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Kathleen Muriel Worthylake
Aspen Antibodies, Inc., McCammon, Idaho

Peter Hovingh
University of Utah

MASS MORTALITY OF SALAMANDERS (*AMBYSTOMA TIGRINUM*) BY BACTERIA (*ACINETOBACTER*) IN AN OLIGOTROPHIC SEEPAGE MOUNTAIN LAKE

Kathleen Muriel Worthylake[1] and Peter Hovingh[2]

ABSTRACT.—Of the 13 lakes in the central Wasatch Mountains with tiger salamander (*Ambystoma tigrinum*) populations, salamander mass mortalities occurred in four seepage lakes that experienced extensive lowering of the water levels during the summer. The largest of these lakes, the oligotrophic Desolation Lake, was studied to determine the cause of the mortality phenomenon. The recurrent annual mass mortality involved both breeding adults and young-of-the-year. Rate kinetics suggest that mortality rate doubles with a fivefold increase in the number of aquatic young salamanders. The proximate cause of the mass mortality was identified as a bacterium, *Acinetobacter* sp. Desolation Lake and a seepage lake that did not experience the mass mortality were studied for the presence of *Acinetobacter*. Both lakes experienced two bacteria cycles: the first in early summer involved gram-positive bacteria, and the second in late summer involved gram-negative bacteria (mostly coliform bacteria and *Acinetobacter*). The mass mortalities were associated with the late-summer gram-negative bacterial bloom, and *Acinetobacter* was found in large numbers in Desolation Lake. Algae, as well as other photosynthetic plants, were not present in large numbers. Since these lakes are growth-limited with respect to nitrogen and not with respect to phosphate, and since Desolation Lake had extensive lowering of the lake level during the summer, the biological cycle of the lake is proposed to be due to atmospheric and sheep-produced nitrogen products within the watershed. In this unstable lake system, bacteria are primarily responsible for incorporation of nitrogen in the food chain. The timing of the cyclical events depends on total winter precipitation.

The biology of the tiger salamander (*Ambystoma tigrinum*) has been extensively described (Anderson et al. 1971, Collins 1981, Hassinger et al. 1970, Rose and Armentrout 1976, Semlitsch 1983, Sexton and Bizer 1978, and Tanner et al. 1971), but only one population has been described with pathological symptoms (Rose and Harshbarger 1977). This may not be unexpected in view of the fact that natural amphibian populations may not be prone to mass pathological events as shown by the scarcity of literature reports (Reichenbach-Klinke and Elkan 1965). Recently, a report of mass mortality of larval *Rana sylvatica* was attributed to the bacterium *Aeromonas* (Nyman 1986).

This paper describes the recurrent mass mortality of the tiger salamander in an oligotrophic seepage lake in the Wasatch Mountains, Utah. We found that the proximate cause of the deaths was due to the bacterium *Acinetobacter*. Because the mass mortality was recurrent, we were able to study the kinetics of the die-off as well as the presence of *Acinetobacter* in the lake. Furthermore, we suggest that the die-off of the tiger salamander in the seepage lake is in part attributed to the extensive lowering of the lake levels as well as the concentration of nitrogen in the snow and from the watershed. The combined information describes some of the biological features of seepage lakes in the Wasatch Mountains.

METHODS

Water sampling in the hypolimnion utilized the Kemmerer water sampling bottle. Water samples from the surface waters were taken from two points in the lake and combined. The samples were collected in the afternoon, placed on ice in the dark two hours after the collection time, and delivered the subsequent morning for bacterial, algal, or chemical analysis. Snow sampling occurred in early April from three points around Desolation Lake. The samples were analyzed for nitrites and nitrates (test kits from Aquarium Pharmaceuticals, Inc., Perkasie, Pennsylvania) and sulfates (Terho and Hartiala 1971) after the addition of 10 mg sodium carbonate (to prevent volatilization of the nitrites and nitrates), and concentrated to about 25 ml. Climatic data came from U.S. Climatological Data (1980–1986) taken at Silver Lake, Brighton (lat. 40° 36′ North, long. 111° 35′ West), at 2,666 m elevation in Big Cottonwood Canyon, Wasatch Mountains.

[1]Aspen Antibodies, Inc., 9866 South Marsh Creek Road, McCammon, Idaho 83250.
[2]Division of Dermatology, College of Medicine, University of Utah, Salt Lake City, Utah 84132.

The monitoring of dead adult salamanders involved the inspection of the lake, the shoreline, and the area beneath logs on the shoreline. The numbers of dead adults are thus minimal figures since some adults may have died undetected in the lake and under logs in the adjacent forest. The aquatic larval salamanders were counted as they were stranded on the shore and in the adjacent waters. Most of the salamanders could be counted in this manner, and very few were observed in the lake bottoms. The exception to this occurred in 1986 when many of the aquatic young salamanders sank to the bottom and could not be inventoried. The inventory involved the relocation of all dead salamanders such that they would not be counted twice.

Histological analysis was performed by Dr. J. Harshbarger at the Registry of Tumors in Lower Animals, Smithsonian Institution, Washington, D.C. Algal analysis involved both the determination of chlorophyll *a* (corrected for the presence of degradation products) by M. A. Nielsen, Ecosystem Research Institute, Logan, Utah, and algal counts and identification by Dr. R. I. Lynn, Department of Biology, Utah State University, Logan, Utah. Chemical analysis was performed by Utah State University Water Research Laboratory, Logan, Utah.

Bacteria were enumerated as viable counts by the plate count technique using 5% sheep blood in tryptic soy agar. Identification of bacteria was by standard biochemical methods (Lennette et al. 1985). The plate count method was selected as we were examining the cause of the salamander deaths. We then extended the observations to the lake with the same plate count technique to determine the presence of these bacteria in the lake. Although viable counts have limitations as to "seeing" fast-growing colonies at the expense of slow-growing colonies, two circumstances justify the use of viable counts in this research: (1) specific groups (gram-negative versus gram-positive and the determination of the pathogens *Acinetobacter*, *Pseudomonas*, and coliform bacteria) were counted, and (2) a perturbation in the lake (mass mortality of the salamanders) was being monitored. Thus, two of the three acceptable circumstances for using viable count methodology were met (Fry 1982).

To demonstrate that *Acinetobacter* was the cause of the salamander mortality, we placed two salamanders (obtained from lakes that did not experience the mass mortality) in 8 liters of water. The water was inoculated with approximately 300 million colony-forming, live bacteria obtained from diseased salamanders or from Desolation Lake in the case of *Escherichia coli*. After the disease developed, the bacteria were identified from the salamander. Koch's postulates for the cause of a disease from terrestrial animals require that bacteria be taken from a lesion of the diseased animal and that after inoculation of a healthy animal this same bacteria be isolated from the new lesions. Aquatic animals present a variation on this procedure since the lesion is in water and water may contain other pathogens and bacteria. Thus, the bacteria were isolated from the peritoneum of the salamander and inoculated in the water that contained the salamanders.

DESCRIPTION OF THE LAKES AND SALAMANDER BIOLOGY IN THE CENTRAL WASATCH MOUNTAINS

Desolation Lake is the largest of the seepage lakes under study in the Wasatch Mountains. Desolation Lake has no surface inflows and outflows (definition of seepage lake, Pennak 1968), receives all its water from snowmelt, has extensive shorelines of soil, and has a paucity of contiguous rock abutments. It occurs in Big Cottonwood drainage at 2,820 m elevation and covers approximately 0.042-km^2 surface area in a 0.7-km^2 glaciated bowl.

During the atypically wet years of 1983 and 1984 the 10-m-deep lake dropped 4 m during the summer (Fig. 1). During a more average year (1985) it dropped 2 m while starting at a lower level in July. The lake dropped at a rate of 5 cm per day in July of the wet years until it reached the more typical level, at which time the lake continued to drop 2 cm per day for the remainder of summer. In early summer thermostratification occurs at the 6-m-deep level; this thermostratification disappears as the lake level drops. For two months the surface temperature of the lake is above 15 C (Fig. 1).

The total dissolved solids (46 mg/ml) consisted mostly of calcium bicarbonate (40.3 mg/l) in the surface waters in 1983. The pH varied from 6.9 to 7.7, and the dissolved

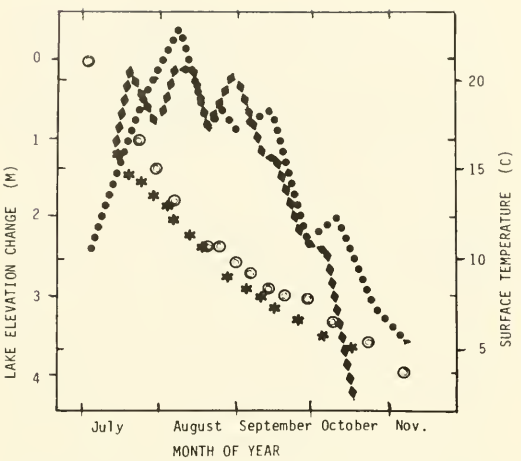

Fig. 1. Lake levels and surface temperature values for Desolation Lake for two wet years. Solid circles (1983) and diamonds (1984) depict the surface temperatures. Open circles (1983) and stars (1984) depict the lake level drop.

oxygen measured 6.6 mg/l. Nitrogen (greater than 60 ug N/l) was growth-limiting with respect to phosphorus (8–12 ug P/l) in the epilimnion. The hypolimnion contained more balanced amounts of phosphates and nitrogen and tended to have an increase in ammonia, carbon dioxide, phosphate, and many of the cations (zinc, iron, mercury, barium, and hexavalent chromium) and a decrease in oxygen (2.4 mg/l).

Desolation Pond is in the same basin as Desolation Lake and hence receives some of the snow from the same watershed. This pond is small, with a diameter of 20 m, a surface of less than 0.001 km^2, and during the wet year of 1984 a depth of 3 m. This pond dries in most years. Magnesium and calcium are the major cations and bicarbonate the major anion. Biological growth is limited with respect to nitrogen but not with respect to phosphorus.

Snow analysis in the Desolation Lake area indicated that this drainage basin received 52 kg sulfate, 4 kg nitrite N, and 41 kg nitrate N. The nitrogen value from atmospheric sources is equivalent to the nitrogen contribution of 100 sheep per 10 days in the basin (Darling 1973). This number of sheep in the basin is usually exceeded during most summers. Thus, both sheep and atmospheric sources of nitrogen can contribute significantly to the biological growth in Desolation Lake.

Thirteen of the 41 lakes examined in the central Wasatch mountains contained salamanders. Dog Lake was used in this study as an example of a seepage lake that did not experience a die-off. It is a seepage lake in Big Cottonwood Canyon with a surface area of approximately 0.007 km^2 within a drainage basin of 0.14 km^2. The lake is at 2,660 m elevation, has a depth of 2.4 m, and lowers in elevation during the summer approximately 0.6 m. Dog Lake has an abundance of aquatic insects such as Gerridae and Odonata and the leech *Batracobdella picta* (identified by Dr. D. J. Klemm). The abundance of macroinvertebrates in Dog Lake sharply contrasts with the paucity of macroinvertebrates in Desolation Lake.

Other seepage lakes that were observed included Red Pine Lake (elevation 2,684 m) and Shadow Lake (elevation 2,715 m) in the Weber River drainage and Lower Lake Solitude (elevation 2,684 m) in the Big Cottonwood drainage. Both Shadow Lake and Lower Lake Solitude were formed behind man-made dams and dried up in late summer. They also experienced salamander die-off, but since these lakes were very small, the die-off, although it may have been all-inclusive, did not involve large numbers. Only Lower Lake Solitude drainage was prohibited to the grazing of sheep. The drainage lakes did not experience any mass mortality.

Salamander populations in the high mountain lakes of Utah follow the life-history pattern described as "two size classes of larvae with metamorphosis occurring in the second warm season" (Sexton and Bizer 1978). Eggs are laid in these mountain lakes during July, although some lakes are utilized by breeding salamanders in August. Larval growth rates of 1.2 mm per day allow the young to reach the pretransformation snout-vent length of 55–70 mm by September (Fig. 2). Should the larvae reach a snout-vent length of about 40 mm by early August, they may metamorphose at that time, as indicated by Tanner et al. (1971). The aquatic young feed predominantly on crustaceans, including *Branchinecta paludosa*, *Gammarus lacustris*, and cladocerans. Transformed adult salamanders in the Desolation Lake region can arrive from populations derived from the drainage lakes of Willow Lake (2.4 km distant, ridgeline pass of 2,928 m, Big Cottonwood Creek drainage), Dog

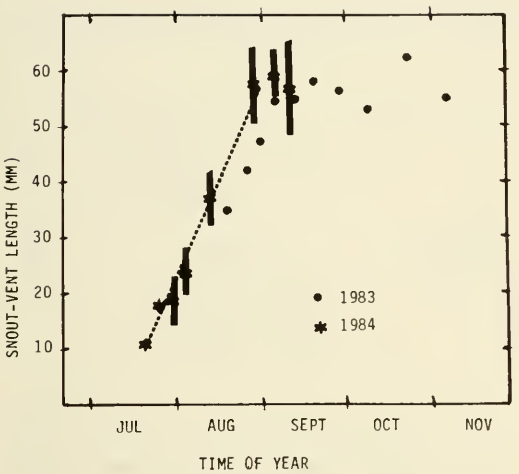

Fig. 2. Growth rates of aquatic larval salamanders in Desolation Lake. Stars represent average snout-vent length, with the bars representing one standard deviation for 1984. Solid circles represent average snout-vent length in 1983. The line represents a growth rate of 1.2 mm per day as determined by linear regression analysis from the data of 1984.

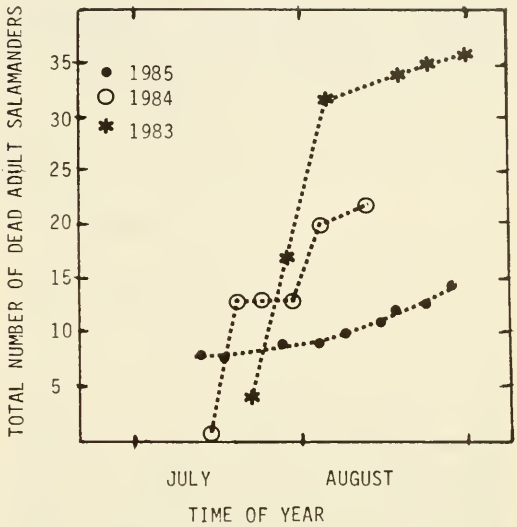

Fig. 3. Terrestrial adult mortality at Desolation Lake for three years.

Lake (3.3 km distant, same tributary of Big Cottonwood Creek, no pass), and Red Pine Lake (0.8 km distant, ridgeline pass of 2,952 m, Weber River drainage).

RESULTS

Adult transformed salamanders die in July and August (Fig. 3). At least 33 (1982), 37 (1983), 22 (1984), 15 (1985), and 12 (1986) adults were observed dead in the water, on the shoreline, and under logs adjacent to the shoreline. The adults died in a rapid mode (1983), in a biphasic mode (1984), or in an extended mode (1985).

The larval die-off at Desolation Lake started during mid-August 1983 and 1984 and the end of July 1985 (Fig. 4). A total of 4,949 (1983), 12,703 (1984), and 26,780 (1985) dead were counted. The mortality for these years was logarithmic, with its slope (determined by linear regression analysis of the accumulated sum of dead salamanders versus time) being 0.19 (1983), 0.28 (1984), and 0.40 (1985). Correlation coefficients were 0.99. The doubling time for the accumulation of dead salamanders was 3.6 (1983), 2.5 (1984), and 1.8 (1985) days. This doubling time was inversely proportional (slope −0.93) to the logarithm of the

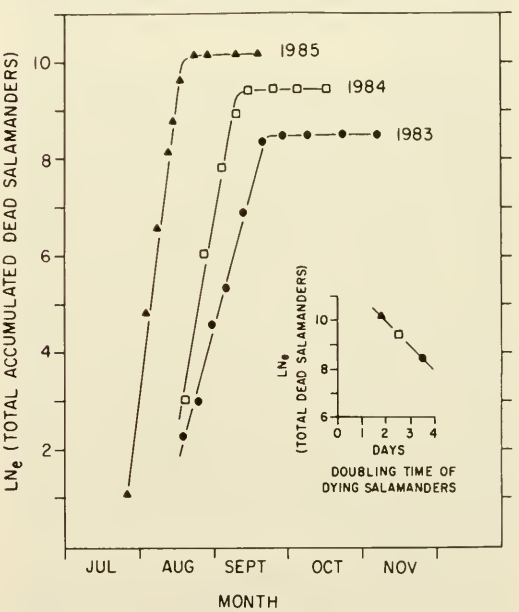

Fig. 4. The kinetics of the aquatic young salamander mortality in Desolation Lake for three years. The inset shows the relationship of the death rate to the total population.

total dead salamanders (correlation coefficient 1.0) (Fig. 4 inset). The doubling of the mortality rate coincided with a fivefold increase in the total number of salamanders. Once the larval die-off started, the time for 50% of the salamanders to die was 13–16 days. In 1981 and 1982 the die-off occurred during this

TABLE 1. Experimental infection of healthy larval salamanders with *Acinetobacter calcoaceticus*, *Pseudomonas aeruginosa*, and *Escherichia coli*; *A. calcoaceticus* and *P. aeruginosa* were isolated from infected salamanders and *E. coli* from Desolation Lake. Approximately 300 million colony-forming bacteria were added to 8 liters of water in aquaria. Two salamanders were in each aquarium.

Aquarium No.	Inoculant	Disease developed	Inoculant recovered from salamander
1	None	No	Not applicable
		No	Not applicable
2	*A. calcoaceticus*	Yes (18 days)	Yes
		Yes (28 days)	Yes
3	*P. aeruginosa*	Yes (33 days)	Yes
		No	No
4	*E. coli*	No	No
		No	No

same period, but in 1980 it was completed by early August with much smaller young. The shift in the time of the die-off (Fig. 4) is related to the amount of winter (October to June) precipitation (correlation coefficient 0.86), which secondarily influences the depth of water in Desolation Lake and perhaps the time of year the adult salamanders travel to and breed in the lake.

To determine the etiology of the process, we searched for the proximate cause of the aquatic salamander deaths. Histological analysis indicated that salamander livers did not contain lesions attributable to organic compounds or heavy metals. Algal analysis indicated that the seepage lakes did not experience an algal bloom and that the lakes were oligotrophic during the summer. Water analysis did not turn up any unusual concentrations of heavy metals. Transferring salamanders from the lake in July and in August to aquaria showed that the July salamanders remained healthy throughout the summer and the August salamanders died at the same time as the lake population. This suggested an infectious etiology.

Microbiological evaluation disclosed that *Acinetobacter* could be isolated from the peritoneum, skin, and gills of recently expired specimens from Desolation Lake. We found only gram-positive bacteria (*Corynebacteria, Bacillus, Micrococci, Staphlococci,* and *Streptococci*) on the salamander gills and skin from specimens taken from three different Wasatch Mountain ponds and lakes where the salamanders completed their life cycle. Although some gram-positive bacteria could be isolated from ill salamanders, all the infected salamanders contained an *Acinetobacter* sp. on their skin and gills and only this species in the peritoneum. *Pseudomonas aeruginosa* could be isolated from some infected individuals along with *Acinetobacter*. *Acinetobacter* sp. was identified as *Acinetobacter calcoaceticus;* but since the taxonomy has been updated (Bouvet and Grimont 1986), the bacterium involved may be *A. haemolyticus* because the isolates from the salamanders and from Desolation Lake were strongly hemolytic and some isolates hydrolyzed gelatin. Furthermore, *Acinetobacter* sp. could not utilize nitrates in complex media.

Acinetobacter isolates from infected salamanders, upon inoculation of aquarium waters, did cause the mortality of salamanders (Table 1). *Acinetobacter* was then isolated from these diseased salamanders. Thus, our data satisfy Koch's postulates for the identification of *Acinetobacter* as the proximate cause of the aquatic salamander's deaths. *Pseudomonas* caused the disease in one of two animals by these same criteria. Because of its limited numbers, *Pseudomonas* may cause secondary infections in the diseased salamanders. *Escherichia coli* taken from Desolation Lake was ineffective in causing symptoms or mortality in aquaria. The infected salamanders from aquaria and from Desolation Lake at the time of death had red, swollen hind legs and vent region. The throat skin, abdominal skin, kidney, spleen, and stomach were major organs affected with diffuse hemorrhage—possibly due to the hemolytic nature of the *Acinetobacter* strain.

After establishing the proximate cause of the salamander deaths, we examined Desolation Lake for bacteria and algae. Bacterial analysis in Desolation Lake in August 1983 showed

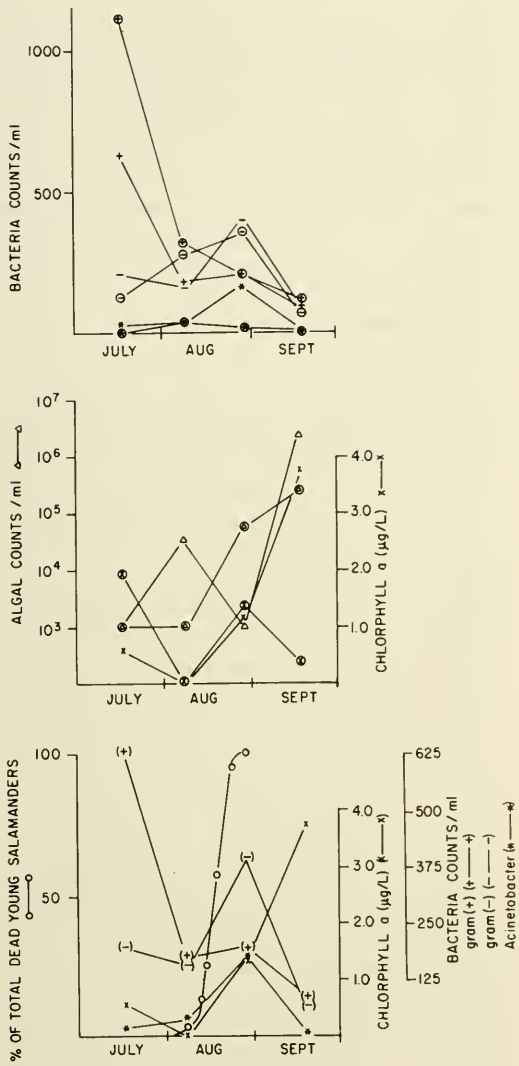

Fig. 5. Bottom: Desolation Lake salamander deaths in association with gram-positive (+) and gram negative (−) bacteria, *Acinetobacter* (star) levels, and chlorophyll *a* levels. Middle: Desolation and Dog lakes comparison (1985) of chlorophyll *a* and algal counts. Top: Desolation and Dog lakes comparison (1985) of gram-positive (+), gram-negative (−), and *Acinetobacter* (star) bacteria. The circled symbols represent Dog Lake. Algal counts of 10^3/ml were the minimum values detected. The difference between *Acinetobacter* levels and total gram-negative bacteria values is largely due to coliform bacteria.

gram-negative bacteria in the surface water (100/ml, only *Acinetobacter* sp.) and in the hypolimnion (greater than 1,000/ml with 100/ml as coliform and the remainder as *Acinetobacter*). In 1984 bacteria were undetected in the surface waters in our July sample (perhaps reflecting the snowmelt addition to the lake). The August sample was equally divided between gram-positive (190/ml) and gram-negative (220/ml). The September sample was entirely gram-negative (2,000/ml) and mostly *Acinetobacter*. Desolation Lake was largely void of bacteria in October.

In 1985 both Desolation and Dog lakes were examined at three-week intervals (Fig. 5). High numbers of gram-positive bacteria occurred in July and decreased in August and September. The gram-negative bacteria were present at low levels in July but peaked in late August at the end of the salamander die-off. By mid-September both Desolation and Dog lakes had a large decrease in gram-negative bacteria. Only in Desolation Lake did an *Acinetobacter* bloom occur, and this occurred at the same time as the salamander die-off. The remainder of the gram-negative bacteria in Dog and Desolation lakes were largely coliform bacteria.

Chlorophyll *a* and algal counts and identification showed that both Dog and Desolation lakes were oligotrophic at least until the end of summer (Fig. 5). An algal bloom might be considered as occurring in mid-September after the bacteria and salamander cycles occurred in Desolation Lake. This bloom was a result of the presence of the diatom *Cyclotella* (381×10^4/ml). In Dog Lake at this same time the diatom *Navicula* dominated (11.4×10^4/ml). Blue-green algae (*Anabaena*, *Oscillatoria*, and *Merismopedia*) occurred in both Dog and Desolation lakes, with *Chroococcus* occurring additionally in Desolation Lake. The two diatoms *Navicula* and *Cyclotella* occurred in both lakes. Green algae (*Chlamydemonas*-like) occurred in both Dog and Desolation lakes, with *Scenedesmus* occurring, in addition, in Desolation Lake and *Closterium* in Dog Lake. These studies indicate that the algal components of Desolation Lake did not contribute to the bacteria cycles in the lake or to the die-off of the salamanders.

Desolation Pond, adjacent to Desolation Lake, also experienced the salamander mass mortality. This pond is very small, a breeding ground for probably no more than one salamander pair. The die-off generally preceded the Desolation Lake mass mortality. In 1986 there was no die-off in Desolation Pond, while

the die-off in Desolation Lake occurred in a typical manner.

DISCUSSION

Although the initial purpose of this study was to determine the proximate cause of the mass mortality in the salamander populations in Desolation Lake, observations of the bacteria, algae, and salamanders in seepage lakes did raise additional questions. Since these seepage lakes may all be nitrogen-limiting with respect to biological growth and this would extend previous observations for the region (Wurtsbaugh 1986), any addition of nitrogen, from grazing sheep in the watershed or from atmospheric sources, to the aquatic systems will alter the abundance and diversity of the microorganisms. To what extent this is occurring in Desolation Lake would require more studies as to the available nutrient and biological productivity at the bacteria level and at the aquatic insect level. With the average weight of an aquatic salamander in early autumn being 13.2 g (average snout-vent length of 67 mm), the total salamander productivity of Desolation Lake varied from 66 (1983) to 330 (1985) kg of wet weight. It would be interesting to note the biological productivity of salamanders in Desolation Lake without the external sources of nitrogen.

It is not known why *Acinetobacter* was dominant in Desolation Lake, nor is it known how the crustaceans in the lake influence the bacterial composition or how the aquatic salamanders affect the crustacean populations. It is known that salamanders affect the species and numbers of crustaceans in ponds (Dodson 1971, Giguére 1970). Since the crustaceans are large feeders of microorganisms, the salamanders may be contributing to their own deaths by allowing more bacteria to survive. Riemann (1985) noted that fish predation on zooplankton increased the bacterial numbers in a lake. Salamander predation on zooplankton should likewise allow for bacterial numbers to increase.

In Desolation Lake only large Dystiscidae have the capability to preying on the aquatic salamanders, but their numbers were low in comparison to other lakes in both the Wasatch and Uinta mountains. Most aquatic salamanders probably escape predation in Desolation Lake. In this study, then, the total dead aquatic salamanders for the year accounts for the total number of eggs laid. The ratio of total aquatic larvae to total adult (dead) salamanders varied from 137 (1983) to 577 (1984) and 1,912 (1985). If one assumes that an adult female lays from 250 to 350 eggs (Anderson et al. 1971) and that half of the dead adults were females, one can conclude that in 1983 all the adults died that bred in Desolation Lake. It is apparent that surplus adults can come from the region and adjacent lakes to breed in Desolation Lake.

Acinetobacter can act as a pathogen for other vertebrates. It is not known whether *Acinetobacter* was responsible for the deaths of other vertebrates at Desolation Lake. Besides the dead salamanders, 2 sheep, 2 mice, 2 ground squirrels, 1 porcupine, and 10 songbirds were found dead along the shore. Again, other lakes in these mountains did not reveal such numbers of dead mammals and birds; in fact, no dead mammals or birds could be found at the other lakes. The dead salamanders, birds, and mammals were fed upon by *Gammarus*, Trichoptera larvae, Dystiscidae, fungus, snails (Physidae), Hymenoptera, Diptera, and Coleoptera.

A proposed hypothesis to explain the seasonal salamander mortality states that the lake starts with fresh snow water in the epilimnion and old water from the previous year in the hypolimnion. The adult salamanders arrive at the lake in July to breed and feed in the bacteria-rich hypolimnion. While feeding, they become infected and die. The lake then experiences a gram-positive bloom and subsequent decline, attributed to the nitrate from snowmelt in the epilimnion. The lake level lowers and in this process the hypolimnion disappears. This allows the supply of gram-negative bacteria, including *Acinetobacter* which wintered in the hypolimnion, to bloom in the lake. At this time the aquatic young salamanders that have been growing all season long become infected and begin dying. At the peak of this salamander mortality, the gram-negative bacteria decline and subsequently the diatom bloom occurs. The timing of the seasonal cycles is related to the amount of winter precipitation, which directly affects the level of the lake in July.

CONCLUSIONS

We have described a recurrent annual salamander die-off attributed to *Acinctobacter* sp. in an oligotrophic lake. Not only was *Acinetobacter* sp. found to be the proximate cause by Koch's postulates, but it was also found to reach its greatest numbers in the lake at the peak of the mass mortality. Since the lake was found to be growth-limiting with respect to nitrogen, the addition of nitrogen from atmospheric sources and from sheep grazing in the watershed contributed to the abundance of bacteria and hence may be the ultimate perturbation of the lake system responsible for the salamander mass mortality. The seepage lakes with mass mortality had extensive lowering of the water levels. The seasonal timing of the biological cycles in the lake was correlated with the total snowfall.

This paper thus describes some of the biological properties of seepage lakes as well as some aspects of salamander biology as they relate to seepage lakes. It is apparent that such ecological disasters as salamander mass mortality can occur in aquatic systems without either acidification or eutrophication.

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LITERATURE CITED

ANDERSON, J. D., D. D. HASSINGER, AND G. H. DALRYMPLE. 1971. Natural mortality of eggs and larvae of *Ambystoma t. tigrinum*. Ecology 52: 1107–1112.

BOUVET, P. J., M. M., AND P. A. D. GRIMONT. 1986. Taxonomy of the genus *Acinctobacter* with the recognition of *Acinctobacter baumannii* sp. nov., *Acinctobacter haemolyticus* sp. nov., *Acinctobacter johnsonii* sp. nov., and *Acinctobacter junii* sp. nov. and emended descriptions of *Acinetobacter calcoaccticus* and *Acinetobacter lwoffii*. Inter. J. Sys. Bact. 36: 228–240.

COLLINS, J. P. 1981. Distribution, habitats and life history variations in the tiger salamander, *Ambystoma tigrinum*, in east-central and southeast Arizona. Copeia 1981: 666–675.

DARLING, L. A. 1973. The effects of livestock grazing on wildland water quality. Unpublished thesis, Utah State University, Logan.

DODSON, S. I. 1970. Complementary feeding niches sustained by size-selective predation. Limnol. Ocean. 15: 131–137.

FRY, J. C. 1982. The analysis of microbial interactions and communities *in situ*. Pages 103–132 *in* A. T. Bull and J. H. Slater, eds., Microbial interactions and communities. Vol. 1. Academic Press.

GIGUÉRE, L. 1979. An experimental test of Dodson's hypothesis that *Ambystoma* (a salamander) and *Chaoborus* (a phantom midge) have complementary feeding niches. Canadian J. Zool. 57: 1091–1097.

HASSINGER, D. D., J. D. ANDERSON, AND G. H. DALRYMPLE. 1970. The early life history and ecology of *Ambystoma tigrinum* and *Ambystoma opacum* in New Jersey. Amer. Mid. Nat. 84: 474–495.

LENNETTE, E. H., A. BALOWS, W. J. HAUSLER, AND H. G. SHADOMY. 1985. Manual of clinical microbiology. 4th ed. Amer. Soc. Microbiology, Washington, D.C. 1,149 pp.

NYMAN, S. 1986. Mass mortality in larval *Rana sylvatica* attributable to the bacterium, *Aeromonas hydrophila*. J. Herp. 20: 196–201.

PENNAK, R. W. 1968. Colorado semidrainage mountain lakes. Limnol. Ocean. 14: 720–725.

REICHENBACH-KLINKE, H., AND E. ELKAN. 1965. Pages 233–236 *in* Diseases of amphibians. T.F.H. Publications, Neptune City, New Jersey.

RIEMANN, B. 1985. Potential importance of fish predation and zooplankton grazing on natural populations of freshwater bacteria. Appl. Environ. Microbiology 50: 187–193.

ROSE, F. L., AND D. ARMENTROUT. 1976. Adaptive strategies of *Ambystoma tigrinum* Green inhabiting the llano estacado of west Texas. J. Animal Ecol. 45: 713–729.

ROSE, F. L., AND J. C. HARSHBARGER. 1977. Neoplastic and possibly related skin lesions in neotenic tiger salamanders from a sewage lagoon. Science 196: 315–317.

SEMLITSCH, R. D. 1983. Structure and dynamics of two breeding populations of eastern tiger salamander, *Ambystoma tigrinum*. Copeia 1983: 608–616.

SEXTON, O. J., AND J. R. BIZER. 1978. Life history patterns of *Ambystoma tigrinum* in montane Colorado. Amer. Mid. Nat. 99: 101–118.

TANNER, W. W., D. L. FISHER, AND T. J. WILLIS. 1971. Notes on the life history of *Ambystoma tigrinum nebulosum* Hallowell in Utah. Great Basin Nat. 31: 213–222.

TERHO, T. T., AND K HARTIALA 1971. Method for determination of the sulfate content of glycosaminoglycans. Anal. Biochem. 41: 471–476.

WURTSBAUGH, W. A 1986. Limiting factors for algal growth and nitrogen fixation in the Great Salt Lake drainage, Utah. Pages 121–132 *in* Proc. Bonneville Chapter of the Amer. Fisheries Soc.