servation of Schmid-Schoenbein *et al.* (*10*) that changes to a new level of shearing quickly established a new dimension for red cell aggregates. Each successive equilibration of aggregate size and number could explain the rapidly occurring stablity of echo intensity. With complete stoppage of blood flow (zero shear), red cell aggregation would become maximal. Under these circumstances, red cell aggregates produce a continuous network that is partly suspended by the walls of the blood container (*9*). This process may require more time than under conditions of blood flow, thus explaining the longer interval needed to produce maximum echogenicity following the onset of stasis.

In previous experiments with static liquid blood, we found that echogenicity of blood is a thixotropic phenomenon that disappears with mechanical agitation and develops seconds after the onset of stasis (*13*). We also demonstrated that the echogenicity of static blood is directly related to temperature and the concentrations of red cells and macromolecules (such as fibrinogen and gelatin) (*14*). Finally, we found that peripheral blood from myeloma patients—blood showing increased numbers of rouleaux in smears—is more intensely echogenic than normal blood (*14*). We conclude that red cell aggregation is an important cause of ultrasound echogenicity in both static liquid blood and flowing blood.

Red cell aggregation may thus explain the observed differences in ultrasound echogenicity in different vessels and in the same vessels. The variable echogenic zones in the inferior vena cava and portal vein may be due to differences in red cell aggregation. Factors such as hematocrit, plasma macromolecules, and shear rate may create differences in red cell aggregation in tributary veins that in turn produce the echogenic variability.

Blood echogenicity could be used as the basis for observing mixing of tributary blood in veins and for noninvasively determining red cell aggregation in vivo in blood vessels opaque to light. Present optical methods of observing aggregation are applicable only to microcirculation. Further study of the relation of echogenicity to red cell aggregation could result in the use of echogenicity or other properties of ultrasonography (such as attenuation of the transmitted sound beam) as a more quantitative measure of aggregation. Finally, these findings may have clinical applications. By controlling the degree of red cell aggregation (as by adjusting the concentration of macromolecules in the circulation), ultrasonic images of blood vessels and perfused organs may be enhanced. Blood could thus act as a "contrast medium" during ultrasound imaging in medical diagnosis.

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Yeast Mating Pheromone Activates Mammalian Gonadotrophs: Evolutionary Conservation of a Reproductive Hormone?

Abstract. *α-Factor, a tridecapeptide mating pheromone of yeast* (Saccharomyces cerevisiae)*, has extensive sequence homology with the hypothalamic decapeptide gonadotropin-releasing hormone (GnRH). Both synthetic and natural preparations of α-mating factor were found to bind specifically to rat pituitary GnRH receptors and to stimulate the release of luteinizing hormone from cultured gonadotrophs. The ability of the yeast pheromone to reproduce the biological actions of GnRH in the mammalian pituitary gland indicates that the structural and functional properties of GnRH-related peptides may have been highly conserved during evolution.*

Unicellular organisms and invertebrates produce a variety of molecules with structural or conformational similarities to the vertebrate hormones and neurotransmitters (*1, 2*). These include a yeast pheromone that resembles the central regulatory hormone for mammalian reproduction, the hypothalamic neuropeptide known as gonadotropin-releasing hormone (GnRH) or gonadoliberin. This decapeptide is secreted into the pituitary portal system for transport to the adenohypophysis (*3*), where it binds to plasma-membrane receptors on the gonadotrophs (*4*) and activates the calcium-dependent release (*5*) of glycoprotein hormones [luteinizing hormone (LH) and follicle-stimulating hormone (FSH)] which control the endocrine and reproductive functions of the testis and ovary. Immunoreactive GnRH is also present in the brain (*6*), placenta (*7*), and possibly the gonads (*8*) and milk (*9*) of mammals. Recently, extrapituitary receptors and actions of GnRH have been described in the rat ovary and testis (*10*) and in the human placenta (*11*).

In terms of phylogenetic distribution, mammalian GnRH-like peptides have been described in the hypothalamus and other regions of the nervous system of birds, reptiles, amphibia, and fish. Some of these peptides exhibit minor differences in their amino acid sequence from mammalian GnRH and behave as weak agonists for LH release in cultured rat pituitary cells (*6, 12*). In bullfrog sympathetic ganglia, the target neurons for an endogenous GnRH-like peptide recognize mammalian GnRH and its potent

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synthetic agonists. Such sites also discriminate between an agonist and an antagonist, indicating their conformational similarity to the mammalian GnRH receptor (*13*).

One of the phylogenetically oldest reproductive hormones to be chemically characterized is the yeast mating pheromone, α-factor. This tridecapeptide is secreted by *Saccharomyces cerevisiae* cells of the α-mating type, and acts on haploid cells of the opposite (a) mating type to cause arrest in the G_1 stage of the cell cycle and "gamete" formation prior to fusion with α cells to produce diploid zygotes (*14*). In α-factor, Trp-*His-Trp*-Leu-Gln-*Leu-Lys-Pro-Gly*-Gln-Pro-Met-Tyr, the nine *N*-terminal amino acid residues exhibit significant sequence homology (*italics*) with the mammalian GnRH molecule, pGlu-*His-Trp*-Ser-Tyr-Gly-*Leu-Arg-Pro-Gly*-NH_2, to the extent of about 80 percent (*15*). The existence of such structural and functional similarities among hormones and peptides from distantly related eukaryotes has been emphasized by Hunt and Dayhoff (*15*), who noted the remarkable degree of sequence homology between mammalian GnRH and the yeast α-factor.

To evaluate the biological significance of this structural similarity, we investigated the ability of the yeast α-factor to recognize and activate mammalian GnRH receptor sites. When synthetic α-factor from two sources and the purified natural pheromone (*16*) were analyzed by a radioactive ligand-receptor assay with rat pituitary cells, all three preparations caused dose-dependent inhibition of the specific binding of [^{125}I]GnRH agonist (Fig. 1A). The synthetic peptides were about 9000-fold less active than native GnRH (dissociation constant, K_d $4.5 \times 10^{-5} M$ compared to $5 \times 10^{-9} M$ for GnRH), and the natural α-factor was about twofold more potent. Thus, the yeast pheromone behaved as a low-affinity ligand for the rat pituitary GnRH receptor. In cultured rat pituitary cells, both the synthetic and natural preparations (*17*) of α-factor caused dose-dependent release of LH into the incubation medium (Fig. 1B). The dose-response curves for α-factor were similar to those obtained with hypothalamic GnRH but were shifted to the right by a factor of 10,000, in keeping with their receptor-binding activities. The effects of the synthetic and natural α-factors on gonadotropin release were prevented by the addition of a specific GnRH antagonist (*16*). High concentrations ($10^{-5} M$) of other small hormonal peptides, including angiotensin II and somatostatin, did not induce LH release.

The validity of the effect of α-factor on LH release from pituitary cells was established in several ways. In particular, the possibility that the activity of the synthetic peptide could arise from contamination with a small amount of GnRH agonist (*18*) was excluded by the similarity in the potencies of two synthetic peptides, from different suppliers, and the natural peptide. The absence of contamination by GnRH analogs used in our laboratory was ensured by the use of disposable materials and repetition of the assays with new preparations of peptide, and was indicated by the lack of activity of nonrelated peptides handled under the same conditions. In addition, the absence of LH release by angiotensin II and somatostatin, hormonal peptides that bind to specific receptors in other pituitary cell types, indicates that high concentrations of peptides regulating adjacent cells do not activate the gonadotroph receptors for GnRH.

The ability of α-factor to stimulate LH release from pituitary cells is dependent on binding of the yeast peptide to GnRH receptors. The mating peptide not only displaced the labeled GnRH agonist from its receptor sites but, as observed for other GnRH analogs (*19*), its binding affinity was proportional to its biological potency. The ability of a specific GnRH antagonist to abolish the releasing activity of yeast α-factor further validates the receptor-mediated nature of this effect. Taken together, these observations indicate that the sequence homology between GnRH and the yeast α-factor is not a coincidence. With the yeast α pheromone, GnRH and its natural analogs in reptiles, fish, and birds, appear to form a highly conserved line of peptides throughout evolution. Our data suggest that the receptor corresponding to the ligand is also preserved. Although little is known about the yeast receptor site for α-factor, both the mating pheromone and GnRH inhibit adenylate cyclase in yeast (*20*). It is interesting that GnRH agonists inhibit the production of adenosine 3′,5′-monophosphate in gonadal tissues (*21*).

It is intriguing that a pheromone responsible for mating and zygote formation in a unicellular organism is both structurally and functionally related to the peptide serving a key function in mammalian reproduction. Although several unicellular organisms contain or produce peptide or protein hormones similar to human chorionic gonadotropin, insu-

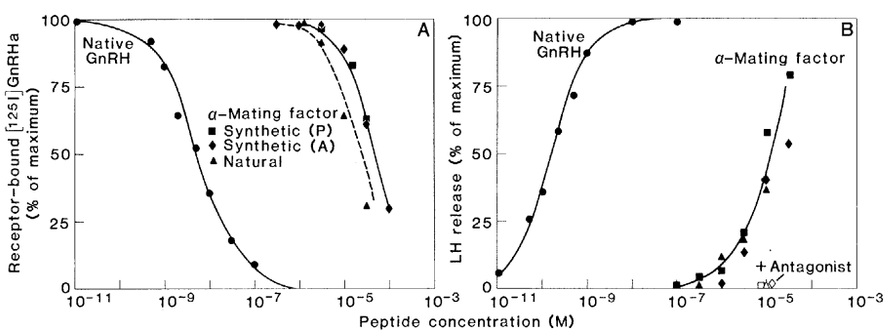

Fig. 1. (A) Inhibition of [^{125}I]GnRH agonist binding to rat pituitary particles by native mammalian GnRH (●), two preparations of synthetic α-factor (■, Peninsula Laboratories; ◆, Accurate Chemical), and purified natural α-factor (▲). The particulate pituitary preparation was a 12,000*g* fraction of rat pituitary homogenate from a female rat (*4*). The binding assay was performed at 0°C in 10 m*M* tris-HCl buffer containing 1 m*M* dithiothreitol and 0.1 percent bovine serum albumin (BSA), in a final volume of 0.3 ml. We used radioiodinated [D-Ala^6]des-Gly^{10}-GnRH *N*-ethylamide (30,000 count/min; ~ $3 \times 10^{-11} M$) as the labeled ligand. Incubations were terminated by dilution with 4 ml of ice-cold phosphate-buffered saline, *p*H 7.4, followed by immediate filtration under vacuum through glass-fiber filters presoaked in 2 percent BSA solution (GF/C Whatman Inc.). The filters were then washed three times with 4 ml of phosphate-buffered saline and the bound radioactivity was determined by γ-spectrometry. The maximum bound ligand was 7000 count/min in the absence of added peptide, and the nonspecific binding in the presence of 10^{-7} *M* GnRHa was 900 count/min. (B) Stimulation of LH release by mammalian GnRH (●), two preparations of synthetic α-factor (■, Peninsula Laboratories; ◆, Accurate Chemical), and natural α-factor (▲) (*16*). Cells were also incubated with GnRH or the α-factors in the presence of the potent GnRH antagonist [Ac-D-p-Cl-Phe^{1,2},D-Trp^3,D-Lys^6,D-Ala^{10}]GnRH (□, ◇, and △) at a final concentration of $10^{-7} M$. Pituitary cells were isolated by trypsin dispersion of glands from adult female rats (*22*). The isolated cells were cultured in multiwell plates (2×10^5 cells per 16-mm dish) for 3 days at 37°C under 5 percent CO_2 in air, in Medium 199 containing 1.4 g of sodium bicarbonate per liter, 10 percent horse serum, 100 U of penicillin, and 100 μg of streptomycin per milliliter. They were then incubated for 4 hours, with GnRH or α-factor in 0.5 ml of Medium 199 containing 0.25 percent BSA, and LH release was measured by radioimmunoassay of the medium in terms of the RP-1 rat LH standard provided by the National Pituitary Agency. The basal LH release was 80 ng/ml, and the maximum response to GnRH was 600 ng/ml.

lin, somatostatin, adrenocorticotropin, β-endorphin, arginine vasotocin, cholecystokinin, glucagon, and salmon calcitonin (*1*, *2*), none of these substances has been sequenced, and in most cases no function has been attributed to them. In the present case, the combination of sequence homology and analogous biological properties of the two peptides supports the interpretation that their similarities are not simply due to chance. Even so, it has yet to be established whether the structural and functional resemblances between the yeast pheromone and mammalian GnRH are indeed adequate to establish an evolutionary relationship between the two peptides. This reservation must be noted because the lengths of the peptides are not sufficient to permit the derivation of a compelling statistical analysis of their evolutionary relatedness. Nevertheless, GnRH and the yeast α-mating factor appear to represent a highly conserved effector system which includes the peptide ligand, the cell-surface receptor, and the physiological regulation of reproductive function.

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17. The binding-inhibition potency of the natural mating factor in the GnRH radioligand-receptor assay was similar to that of the synthetic preparations, but on two occasions the natural peptide showed extremely low bioactivity in cultured pituitary cells that responded well to GnRH. Since the methionine residue of α-factor is necessary for full biological activity and can undergo oxidation during or after preparation of the peptide (*14*), we also analyzed the activity of the natural peptide after reduction to obviate any such effect of oxidation. For this purpose, a portion of the peptide solution was incubated overnight at room temperature with an excess of mercaptoethanol at *p*H 8.5 and was then repeatedly lyophilized to remove the reducing agent. After this procedure, the reduced natural peptide stimulated the release of LH with a potency similar to that of the synthetic mating-factor preparations (Fig. 1B).
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Dual Task Interactions Due Exclusively to Limits in Processing Resources

Abstract. *Information presented to each hemisphere of the commissurotomy patient is available only to the stimulated hemisphere. Despite this, the hemispheres have access to a common pool of processing resources, which, under conditions of demanding bilateral stimulation, is distributed between the hemispheres.*

The concept of "limited resources" is central to most contemporary theories of human information processing; performance of a particular task is thought to depend on both the efficacy of processing strategies and the degree of "effort" or "resource" directed toward its solution (*1*). Empirical evidence that implies a fixed resource capacity is based primarily on demonstrations of performance decrements when two tasks are performed concurrently: in general, performance of each of two concurrently presented tasks is inferior to that achieved when each task is presented alone. In addition, when the difficulty of one task is varied, a reciprocal change in performance of the unchanged task is observed (*2*). Alternatively, concurrent task performance may reflect limits in processes that are peculiar to dual tasks, termed "emergent processes" by Duncan (*3*). Emergent processes have been demonstrated for a variety of stimulus and response measures and experimental contexts (*3*). For example, the physical attributes (for example, form and color) of simultaneously presented stimuli are to some degree perceptually interchangeable; that is, one stimulus can be erroneously perceived to have characteristics that in fact belong to the other. Under single-stimulus conditions, of course, errors of this kind could not occur. Thus, variations in the performance of concurrent tasks may be due to limits in the efficiency of perceptual segregation rather than resource limitations. We attempted to avoid the possibility of performance trade-offs due to perceptual

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