiorbitalis acs.	9	2.33	0.17	0.22	0.22	3.00	0.72	9.00	1.40
F. dorsal condylar	10	1.60	0.18	0.20	0.24	– 0.40	0.69	– 1.07	1.33

Appendix 2. Descriptive data of asymmetry.

Trait		(*R* + *L*)/2		*R* – *L*						
	N	Mean	*S.E.*	Mean	*S.E.*	Skew	*S.E.*	Kurtosis	*S.E.*	Slope
Teeth 1889–90										
Lower 2. molar	7	7.70	0.19	0.13	0.04	0.51	0.794	– 1.32	1.587	0.01
Lower 3. molar	33	8.98	0.09	0.05	0.02	0.62	0.409	– 0.23	0.80	0.05
Upper 2. molar	16	8.66	0.16	0.07	0.06	0.64	0.58	– 0.13	1.121	– 0.13
Upper 3. molar	28	8.70	0.11	– 0.04	0.03	– 0.38	0.448	0.00	0.872	– 0.01
Teeth 1955–87										
Lower 2. molar	26	7.95	0.10	0.04	0.07	– 0.27	0.456	– 0.58	0.887	0.09
Lower 3. molar	69	8.96	0.06	– 0.03	0.02	– 0.54	0.293	1.68	0.578	0.02
Upper 2. molar	29	8.64	0.10	0.00	0.04	0.8	0.434	0.94	0.845	– 0.03
Upper 3. molar	41	8.93	0.07	0.04	0.03	0.6	0.369	0.81	0.724	– 0.07
Teeth 1988										
Lower 2. molar	33	7.53	0.08	0.06	0.04	– 0.67	0.409	1.56	0.80	– 0.05
Lower 3. molar	41	8.64	0.08	0.01	0.02	0.25	0.369	0.11	0.724	0.02
Upper 2. molar	45	8.22	0.09	0.05	0.04	1.72	0.354	3.6	0.695	0.04
Upper 3. molar	39	8.62	0.08	0.00	0.03	– 0.24	0.378	0.11	0.741	0.03

(continues …)

Appendix 2. Continues.

Fractal asymmetry of *maxillo palatinae* suture

Period	*N*	Mean	*S.E.*	Mean	*S.E.*	Skew	*S.E.*	Kurtosis	*S.E.*
1889–90	9	1.045	0.003	0.013	0.005	0.510	0.717	– 0.700	1.399
1955–87	20	4.055	0.004	– 0.003	0.006	– 0.131	0.512	0.724	0.992
1988	21	1.052	0.002	0.004	0.00	0.498	0.501	– 0.470	0.972

R = right side measurement, *L* = left side measurement; slope– slope of regression of $R - L$ against $(R + L)/2$