tin subunit, thereby increasing the distance between successive synemin binding sites.

The transient expression of the neurofilament polypeptide in erythrocytes is unlike the usual program of intermediate filament expression. Typically, in neurons, astrocytes, and muscle cells, vimentin is expressed initially but is largely or completely replaced during differentiation by NF70, glial filament protein, or desmin, respectively (*16, 17, 20*), except for some fetal ependymal cells in which glial filament protein is only transiently expressed (*21*). However, these cases are not strictly comparable to that of erythrocytes, since the neural, glial, and muscle changes involve the differentiation of individual cells rather than populations. Our results suggest that individual erythroid cells do not modulate their ratios of vimentin to E70 during differentiation, but that this ratio changes as the cell population changes during development and growth of the organism, culminating in the virtual absence of E70 from most cells in the adult. This is supported by the observations that phenylhydrazine-induced anemia in adult chickens does not result in circulating erythroblasts with increased amounts of E70, and that bone marrow from anemic and normal adult chickens contains little erythroid E70, as assayed by immunofluorescence (data not shown). Precedents exist for this time course of expression of avian erythroid components: A hemoglobin variant (*22, 23*) and certain cell surface antigens (*24, 25*) are present in embryos but disappear within a few months of hatching; furthermore, induced anemia does not result in their reappearance (*23, 25*). The observed range in the amount of E70 present in different cells from a given individual (regardless of age) suggests that synthesis of E70 may be influenced by hemopoietic microenvironments or may be a function of stem cell heritage.

The role of E70 in erythrocytes has not been discerned; it may modulate the structure or function of the vimentin-synemin filament network in these cells, or merely be an ontogenic vestige of gene expression. In the context of current concepts of intermediate filament expression, the existence of the neurofilament polypeptide in a nonneuronal cell type is surprising. Accumulating evidence that intermediate filament expression cannot always be correlated with the histological classification or embryonic derivation of cells indicates that, despite the general utility and validity of such a scheme, until the apparent exceptions are more fully understood, intermediate filament polypeptides may not always be suitable markers or indicators of cell type or state of differentiation.

Many unanticipated similarities between the nervous and hemopoietic systems have been described, such as an abundance of hemopoietic stem cells in the brain (*26*), common cell surface antigens, and responsiveness to neurohormones (*27*). Although the underlying reasons for these links remain elusive, the major neurofilament subunit can now be recognized as part of this phenomenon.

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Potent Interaction Between Glucocorticoids and Growth Hormone–Releasing Factor in vivo

Abstract. *Administration of dexamethasone significantly enhanced the pituitary growth hormone response to growth hormone–releasing factor in intact as well as adrenalectomized rats. Thus the inhibitory effects of glucocorticosteroids on somatic growth which involve an interaction of these steroids and growth hormone at a peripheral level may also involve a modification of pathways within the central nervous system that regulate normal growth hormone secretion.*

One of the most overt features of long-term treatment with glucocorticosteroids is the resultant inhibition of somatic growth in both man (*1*) and laboratory animals (*2*). Clinically, glucocorticoids suppress the pituitary growth hormone (GH) response to various stimuli (*3*). Yet these steroids increase the synthesis and content of GH in dispersed pituitary cells in vitro as well as sensitize the response of the somatotrophs to release GH after stimulation (*4*). As a result of the recent isolation and characterization of a growth hormone–releasing factor from a human pancreas tumor (hpGRF-44) which had caused acromegaly (*5*), we have been able to study this apparent dichotomy in the action of glucocorticoids on GH secretion. Rats subjected to adrenalectomy or sham operations were given long-term treatment with either saline or the synthetic glucocorticoid dexamethasone, and were then injected with two doses of hpGRF-44. Adrenalectomy, without steroid replacement therapy, significantly decreased the pituitary response to a submaximal dose of hpGRF-44, and the increase in plasma GH concentrations following the intravenous administration of hpGRF-44 was significantly greater in rats treated with dexamethasone. These results show that glucocorticosteroids enhance the GH response of the pituitary to hpGRF-44 in

vivo, and suggest that some of the catabolic effects of these steroids observed in vivo, although due in part to their direct action on peripheral tissues (*6*), may also be due to their capacity to modify the secretory pathway of GH in the central nervous system.

Male Sprague-Dawley rats weighing 250 g were housed in a temperature-controlled (19° to 22°C) and humidity-controlled vivarium and exposed to a schedule of 14 hours of light and 10 hours of darkness (lights on at 0600 hours). Food and water or 0.9 percent saline, where appropriate, were always available. The animals received sham operations or were bilaterally adrenalectomized under sodium pentobarbital anesthesia (50 mg/kg, injected intraperitoneally). After the surgery, six rats with sham operations and six adrenalectomized rats were injected intraperitoneally with 40 μg of dexamethasone daily; an equal number of animals received saline. Seven days later, all animals were anesthetized with an intraperitoneal injection of sodium pentobarbital (60 mg/kg) and fitted with a catheter placed in the superior vena cava via an external jugular vein. An initial blood sample (0.15 ml) was drawn 15 minutes after pentobarbital administration and was immediately followed by the intravenous administration of hpGRF-44 (500 ng/kg) with subsequent blood sampling for the measurement of GH (*7*). The injection of dexamethasone or saline was continued for seven more days, after which time the animals were again injected intravenously with hpGRF-44 (25 μg/kg). The 500 ng/kg dose of hpGRF-44 was chosen to evaluate whether glucocorticoids alter pituitary sensitivity because this dose elicits a submaximal response (*5, 8*). The 25 μg/kg dose of hpGRF-44 was chosen to evaluate the readily releasable pool of GH since this dose is several times the concentration of hpGRF-44 required to elicit a maximal response (*5, 8*). Significant treatment effects were evaluated by analysis of variance (*9*).

Treatment of the sham-operated and adrenalectomized rats with dexamethasone significantly enhanced ($P < 0.05$) the pituitary GH response to hpGRF-44. The 500 ng/kg dose resulted in mean plasma GH concentrations of 987 ± 80 ng/ml in all rats receiving dexamethasone ($N = 12$) and 537 ± 48 ng/ml in all rats receiving saline ($N = 12$). After the 25 μg/kg dose, GH concentrations in the two groups were 4121 ± 352 and 2326 ± 272 ng/ml, respectively. This is consistent with previous reports showing that corticosteroid treatment increases pituitary GH content in vivo (*10*).

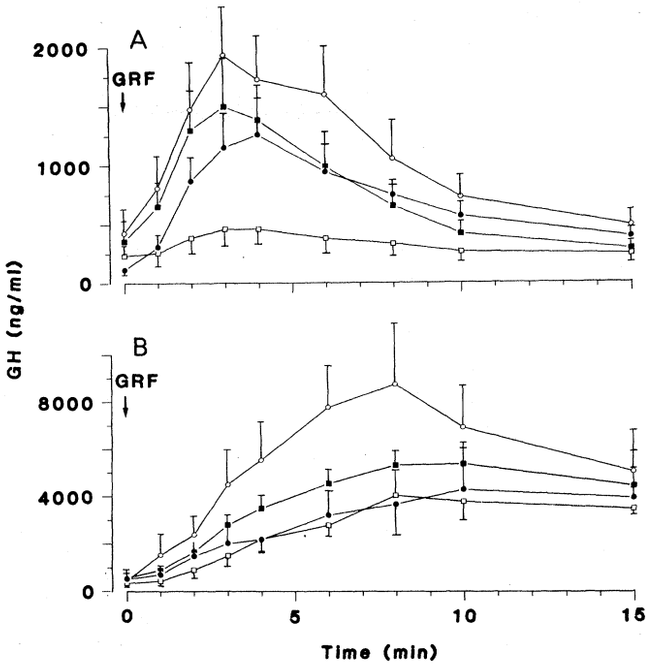

Fig. 1. Plasma GH concentrations in response to an intravenous injection of hpGRF-44 in male rats that received sham operations or were adrenalectomized and received long-term replacement therapy of either saline or 40 μg of dexamethasone intraperitoneally. (A) The response to a 500 ng/kg bolus injection immediately after the blood sample was obtained at time 0. (B) The response to a 25-μg bolus injection immediately after the blood sample was obtained at time 0. Symbols: ●, sham-operated plus saline; ○, sham-operated plus dexamethasone; □, adrenalectomized plus saline; and ■, adrenalectomized plus dexamethasone.

As shown in Fig. 1A, the mean increase in plasma GH concentrations after the 500 ng/kg dose of hpGRF-44 was significantly less ($P < 0.05$) in adrenalectomized rats (336 ± 35 ng/ml, $N = 6$) than sham-operated animals (705 ± 76 ng/ml, $N = 6$). This decrease in response in adrenalectomized rats could be reversed by long-term administration of dexamethasone (837 ± 103 ng/ml, $N = 6$). In contrast, the mean increase in plasma GH concentrations after the 25 μg/kg dose of hpGRF-44 (Fig. 1B) was not different between adrenalectomized (2165 ± 318 ng/ml, $N = 6$) and sham-operated animals (2447 ± 418 ng/ml, $N = 6$) indicating that the readily releasable pool of GH is not altered by adrenalectomy. Thus, the potent effects of dexamethasone in restoring pituitary response to the 500 ng/kg dose of hpGRF in adrenalectomized rats appear to be due to an increased pituitary sensitivity, not to an altered releasable pool of GH.

The inhibition of somatic growth as a result of either endogenous or exogenous hypercorticism is well documented (*1, 2, 11*). That these steroids may be interfering with normal GH release is supported by numerous clinical studies (*3*) in which long-term glucocorticoid treatment suppresses the pituitary GH response to various stimuli which are known to increase GH through pathways that require participation of the central nervous system (*12*). Yet it remains unclear whether or not the administration of GH can restore normal growth during periods of hypercorticism (*13*). Until now the inhibition of growth in vivo has been difficult to reconcile with the observations that, in vitro, glucocorticoids increase GH gene transcription (*14*) as well as GH release. Our present observations demonstrate that these steroids enhance the pituitary GH response to growth hormone–releasing factor in vivo. These differences in the actions of glucocorticoids lead to the interesting hypothesis that adrenocortical steroids may be positive modulators of the GH response at the pituitary level but negative modulators of the same response within the central nervous system.

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Intraspecific Deception by Bluffing: A Defense Strategy of Newly Molted Stomatopods (Arthropoda: Crustacea)

Abstract. *After molting, stomatopods can be evicted easily from home cavities by conspecifics because these marine crustaceans lose temporarily their body armor and the use of their raptorial appendages. Some newly molted stomatopods defend their cavities with a meral spread display, a signal correlated with attack when used by animals between molts. The use of the meral spread display actually increases after molting. Since new molts cannot fight, their use of meral spread appears to be a bluff.*

Animal studies have shown many deceptions used between species (*1*) but few examples from intraspecfic interactions (*2, 3*). There is even a question of whether deception can be maintained in this context since it may be evolutionarily unstable (*2–4*). One form of deception is a bluff, where fighting ability or the tendency to persist in or escalate a contest is misrepresented. We report that newly molted individuals of the marine crustacean *Gonodactylus bredini* bluff conspecific opponents.

Gonodactylus bredini lives in the Caribbean and defends cavities in hard substrata. The second maxillipeds are enlarged to smash hard-shelled prey and, apparently in conjunction with the evolution of these weapons, *G. bredini* has evolved armor and a complex repertoire of agonistic displays that it uses during contests for cavities (*5*). Like other crustaceans, *G. bredini* molts to grow and repair the exoskeleton. After molting, the cuticle is soft for at least 3 days, providing little protection from predators or competitors (*6*), and the raptorial appendages are not effective for up to 4 days. Thus, molting periodically destroys a stomatopod's fighting ability or resource holding power (RHP) (*7*).

Table 1. Cavity defense tactics used by *G. bredini* residents during day 1 contests. After an intruder is detected, residents either flee or stay and attempt to retain the cavity. If they remain, residents hide deep inside the cavity and give no displays, or they actively defend the cavity by displaying to intruders.

Tactic	Won	Lost	Total
	New molts		
Flee		13	13
Hide	7	23	30
Display	9	8	17
	Controls		
Flee		1	1
Hide	2	3	5
Display	17	2	19

Because maintenance of a home cavity dominates the biology of gonodactylids, we examined how newly molted residents defend cavities and how aggressive behavior changes as RHP returns. Each new molt ($N = 60$) was placed into an arena 30 cm in diameter with a piece of coral rubble, where it established residency in a cavity that a stomatopod of similar size had occupied in the field. Less than 12 hours after the resident had molted (day 1), we introduced an intruder that was between molts (intermolt) into the arena and recorded the interaction until one of the contestants left the vicinity of the cavity. Trials against different intruders were staged on days 2, 3, 4, 5, 7, and 10. Intermolts ($N = 25$) were used as residents for the control series. All opponents were matched according to their size and sex. The data were pooled for males and females since we detected no differences in their behavior. On average, 74 percent of controls retained their cavities during a contest. New molts were less successful on days 1 through 5 (G-test; all $P < 0.05$) (*8*) but recovered RHP at least to premolt levels 7 to 10 days after ecdysis.

Some new molts were able to retain their cavities only hours after ecdysis by aggressively displaying to intruders (Table 1) (*9*). Residents that display typically use five agonistic acts: appear, lunge, meral spread, strike-cavity, and strike-opponent (Fig. 1). Controls tended to attack and used strike-opponent most frequently. New molts, which could not strike, used meral spread in 15 out of 17 contests while controls used it in only 4 of 19 contests (G-test, $P < 0.001$) (*10*). These new molts apparently were attempting to defend their cavities by bluffing.

To present a meral spread, *G. bredini* and other gonodactylids lean out of their cavity and while facing an opponent, raise and laterally spread the raptorial appendages. At times, the magnitude of the spread is increased during an exchange. Meral spread by intermolts has been described as a conventional threat display and linked statistically to escalation by the signaler and to inhibition of attack in opponents (*5*). Meral spread provides information about size that intruders could use to assess fighting ability. The data on subsequent behavior and the sometimes graded nature of the display indicate that meral spread also may signal motivation. Regardless of whether meral spread signals the tendency to