similar analogs in the treatment of T cell lymphoproliferative diseases or as immunosuppressive agents in the treatment of autoimmune disorders has yet to be explored. However, the results of this study suggest that 8-aminoguanosine could be both extremely effective as a lympholytic agent and more specific for T lymphocytes than are other agents that are currently available.

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References and Notes

1. E. R. Giblett, A. J. Ammann, D. W. Wara, R. Sandman, L. K. Diamond, *Lancet* **1975-I**, 1010 (1975); J. W. Stoop *et al.*, *N. Engl. J. Med.* **296**, 651 (1977).
2. A. Cohen, L. J. Gudas, A. J. Ammann, G. E. J. Staal, D. W. Martin, *J. Clin. Invest.* **61**, 1405 (1978).
3. B. S. Mitchell, E. Mejias, P. E. Daddona, W. N. Kelley, *Proc. Natl. Acad. Sci. U.S.A.* **75**, 5011 (1978).
4. J. M. Wilson, B. S. Mitchell, P. E. Daddona, W. N. Kelley, *J. Clin. Invest.* **64**, 1475 (1979); B. Ullman, L. J. Gudas, S. M. Clift, D. W. Martin, *Proc. Natl. Acad. Sci. U.S.A.* **76**, 1074 (1979).
5. E. R. Giblett, J. E. Anderson, F. Cohen, B. Pollara, H. J. Meuwissen, *Lancet* **1972-II**, 1067 (1972); H. J. Meuwissen, B. Pollara, R. J. Pickering, *J. Pediatr.* **86**, 169 (1975).
6. M. S. Coleman, J. Donofrio, J. J. Hutton, L. Hahn, A. Daoud, B. Lampkin, J. Dyminski, *J. Biol. Chem.* **253**, 1619 (1978); A. Cohen, R. Hirschhorn, S. D. Horowitz, A. Rubenstein, S. H. Polmar, R. Hong, D. W. Martin, *Proc. Natl. Acad. Sci. U.S.A.* **75**, 472 (1978).
7. D. A. Carson, J. Kaye, J. E. Seegmiller, *J. Immunol.* **121**, 1726 (1978).
8. M. S. Hershfield, *J. Biol. Chem.* **254**, 22 (1979).
9. B. S. Mitchell, C. A. Koller, R. Heyn, *Blood* **56**, 556 (1980).
10. R. E. Holmes and R. K. Robins, *J. Am. Chem. Soc.* **87**, 1772 (1965).
11. J. D. Stoeckler, C. Cambor, V. Kuhns, S. H. Chu, R. E. Parks, *Biochem. Pharmacol.*, in press.
12. M. R. Sheen, B. K. Kim, R. E. Parks, *Mol. Pharmacol.* **4**, 293 (1968).
13. D. A. Carson, J. Kaye, S. Matsumoto, J. E. Seegmiller, L. Thompson, *Proc. Natl. Acad. Sci. U.S.A.* **76**, 2430 (1979); R. L. Wortmann, B. S. Mitchell, N. L. Edwards, I. H. Fox, *ibid.*, p. 2434; D. A. Carson, J. Kaye, D. B. Wasson, *J. Immunol.* **126**, 348 (1981).
14. I. H. Fox, C. M. Andres, E. W. Gelfand, D. Biggar, *Science* **197**, 1084 (1977).
15. C. A. Koller, P. L. Stetson, L. D. Nichamin, B. S. Mitchell, *Biochem. Med.* **24**, 179 (1980).
16. M. B. Van der Weyden, R. H. Buckley, W. N. Kelley, *Biochem. Biophys. Res. Commun.* **57**, 590 (1974).
17. We thank E. Ashcraft for technical assistance and J. A. Kaminska and I. H. Fox for performance of *S*-adenosylhomocysteine hydrolase assays. This research was supported by grant AM 19045 from the National Institutes of Health, by grants CA 26284 and CA 26032 from the National Cancer Institute, and by grant CH-183 from the American Cancer Society. B.S.M. is the recipient of NIH clinical investigator award IK08 AM00442.

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Vibrio damsela, a Marine Bacterium, Causes Skin Ulcers on the Damselfish *Chromis punctipinnis*

Abstract. *A previously undescribed marine bacterium,* Vibrio damsela, *was isolated from naturally occurring skin ulcers on a species of temperate-water damselfish, the blacksmith* (Chromis punctipinnis). *Laboratory infection of the blacksmith with* Vibrio damsela *produced similar ulcers.* Vibrio damsela *was pathogenic for four other species of damselfish but not for members of other families of fish. The bacterium has also been isolated from water and from two human wounds and may be a cause of human disease.*

During the summer and fall spawning season in southern California, the blacksmith, a temperate-water damselfish (*Chromis punctipinnis*), may have irregular skin ulcers along its flanks (Fig. 1). There has been much speculation on the cause of these lesions, which are rare among naturally occurring marine fish. We now report that these skin lesions are caused by a newly described marine bacterium, *Vibrio damsela* sp. nov. (*1*), and we discuss the possible role of *V. damsela* in human wound infections.

The blacksmith is a small (total length, up to 35 cm), schooling, mid-water planktivore that lives along inshore reefs, to depths of 33 m, from Baja California to Monterey, California (*2*). Ulcerated *C. punctipinnis* have been observed from August through October in King Harbor, Redondo Beach, California, and from June through August off Ship Rock, Catalina Island (*3*). Although ulceration rates are often around 10 percent, in some aggregations more than 70 percent of the individuals had ulcers.

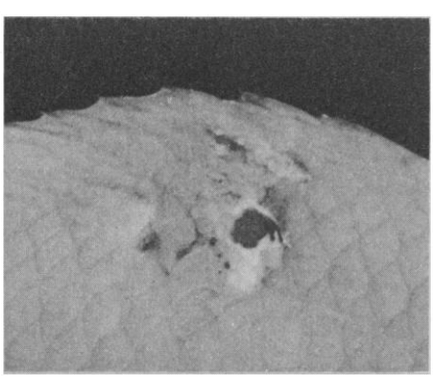

Fig. 1. Ulceration on skin of garibaldi (*Hypsypops rubicunda*) caused by laboratory infection with *Vibrio damsela*.

The ulcers are 0.5 to 2.0 cm in diameter and are usually near the pectoral fin and on the caudal peduncle. Histopathological examination of the lesions indicates a granulomatous ulcerative dermatitis. Lesions are characterized by muscle lysis and by histiocytes present in the dermis and skeletal muscles (*4*).

Bacterial isolations were made by swabbing the ulcerated lesions with a sterile Dacron swab and then streaking a brain-heart infusion agar culture medium containing 5 percent sheep blood. All bacterial incubations were carried out at 25°C unless otherwise stated. Cultures from the lesions yielded a number of different bacterial species and protozoan parasites, so that it was initially difficult to determine which, if any, was causing the lesion. Thirteen different bacteria were obtained in pure culture and tested for ability to produce ulcers. Healthy specimens of *C. punctipinnis* were collected from King Harbor and maintained in the laboratory (*5*). Eight fish were anesthetized with tricaine methane sulfonate (MS-222; 1:5000 in seawater). Four to six scales were removed from the flanks of each fish and the dermis scarified. The resulting lesions of four fish were swabbed with 10^7 to 10^8 viable cells. Four control fish were swabbed with sterile medium. Only *V. damsela* proved pathogenic.

Vibrio damsela produced large ulcers within 3 days and caused death in all animals within 4 days. Experimentally induced wounds in control animals had healed completely by this time. At the time of death, the ulcers in the fish inoculated with *V. damsela* were larger than the area originally scarified. Cultures from each ulcer yielded *V. damsela*. One of these cultures was reintro-

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duced (using the techniques of the first experiment) into ten blacksmith. All developed ulcers, and the bacterium again was cultured from all ten.

It was not necessary to scarify the dermis to induce ulcers. Cultures of *V. damsela* were swabbed onto the flanks of ten unscarified blacksmith and five of these developed ulcers. Again, the bacterium was cultured from all five. In all instances, diseased individuals died within 4 days of infection (*6, 7*).

Underwater fish surveys conducted over a 10-year period at both King Harbor and Catalina Island have indicated that fish other than blacksmith rarely have ulcers. Moreover, we were unable to infect representative members of six other families: Girellidae (opaleye, *Girella nigricans*); Atherinidae (top smelt, *Atherinops affinis*); Clinidae (spotted kelpfish, *Gibbonsia elegans*); Cottidae (wooly sculpin, *Clinocottus analis*); Embiotocidae (shiner perch, *Cymatogaster aggregata*), and Gobiidae (bluebanded goby, *Lythrypnus dalli*), all of which are found in or over temperate reefs frequented by blacksmith. Natural *V. damsela* infections seem to be nearly, or perhaps entirely, limited to *C. punctipinnis* populations.

The seasonal infectivity of *V. damsela* may be the result of elevated water temperatures that allow the buildup of sufficient bacterial populations to cause disease (*8*). Blacksmith create and defend nests during this period, and this (together with the physiological requirements needed to develop gonadal tissue) may lower host resistance. Indirect evidence comes from our success in experimentally infecting both immature and mature individuals, although only mature infected blacksmith have been observed in reef surveys. We have isolated *V. damsela* from marine algae, and perhaps blacksmith encounter *V. damsela* on adjacent nest sites. Further, the characteristic behavior of blacksmith to seek shelter at night in reef crevices with other blacksmith (*2*) may aid in disease transmittal. In garibaldi, natural infections appear to be rare (*3*), although we readily infected garibaldi with *V. damsela* in the laboratory. Garibaldi are solitary, and this behavior may reduce disease transmission. In addition, garibaldi spawn in the spring when environmental conditions may not be as conducive to bacterial growth.

Whether *V. damsela* can cause human infections is a question that cannot be answered with certainty (*9*), but *V. damsela* has been isolated from wounds of patients from coastal states. The importance of halophilic vibrios in human disease is now recognized (*10*). *Vibrio damsela* was definitely excluded from belonging to the species usually associated with human infection—*V. cholerae, V. parahaemolyticus, V. alginolyticus, V. vulnificus, V. metschnikovii,* and *V. fluvialis*. Three of the isolates from water or clinical specimens were almost identical phenotypically to *V. damsela* (strain 2588-80). The first isolate (number 0183-79; ATCC 33537) was from a puncture wound of a 32-year-old man in Hawaii. The second isolate (number 1958-80; ATCC 33538) was from the leg wound of a 47-year-old man from Florida. A third isolate (0023-81; ATCC 33536) was from a water sample taken on the Gulf coast of Florida. By DNA-DNA hybridization these isolates were so closely related to *V. damsela* (> 75 percent at 75°C, hydroxyapatite method) that they clearly belong to the same species. These data indicate that *V. damsela* may be able to infect man as well as marine fish.

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References and Notes

1. The following description is required by the Bacteriological Code for valid announcement of a new species. The name *Vibrio* is a modern Latin masculine noun. The species name *damsela* is taken from the modern zoological term damselfish and is treated as a modern Latin substantive in opposition to the name *Vibrio*. Thus, the species name does not have to agree in gender with the genus name. The type strain of *V. damsela* is designated by Centers for Disease Control number 2588-80, which was deposited in the American Type Culture Collection, Rockville, Md., as ATCC 33539. *Vibrio damsela* shares the properties of the genus *Vibrio*; its general properties are (all data are based on the type strain incubated at 25°C unless otherwise indicated): Gram-negative rod; oxidase-positive; requires addition of NaCl to peptone media for growth; grows better at 25°C than at 37°C on initial isolation; grows well on thiosulfate citrate bile salts agar as 2- to 3-mm green colonies; guanine plus cytosine content of DNA (buoyant density method) is 43 mole percent; and is inhibited by 0/129 disks (22-mm zone). The properties that differentiate *V. damsela* from other *Vibrio* species are indole-negative; urease-positive; methyl red– and Voges-Proskauer-positive; arginine dihydrolase (Moeller's method)–positive; lysine and ornithine decarboxylase–negative; gas produced during fermentation of carbohydrates; weakly motile; not bioluminescent; citrate not used as the sole source of carbon and energy; and only D-glucose, D-mannose, and maltose are fermented. These properties are unique to *V. damsela* among the named species of *Vibrio* and *Photobacterium* [P. Baumann, L. Baumann, S. S. Bang, M. J. Woolkalis, *Curr. Microbiol.* **4**, 127 (1980)]. In addition, *V. damsela* also is distinct from all of the named species by DNA-DNA hybridization. Other properties include positive results in tests for deoxyribonuclease production, nitrate reduction to nitrite, and growth in the presence of 1, 3.5, and 6 percent NaCl (weight by volume, in nutrient broth). Tests that produced negative results include hydrogen sulfide production on triple-sugar iron agar; phenylalanine deaminase; utilization of malonate or acetate; gelatin hydrolysis; growth in the presence of potassium cyanide; lipase (corn oil) production; the *O*-nitrophenyl-β-D-galactopyranoside test; the string test; fermentation of D-adonitol, L-arabinose, D-arabitol, cellobiose, dulcitol, erythritol, *i*-inositol, lactose, D-mannitol, melibiose, α-methyl-D-glucoside, mucate, raffinose, L-rhamnose, salicin, D-sorbitol, sucrose, trehalose, and D-xylose; and growth in the presence of 0, 8, 10, and 12 percent sodium chloride (weight by volume, in nutrient broth).
2. R. Bray. *U.S. Fish Wildl. Serv. Fish. Bull.* **78**, 829 (1981); D. J. Miller and R. N. Lea, *Calif. Dep. Fish Game Fish Bull.* **157**, 42 (1971).
3. J. S. Stephens, Jr., personal communication; R. Given, personal communication.
4. J. O. Britt, personal communication.
5. After collection by scuba divers, fish were transported immediately to the laboratory and maintained in 100-liter polypropylene holding tanks immersed in a temperature-controlled water bath. Each tank was supplied with a once-through continuous flow of filtered (5 μm), ultraviolet-sterilized, aerated harbor water. Fish were acclimated to laboratory holding facilities (at 16.0° to 16.6°C) for at least 1 week before experimentation and were fed daily with brine shrimp. Tanks were illuminated for the natural photoperiod.
6. These experiments strongly indicate that *V. damsela* was actually causing the infections; however, multiorganism infection with *V. damsela* as the precipitating factor cannot be ruled out completely. In this model, a second organism, such as a bacterium, parasite, or virus, could be present in the fish's skin (although none was detected with light microscopy) and, with the introduction of *V. damsela*, a skin lesion of polymicrobial origin would result.
7. To determine whether ulcers were limited to blacksmith, we infected another temperate-water damselfish, the garibaldi (*Hypsypops rubicunda*) with *V. damsela*. Two species that had been scarified and inoculated with *V. damsela* showed characteristic ulcers that yielded *V. damsela*, and the fish died 1 to 2 weeks after infection. An ulcer was produced on one of two garibaldi that had been swabbed (but not scarified) with *V. damsela*. We purchased three other species of tropical damselfish, maintained them at 23°C, and applied *V. damsela* after scarification. Four of five *Chromis caerulea*, two of three *Dascyllus trimaculatus*, and two of five *Abudefduf uniocellatus* developed ulcers. All infected fishes died within 24 hours of experimental infection, and *V. damsela* was recovered from all ulcerations.
8. *Vibrio anguillarum* infections are most prevalent in late summer [C. J. Sindermann, *Adv. Mar. Biol.* **4**, 1 (1966)].
9. Case histories of seven patients with wound infections positive for *V. damsela* have been analyzed (J. G. Morris, personal communication).
10. P. A. Blake *et al.*, *N. Engl. J. Med.* **302**, 305 (1980).
11. We thank M. Mandel for guanine plus cytosine determinations, J. Britt for preparing and interpreting histology specimens, and J. Stephens for encouragement. P. Morris, K. Zerba, and C. Rand first brought this condition to our attention, and they, along with E. Taylor, K. Shriner, D. Wakamatsu, T. King, and L. Purcell, collected specimens. Helpful comments and criticisms came from A. W. Ebeling and R. Lavenberg. Garibaldi were obtained from P. Haaker, California Fish and Game Department. We thank P. Baumann, A. L. Furniss, and R. Gherna for furnishing cultures of all the named species of *Vibrio* and *Photobacterium* and K. A. Kelley of Emory University for helping with the coining of the scientific name.

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