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# High frequency of chick diseases in nominate Lesser Black-backed Gulls *Larus f. fuscus* from the Gulf of Finland

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Diseases due to degeneration and inflammation of various internal organs were an important cause of death in Lesser Black-backed Gull chicks at Söderskär, central Gulf of Finland, from 1991 to 1993. Chicks either died in the nest within four days of hatching (17%) or, after having first grown normally, at the age of one to three weeks (mean 15 days, 13%). The former ones lost, on average, 15% of their body weight during the two days prior to death and the latter ones 23% in three days. Pathological findings were similar in both groups: degenerations in liver, cardial muscle and kidneys, inflammation in the intestine, and sepsis, the final cause of death. About half of the total number of chicks under surveillance, however, disappeared; their growth rate resembled that of the latter diseased group. Predation by Herring Gulls *L. argentatus* took 10% of the Lesser Black-back chicks, mostly from the best-growing group. A similar differentiation in chick fates was apparent in local Common Gulls *L. canus*, although Herring Gull predation partly masked the effect (35% depredated). In Herring Gulls, however, the latter diseased group was totally absent. The outbreak of diseases did not seem to be starvation-induced. Supplementary fed Lesser Black-back chicks died in the same proportion as chicks that were not given extra food. It is concluded that rapid weight loss is a symptom of a wide variety of physiological disorders and could not be used unequivocally as an indicator of food deficiences in the local environment.

## 1. Introduction

Slow growth and low body weight occur along with elevated chick mortality in many studies on gull reproduction. Chicks that lose weight and die in the nest within a few days are commonly viewed as starving to death. Although never actually verified, the starvation effect has been proposed to stem from parents' inability to find food for the whole brood (e.g. Harris 1964, Haycock & Threlfall 1975, Barrett & Runde 1980, Galbraith 1983, Thomas 1983, Murphy et al. 1984, Hébert & Barclay 1986, Pugesek 1993), or to their behavioural anomalies in brood care (Kadlec et al. 1969, Hario 1990). Whatever the causes, the only evidence of starvation is usually the weight loss of moribund chicks. In some cases, necropsy has revealed an empty alimen-

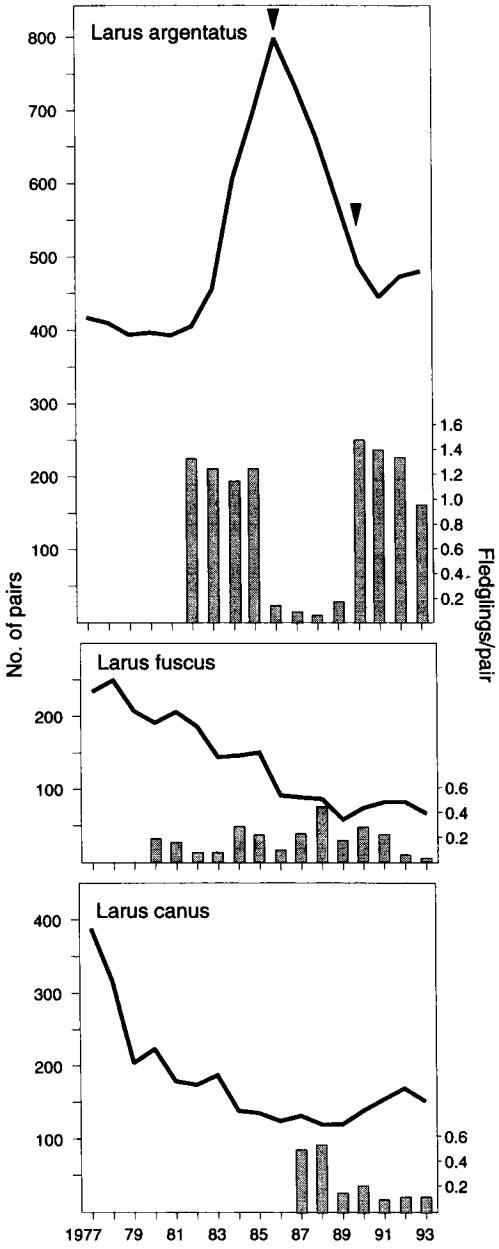


Fig. 1. Population trends of Herring Gulls, Lesser Black-backed Gulls and Common Gulls at Söderskär for 1977–93 (no. of pairs, graphs). Bars indicate the mean numbers of fledglings per nest in different years (scale to the right). Arrows give the period of egg culling on Herring Gulls (1986–89).

tary tract, this being a further example of starvation (Hario 1990, Strann & Vader 1992).

During the last two to three decades, the populations of the nominate race of the Lesser Black-backed Gull (*Larus fuscus fuscus*) have experienced a drastic decline in northern Scandinavia and the Baltic, i.e. over most of the subspecies' breeding range (Hario 1990). Gulleries in the Gulf of Finland suffer from a high rate of chick mortality, being able to produce less than 0.2 fledglings per pair (Hario 1994). Chick deaths in nominate Lesser Black-backed Gulls have been linked with presumed depletion of gulls' food stocks in northern Norway (Bevanger & Thingstad 1990, Strann & Vader 1992). In the Gulf of Finland, food itself is not scarce, but nevertheless the chick mortality is exceedingly high (Hario 1990, 1994).

In this study, we assess the validity of chick weight loss as an indicator of starvation and a predictor of prevailing feeding conditions. We compare breeding parameters of three species of gulls living in the same local area in the Gulf of Finland: Herring Gull, Lesser Black-backed Gull of the nominate race and Common Gull. The emphasis is on the nominate Lesser Black-backed Gull, this form being distinctly different from the western race *graellsii* in many aspects (for a review, see Strann & Vader 1992).

## 2. Materials and methods

### 2.1. Ecological data

The field data were collected during 1991–93 at the Söderskär Game Research Station, central Gulf of Finland (c. 60°N,25°E). The study area is a bird sanctuary, located in the outermost archipelago, 25 km southeast of Helsinki. It consists of 25 islets, all rich in seabirds (detailed in Hario et al. 1986). The population trends of the three gull species studied are presented in Figure 1. The nominate Lesser Black-backed Gull has decreased by 72% since 1977. Common Gulls have declined by 61% during the same time. The population trend of the Herring Gull has been affected by culling programs and is presently increasing again (detailed in Hario 1994).

In each of the three main study colonies (one for each species) nests were marked and mapped as soon as they were located, and their contents were surveyed at 1-2(3)-day intervals during laying. Timing of breeding was defined as the date of the

laying of the first egg in a nest. Eggs were marked (on the blunt end) in water-proof ink with their ordinal number in the clutch (henceforth: A, B and C-egg). Their laying sequence was determined from direct observations or from the hatching order. The eggs were measured to the nearest 0.1 mm with a sliding caliper, and the volume was calculated from the equation of Harris (1964, see also Bolton 1991). The percentage deviation of the C-egg volume from the volume of the A-egg was used to express the so-called egg size asymmetry within the clutch, an important predictor of the fate of the C-chick (see e.g. Kilpi 1995).

The fate of the chicks was monitored during the whole chick-rearing period. This was done 1) by ringing each chick soon after hatching, 2) by locating the preyed-on chicks' rings in predatory gulls' territories (in food remnants), 3) by weighing each chick whenever it was found (to the nearest gram with a spring balance), and 4) by collecting carcasses of dead chicks for further pathological studies. The growth data were completed with additional monitoring of 156 Lesser Black-back chicks in four other colonies in the Söderskär archipelago during 1991-93. The total ecological data consist of the growth and/or fate of 277 Lesser Black-back chicks, 185 Common Gull chicks and 137 Herring Gull chicks.

Survival of the chicks of the three main study colonies was examined in relation to the hatching sequence, the egg size asymmetry within the clutch, the hatchling weight and the timing of breeding.

### 2.2. Pathological studies

Chicks found dead in the study colonies and suitable for examination were taken for post mortem at the National Veterinary and Food Research Institute in Helsinki. Necropsy was performed on most specimens found dead in 1991 and 1993; in 1992 the corpses were left in position in order to estimate the effect of scavenging on the predation rates (see Hario 1994).

Chicks were examined for cause of death by routine necropsy followed by histological investigations. Bacteriological examination was always done on the lung and intestine and on other organs according to gross findings. Sample sizes were as follows: 31 Lesser Black-back chicks, 8 Common Gull chicks and 5 Herring Gull chicks, i.e. 35% of the total number of chicks found dead. The small sample sizes resulted from difficulties in logistics. To be suitable for bacteriological examination a chick's corpse could not be frozen, and many were badly decomposed already when found, especially during warm weather periods. The shipping from Söderskär to Helsinki, a 25 km long sea voyage, posed further problems, especially during windy weather.

### 2.3. Supplementary feeding

In order to pinpoint the reasons behind the rapid death (i.e. factors inducing intestinal inflammation with accompaning sepsis, the final causes of death, see later), 19 newly-hatched Lesser Black-back chicks were supplementary fed for five days by dosing the food directly into the gizzard. The food load consisted of 30 ml ground fish (Baltic Herring *Clupea harengus*) mixed with water, an amount sufficient for the maintenance of the Herring Gull chicks (own obs., see also Kahru & Keskpaik 1978). The supplements were given once a day during the normal colony visits; chicks were not taken away from their nest sites. The growth and fate of this test group was compared with those of a control group of 20 similar-aged Lesser Black-back chicks, not supplementary fed. The samples were randomised by assigning a number to each nest and then picking a sample of numbers randomly. Then, every other C-hatchling was taken as a test chick and every other C-hatchling as a control chick. The test was done in 1995 over the entire Söderskär area (52 nests under surveillance). Chicks found dead were necropsied in the same manner as the chicks studied during 1991-93. The weather in the 1995 hatching season was unusually warm and sunny; thus, of the 19 dead chicks only four were suitable for examination.

## 3. Results

### 3.1. Overall breeding success

In the three main study colonies, egg hatchability did not deviate between species (Table 1, $G^2 = 1.25$, $df = 2$, n.s.). Common Gulls experienced a slightly higher rate of egg mortality in the form of addled

eggs, but the difference was not significant ($G^2 = 2.73$, n.s.). The fertility of addled eggs could not be assessed.

Mortality of pipped eggs was extremely low, 0.7–0.9% in the three species.

Final fledging success was highest in Herring Gulls and lowest in Common Gulls: Herring Gulls produced 1.20 fledglings per pair, Lesser Black-backed Gulls 0.12 and Common Gulls none ($G^2 = 153.2$, df = 2, $P < 0.001$).

### 3.2. Chick mortality

The striking differences in fledging success were an outcome of differences in chick mortality among species. Of the verified losses compiled in Table 2, the category "intact corpse found" refers to chicks found dead in the colony, with no injuries to the body nor signs of pecking, attributable to adult aggression. There were a few such pecked chicks, but they were all Herring Gulls, assigned as victims of territorial aggression in Table 2.

It was the "intact corpse found" category from which individuals were taken for pathological investigations. In the following, only the chicks of this category will be treated along with the "disappeared" ones. The predation and the territorial aggression have been dealt with elsewhere (Hario 1994).

### 3.3. Timing of death and sequence in the clutch

Lesser Black-back chicks of "intact corpse found" category could be divided into two gross "death waves":

1) those that died in the nest within four days of hatching, showing poor weight gain or none at all, and
2) those that first grew normally, but at the age of one to three weeks (mean 15.1 days, SD = 6.6, median 14 days, N = 35) died of similar symptoms as the former group, and, equally suddenly, within four days after the first weight loss (see Fig. 2).

Nine chicks or 19% of the first "death wave" died on day 0, only a few hours after hatching, some even as hatchlings (being still "wet").

In the Common Gull, the same pattern of chick mortality was discernable although not as distinctly (Fig. 2). This was due to Herring Gull predation partly masking the effect. An edge-nesting pair of Herring Gulls effectively took Common Gull chicks from the study colony irrespective of chicks' condition and nest location (see Hario 1994). At most, the food remnants of this predatory pair included 40 rings of Common Gull chicks from the neigh-

Table 1. Certain breeding parameters in the three main study colonies at Söderskär during 1991–93 (combined).

| | Herring Gull | Lesser Black-backed Gull | Common Gull |
|---|---|---|---|
| No. of nests | 54 | 49 | 74 |
| Eggs laid | 153 | 139 | 216 |
| Mean clutch size | 2.83 | 2.84 | 2.92 |
| Hatching rate % (N) | 90 (137) | 87 (121) | 86 (185) |
| Addled eggs % | 5.2 | 4.3 | 8.3 |
| Mortality of pipped eggs % | 0.7 | 0.7 | 0.9 |
| Nest success % | 94 | 90 | 95 |
| Mean brood size | 2.54 | 2.47 | 2.50 |
| Fledging rate % (N) | 47 (65) | 5 (6) | 0 |
| Nest success % | 63 | 10 | 0 |
| Fledglings/pair | 1.20 | 0.12 | 0 |

Table 2. Numbers of chicks lost to various causes at Söderskär during 1991–93 (combined). Data of Lesser Black-backed chicks stem from five colonies (main study colony included), those of Herring Gulls and Common Gulls each from one colony. Percentages in parentheses. N = no. of chicks hatched.

| | Herring Gull N = 137 | Lesser Black-back N = 277 | Common Gull N = 185 |
|---|---|---|---|
| Verified predation [1] | 4 (2.9) | 27 (9.7) | 65 (35.1) |
| Intact corpse found | | | |
| 0–4 days | 11 (8.0) | 48 (17.3) | 14 (7.6) |
| >4 days | 0 | 35 (12.6) | 16 (8.6) |
| Accidents & other causes [2] | 6 (4.4) | 4 (1.4) | 1 (0.5) |
| Territorial aggression | 12 (8.8) | 0 | 0 |
| Disappeared | 36 (26.3) | 147 (53.1) | 89 (48.1) |
| Total losses | 69 (50.4) | 261 (94.2) | 185 (100) |

[1] Chick's ring and remnants found at predator's nest site.
[2] Chicks fallen in crevices, entangled in vegetation, lost to flooding, etc.

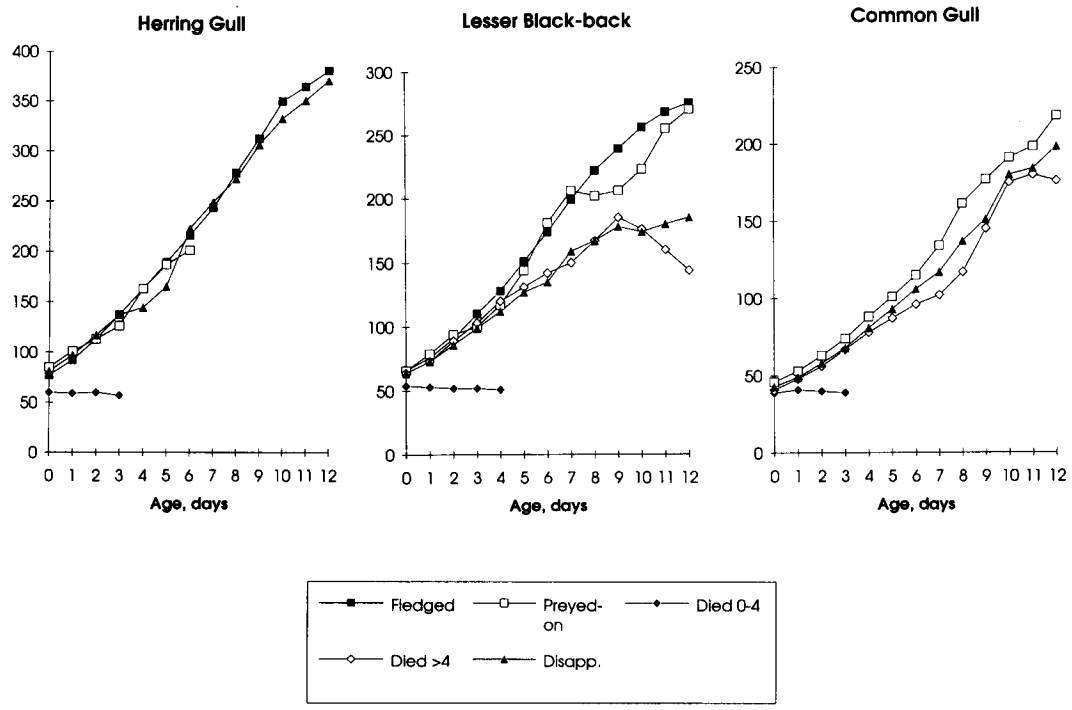


Fig. 2. Weight gain of gull chicks according to age at Söderskär during 1991–93. Running 3-day means. Sample sizes given in Table 2. Weights of dying chicks excluded. Curves of "Died 0–4 days" and "Died > 4 days" refer to the first and second "death wave", respectively.

bourhood, i.e. 63% of that year's (1992) production of the study colony.

Only one Common Gull chick died as a hatchling, on day 0. In the Herring Gull, the latter "death wave" was totally absent, and only one chick died on day 0.

The data are heavily skewed to the C-chicks, the last-hatched chick in a clutch. Of the Lesser Black-back chicks of the "intact corpse found" category, 75% were C-chicks, of Herring Gulls 73% and of Common Gulls 50% ($G^2 = 4.54$, n.s.). Timing of breeding did not explain the occurrence of these chicks: they were not the offspring of late or early breeders (Table 3). The same applies to the egg size asymmetry within a clutch (the volume of the C-egg as percentage of the volume of the A-egg, Mann-Whitney U-test, $P > 0.1$), although the C-egg tended to be proportionally smaller in clutches of "intact corpse found". Also, body weight at hatching did not differ between chicks found dead and healthy chicks in any of the categories (A-, B- and C-chicks within species), except in Common Gull C-chicks (Mann-Whitney, $U = 2.96$, $P = 0.003$).

Hence, no external factor measured (timing of breeding, egg size, chick weight) could predict the fate of the chick, only the sequence in the clutch (= C).

### 3.4. Chick diseases

All chicks necropsied, irrespective of gull species or the sequence in the clutch, had similar pathological changes: degenerations in liver, cardial muscle and kidneys, inflammations (mostly in lungs and intestine) and sepsis (Table 4). The alimentary tract was empty, and the final cause of death was sepsis. Frequency of sepsis did not deviate from the occurrences of intestinal inflammation ($G^2 = 1.23$) and liver degeneration in chick deaths ($G^2 = 0.98$, Table 4).

Chick death was always preceded by a marked reduction in body weight. Chicks of the first "death wave" lost, on average, 15.5% of their body weight (SD = 9.8, median 16.7, N = 26) during the two days they stayed alive after the first signs of weight loss (mean $1.8 \pm 1.0$ days, median 2, N = 26). The

corresponding figures for the second "death wave" were 23.4 ± 7.6% (median 22.6, N = 10) and three days (3.3 ± 2.1, median 3, N = 10). These data refer to corpses sufficiently fresh for necropsy (in Table 4) and, consequently, possibly least prone to weight loss through dehydration. They, of course, exclude dead hatchlings.

### 3.5. Supplementary feeding

There was no difference in mortality between supplementary fed Lesser Black-back chicks and controls; about half of the chicks died in both groups ($G^2 = 0.024$ n.s., Table 5). Nor was there any difference in hatching weight between surviving and succumbing chicks in the two groups (two-way ANOVA, $F = 2.74$, $P = 0.11$). In contrast, weight at the age of three to four days differed significantly between survivors and moribund chicks in both groups, survivors having gained weight and moribund chicks having lost weight (Table 5, $F = 40.27$, $P < 0.001$, interaction fate × treatment $F = 0.23$, $df = 1, 24$, $P = 0.64$). The weight gain of the supplementary fed survivors tended to be greater than that of the surviving controls, but the difference was not statistically significant ($t = -2.10$, $P = 0.104$).

The pathological changes in the few necropsied test chicks were basically similar to those in the wild chicks studied during 1991–93 (Table 4).

### 3.6. Chick disappearances

The final "fate" of most chicks under surveillance was disappearance (Table 2), a common observation in all studies on ground-nesting larids conducted so far. Lesser Black-back chicks that disappeared showed a growth rate grossly similar to that of the latter "death wave", deviating markedly from the steady growth of fledglings and most preyed-on chicks (Fig. 2). This suggests that a substantial proportion of them were in fact moribund and died hidden in vegetation, remaining undiscovered. The size of this group cannot be assessed accurately. Note that the growth curve of the preyed-on Lesser Black-back chicks on days 8 to 10 also approached that of the second "death wave" (Fig. 2), indicating that there might have been some moribund chicks included in them, too. The slight terminal up in the second "death wave" curve in Figure 2 was only temporal, levelling off the next day, but the material became too scanty to allow for a further extension of the graph. In Herring Gulls, the disappeared chicks gained weight equally well as those subsequently fledging. This implies that they were equally healthy and equally prone to predation. In the Common Gull, the picture is obscure, mainly due to the heavy predation rate.

## 4. Discussion

### 4.1. Recognising the diseased chicks

Judged merely from their weight loss and the empty alimentary tract the necropsied chicks in this study would have conventionally been considered as starving to death. Rapid weight loss, however, is a symptom of a wide variety of physio-

Table 3. Differences in timing of breeding, hatchling weight and egg size asymmetry between broods of healthy chicks and those containing at least one diseased chick at Söderskär during 1991–93 (combined). Mean (SD), N = no. of broods. The sample sizes vary according to data available.

| | Timing of breeding, date (SD) | | Hatchling weight of the C-chick, g (SD) | | Egg size asymmetry % (SD) | |
|---|---|---|---|---|---|---|
| | Diseased | Healthy | Diseased | Healthy | Diseased | Healthy |
| Herring Gull | 27.4. (6) | 28.4. (5) | 65.5 (6.2) | 64.5 (5.3) | 8.8 (0.6) | 7.3 (0.6) |
| (N) | (10) | (28) | (6) | (18) | (9) | (29) |
| Lesser Black-backed Gull | 16.5. (4) | 17.5. (4) | 53.5 (5.3) | 55.3 (3.6) | 6.2 (0.7) | 4.2 (0.3) |
| (N) | (22) | (16) | (13) | (15) | (22) | (15) |
| Common Gull | 14.5. (6) | 13.5. (4) | 35.4 (2.6) | 38.8 (3.1) | 8.2 (0.5) | 7.2 (0.4) |
| (N) | (20) | (32) | (11) | (32) | (19) | (28) |

logical disorders and stems much from the diminishing water content of the body. As the 0-day-old chicks examined (having only lived a few hours) also had the diseases it seems that the degenerations and inflammations are possibly innate, affecting particularly the last hatching chick. They may stem from the biochemical processes embedded in vitamin mobilisation in the body, disorders of which cause increased susceptibility to infections. Breakdowns in the enzyme repertoire may also be involved.

However, the biochemical pathway of these disorders cannot be studied without an experimental approach. The diseases described here are by no means unique in the wild, but the etiology of the causal agent involved (entering the chick from the female via the yolk sac) is hard to study (see, e.g. Giesy et al. 1994).

One might protest, however, that the intestinal inflammation of older chicks may have been induced by starvation, this in turn leading to difficulties in digestion, accelarated weight loss, sepsis and rapid death. In semiprecocial gulls, this means parental failure in feeding. Yet, in our feeding experiment, chicks that received extra food died in equal proportion to chicks that were not given extra food. An empty alimentary tract *per se* was clearly not an inductor of intestinal inflammation and the accompanying sepsis.

However, one could further argue that an extra food load once a day is too small an amount to save a chick if parents systematically expell the C-chick at every feeding bout. There seems to be a common consensus about the death of the C-chick being an outcome of preferential provision for larger chicks by the parents (e.g. Parsons 1975, Hébert & Barclay 1986), i.e. letting the C-chick starve at an early age when little investment has been allocated to the brood. Starvation is considered the functional factor affecting the pruning of C-chicks, even in cases of high parental quality (Bolton 1991). In our study, however, also 2-week-old chicks perished of the same symptoms after a period of good growth and a lot of parental investment. We will refer further to the above mentioned newly-hatched chicks, whose death seemed to be too early to be induced merely by systematic expelling.

Table 4. Occurrence of various pathological lesions in dead chicks of the three gull species, ranked according to the sequence in the brood (A, B and C-chicks) and the age at death (0-4-day-old and 2-week-old referring to the first and second "death wave", respectively). Data for experimental chicks of 1995 also included; "fed" means chicks that received food supplements, "controls" were not given extra food. N = no. of chicks studied.

| | Herring Gull | | | Lesser Black-backed Gull | | | | | | Common Gull | | | | | Experimentals | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0-4-d | | | 0-4-d | | | 2-wk | | | 0-4-d | | | 2-wk | | Fed | Controls |
| | A | B | C | A | B | C | A | B | C | A | B | C | A | B | C | C |
| N | 1 | 3 | 1 | 2 | 4 | 11 | 4 | 4 | 2 | 1 | 2 | 4 | | 1 | 1 | 3 |
| Inflammations | | | | | | | | | | | | | | | | |
| intestine | 1 | 2 | 1 | 1 | 2 | 4 | 4 | 2 | 2 | | 2 | 3 | | 1 | 1 | 3 |
| liver and kidneys | | | | | | | | | | 1 | 3 | 1 | | | | |
| lungs | | | | | 1 | 5 | 3 | 1 | 1 | | 1 | | | | 1 | |
| cardial muscle | | 1 | | | | 1 | | | | | | | | | | |
| umbilicus | | | | | 1 | 1 | | | | | | 1 | | | | 1 |
| Degenerations | | | | | | | | | | | | | | | | |
| liver | 1 | 3 | 1 | 2 | 4 | 11 | 4 | 4 | 2 | | 2 | 4 | | | | 3 |
| cardial muscle | 1 | 1 | 1 | 2 | 4 | 9 | 4 | 2 | 1 | | 2 | 4 | | 1 | | 1 |
| kidneys | 1 | 2 | 1 | 1 | 4 | 10 | 1 | 3 | 1 | | 2 | 4 | | 1 | | |
| lungs | | | | | | | | | | | 2 | 3 | | 1 | | |
| Sepsis | 1 | 3 | 1 | 2 | 4 | 11 | 4 | 3 | 2 | | 2 | 4 | | 1 | 1 | 3 |
| Other symptoms | | | | | | | | | | | | | | | | |
| anemia | | | | | | 2 | | | | | | | | | | 1 |
| persistent yolk sac | | | | | 1 | 3 | | | | | | | | | | |

Why should C-chicks in particular suffer from diseases? Would it be more evident that they at first suffer merely from undernourishment due to their poor competitive ability in relation to their sibs? Then, this "third chick disadvantage" (e.g. Parsons 1970, Lundberg & Väisänen 1979) might lead to an early outbreak of diseases. Undeniably, last is the least in most broods and litters throughout the animal kingdom. However, this wasting syndrome due to diseases applies to fully precocial species as well (e.g. Milonoff & Lindén 1989).

In our study, however, not all diseased chicks were the "least ones". Their hatching weight was not significantly smaller than that of the healthy C-chicks. Nor was the egg size asymmetry within their brood particularly large. Besides, many diseased chicks were B or even A-chicks.

### 4.2. Concluding remarks

We can offer no final solution to the problem of gull chick diseases. It seems that their etiology as a whole is poorly understood (see e.g. Gilbertson 1983, Gilbertson et al. 1991). Thirty-five percent of chicks found dead in this study were necropsied, and they all had died of degeneration and inflammation of various internal organs. Therefore, we cannot consider every gull chick losing weight in our study area as being expelled by their parents, in contrast to many other studies made on the western race *graellsii*. Consequently, low overall breeding success with high chick mortality does not necessarily mean that food is scarce. A hatchling suffering from diseases may die soon regardless of the overall feeding conditions for the parents. The spawning stock biomass of the Baltic Herring, the preferred food of fish-eating seabirds in the Gulf of Finland, has increased tremendously being now close to the highest levels recorded (Anon. 1993). The populations of all the other seabirds in this sea area are thriving (Hildén & Hario 1993). The nominate Lesser Black-backed Gull is nowadays the most striking exception from this overall welfare. The frequency of chick diseases seems to be a prime contributor to the population decline, notwithstanding the high occurrence of chick disappearances in this study.

Table 5. Body weight (g) of supplementarily fed vs. control Lesser Black-back chicks according to subsequent fate. Mean ± SD.

| | Died | Survived |
|---|---|---|
| Supplementarily fed | N = 10 | N = 9 |
| at hatching | 56.1 ± 4.4 | 56.3 ± 3.5 |
| on days 3–4 | 51.3 ± 7.3 | 92.5 ± 20.0 |
| Controls | N = 9 | N = 11 |
| at hatching | 59.0 ± 5.8 | 58.5± 3.0 |
| on days 3-4 | 54.5 ± 10.6 | 90.4 ± 20.9 |

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## Selostus: **Selkälokin poikasten korkea tautikuolleisuus Suomenlahdella**

30% selkälokin poikasista löytyi kuolleina Söderskärin yhdyskunnista vuosina 1991–93 (taulukko 2). Kalalokin vastaava osuus oli 16% ja harmaalokin 8%. Kaikki tutkitut lokinpoikaset (yht. 35% kuolleina löytyneistä) olivat menehtyneet sisäelinsairauksiin. Yleisin tautitila käsitti maksarappeutuman ja suolistotulehduksen, ja poikasen lopullinen kuolinsyy oli verenmyrkytys (taulukko 4). Munien kuoriutuvuudessa ei ollut lajien välillä merkitseviä eroja (taulukko 1). Tutkimusyhdyskunnan selkälokit tuottivat 0.12 lentopoikasta/pari, harmaalokit 1.20 ja kalalokit eivät yhtään vuosina 1991–93. Kalalokin täydellisen epäonnistumisen aiheutti ennen muuta harmaalokin saalistus. Huonon poikastuoton johdosta selkä- ja kalalokin kannat ovat selvässä laskussa tutkimusalueella (kuva 1).

Selkälokin ja kalalokin poikaset menehtyivät tauteihin kahdessa vaiheessa: joko heti kuoriuduttuaan tai vasta kahden viikon ikäisinä (kuva 2). Molemmissa tapauksissa poikasten tautitila oli yllä kuvatunlainen. Harmaalokin poikasilta puuttui jälkimmäinen "kuolemanaalto"; sen harvat kuolleet poikaset poimittiin pesiltä pian kuoriuduttuaan.

Kaikkien kolmen lajin pesyeissä viimeisenä kuoriutuneen poikasen kuolevuus oli suurinta (taulukko 3). Ruokintakokeissa selkälokin kuopuksen lisäruokkiminen ei estänyt tautitilan puhkeamista viiden ensimmäisen elinpäivän aikana. Siten tautitilat eivät olleet seurausta nälkiintymisestä, vaan verenmyrkytykseen johtavat tulehdukset näyttivät

syntyvän ravitsemuksesta riippumatta, ja niille altistava tekijä on poikasella ilmeisesti jo syntymästään. Siten selkälokkiemot eivät tarkoituksellisesti eliminoineet nuorinta poikastaan jättämällä sen ravinnotta, mikä on vastoin pesyeen pienentämishypoteesin eräitä olettamuksia. Ei liioin pesimäalueiden yleistä ravintotilannetta voida pitää selkälokin poikastappioiden syynä (ravintokalakannat ovat päinvastoin hyvin suuret).

Poikasten elinsairaudet saattavat olla määrällisesti merkittävin tappiotekijä selkälokin pesinnässä Söderskärillä. Harmaalokin saalistus on paikallinen ongelma, koska se vie poikasista myös laadullista parhaimmistoa eli niitä, jotka ovat välttyneet taudeilta. Elinsairauksien alkusyytä ei toistaiseksi tunneta eikä sitä, miksi selkälokki altistuu niille herkemmin kuin muut saariston lokit.

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