8. Fruit color was matched against the *p*H reference color scale on Riedel–De Haën (AG) wide range *p*H indicator paper, *p*H 0.5 to 5.5 and *p*H 5.5 to 9.0. Colors not on the *p*H reference scale were compared to available constant reference objects (for example, a red Victorinox Swiss Army knife). Dark shades of purple or blue were classified as black. Mixed-color fruits have two or more colors present in the ripe fruit, including the support structures.
9. All statistical methods follow S. Siegel [*Nonparametric Statistics for the Behavioral Sciences* (McGraw-Hill, New York, 1956)], except where noted otherwise. All chi-square tests used Yates correction for continuity [R. R. Sokal and F. J. Rohlf, *Biometry* (Freeman, San Francisco, 1969), p. 590].
10. Chi-square test for heterogeneity, $\chi^2(8) = 34.48$, $P < .001$. For the categories of color and morphology in Fig. 1, 132 genera had no variation among species.
11. Groups of color categories were defined by a test for homogeneity of subsets [see R. R. Sokal and F. J. Rohlf, *Biometry* (Freeman, San Francisco, 1969), pp. 575–585]. I used the .05 probability level for evaluating statistically significant heterogeneity among subsets of the color categories, which were ranked by percentage of protected fruits.
12. The affinity of blue and purple with type A is supported by the high percentage of unprotected fruits of these colors, by the grading of these colors into black (*8*), and by the fact that four of five occurrences of these colors in the mixed-color category were combinations with type A colors.
13. Because of the small number of variable genera, colors are combined into type A or type B. For the resulting four categories of color and protectedness, 15 variable genera have 53 fruit forms. In the 11 genera in which only color varies, nine of ten protected forms fall into type B colors, whereas only 11 of 30 unprotected forms are type B colors [$\chi^2(1) = 6.53$, $P < .01$]. For four genera in which both color and construction vary, five of seven protected forms belong to type B colors and none of six unprotected forms are type B colors [$\chi^2(1) = 4.28$, $P < .05$]. No genera show variation only in construction.
14. Kolmogorov-Smirnov two-sample test, $D = .651$, $N = 258$, $P < .001$. Species are considered independent samples because even otherwise morphologically constant genera show appreciable variation in size among species.
15. Most fruit-eating birds in Peru weigh less than 200 g, whereas the major mammalian fruit-eaters, monkeys, weigh from 400 to 8000 g. Weights are from live measurements of birds and tamarins in the study site (J. Terborgh, personal communication) or from J. F. Eisenberg [*The Mammalian Radiations* (Univ. of Chicago Press, Chicago, 1981), p. 468].
16. Mammals usually have poor color vision, whereas birds have high sensitivity over the visible spectrum [G. L. Walls, *The Vertebrate Eye and Its Adaptive Radiation* (Hafner, New York, 1963)]. Neotropical monkeys see well in the green-yellow-orange range but have low sensitivity to and ability to discriminate among reds [R. L. De Valois and G. H. Jacobs, *Science* **162**, 533 (1968); D. M. Snodderly, in *The Behavioral Significance of Color*, E. H. Burtt, Ed. (Garland, New York, 1979), pp. 237–279].
17. Mammals have bony jaws, complex teeth, and manipulative tongues that aid in processing fruits (C. Janson, personal observation). Most fruit-eating birds have little ability to manipulate fruits with precision; the major exception, parrots, are mostly seed predators [D. H. Janzen, *Auk* **98**, 841 (1981)].
18. Chi-square one-sample test, $\chi^2(1) = 10.98$, $P < .001$. Birds were observed to eat fruits opportunistically during other studies (C. Janson, personal observation). Although not a complete description of the diets of fruit-eating birds, these observations should not be biased toward small unprotected fruits.
19. Chi-square one-sample test, $\chi^2(1) = 5.91$, $P < .02$. Fruit-eating by monkeys was recorded from systematic study of seven species in the area.
20. It is likely that fruit species not included in the two major classes show morphological adaptation to other dispersers. In particular, a number of species known to be eaten by bats are included in the class of large unprotected type B–colored fruits (Table 1).
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22. R. R. Sokal and F. J. Rohlf, *Biometry* (Freeman, San Francisco, 1969), pp. 601–607. The *G* statistic to test joint independence of all three characters is 118.1, allowing rejection of independence ($P < .001$ with 4 degrees of freedom, $N = 172$). The *G* statistics to test independence of color and protection, color and size, and size and protection are 49.4, 61.2, and 29.3, respectively. All allow rejection of independence ($P < .001$ with 1 degree of freedom, $N = 172$).
23. I thank R. Foster for invaluable instruction in plant identification at the study site and the Peruvian Ministry of Agriculture, Forestry Institute and ORDEMAD for support and permission to work in the Manu National Park. Several researchers contributed plant specimens and feeding observations: P. C. Wright, A. Wilson, C. Munn, S. Robinson, B. Mitchell, P. Daniels, G. Stuart, and especially R. Foster. For stimulating discussions, I am grateful to R. Foster, J. Terborgh, G. Orians, N. Wheelwright, P. Harvey, and M. Slatkin. Supported by NSF grant BNS8007381 and Sigma Xi grant in aid of research.

26 April 1982; revised 3 August 1982

Immunoreactive Dynorphin-(1–8) and Corticotropin-Releasing Factor in Subpopulation of Hypothalamic Neurons

Abstract. *Immunoreactive corticotropin-releasing factor (CRF) and dynorphin-(1–8) were visualized in rat hypothalamus by immunohistofluorescence with specific antibodies. In brains from colchicine-treated, adrenalectomized rats, neuronal perikarya with immunoreactive CRF were observed in the paraventricular nucleus of the hypothalamus. The CRF occurred together with the dynorphin-(1–8). However, the CRF immunoreactivity occurred only in a subpopulation of the dynorphin-(1–8) immunoreactive cells. These findings suggest that there may be a functional interrelationship of CRF with dynorphin-related opioid peptides and provide further evidence that neurons may contain more than one bioactive substance.*

Corticotropin-releasing factor (CRF), which consists of 41 amino acid residues, was isolated from ovine hypothalamus extracts (*1*). The peptide is a potent stimulator of adrenocorticotropic hormone (ACTH) release in vivo (*2*) and of ACTH-like and β-endorphin-like immunoreactivities in cultured pituitary cells (*1*). It has been reported that CRF originates in perikarya of the paraventricular nucleus of the hypothalamus (*3*). The paraventricular nucleus is one of the main synthesis and storage sites of vasopressin in mammalian hypothalamus (*4*). The same neurons in the paraventricular nucleus which manufacture vasopressin also contain dynorphin-(1–17) and α-Neo-endorphin, two leucine-enkephalin–related opioid peptides (*5–7*). These two opiate active substances also occur together in brain areas other than hypothalamus (*8*). We have recently demonstrated that one of the major peptide products of this α-Neo-endorphin/dynorphin neuronal system is dynorphin-(1–8) (*9*), an amino-terminal fragment of dynorphin-(1–17), which is present in much higher concentrations in brain than dynorphin-(1–17). In subsequent immunohistochemical studies, very intense dynorphin-(1–8)-like staining occurred in the same neurons that were previously demonstrated to contain α-Neo-endorphin, dynorphin-(1–17), and vasopressin immunoreactive material (*10*).

In the studies described here we investigated whether the CRF-containing neurons in the paraventricular nucleus are related to those containing the opioid peptides. To examine this question we developed a specific antiserum to CRF and compared the immunostaining produced by this antiserum with the distribution of dynorphin-(1–8) immunoreactive material. The studies were carried out on brains from colchicine-treated, adrenalectomized rats, and we found that CRF immunoreactivity is present in a subpopulation of the dynorphin-(1–8) immunoreactive neurons in the paraventricular nucleus.

The antiserum to CRF was raised in rabbits against the synthetic peptide (*11*). In a radioimmunoassay (RIA) (*12*) this antiserum bound 30 percent of a trace amount of [^{125}I-Tyr°]CRF at a dilution of 1 in 100,000. A 50 percent inhibition of this binding of [^{125}I-Tyr°]CRF to the CRF antibodies occurred by addition of a 400 p*M* concentration of authentic CRF. No inhibition of the labeled CRF occurred when 1 μ*M* sauvagine, α-Neo-endorphin, dynorphin-(1–8), dynorphin-(1–17), vasopressin, or oxytocin were added. The RIA specificity of the dynorphin-(1–8) antiserum was established in studies previously described (*9*).

Since RIA's are performed with a much higher antiserum dilution than immunohistochemistry, other populations of antibodies may be active in the latter method, therefore the antiserum specificity data obtained with the RIA are only of limited value in interpreting immunohistochemical results obtained with these same antiserums. Therefore, we also subjected the two antiserums used to immunohistochemical blocking controls, which were performed by adding an excess of various synthetic peptides

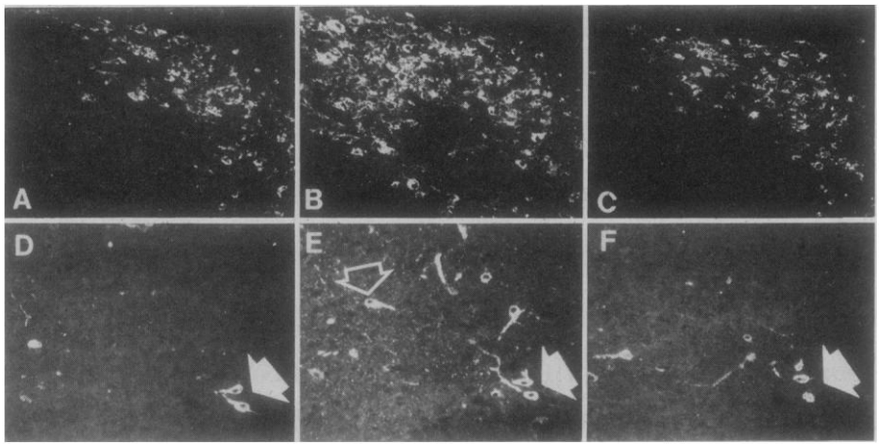

Fig. 1. Photomicrographs of CRF and dynorphin-(1–8) immunoreactive neuronal elements in rat brain. (A to C) One series, and (D to F) another series of consecutive sections (2.5 μm) through the paraventricular nucleus of a colchicine-treated, adrenalectomized rat. (A and C) Sections incubated with antiserum to CRF. (B) Section incubated with antiserum to dynorphin-(1–8). Immunoreactive CRF appears to be in a subpopulation of the immunoreactive dynorphin-(1–8) cells in the paraventricular nucleus. (D and F) Sections incubated with antiserum to CRF. (E) Section incubated with antiserum to dynorphin-(1–8). The solid arrow indicates two cells that can be visualized in all three sections. The open arrow indicates a cell that was stained by antiserum to dynorphin-(1–8) but not by antiserum to CRF.

to the antiserum immediately prior to incubation on the tissue slice. Using these two criteria for specificity we were able to determine that the two antiserums were specific for their respective antigens. Thus, the specific immunofluorescence produced by each antiserum was blocked by excess concentration of its respective antigen but not by excess concentration of various other synthetic peptides whether structurally related or not (Table 1).

To examine the possibility that CRF immunoreactivity occurred together with dynorphin-(1–8) immunoreactivity in hypothalamic neurons, we used paraformaldehyde-fixed brains from animals that had been adrenalectomized 7 days previously and treated with colchicine (*13*). Colchicine inhibits the axonal flow of secretory vesicles, thus producing a buildup of intraneuronally transported substances within the perikarya. This method allows visualization of neuronal perikarya that are often not seen in normal animals (*14*). Serial sections (2.5 μm) were stained alternately with the two antiserums so that identical cells in immediately adjacent sections would be stained by different antiserums (*15*).

We found that CRF immunoreactivity occurred in a subpopulation of dynorphin-(1–8) immunoreactive cells (Fig. 1, A to F) that did not include the supraoptic nucleus, even though this nucleus contains extensive systems of dynorphin-(1–8) neurons (*10*).

In the brains of adrenalectomized rats, immunoreactive neurophysin/vasopressin and CRF occur together both in terminals of the zona externa of the median eminence (*16*) and in perikarya of the paraventricular nucleus (*17*). Thus it appears that at least three separate peptide systems of apparently distinct function—opioid, CRF, and vasopressin—coexist in a set of neurons that originate in the paraventricular nucleus.

The coexistence of CRF and vasopressin is of interest in view of previous observations of a CRF-like activity of vasopressin on ACTH release from anterior pituitary cells (*18*). A synergistic action of a number of hormonal peptides that occur in the same cells has recently been demonstrated (*19*). That CRF occurs in the same cells as opioid active peptides is important since it has been known for many years that CRF activity in the hypothalamus is strongly influenced by opioid substances (*20*). For example, morphine stimulates hypothalamo-pituitary-adrenocorticotropic activity apparently by acting on hypothalamic opioid receptors. It is possible that the ACTH-releasing activity of CRF is potentiated by a concerted action of vasopressin and dynorphin-related peptides acting at hypothalamic or anterior pituitary receptor sites with all three substances functioning as neuroregulators (*21*) and being released simultaneously by the same nerve terminals. Moreover, our finding that immunoreactive CRF and dynorphin-(1–8) coexist in some cells of the paraventricular nucleus suggests that at least one of the several opioid peptide systems that have now been demonstrated in brain (*22*) may be under partial influence of the pituitary-adrenal axis. This possibility is important in view of recent demonstrations that a form of stress-induced analgesia which is opioid-mediated is controlled by corticosteroids (*23*) from the adrenal cortex.

Thus the findings reported here open the way for further studies on the interaction of leucine-enkephalin–related opioid peptide systems with the neuroendocrine system that mediates many responses to stressful stimuli.

Table 1. Immunohistochemical specificity of CRF and dynorphin-(1–8)-like immunostaining in hypothalamic neurons. Tissue sections were incubated with antiserum diluted 1 in 400 in the presence of a 5 to 50 μ*M* concentration of the synthetic peptides. They were then washed and incubated with FITC-labeled sheep antiserum to rabbit immunoglobulin G conjugated to fluorescein isothiocyanate. Plus signs indicate that immunofluorescence was blocked by a 5 μ*M* concentration of the peptide; minus signs indicate that the immunostaining was not blocked by a 50 μ*M* concentration of the peptide; NT, not tested.

Peptides tested in immunohistochemical blocking controls	Antiserum to	
	CRF (code R3-3)	Dynorphin-(1–8) (code R2-3)
CRF	+	–
Dynorphin-(1–8)	–	+
Leu-enkephalin	–	–
Dynorphin-(1–6)	NT	–
Dynorphin-(1–7)	NT	–
α-Neo-endorphin	–	–
Vasopressin	–	–
Oxytocin	–	–
Sauvagine	–	–

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13. Male Sprague-Dawley rats (180 to 300 g) were adrenalectomized 7 days before the experiments and treated with colchicine (50 μg in 25 μl of water, injected intraventricularly) 48 hours before they were killed. Their brains were fixed and processed for immunohistochemistry as described [E. Weber *et al.* (*8*)]. The CRF (R3-3) and dynorphin-(1–8) (R2-3) antiserums were used at a dilution of 1 in 400.
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17 May 1982; revised 20 August 1982

Red/Green Color Opponency at Detection Threshold

Abstract. *By means of visual stimulus without temporal or spatial edges, we have achieved better isolation of chromatic signals at detection threshold than has been reported previously. Under various states of adaptation, the spectral sensitivity of the chromatic mechanism detecting middle- and long-wavelength lights corresponds with that deduced from suprathreshold red/green hue equilibriums.*

The concept "color opponency" originated with Hering, who proposed that chromatic sensations occur along two independent, bipolar dimensions: red/green (R/G) and yellow/blue (Y/B) (*1*). Lights that cause neither sensation of an opponent pair are said to be in equilibrium with respect to that dimension. For example, equilibrium or unique yellow is the long-wave spectral light that appears neither reddish nor greenish. Hurvich and Jameson developed Hering's opponent process theory and applied it to a wide range of phenomena (*2*). Their hue cancellation procedure is the basis of quantitative opponent colors theory (*3, 4*). This procedure determines for each opponent system a subtractive coding of cone signals: the R/G (or Y/B) code assigns the value zero to a light if and only if that light is in R/G (or Y/B) equilibrium. Threshold experiments, which require no judgments of hue quality, have confirmed the existence of subtractive interactions between cone signals (*5–7*), but have not of themselves implicated the particular coding scheme hypothesized by opponent colors theory.

Figure 1 shows a series of spectral sensitivity curves. Curves A through C represent sensitivity to a novel test stimulus, and curve D is previously published data obtained with a conventional stimulus (*5*). The unique features of the new stimulus were its shape and time course: its temporal wave form was one period (trough-to-trough) of a 2-Hz cosine wave; its spatial profile was a radially symmetric Gaussian with a full bandwidth of 3°. This test will be dubbed the "low-frequency" test because its temporal and spatial Fourier spectra show that most of its energy is concentrated at low frequencies. Previous evidence indicated that chromatic visual channels are relatively more sensitive at low spatial and temporal frequencies, whereas achromatic or luminance channels are most sensitive at higher frequencies (*8*). The three bottom curves are estimates (*9*) of the spectral sensitivities of the three human cone pigments with peak sensitivities near 440, 540, and 570 nm (here labeled P_{440}, P_{540}, and P_{570}).

A notable feature of all the threshold spectral sensitivities is their peaks and troughs. Below 500 nm is a peak that can be attributed to P_{440} alone. Above 500 nm there are two peaks [or one shoulder and one peak (*10*)] whose location and bandwidth clearly do not correspond to those of the underlying cone spectra. These narrowed peaks have been attributed to linear subtractive interaction between the cones containing P_{540} and the cones containing P_{570} (*5*). An important quantitative feature of the new curves A through C is the magnitude of the troughs (arrows). Using the low frequency stimulus produced troughs more than twice as deep as that in curve D, the deepest trough previously reported for normal human observers.

Figure 2 shows another way to quantify the sensitivity loss revealed by the troughs in Fig. 1. Here curves B and D of Fig. 1 are transformed and replotted as equivalent threshold mixtures of two "primaries" (*11*). In the Rayleigh region of the spectrum (wavelengths longer than 540 nm) the normal eye is dichromatic: each spectral light in that region can be matched exactly (in photons caught by the cones) by a unique mixture of two spectral lights (primaries), one chosen from each extreme of the region. Plotted in this way, the data directly demonstrate a subtractive interaction of the signals generated by the two primaries: more of one primary necessitates more of the other to attain threshold. The topmost point for the low-frequency test shows that 6 times the threshold amount of 650 nm added to 3.5 times the threshold amount of 540 nm is just barely at threshold. To our knowledge, the inhibitory interaction revealed by the low-frequency test is more extreme than any previously observed in experiments measuring threshold for bichromatic mixtures with a more conventional stimulus (*6, 7*). The slopes of the linear portions of the threshold contours give the relative sensitivity of the detecting mechanism to the two primaries. The parallelism of the two sides of the low-