related to its ability to bind specifically to basement membranes. This binding capacity could contribute significantly to the organism's extreme virulence.

References and Notes

1. E. H. Beachey, in *Bacterial Adherence*, E. H. Beachey, Ed. (Chapman & Hall, London, 1980), p. xi; E. H. J. Beachey, *Infect. Dis.* **143**, 325 (1981).
2. R. Timpl *et al.*, *J. Biol. Chem.* **254**, 9933 (1979).
3. H. K. Kleinman, P. J. Klebe, G. R. Martin, *J. Cell Biol.* **88**, 473 (1981); E. G. Hayman *et al.*, *ibid.* **92**, 28 (1982).
4. H. Lesot, U. Kuhl, K. von der Mark, *EMBO J.* **2**, 861 (1983).
5. H. L. Malinoff and M. S. Wicha, *J. Cell Biol.* **96**, 1475 (1983); V. P. Terranova *et al.*, *Proc. Natl. Acad. Sci. U.S.A.* **80**, 444 (1983).
6. M. S. Wicha, H. L. Malinoff, T. K. Huard, *Proc. Am. Assoc. Cancer Res.* **24**, 35 (1983).
7. M. M. Brentani, V. R. Martins, R. R. Brentani, *ibid.* **25**, 54 (1984).
8. M. H. Kaplan and M. J. Tenenbaum, *Am. J. Med.* **72**, 248 (1982).
9. G. P. Youmans, P. Y. Paterson, H. M. Sommers, in *The Biologic and Clinical Basis of Infectious Disease* (Saunders, Philadelphia, 1975), p. 596.
10. R. A. Forse *et al.*, *Surgery* **86**, 507 (1979).
11. J. N. Sheagren, *N. Engl. J. Med.* **310**, 1368 (1984).
12. C. Scatchard, *Ann. N. Y. Acad. Sci.* **51**, 660 (1949).

12a. P. L. Ey, S. J. Prowse, C. R. Jenkin, *Immunochemistry* **15**, 429 (1978).

13. L. M. Switalski *et al.*, *J. Biol. Chem.* **259**, 3734 (1984).
14. E. Plessmann Camargo *et al.*, *Trans. R. Soc. Trop. Med. Hyg.* **77**, 825 (1983).
15. R. A. Proctor, D. F. Mosher, P. J. Olbrantz, *J. Biol. Chem.* **257**, 14788 (1982).
16. P. G. Garrison and L. R. Freedman, *Yale J. Biol. Med.* **42**, 394 (1970); A. A. August and M. Oka, *J. Am. Med. Assoc.* **183**, 249 (1963); C. Weinstein and J. J. Schlesinger, *N. Engl. J. Med.* **291**, 832 (1974).
17. J. E. Doran, A. R. Mansherger, A. C. Reese, *J. Reticuloendothel. Soc.* **27**, 471 (1980); M. P. Bevilacqua *et al.*, *J. Exp. Med.* **153**, 42 (1981).
18. V. P. Terranova *et al.*, *Science* **226**, 982 (1984).
19. V. P. Terranova *et al.*, *Cancer Res.* **42**, 2265 (1982); L. A. Liotta, *Am. J. Pathol.* **117**, 339 (1984); S. A. Barsky *et al.*, *J. Clin. Invest.* **74**, 843 (1984).
20. S. Arvidson *et al.*, *Acta. Pathol. Microbiol. Scand. Sect. B* **79**, 399 (1971).
21. S. W. Kessler, *J. Immunol.* **115**, 1617 (1975).
22. J. W. Goding and E. Handman in *Genes and Antigens of Parasites, a Laboratory Manual*, C. M. Morel, Ed. (Fundacao Oswaldo Cruz, Rio de Janeiro, 1984), p. 373.
23. Bacterial cell pellets from 1-liter cultures were suspended in 8.0 ml of 50 m*M* tris-HCl (*p*H 8.0) containing 25 percent sucrose, 1 m*M* *N*-ethylmaleimide, 1 m*M* phenylmethylsulfonyl fluoride, and 1 m*M* EDTA. Lysozyme (3 mg) and Nonidet P-40 (final concentration 1 percent) were added and the suspension was incubated at 37°C for 60 minutes. Cell extracts were used for SDS-PAGE (*22*). After blotting, filters were incubated for 60 minutes with 3 percent BSA in PBS containing 0.03 percent Tween 20, then immersed in 25 m*M* tris-HCl (*p*H 7.4) containing unlabeled laminin (20 μg/ml), 5 m*M* $CaCl_2$, 5 m*M* $MgCl_2$, and 0.15*M* NaCl for 120 minutes at room temperature and washed in the same buffer without laminin. Affinity-purified rabbit antibody to mouse laminin, prepared in our laboratory by intramuscular injections of laminin in complete Freund's Adjuvant into New Zealand rabbits, was diluted 20-fold and added to the filters. The filters were incubated for 120 minutes at room temperature and developed with a horseradish peroxidase–conjugated goat antibody to rabbit IgG (Miles Laboratories) as described (*22*).

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Induced Expression of the Glucocorticoid Receptor in the Rat Intermediate Pituitary Lobe

Abstract. *Synthesis and release of pro-opiomelanocortin–derived peptides are under differential regulation in the anterior and intermediate lobes of the pituitary. Glucocorticoids inhibit synthesis of pro-opiomelanocortin–related peptides in the anterior lobe but not in the intermediate lobe. These two lobes are also characterized by differences in neural innervation and blood flow, both of which may represent routes of access for regulatory factors (the intermediate lobe is avascular). Immunoreactive glucocorticoid receptor, which can be demonstrated in many tissues, is absent from the intermediate lobe. Immunocytochemistry was used to demonstrate the presence of immunoreactive glucocorticoid receptor in the intermediate lobe after pituitary stalk transection, neurointermediate lobe grafts to kidney capsule, or monolayer culture of neurointermediate pituitary cells. This appearance of the glucocorticoid receptor is presumably a consequence of removal of intermediate pituitary cells from neural influences that may be responsible for inhibiting their expression under normal conditions in vivo.*

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Adrenocorticotropic hormone (ACTH) and lipotropic hormone (β-LPH), initially isolated from anterior pituitary lobe (AL) extracts, are derived from a common precursor molecule, pro-opiomelanocortin (POMC) (*1*). This precursor molecule is also present in the intermediate pituitary lobe (IL), in which ACTH and β-LPH are further processed to generate α-melanocyte stimulating hormone (α-MSH) and β-endorphin–related peptides (*2*). There is a differential regulation of synthesis and release of POMC and its derived peptides in AL and IL (*3*). Whereas the AL of virtually all species is regulated by way of central nervous system–derived (humoral) factors (peptides, biogenic amines) that are transported by the hypophyseal portal system, the only nerve supply to AL is a limited one, arriving via sympathetic innervation of blood vessels. Abundant serotonergic fibers were found in rat AL (*4*), although this was not confirmed in another study (*5*). Muscarinic (*6*), dopaminergic (*7*), and adrenergic receptors (*8*) are present in AL, and serotonergic uptake mechanisms (*9*, *10*) are reported to be present therein.

In contrast to the AL, the IL is poorly vascularized and receives essentially a dopaminergic and serotonergic innervation arising from the hypothalamus (*11*). Glucocorticoids, which are potent inhibitors of stimulated ACTH secretion from AL, act at a transcriptional level to inhibit both synthesis and release of POMC-related peptides in AL (*12*). Similar glucocorticoid treatment of AL and IL resulted in suppression of POMC messenger RNA (mRNA) in IL that was only one-tenth of that seen in AL (*13*); in another study, no suppression of POMC mRNA was observed (*14*). Glucocorticoid effects on AL, as in all other glucocorticoid-sensitive tissues, are mediated through interaction with a cellular receptor (*15*). The lack of effect of glucocorticoids on IL POMC levels is thought to be secondary to the lack of detectable glucocorticoid receptors in this lobe (*16*, *17*). Using immunocytochemistry, we localized the glucocorticoid receptor in the AL but not in adjacent cells of the IL (*16*). In other instances of glucocorticoid resistance, the glucocorticoid receptor decreased (*18*). In contrast to glucocorticoid, dopamine inhibited the release of POMC-derived peptides from IL but not AL (*19*). Administration of dopamine antagonists and agonists, respectively, increases and decreases POMC mRNA in neurointermediate lobe (NIL) but has no effect on the concentration of POMC mRNA in AL (*19*).

Because glucocorticoid receptors are found in nearly all tissues studied to date (*15*), and because of the wide range of metabolic actions of glucocorticoids subserved by such receptors, we investigated the reasons that glucocorticoid receptors are not detected in IL. One possibility is that glucocorticoid receptor expression is suppressed in IL. The release of IL peptides and the synthesis of IL POMC, as noted above, as well as IL cell growth (*20*) appear to be inhibited by

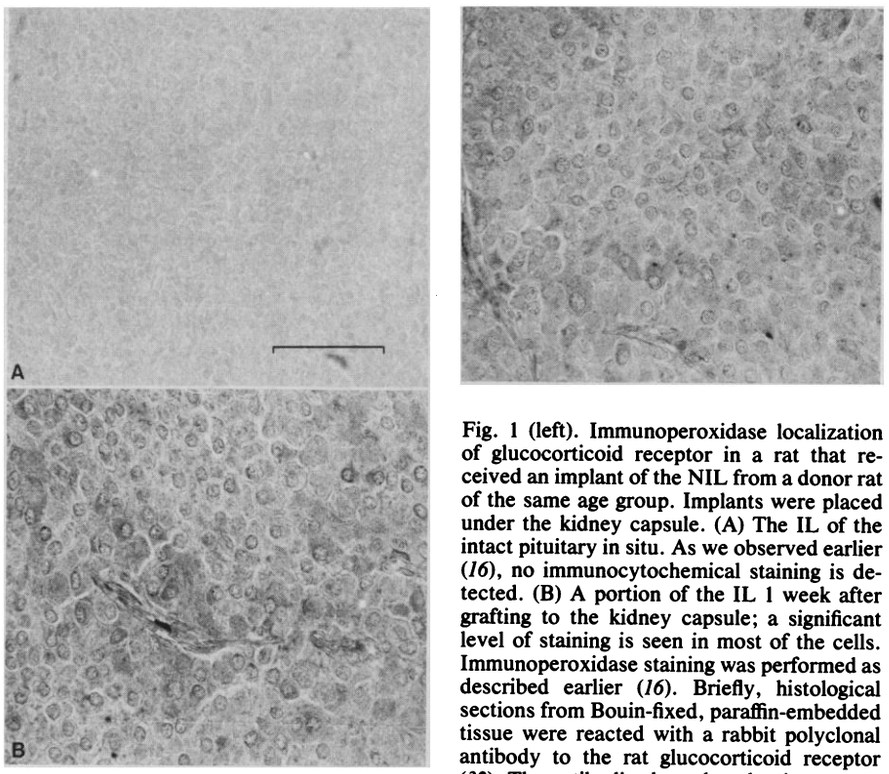

Fig. 1 (left). Immunoperoxidase localization of glucocorticoid receptor in a rat that received an implant of the NIL from a donor rat of the same age group. Implants were placed under the kidney capsule. (A) The IL of the intact pituitary in situ. As we observed earlier (*16*), no immunocytochemical staining is detected. (B) A portion of the IL 1 week after grafting to the kidney capsule; a significant level of staining is seen in most of the cells. Immunoperoxidase staining was performed as described earlier (*16*). Briefly, histological sections from Bouin-fixed, paraffin-embedded tissue were reacted with a rabbit polyclonal antibody to the rat glucocorticoid receptor (*32*). The antibodies bound to the tissue were revealed by an antibody to rabbit immunoglobulin G (IgG) labeled with peroxidase (*16*). When the sections were stained with either a monoclonal antibody (*30*) or a polyclonal (*31*) antibody to the human glucocorticoid receptor, which also reacts with the rat receptor (*31*), similar results were obtained. Scale bar, 50 μm for this and all subsequent figures. Fig. 2 (right). Immunoperoxidase localization of glucocorticoid receptor in the IL of the pituitary of a rat with pituitary stalk transection. The animal was killed 2 weeks after the surgery. This tissue section was processed for the localization of the glucocorticoid receptor, as in Fig. 1, with a polyclonal antibody to the rat receptor (*32*). A significant immunoreactivity is observed in these IL cells as compared to those in intact pituitary IL (Fig. 1A). Nuclear and cytoplasmic staining can be seen.

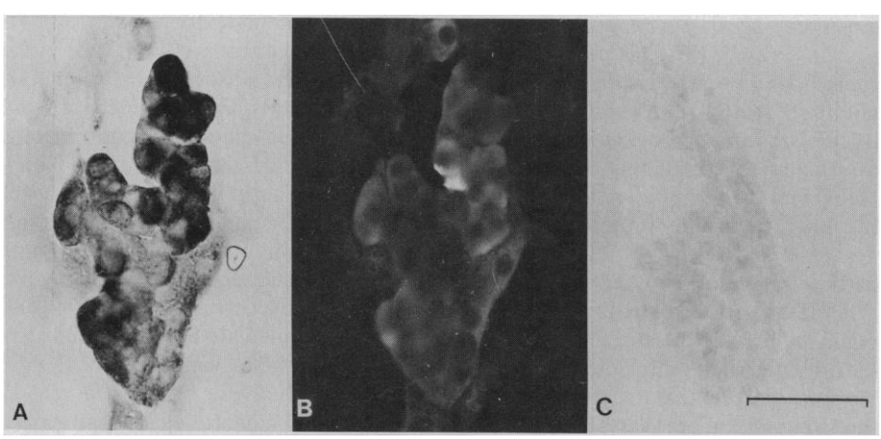

Fig. 3. (A) Immunoperoxidase localization of glucocorticoid receptor (*16*) in NIL pituitary cells maintained in primary monolayer culture as described (*22, 23*). After 6 days in culture, the cells were fixed and processed (*23*) for immunoperoxidase localization of glucocorticoid receptor (*16*), with a monoclonal antibody to the human receptor (*30*). When either one of the two polyclonal antibodies (*31, 32*) or a monoclonal antibody (*33*) was used to localize the receptor, similar results were obtained. (B) To demonstrate that the cells that stain with the receptor antibodies are indeed IL cells, we used a double-labeling procedure (*23*). The cells previously stained by immunoperoxidase as in (A) were further incubated with rabbit antibody to α-MSH (*27*). The bound antibodies were revealed by antibodies to rabbit IgG labeled with fluorescein (Boehringer Mannheim). As observed in a fluorescence microscope, the cells shown in (B) also stain with antibody to α-MSH. (C) Immunocytochemical control in which the cells were incubated with the antibody to the receptor previously absorbed overnight with 0.1 μg of purified receptor (*32*) per milligram of IgG. No staining is observed.

neural mechanisms, the molecular bases of which are unknown. We therefore questioned whether neural mechanisms might regulate the expression of IL glucocorticoid receptors, in which case denervation of IL might facilitate the detection of glucocorticoid receptors.

We used three models of denervated IL cells, namely (i) grafting of IL tissue into a site remote from the pituitary (under the kidney capsule) (*21*), (ii) stalk transection, and (iii) culture of the IL cells in vitro (*22, 23*). Both of the surgical procedures were associated not only with viability of IL tissue but with evidence of increased or maintained growth (*24*). At 12 days after stalk section, no change was seen in pituitary protein concentration (*25*). Other investigators, using grafted NIL, have obtained histological evidence of increased secretory activity (*25*) and enhanced secretion, as measured by the appearance of bioassayable MSH-like activity in plasma (*23, 25*).

The glucocorticoid receptor in grafted IL cells was detected as early as 1 week after surgery and was present in grafted IL cells 2 and 4 weeks after surgery, the early time point being the first such data point obtained (Fig. 1B). In all instances in which glucocorticoid receptor was present in the grafted IL ($n = 6$), the intact IL of the same animal was devoid of immunoreactive glucocorticoid receptor (Fig. 1A), in agreement with our previous observations (*16*). In all instances, abundant glucocorticoid receptor was present in the AL [for staining of AL cells, see (*15*)]. To confirm the nature of the grafted tissue, we stained serial sections with an antibody to α-MSH (*26*). The grafted tissue showed staining typical of the IL.

In the second set of experiments, we studied the localization of glucocorticoid receptor in stalk-sectioned animals. In all instances, glucocorticoid receptor was detected in IL obtained 2 weeks after the animals ($n = 6$) were killed, the only time point studied (Fig. 2). Earlier studies showed that serotonin-immunoreactive fibers almost disappeared from IL 7 days after pituitary stalk transection (*24*). Serotonin concentrations decreased by 44 percent and dopamine levels decreased by 77 percent in the IL of the stalk-transected animals. These data suggested that removal of the IL from its hypothalamic neural connections induced the expression of the glucocorticoid receptor, which was previously repressed. To validate our in vivo observations, we used in vitro experiments in which we examined NIL cells after

monolayer culture (*21, 22*). Glucocorticoid receptor was detected in cells that were immunoreactive for α-MSH (Fig. 3), an indication that the immunoreactive cells were IL POMC–containing cells. The presence of glucocorticoid receptor was evident in cells after 3 days in culture, the earliest time point examined, and could still be detected after 12 days in culture.

In all of these experiments, rigorous immunocytochemical control experiments, similar to those described in Fig. 3C, were performed to determine the specificity of the immunocytochemical staining. In addition, we used two different monoclonal and two different polyclonal antibodies to glucocorticoid receptor in all of these studies and obtained similar results with all of them. This strongly indicates that the antigen detected in the IL was indeed the glucocorticoid receptor. Furthermore, in parallel experiments (*28*), we used Western blot analysis (*29*) to show that the immunoreactive glucocorticoid receptor found in the NIL cultures had a molecular weight identical to that of the receptor in AL and liver. In other parallel studies, we found that incubation of primary NIL cultures with 100 n*M* dexamethasone for 30 minutes resulted in a significant attenuation (30 to 50 percent) of corticotropin-releasing factor (CRF)–stimulated secretion (10 n*M* CRF for 30 minutes) of immunoreactive β-endorphin and α-MSH from primary NIL cultures (4 days). In contrast, less than 10 percent inhibition occurred in 1-day NIL cultures. These data demonstrate that the induced receptor is functional.

We have shown that glucocorticoid receptor expression can be induced in a normally nonexpressing tissue. This finding may provide a working hypothesis for the study of the molecular basis of glucocorticoid resistance in normal and neoplastic tissues.

References and Notes

1. R. E. Mains *et al.*, *Proc. Natl. Acad. Sci. U.S.A.* **74**, 3014 (1977); J. L. Roberts and E. Herbert, *ibid.*, p. 5300; E. Herbert, *Trends Biochem. Sci.* **6**, 184 (1981).
2. D. T. Krieger *et al.*, *Recent Prog. Horm. Res.* **36**, 272 (1980).
3. F. E. Yates and J. W. Maran in *Handbook of Physiology* (American Physiology Society, Washington, D.C., 1974), vol. 4, part 2, sect. 7, p. 367; W. Vale *et al.*, *Science* **213**, 1394 (1981); F. Berbosch *et al.*, *Life Sci.* **29**, 2449 (1981); M. Munemura *et al.*, *Endocrinology* **106**, 1795 (1980); I. Vermes *et al.*, *Life Sci.* **27**, 1761 (1980); J. M. Farah, Jr., *et al.*, *Endocrinology* **110**, 657 (1982); J. Lepine and A. Dupont, *Endocr. Soc. Abstr.* **108**, 385 (1981); J. Axelrod and T. D. Reisine, *Science* **224**, 452 (1984); D. T. Krieger, *Clin. Res.* **31**, 342 (1983); C. L.-C. Chen *et al.*, *Proc. Natl. Acad. Sci. U.S.A.* **80**, 2211 (1983); V. Hollt and I. Haarmann, *Biochem. Biophys. Res. Commun.* **124**, 407 (1984).
4. K. N. Westlund and G. V. Childs, *Endocrinology* **111**, 1761 (1982).
5. C. Leranth *et al.*, *Neuroscience* **2**, 289 (1983).
6. R. D. Burt and R. L. Taylor, *Neuroendocrinology* **30**, 344 (1980).
7. G. M. Brown *et al.*, *Endocrinology* **99**, 1407 (1976).
8. L. Beaulac-Baillargeon *et al.*, *Soc. Neurosci. Abstr.* **6**, 28 (1980); T. D. Reisine *et al.*, *J. Neurosci.* **3**, 725 (1983); H. R. Furchgott, in *Catecholamines*, H. Blaschko and E. Muscholl, Eds. (Springer-Verlag, Berlin, 1972), p. 282.
9. M. A. Johns *et al.*, *Endocrinology* **110**, 754 (1982); E. A. Nunez *et al.*, *J. Histochem. Cytochem.* **29**, 1336 (1981).
10. A. Biegon and B. S. McEwen, *J. Neurosci.* **2**, 199 (1982); D. A. Kendall *et al.*, *Science* **211**, 1183 (1981).
11. H. G. Baumgarter *et al.*, *Z. Zellforsch. Mikrosk. Anat.* **126**, 483 (1972); F. J. H. Tilders and P. G. Smelik, in *Frontiers of Hormone Research*, F. J. H. Tilders, D. Swaab, T. J. Van Wimersma Greidanus (Karger, Basel, 1977), p. 80.
12. S. Nakanishi *et al.*, *Proc. Natl. Acad. Sci. U.S.A.* **74**, 3283 (1977); J.-P. Cragner and J. Drouin, *Mol. Cell. Endocrinol.* **40**, 25 (1985).
13. B. S. Schachter *et al.*, *Endocrinology* **110**, 1442 (1982).
14. E. Herbert *et al.*, *Neurosci. Lett.* **12**, 16 (1981); J. H. Eberwine and J. L. Roberts, *J. Biol. Chem.* **259**, 2166 (1984).
15. J. D. Baxter and G. G. Rousseau, Eds., *Glucocorticoid Hormone Action* (Springer-Verlag, New York, 1979).
16. T. Antakly and H. J. Eisen, *J. Cell Biol.* **95**, 437a (1982); paper presented at the annual meeting of the Endocrine Society, 1983 (abstract 592); *Endocrinology* **115**, 1984 (1984).
17. H. D. Rees *et al.*, *Cell Tissue Res.* **182**, 347 (1977).
18. C. D. Bloomfield *et al.*, in *Hormones and Cancer*, S. Iacobelli *et al.*, Eds. (Raven, New York, 1980), p. 345; C. D. Bloomfield *et al.*, *J. Steroid Biochem.* **15**, 275 (1981); C. D. Bloomfield *et al.*, *Cancer Res.* **41**, 4857 (1980); E. B. Thompson and J. M. Harmon, in *Hormones and Cancer*, S. Iacobelli *et al.*, Eds. (Raven, New York, 1980), p. 89.
19. V. Hollt *et al.*, *Endocrinology* **110**, 1885 (1982); C. L.-C. Chen *et al.*, in (*3*); H. Meunier and F. Labrie, *Life Sci.* **30**, 963 (1982).
20. J. Richter and J. Stepien, *J. Endocrinol.* **75**, 443 (1977).
21. Ten adult male inbred rats (CPF, Fisher 334/CRLB BR) (125 to 150 g) received implants of two NIL from similar donor animals. NIL rather than IL alone was used, in view of the difficulty of accurately dissecting IL from NIL. The animals ($n = 6$) were maintained in individual cages with free access to food and water, and with lights on from 0700 to 2100. Six other animals had sham operations. The animals were killed 27 days after placement of grafts.
22. W. Vale and J. Rivier, in *Central Nervous System Effect of Hypothalamic Hormones and Other Peptides*, R. Collu *et al.*, Eds. (Raven, New York, 1979), p. 163; T. Antakly *et al.*, *Proc. Can. Fed. Biol. Soc.* (1979), p. 36.
23. T. Antakly *et al.*, *J. Cell Biol.* **86**, 377 (1980).
24. J. W. Kendall *et al.*, *Endocrinology* **78**, 533 (1966); E. M. Daniel and D. M. N. Pritchard, *Acta Endocrinol. Copenhagen* **201** (Suppl.), 1 (1975).
25. E. Friedman *et al.*, *Endocrinology* **112**, 1943 (1983).
26. S. C. Iturriza *et al.*, *Neuroendocrinology* **22**, 175 (1976).
27. H. Vaudry *et al.*, *ibid.* **27**, 9 (1978).
28. T. Antakly *et al.*, in preparation.
29. H. Towbin *et al.*, *Proc. Natl. Acad. Sci. U.S.A.* **76**, 4530, 1979.
30. J. A. Cidlowski, in preparation; the partially purified receptor is described earlier [J. A. Cidlowsky and V. Richon, *Endocrinology* **115**, 1588 (1984)].
31. J. M. Harmon *et al.*, paper presented at the annual meeting of the Endocrine Society, 1983 (abstract 456); J. M. Harmon *et al.*, *Cancer Res.* **44**, 4540 (1984).
32. H. J. Eisen, *Proc. Natl. Acad. Sci. U.S.A.* **77**, 3893 (1980); _____ *et al.*, *J. Biol. Chem.* **254**, 10378 (1981); H. J. Eisen, *Biochem. Actions Horm.* **9**, 255, (1982).
33. B. Gametchu and R. W. Harrison, *Endocrinology* **114**, 274 (1984).

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Auditory Responses in Avian Vocal Motor Neurons: A Motor Theory for Song Perception in Birds

Abstract. *The hypoglossal motor neurons that innervate the vocal organ (syrinx) of the male zebra finch show a selective, long-latency (50-millisecond) response to sound. This response is eliminated by lesions to forebrain song-control nuclei. Different song syllables elicit a response from different syringeal motor neurons. Conspecific vocalizations may therefore be perceived as members of a set of vocal gestures and thus distinct from other environmental sounds. This hypothesis is an avian parallel to the motor theory of speech perception in humans.*

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Acoustically different components of human speech are at times perceived as a single phoneme. What these components have in common is the articulatory gesture that produces them. This view, known as the motor theory of speech perception, has been advanced to account for the phonetic decoding of speech (*1*). In this context, speech is perceived not just as a sound but as a series of articulatory gestures (*1, 2*). We now report observations on songbirds that suggest that they too may decode conspecific sounds by reference to the vocal gestures used to produce them.

Recordings from the tracheosyringeal (ts) branch of the hypoglossal nerve (NXIIts) of anesthetized adult male zebra finches have shown that units in this nerve and in the XIIts motor nucleus (nXIIts) respond to pure tones (Fig. 1, A and B) (*3*). The latency of this auditory response is 45 to 60 msec (*4*), an order of magnitude greater than the latency of a similar auditory response in the laryngeal motor nucleus of the bat (*5*). This