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MORTALITY AND GILL DAMAGE FROM BERYLLIUM IN ACIDIC WATER: A COMPARISON OF ACUTE AND CHRONIC RESPONSES IN THREE FRESHWATER FISH SPECIES

by

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ABSTRACT

Increased concentrations of toxic trace metals such as Aluminum (Al) are found in waters acidified by acid deposition, and elevated Beryllium (Be) levels occur in some acidic waters in eastern Europe. Beryllium and Al are chemically similar, suggesting that their toxic effects may be similar as well. We exposed fry of roach and two species of perch to Be in soft water (calcium [Ca] = 2 mg/L) at two levels of pH (4.5 and 5.5) using static and flow-through bioassays. In acute toxicity tests with *Perca fluviatilis*, 10 µg/L or more Be at pH 5.5 produced gill abnormalities including chloride cell hyperplasia and hypertrophy, increased mucous production, and hyperplasia of the primary lamellar epithelium. At high concentrations, we observed fusion and loss of secondary lamellae, progressing to fusion of adjacent primary lamellae. Less gill damage occurred at pH 4.5, but mortality was much higher at low Be concentrations. Roach were killed only when Be was >50 µg/L, regardless of pH. Roach gills were damaged by 50 µg/L Be or more at both pH 4.5 and 5.5. With chronic exposure, similar abnormalities were caused in *P. flavescens* gills by 6.25 µg/L or more Be regardless of pH. The different responses observed may represent interspecies variation, but were probably influenced by small differences in age among species. Concentrations of Be similar to those reported in some polluted waters produce gill pathologies indicative of ionoregulatory stress. The effects of Be and Al are analogous, but Be is toxic at lower concentrations.

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INTRODUCTION

The concentration of Be in most surface waters is very low. These levels reflect both the relative insolubility of $BeOH_2$ at circumneutral pH (Baes and Mesmer 1976) and the overall scarcity of Be in the earth's crust (Reeves 1986). Although the average Be content of soils is low, some soils and soft coals are enriched in Be (Wilber 1980) in portions of eastern Europe (Vesely et al. 1989).

Acid deposition increases the mobility of many trace metals (Norton 1982), resulting in elevated concentrations of toxic trace metals such as Al in surface waters (Cronan and Schofield 1979). Dickson (1980) showed that cadmium, magnesium, zinc, and lead levels were elevated in some acid waters, and these trace elements were apparently mobilized by acid deposition. Vesely et al. (1989) reported elevated Be concentrations in acidified waters in eastern Europe and suggested that Be was mobilized by acid deposition. Both Be and Al have comparable speciation properties in water (Baes and Mesmer 1976, Vesely et al. 1989). Given the analogous geochemical properties of Be and Al, Be may also be an important toxic agent in some acidic waters.

Few studies have examined toxic effects of Be on fish. Slonim (1973) showed that relatively high concentrations of Be were toxic to guppies *Lebistes reticulatis* at circumneutral pH in hard water (400 mg/L Ca). Slonim and Slonim (1973) found that Be was more toxic to guppies as Ca levels decreased, but even the lowest Ca concentrations they tested were much higher than those present in most acidic waters. In the very dilute waters typical of acidified lakes and streams, Be would be much more toxic than previously suspected. Fish gills are damaged by Al (Karlsson-Norrgren et al. 1986, Jagoe et al. 1987, Evans et al. 1988, Tietge et al. 1988), which also causes ionoregulatory disturbances (Witters 1986, Booth et al. 1988). By analogy, similar effects might result from Be exposure.

MATERIALS AND METHODS

We performed acute toxicity bioassays at the Institute for the Biology of Inland Waters in Borok, Russia, in June 1989 to evaluate the toxicity of Be in low pH water and to determine effects of Be on gill structure (Jagoe et al. 1992). European perch (*P. fluviatilis*) and roach (*Rutilus rutilus*) from the Rybinsk Reservoir were allowed to spawn in artificial ponds, and fry (perch 0.08-0.15 g and roach 0.7-0.9 g) were collected and acclimated to reconstituted soft water for 72 hours. This water simulated the ionic composition of low-alkalinity lakes in the region (Haines et al. 1992), and was prepared by adding salts to distilled water (final concentrations in mg/L: 2 Ca, 1 sodium, 0.25 potassium, 0.25 magnesium, 1 SO_4, and 2.6 chlorine).

Fish were exposed to Be at two levels of pH in 2 L polyethylene aquaria (n = 10 fish/aquarium), two replicates per treatment, at room temperature (20-22°C). Dilute sulfuric acid was added to the reconstituted soft water to obtain a pH of 4.5 or 5.5, and a $BeSO_4$ solution (nominal concentration 1 mg/mL, measured concentration 0.907 mg/mL, SD = 0.12, N = 3) was added to yield final nominal concentrations of 0, 10, 25, 50, 100 or 150 µg/L Be. Controls (pH 7.0) received neither acid nor Be. Acute exposures were performed sequentially, with perch tested first, followed by roach. Dead fry were counted and removed

at 24-hour intervals, and pH recorded and adjusted if necessary. The cumulative number of fish killed by each treatment after 96 hours exposure were compared by analysis of variance. Separate analyses were conducted for each species and pH level. When significantly different levels of mortality were found among the Be treatments, those treatments which differed significantly from the control were identified by Dunnetts' t-test (SAS 1988).

All fish surviving 96 hours were collected and fixed. During the experiments, some moribund fry were removed, scored as dead, and fixed to obtain samples for microscopy from treatments exhibiting high mortality. Whole fry were fixed in 1% glutaraldehyde and 4% formaldehyde in 0.1 m HEPES buffer, pH 7.4. Only fish that were alive when fixed were used for microscopic examination.

To better understand the consequences of chronic exposure to low levels of Be on fish, we exposed juvenile yellow perch (*P. flavescens*) at pH 4.5 and 5.5 for 30 days in May and June 1990 in laboratory experiments at the Midwest Science Center in Columbia, Missouri. Perch (8-10 months old, average weight 1.5 g) were obtained from the National Biological Survey, National Fisheries Research Center in La Crosse, Wisconsin. Exposures were conducted at 20°C in reconstituted soft water using a flow-through proportional diluter system as previously described (Cleveland et al. 1986). Test water contained 2 mg/L Ca, and was very similar in composition to the water used in acute toxicity experiments conducted in Russia in 1989. The water was acidified to pH 4.5 or 5.5 using a mixture of sulfuric and nitric acid, and Be was added (as $BeCl_2$) to nominal concentrations of 0, 6.25, 12.5, 25, or 50 µg/L. Fish were exposed in 77-L aquaria, and test solutions were renewed at the rate of about 8 L/hour. Control treatments (pH 6.9-7.1) received neither acid nor Be. Fish were fed a commercial fish diet ad libitum three times daily. In each treatment, pH was determined daily; oxygen, alkalinity, and conductivity were determined twice each week; and Be was measured weekly. Initially, 40 fish were placed in each treatment, and 5 were removed from each after 5, 15, and 30 days of exposure and fixed for microscopic examination as described above.

Fish from both sets of experiments were prepared for scanning electron microscopy (SEM) using the same protocols. After at least 24 hours of fixation, the samples were treated with 1% OsO_4 for 1 or 2 hours to increase specimen conductivity, dehydrated using a graded ethanol or ethanol-acetone series, then critical-point dried. European perch and roach fry were dried whole, mounted on aluminum specimen stubs, and the opercula removed using fine-tipped forceps to expose the branchial baskets. For yellow perch, gill arches were removed and dried individually. The specimens were spatter-coated with gold or a gold-palladium mixture, and examined by SEM at accelerating voltages of 5 or 15 kV.

RESULTS AND DISCUSSION

ACUTE TOXICITY

No European perch or roach held at pH 7.0 without Be died during the acute toxicity experiments. Exposure to pH 5.5 and Be $\leq$50 µg/L did not kill any fish within 96 hours (Figs. 1a and 2a). Mortality of perch fry exposed for 96 hours to 100 or 150 µg/L Be was significantly higher than mortality of perch fry exposed to lower concentrations at pH 5.5

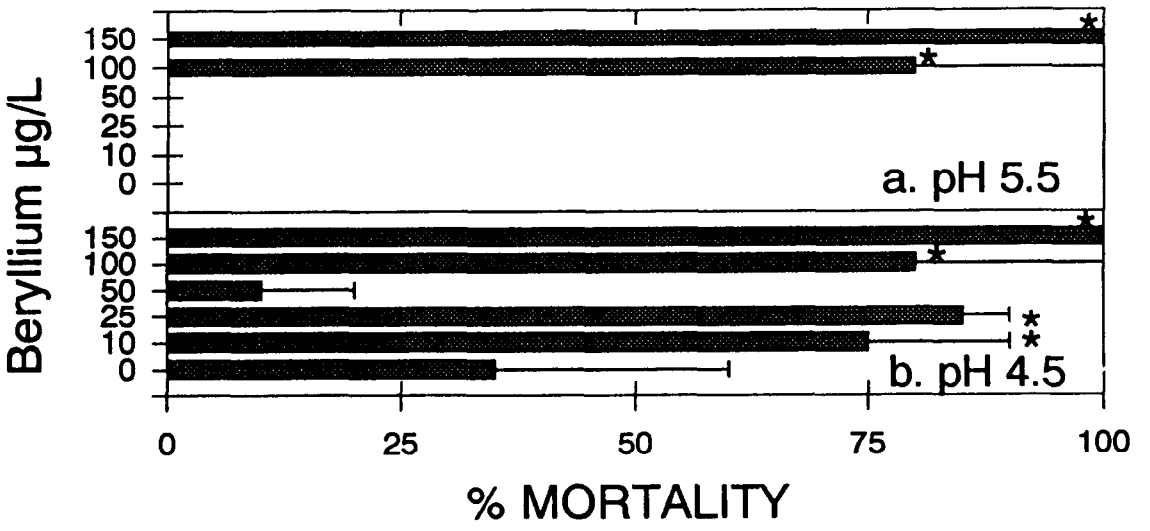

Fig. 1. Mortality of European perch fry exposed to beryllium (Be) at pH 4.5 and 5.5. Values represent the means of two replicates ± standard error. Asterisks indicate significant differences ($p < 0.05$) compared to the control (pH 7.2, 0 µg/L Be) treatment.

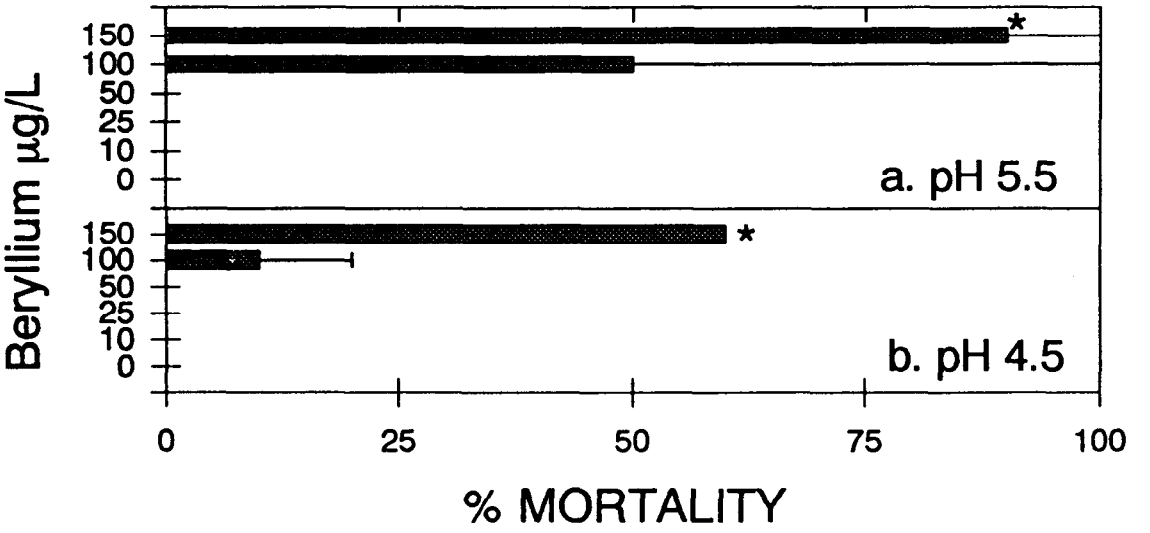

Fig. 2. Mortality of roach fry exposed to beryllium (Be) at pH 4.5 and 5.5. Values represent the means of two replicates ± standard error. Asterisks indicate significant differences ($p < 0.05$) compared to the control (pH 7.2, 0 µg/L Be) treatment.

($p < 0.05$; Fig. 1a). Similarly, roach fry were killed by exposure to 100 and 150 µg/L Be at pH 5.5 (Fig. 2a) for 96 hours. Because of variation among the replicates, at pH 5.5 only mortality at 150 µg/L Be was significantly greater than control mortality in roach ($p < 0.05$).

At pH 4.5, mortality of European perch was lowest in the 0 and 50 µg/L Be treatments (Fig. 1b), which did not differ significantly from each other or the control treatments (pH 7.0, no Be). All other Be concentrations tested at pH 4.5 caused significantly higher mortality of European perch fry after 96 hours ($p < 0.05$). Roach were less sensitive to Be at low pH than the perch. Roach fry were killed only at the highest Be concentrations (100 and 150 µg/L) at pH 4.5, and mortality was significantly greater than control mortality ($p < 0.05$) only at the highest concentration tested.

In European perch, mortality was similar at both levels of pH tested at Be >50 µg/L. Lower concentrations of Be (≤25 µg/L) were more toxic to European perch at pH 4.5 than at pH 5.5. Apparently the pH 4.5 treatment without Be was also stressful to European perch, as over 25% of the fish in this treatment died. The reduction in mortality observed in 50 µg/L Be, pH 4.5, could have resulted from a dilution error; however, this situation is unlikely because reduced toxicity occurred in both replicates and the water in these was prepared independently. A similar response has been observed in Al exposures; survival and body ion content of brook trout (*Salvelinus fontinalis*) fry at low pH were reduced at high and low Al levels, but were enhanced at moderate levels (Wood et al. 1990). Polyvalent Al species present at low pH may stabilize membranes at intermediate concentrations (Baker and Schofield 1982, Wood et al. 1988a) and Be may function in a similar manner.

Dissolved Ca clearly influences Al toxicity at low pH (Brown 1983, Wood et al. 1990), probably by modifying gill epithelial permeability. Previous work on Be toxicity (Slonim 1973, Slonim and Slonim 1973) showed that guppies in hard water (400 mg/L Ca) at circumneutral pH had a 96-hour median tolerance limit for Be of 27 mg/L (Slonim 1973). Toxicity increased as dissolved Ca concentration decreased; the 96-hour median tolerance limit for guppies was about 0.2 mg/L Be when Ca = 22 mg/L. In acidified waters, even less Ca is typically present than used in these previous studies. Our studies yielded 96-hour LC_{50}s of approximately 80 µg/L for European perch fry and 100 µg/L for roach fry at pH 5.5 when Ca = 2 mg/L.

GILL DAMAGE DUE TO ACUTE EXPOSURE

Both juvenile perch and roach held in pH 7.0 water without Be had well-formed gills with no visible abnormalities (Fig. 3a, b). Secondary lamellae appeared regular in size and configuration, and individual cells covered with microridges were observed, with little mucous visible at higher magnification (Fig. 3c). No gross morphological changes were observed at pH 5.5 in the absence of Be in gills of European perch (Fig. 4a) or roach (Fig. 4b). Surface microridges appeared somewhat less abundant, but no swelling or mucous accumulation was evident (Fig. 4c). Exposure to pH 4.5 without Be also had little effect on the gross morphology of European perch gills (Fig. 4d) or roach gills (Fig. 4e), although we observed effects typical of low pH stress, such as greater chloride cell numbers. Swelling of mucous cells indicative of increased mucous production, and many chloride cells with apical crypts were visible at higher magnification (Fig. 4f).

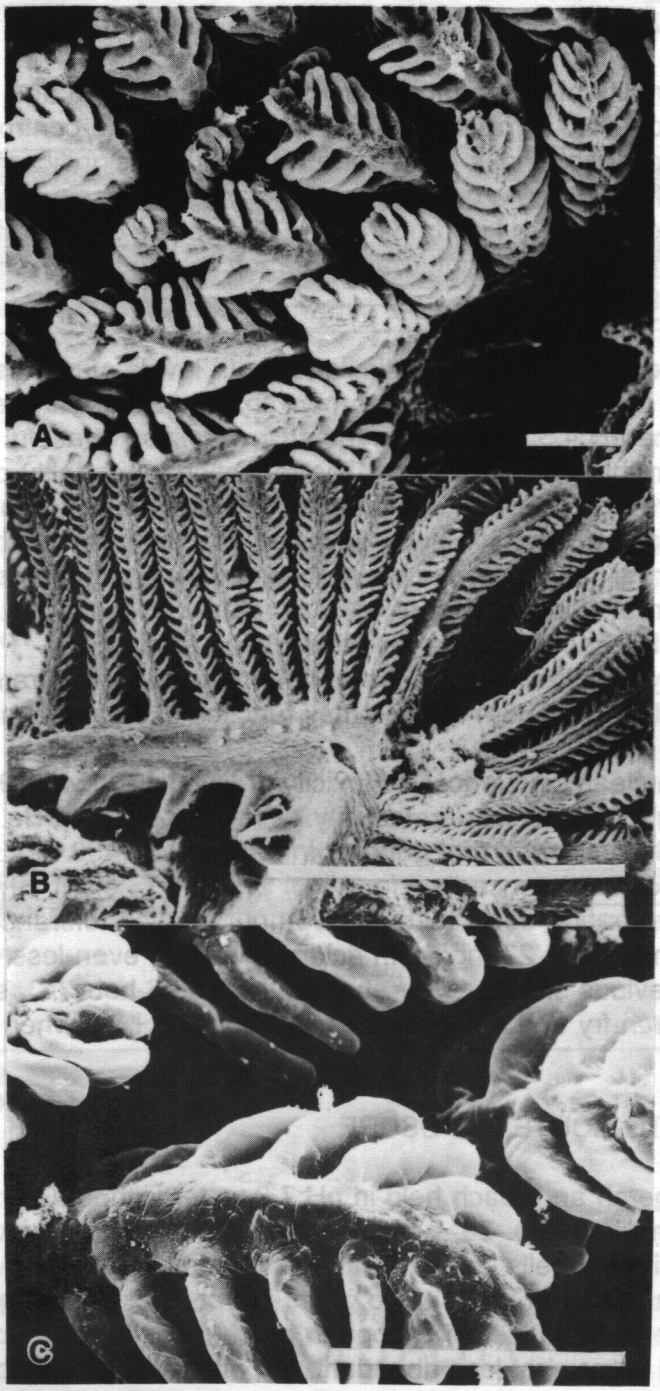

Fig. 3. Scanning electron micrographs of gills at pH 7.0 with no beryllium: a) European perch, scale bar = 100 μm; b) roach, scale bar = 1000 μm; and c) European perch, scale bar = 100 μm.

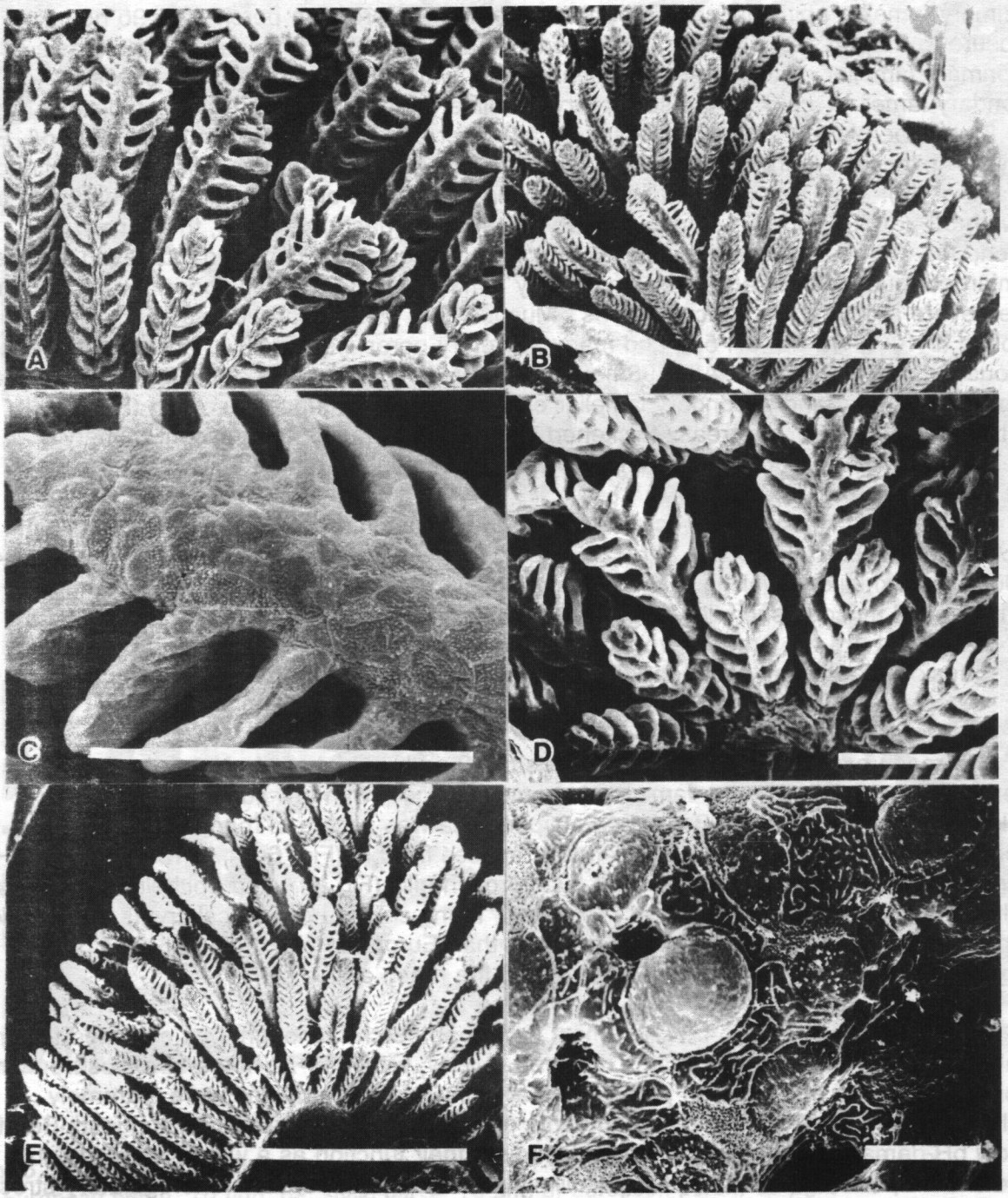

Fig. 4. Scanning electron micrographs of gills of fry exposed to low pH (5.5) without beryllium (Be): a) European perch, scale bar = 100 μm; b) roach, scale bar = 1000 μm; c) European perch, scale bar = 100 μm; d) European perch, scale bar = 100 μm; e) roach, scale bar = 1000 μm; and f) European perch, scale bar = 10 μm.

In European perch, exposure to as little as 10 µg/L Be at pH 5.5 caused changes in gill morphology. Chloride cell apical crypts were present in perch exposed to 10 µg/L Be (Fig. 5a), but not in perch exposed to pH 5.5 without Be. We also found some swelling of epithelial cells and a reduction in microridges on cell surfaces. Microridge loss and swelling of primary lamellar epithelial cells were more marked in European perch after 96 hours exposure to 25 µg/L Be, pH 5.5. Exposure to 50 µg/L Be at pH 5.5 caused hyperplasia of the primary lamellar epithelium in European perch, which filled in the spaces between secondary lamellae, causing the secondary lamellae to appear shorter and less distinct (Fig. 5b). This hyperplasia became more severe at higher Be concentrations; above 100 µg/L Be, massive epithelial hyperplasia fused adjacent primary lamellae in perch gills into a continuous mass (Fig. 5c). Low Be concentrations produced few effects on roach gills at pH 5.5. At pH 5.5 with 50 µg/L Be, primary and secondary lamellae of roach thickened, the density of microridges on the surfaces of epithelial cells was markedly reduced, and increased numbers of enlarged mucous cells appeared on primary lamellae (Fig. 5d). Gill abnormalities became more severe at higher Be concentrations. Mucous cells were enlarged in gills of roach exposed to 100 µg/L Be, pH 5.5, and many secretory pores were visible, indicating increased mucous secretion (Fig. 5e). After 96 hours exposure to 150 µg/L Be at pH 5.5, hyperplasia of both the primary and secondary lamellar epithelia was pronounced in roach (Fig. 5f), resulting in gills appearing greatly thickened and clubbed. Fusion of adjacent primary lamellae, as found in European perch fry, was not observed in roach.

At pH 4.5, high mortality occurred in European perch, but the effects of Be on gill structure were less striking than at pH 5.5. European perch exposed to 50 µg/L Be at pH 4.5 for 96 hours exhibited swelling of the primary lamellar epithelia, some loss of microridges, and increased mucous secretion (Fig. 6a). After exposure to Be at concentrations of 100 µg/L or more at pH 4.5, areas devoid of microridges were visible (Fig. 6b) and hyperplasia caused fusion of adjacent secondary lamellae in a number of regions (Fig. 6c). In this species, the hyperplasia at pH 4.5 was much less severe than at pH 5.5.

In roach exposed to Be concentrations of 50 µg/L or higher at pH 4.5, epithelial hyperplasia occurred. Primary lamellae became enlarged, with few surface microridges visible (Fig. 6d). At 100 µg/L Be, pH 4.5, hyperplasia was severe in roach gills; many secondary lamellae fused as the interlamellar spaces filled in (Fig. 6e). At 150 µg/L Be, pH 4.5, numerous secondary lamellae were lost in roach gills (Fig. 6f), and epithelial hyperplasia resulted in thickened, club-like primary lamellae. Many chloride cell apical crypts and swollen mucous cells were seen. Although fewer roach than European perch died at pH 4.5, gill abnormalities we observed were more severe in the roach at this pH.

Conversely, Be at pH 4.5 killed most European perch, although they exhibited fewer gill abnormalities. Most of the deaths occurred quickly, within 48-72 hours of exposure. The secondary lamellae of European perch dying at high Be concentrations were extremely swollen and irregular, with increased amounts of mucous. This observation suggests Be at the low pH damaged epithelial cells and destroyed their function as a water- and ion-tight barrier. Disruption of this barrier function likely produced rapid, severe ionoregulatory stress that killed the fry before compensatory mechanisms could occur, such as epithelial hyperplasia to increase both chloride cell numbers and the thickness of the diffusion pathway.

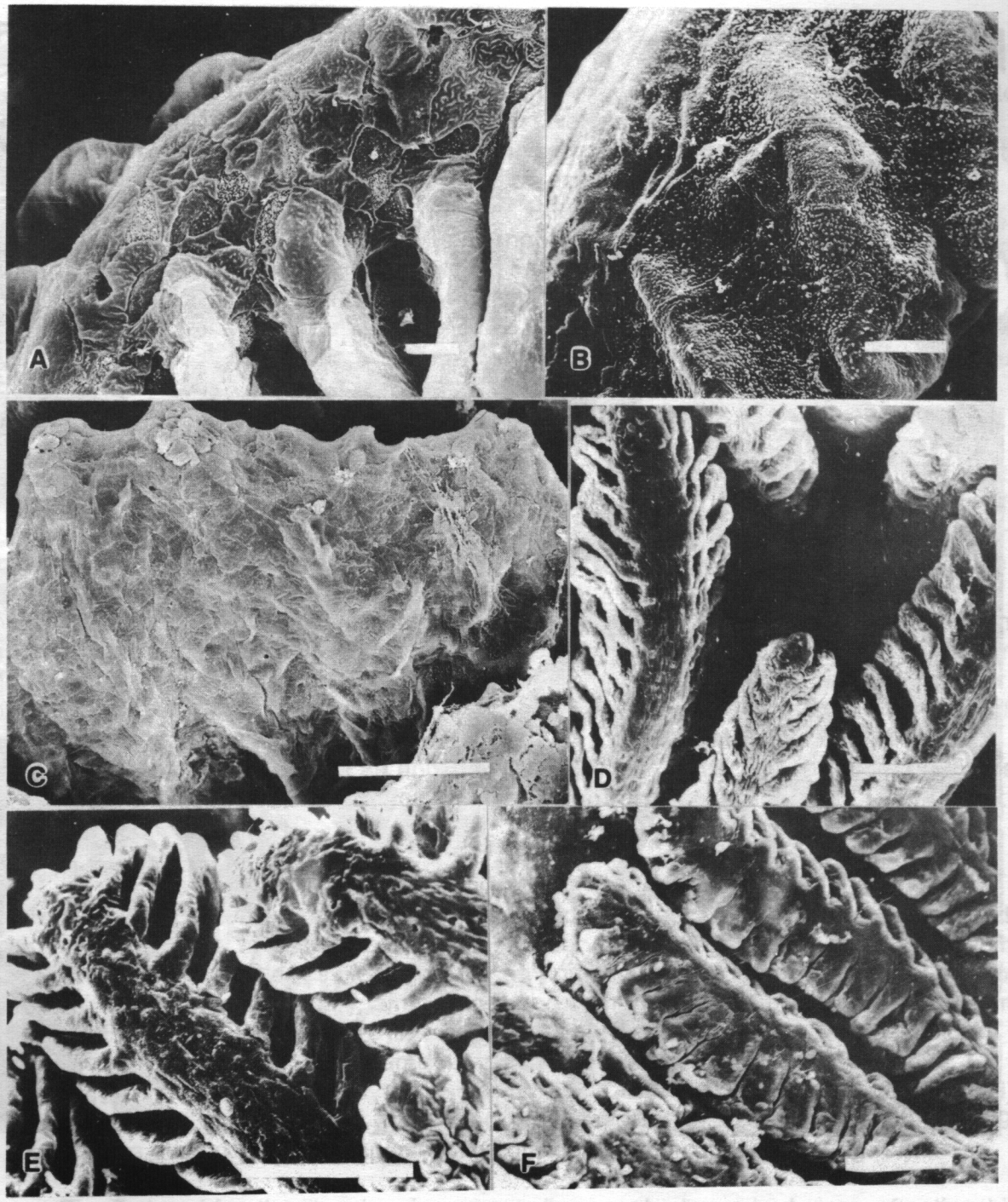

Fig. 5. Scanning electron micrographs of gills of fry exposed to beryllium (BE) at pH 5.5: a) European perch, 10 μg/L Be, scale bar = 10 μm; b) European perch, 50 μg/L Be, scale bar = 10 μm; c) European perch, 100 μg/L Be, scale bar = 100 μm; d) roach, 50 μg/L Be, scale bar = 100 μm; e) roach, 100 μg/L Be, scale bar = 100 μm; and f) roach, 150 μg/L Be, scale bar = 100 μm.

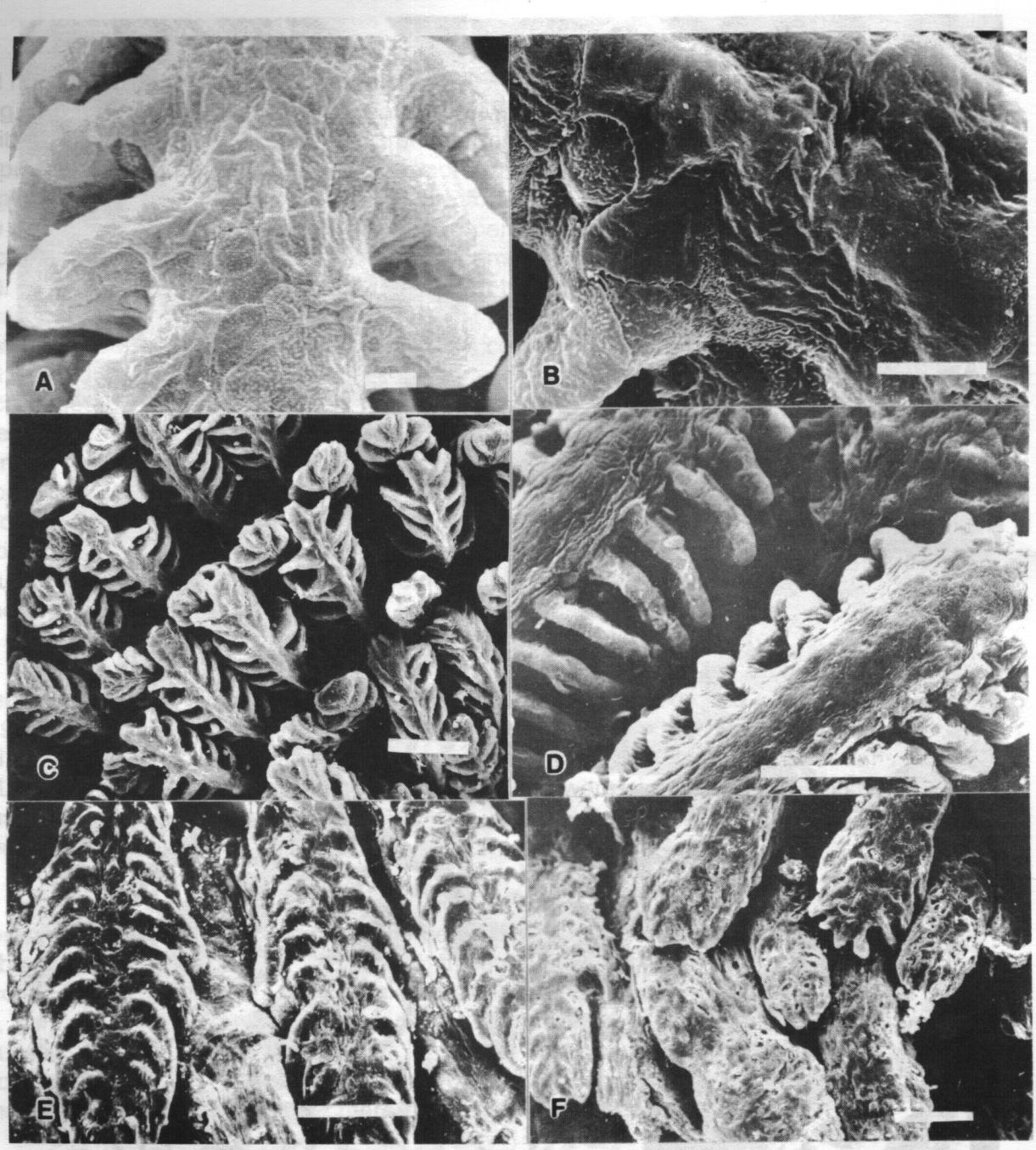

Fig. 6. Scanning electron micrographs of gills of fry exposed to beryllium (Be) at pH 4.5: a) European perch, 50 μg/L Be, scale bar = 10 μm; b) European perch, 100 μg/L Be, scale bar = 10 μm; c) European perch, 150 μg/L Be, scale bar = 100 μm; d) roach, 50 μg/L Be, scale bar = 100 μm; e) roach, 100 μg/L Be, scale bar = 100 μm; and f) roach, 150 μg/L Be, scale bar = 100 μm.

EFFECTS OF CHRONIC EXPOSURE

Survival of yellow perch was not affected by exposure to Be at concentrations up to 50 µg/L at either pH level tested. During the first week of exposure, hyperactivity and abnormal schooling behavior were observed in yellow perch exposed to 50 µg/L Be at both pH 4.5 and 5.5, but these effects disappeared as exposure continued.

The gross morphology of gills was not noticeably different among yellow perch exposed for 30 days to pH 7.0 (Fig. 7a), pH 5.5 (Fig. 7b), or pH 4.5 (Fig. 7c) without Be. Both primary and secondary lamellae were distinct and normal in appearance. Mucous droplets were more abundant after exposure to the lower levels of pH. Numerous chloride cells were present on primary lamellae at all levels of pH, probably resulting from prolonged exposure to relatively soft water. Laurent et al. (1985) and Leino et al. (1987) have also reported chloride cell proliferation in fish held in very soft water.

In contrast, 30 days exposure to Be produced gill abnormalities, even at the lowest concentration tested. Compared to controls (pH 7.0, no Be; Fig. 8a), the secondary lamellae of yellow perch exposed to 6.25 µg/L Be at pH 5.5 (Fig. 8b) were swollen and wrinkled, and increased numbers of chloride cells were present in the secondary lamellar epithelia. The gills of yellow perch exposed to 6.25 µg/L Be at pH 4.5 were coated with mucous that obscured much surface detail, and epithelial hyperplasia had reduced the spaces between adjacent secondary lamellae, making them shorter and less distinct (Fig. 8c). This shortening and loss of secondary lamellae also occurred at 6.25 µg/L Be at pH 5.5, and was most noticeable near the tips of the primary lamellae (Fig. 8d). At both levels of pH, 6.25 µg/L Be caused loss of cell surface microridges (Fig. 8e) and a noticeable increase in chloride cell surface area. We also observed an unusually high number of mucous cell secretory pores, along with bulging, convex chloride cell apices in yellow perch exposed to 6.25 µg/L Be at pH 4.5 (Fig. 8f).

As in acute exposure experiments, gill abnormalities in yellow perch became more severe at higher Be concentrations. Thirty days exposure to 25 µg/L Be at pH 5.5 caused more severe epithelial hyperplasia, causing secondary lamellae to thicken and grow together (Fig. 9a). This hyperplasia resulted in increased numbers of chloride cells as well (Fig. 9b). Hyperplasia leading to secondary lamellar loss and primary lamellar thickening also occurred at 25 µg/L Be at pH 4.5 (Fig. 9c). Abnormalities were not obviously more severe at the highest Be level tested (50 µg/L) than at 25 µg/L Be. Secondary lamellae were also shortened, thickened, and fused by exposure to 50 µg/L Be for 30 days at pH 5.5 (Fig. 9d). Exposure to the 50 µg/L Be at pH 4.5 caused similar effects on secondary lamellae (Fig. 9e, f), and surface microridges were sparse with considerable mucous visible (Fig. 9f).

The first and fourth gill arches in young yellow perch always had a row of noticeably shortened primary lamellae. In fish exposed to pH 7.0 with no Be, the morphology of these short primary lamellae was very similar to the adjacent longer ones (Fig. 10a). Exposure to pH 4.5 without Be for 6 days did not affect the morphology of these shortened lamellae (Fig. 10b), but they were severely damaged by the presence of Be (Fig. 10c). To our knowledge, this report is the first of the presence of these short filaments in the species. Perhaps these primary lamellae are rapidly growing and developing in young yellow perch, as their size and structure is similar to that reported in other young fish (Morgan 1974, El-Fiky et al. 1987),

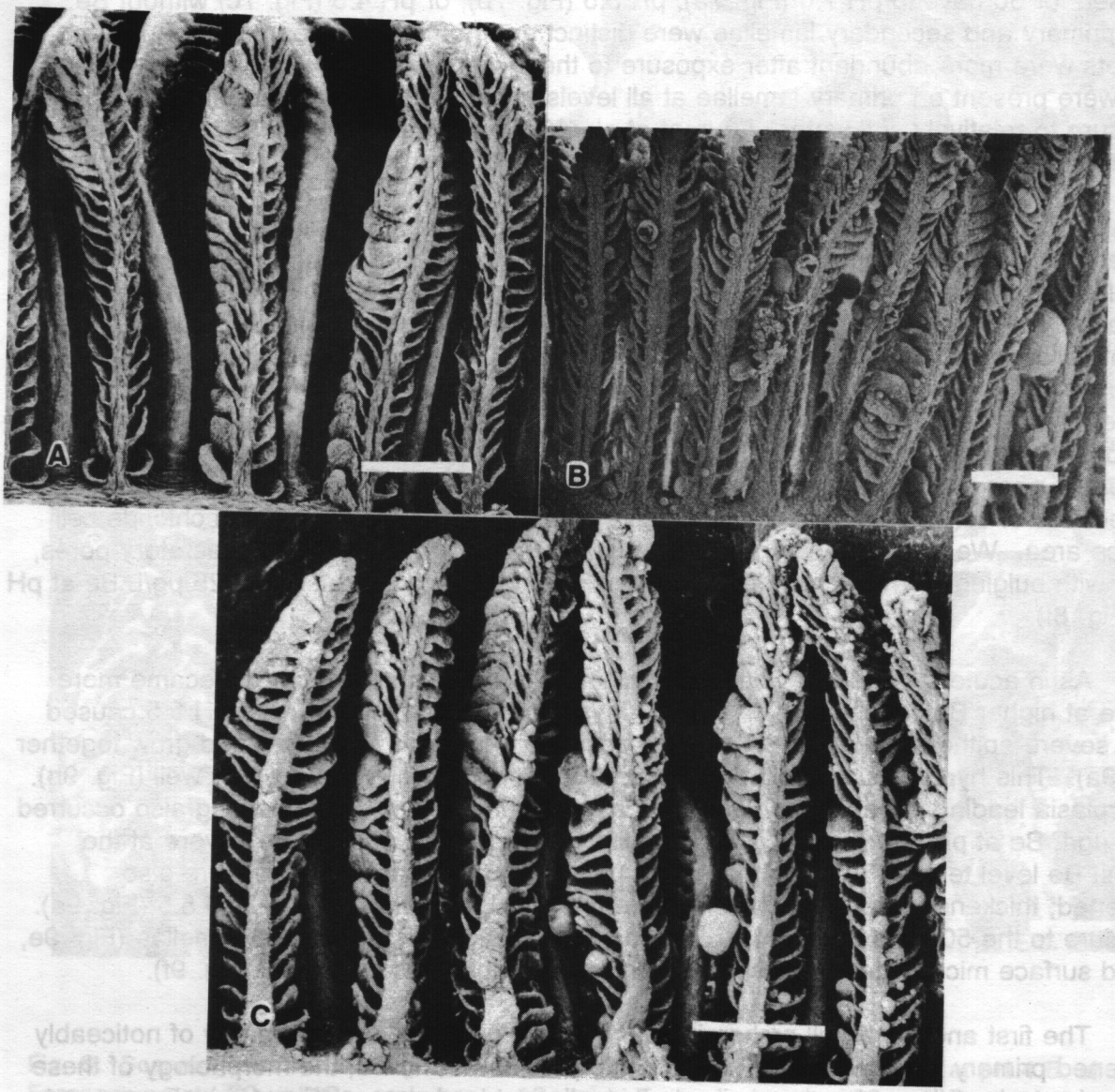

Fig. 7. Scanning electron micrographs of gills of yellow perch fry held at various levels of pH without beryllium (Be); scale bars = 100 μm: a) pH 7.0, b) pH 5.5, and c) pH 4.5.

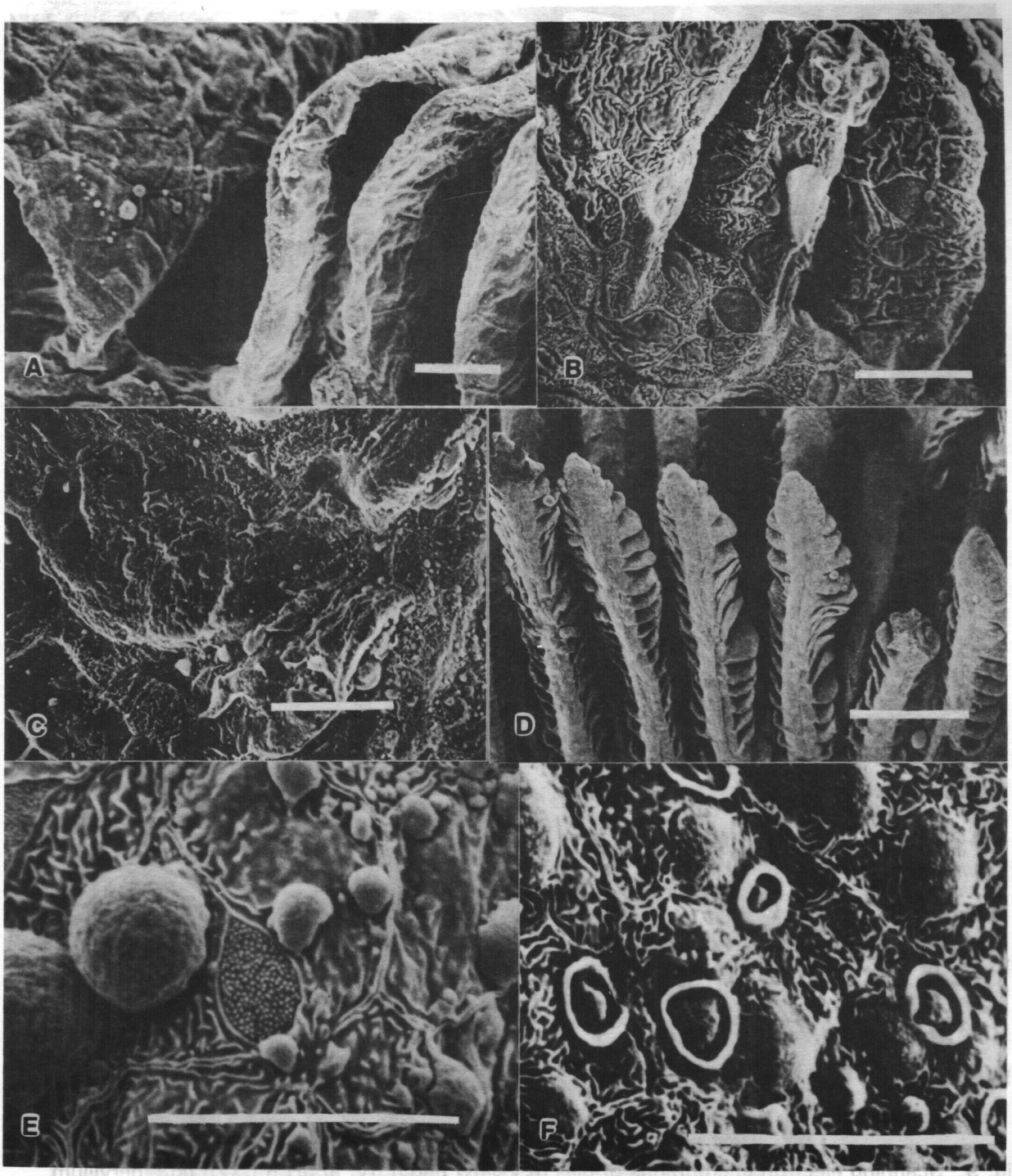

Fig. 8. Scanning electron micrographs of gills of yellow perch fry: a) pH 7.0, no beryllium (Be), scale bar = 10 μm; b) pH 5.5, 6.25 μg/L Be, scale bar = 10 μm; c) pH 4.5, 6.25 μg/L Be, scale bar = 10 μm; d) pH 5.5, 6.25 μg/L Be; scale bar = 100 μm; e) pH 5.5, 6.25 μg/L Be, scale bar = 10 μm; and f) pH 4.5, 6.25 μg/L Be, scale bar = 10 μm.

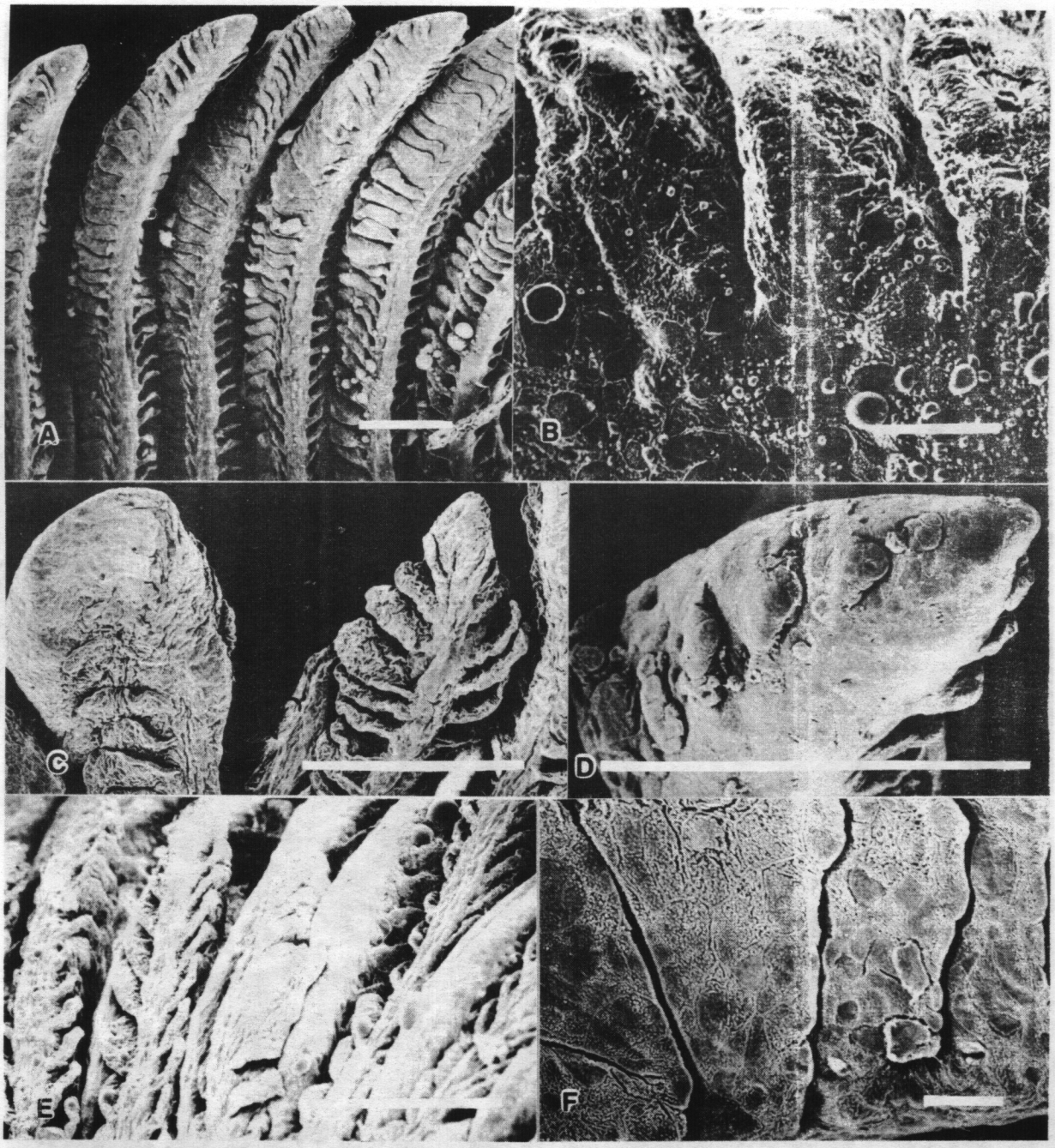

Fig. 9. Scanning electron micrographs of gills of yellow perch fry: a) pH 5.5, 25 μg/L beryllium (Be), scale bar = 100 μm; b) pH 5.5, 25 μg/L Be, scale bar = 10 μm; c) pH 4.5, 25 μg/L Be, scale bar = 100 μm; d) pH 5.5, 50 μg/L Be, scale bar = 100 μm; e) pH 4.5, 50 μg/L Be, scale bar = 100 μm; and f) pH 4.5, 50 μg/L Be, scale bar = 10 μm.

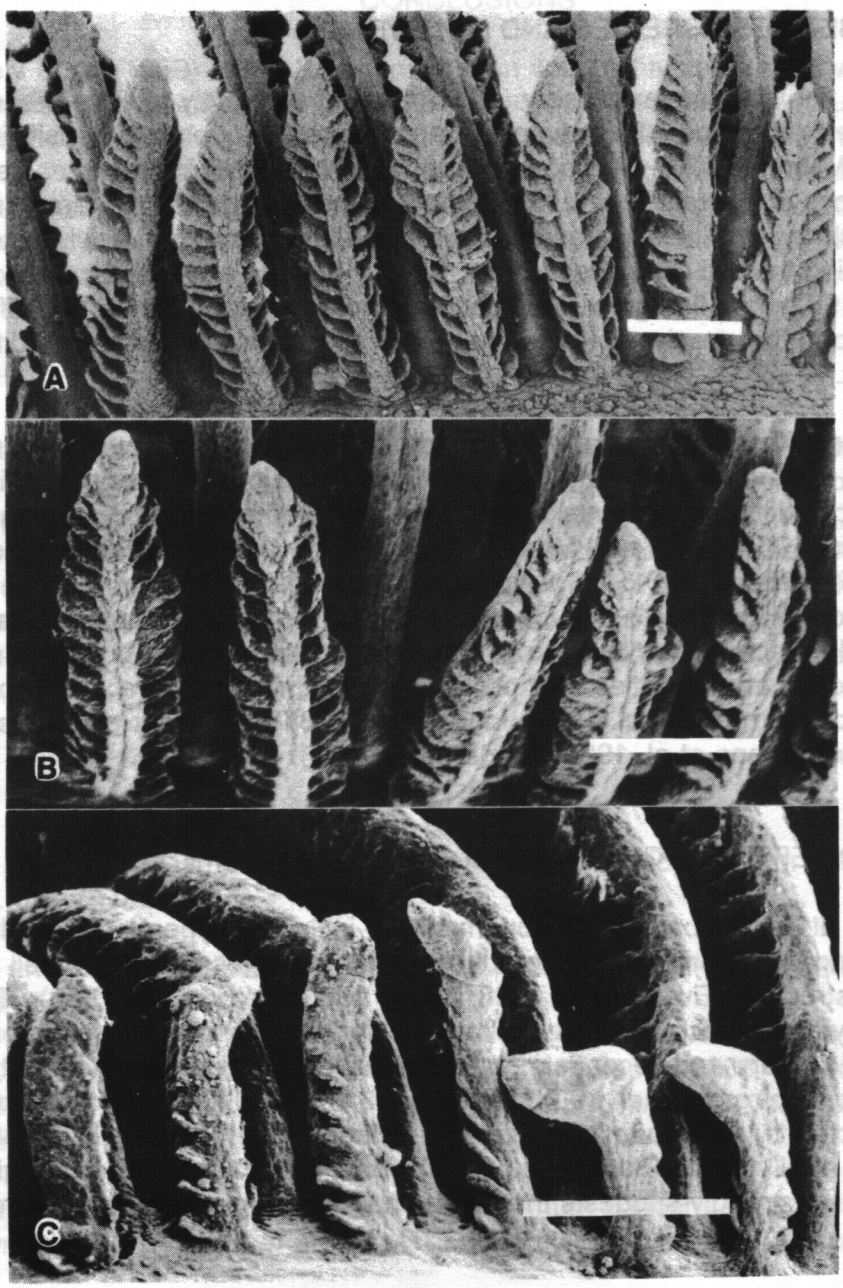

Fig. 10. Scanning electron micrographs of the gills of yellow perch fry showing the shortened row of primary lamellae on the first and fourth gill arches; scale bars = 100 μm: a) pH 7.0, no beryllium (Be), 30 days exposure; b) pH 4.5, no Be, 6 days exposure; and c) pH 4.5, 25 μg/L Be, 6 days exposure.

although these authors do not mention large differences in size among adjacent rows of lamellae. If this situation is true, the rapidly growing structures may be especially susceptible to pollutant damage, as we observed for Be, and may also be a very sensitive indicator of pollutant-induced stress.

COMPARISON BETWEEN SPECIES AND pH LEVELS

The three fish species tested seemed to respond differently to Be. These results may reflect interspecies differences; for example, roach differs from perch in sensitivity to low pH (Johansson and Milbrink 1976). A more thorough comparison of interspecies sensitivity must consider differences in size and age of fish tested. European perch fry were somewhat smaller than roach fry, and both were younger than yellow perch fry. Newly hatched fish and those just beginning to feed are especially sensitive to low pH and metal-induced stress (Peterson et al. 1982). As fish grow, they become more resistant. Thus, the difference in size and age among the species tested makes evaluation of true interspecies differences in sensitivity difficult.

Both Be and Al speciation are pH dependent. Calculation of equilibrium species distributions from thermodynamic data indicated that the major species present in both the acute and chronic toxicity experiments were monomeric Be^{2+} and $BeOH^{+}$, depending on pH (Garrels and Christ 1965, Smith and Martell 1976). Be^{2+} accounts for 88% of the total Be present at pH 4.5, while at pH 5.5, 56% would be in the form $BeOH^{+}$. In European perch, the most striking abnormalities in gills occurred at pH 5.5, when most Be present was in the hydroxide form. In roach and yellow perch, abnormalities occurred at both levels of pH. Aluminum-induced gill abnormalities also occur both at pH levels where AlOH species dominate (Karlsson-Norrgren et al. 1986, Jagoe et al. 1987) and lower pH levels where Al^{3+} becomes important (Evans et al. 1988, Tietge et al. 1988).

COMPARISON OF EFFECTS OF BE AND AL

Aluminum at low pH causes gill abnormalities, including hyperplasia resulting in epithelial thickening, increased mucous production, and changes in chloride cell and mucous cell number and structure (Evans et al. 1988, Jagoe 1988, Tietge et al. 1988). Epithelial hyperplasia, partly due to increased production of chloride cells, can cause fusion and disappearance of primary and secondary lamellae: this greatly reduces the functional surface of the gill (Karlsson-Norrgren et al. 1986, Jagoe et al. 1987). Gill physiological processes known to be affected by Al include ion regulation (Witters 1986, Booth et al. 1988) and gas exchange (Wood et al. 1988b, Playle et al. 1989). Beryllium causes the same gill abnormalities, which strongly suggests that the same physiological processes are affected by the two metals.

The altered chloride cell morphology we observed suggests that Be interferes with ion regulation, because chloride cells are responsible for ion uptake in freshwater fish (Laurent et al. 1985). Also, exposure to Be causes apical crypts to develop in chloride cells. Chloride cell apical crypts normally occur only in seawater-adapted fish (Foskett and Schelley 1982), but also occur in freshwater fish experiencing ionoregulatory disturbances caused by low pH

(Leino and McCormick 1984) and Al (Jagoe et al. 1987). Increased chloride cell production may represent a compensatory response to offset ionic losses if initial exposure to the Be is not acutely lethal. However, such hyperplasia involves a trade-off; in exchange for increased ionic uptake sites, the functional surface area for gas exchange is greatly decreased.

CONCLUSIONS

Vesely et al. (1989) found Be concentration was highly correlated with decreased pH in acidified waters in Czechoslovakia. In the waters they sampled, Be >5 µg/L was found at 27 sites, at concentrations as high as 58 µg/L. This study demonstrates that these Be concentrations would be harmful to fishes, and likely cause gill abnormalities or damage. Our results also suggest that the effects of Be and Al are analogous. In some areas where Be is abundant in soils or because of extensive use of lignite coal usage (Wilber 1980), Be may be an important toxicant whose behavior and effects are still poorly understood.

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