between plots than that predicted from the model (*8*). When the distances between plots were much larger as found in the western Amazon, the model again underestimated the similarity in species composition. Condit *et al.* conclude that dispersal is not the principal event that determines the diversity of tree species in western Amazonia (*9*).

To quantify the relative importance of spatial and environmental events in determining species similarity between plots, we have reanalyzed Condit *et al.*'s Panama plot data. Dispersal is a purely spatial process: Progeny grow close to their parents when dispersal capacity is low. We used straight-line distances between plots to represent the dispersal process. Condit *et al.* provided four crucial environmental variables: elevation, precipitation, age of the forest stand, and the type of bedrock. We used similarities converted from normalized differences between plots to quantify each environmental variable, and the Steinhaus coefficient to quantify species similarity. Distance and the four environmental variables were all significant predictors of species similarity between plots in permutation-based multiple regressions. We then partitioned the variance in species similarity by computing multiple regressions of species similarity against distance only, environmental variables only, and both distance and environmental variables (*10*). Distance alone and environmental variables alone explained minor portions of the variation in species similarity (see the figure). Distance and environment together, however, explained 24% of the variation. The inability to separate distance and environment reflects a strong gradient in rainfall that is highly correlated with distance between Panamanian plots (*11*). Perhaps most important, 59% of the variation in species similarity remained unexplained by either distance or environment. In an analogous study, distance and environment explained just 16% of the variation in upland tree species composition between Colombian forest plots (*12*). This unexplained variance is typical for studies of tree species similarity in tropical forests.

Condit *et al.*'s approach is an important step toward predicting the effects of plant dispersal on species composition in the tropics. However, given that most of the variation in species similarity in tropical forests cannot be explained, there is a clear need for additional data and analyses before we fully understand the events that determine tropical forest diversity.

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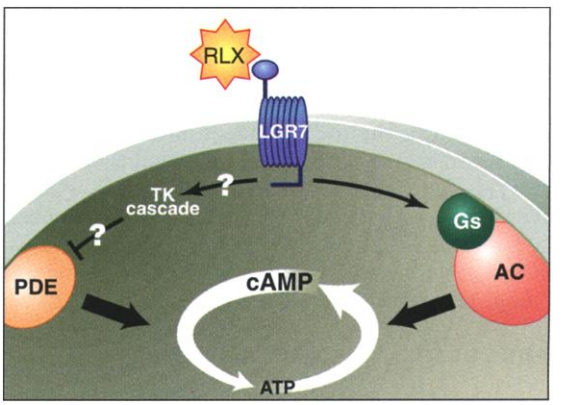
PERSPECTIVES: ENDOCRINOLOGY

This Hormone Has Been Relaxin' Too Long!

Richard Ivell

It is an irony of reproductive endocrinology that we are still seeking a receptor for the pregnancy hormone relaxin, one of the first reproductive hormones to be identified (*1*). Enter Hsu and colleagues (*2*) on page 671 of this issue to remedy the deficit. They describe two G protein–coupled, seven–transmembrane domain receptors (LGR7 and LGR8) that fulfill the requirements of a relaxin receptor.



A receptor for relaxin. The different signal transduction pathways involved in the up-regulation of cAMP by the peptide hormone relaxin (RLX). When relaxin binds to its G protein–coupled receptor, LGR7 or LGR8, a G protein signaling pathway is activated leading to stimulation of adenylate cyclase (AC) and an increase in cAMP (*2*). Binding of relaxin to its receptor also may activate a tyrosine kinase pathway that inhibits the activity of a phosphodiesterase (PDE) that degrades cAMP (*4*).

On the one hand, the result is expected because relaxin is known to cause an increase in the concentration of intracellular cAMP in most (but not all) of its target tissues, consistent with its binding to a G protein–coupled receptor. Furthermore, the structural similarity of LGR7 and LGR8 to receptors for other reproductive peptide hormones, such as luteinizing hormone and follicle-stimulating hormone, suggests that all of these hormone receptors may share a common evolutionary origin. On the other hand, however, the Hsu *et al.* findings are unexpected because relaxin and its relative, relaxin-like factor (RLF/INSL3), structurally belong to another group of peptide hormones that includes insulin and IGF1. Logically, therefore, one might have expected the relaxin receptor to be an orphan membrane–associated tyrosine kinase receptor resembling those that bind to insulin and IGF1. Indeed, pharmacological evidence indicates that inhibitors of tyrosine kinase receptors block signal transduction by the relaxin receptor, and that relaxin can induce tyrosine phosphorylation and inhibition of a cell-specific phosphodiesterase, the enzyme that degrades cAMP (*3*, *4*) (see the figure).

Relaxin regulates the growth and remodeling of reproductive tissues during late pregnancy. In model species, such as the pig, rat, and guinea pig, relaxin promotes expansion of the birth canal (loosening of the pubic symphysis and relaxation of the cervix) during parturition. In rats, relaxin also inhibits both spontaneous and oxytocin-induced contractions of the

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uterus. However, in humans and other primates circulating relaxin is present at 100-fold lower levels compared with that in the rat and pig, and only peaks during the first trimester. In clinical trials with recombinant human relaxin, the peptide hormone failed to show any effect on relaxation of the cervix. Not surprisingly, these results have proved something of a death-knell for interest in relaxin research.

Viewing relaxin as a hormone of late pregnancy is misleading. In humans, the peak in circulating relaxin during the first trimester coincides with implantation of the embryo, and recent experiments show that relaxin appears to be as crucial as progesterone for the induction of decidualization (the differentiation of the uterine stroma to accommodate the implanting embryo) (*5*). Circumstantial evidence supports the notion that disruption of circulating relaxin during early pregnancy is associated with loss of the fetus (*6*, *7*). Relaxin specifically induces the expression of vascular endothelial growth factor (VEGF) in the endometrium and, hence, is responsible for the formation of new blood vessels that are essential for embryonic growth and development (*8*). But the story does not end here for relaxin is secreted not only by the ovary during pregnancy, but also by many other tissues as a paracrine factor involved in the formation of new blood vessels after infarct or during wound healing (*9*). In addition, relaxin has marked effects on the dilation of blood vessels and is locally produced in the human heart to redress the cataclysmic consequences of congestive heart failure (*10*). Relaxin is a vasoactive hormone, but also restricts the formation of fibrotic lesions in humans and in different animal models (*11*). Thus, relaxin is far more versatile than a hormone involved only in reproduction.

In women, most of the relaxin in the circulation derives from the ovary. Like the sex steroid hormones, estrogen and progesterone, levels of relaxin decrease during menopause. Bearing in mind the typical symptoms associated with postmenopausal aging—fibrosis, wound-healing deficits, vasorestriction—we may be overlooking the possible therapeutic benefits of relaxin as a hormone replacement therapy.

In addition to identifying LGR7 and LGR8 as receptors for relaxin, Hsu and colleagues point out that LGR8 also may be the receptor for the closely related hormone RLF (*2*). They make this connection on the basis of the independent discovery that a mutation in the *LGR8* gene in mice results in failure of the testes to descend, the same abnormality that is seen in RLF-deficient mice (*12*–*14*). RLF is synthesized by Leydig cells in the fetal testis and appears to be responsible for the second phase of testicular descent by influencing the growth and differentiation of the cord that connects the testes with the lower abdomen. This peptide hormone is also made in large amounts by the Leydig cells of the adult testis (*15*, *16*), although its function in the adult male is not known. In the female, RLF is made by the theca cells of the ovarian follicles and by other tissues, and may be involved in ovarian follicle selection (*17*). Research on RLF is still in its infancy, but it is already clear that this hormone is secreted by diverse tissues and has several different functions.

The discovery of the receptors for relaxin and RLF should stimulate research into the molecular pharmacology of these hormones for which neither antagonists nor agonists are available.

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PERSPECTIVES: ASTRONOMY

Radio Noise from Dust Grains

Andrea Ferrara

In a galaxy like the Milky Way, about 0.1% of the visible mass resides in "cosmic dust"—tiny, silicate- and carbon-based particles with a mean radius of ~0.1 μm. Although it only makes up such a small fraction of the total mass, this dust plays an important role in galactic evolution: It provides suitable conditions for star formation, acts as a catalyst for the formation of molecules, and shields the molecules from damaging stellar ultraviolet radiation.

Energy absorbed by cosmic dust particles is typically converted into thermal energy and reemitted in the infrared at wavelengths of ~100 μm, where it can be observed and used as a powerful physical indicator of the physical conditions of the source. However, a paper in press in *Astrophysical Journal* reports the detection of a continuum signal at radio wavelengths (~3 cm) that can be attributed to dust beyond any reasonable doubt (*1*). What causes this unusual emission? And what are its implications?

Long before any observations of such emissions were reported, theorists suggested that they might exist. In 1957, Erickson (*2*) suggested a mechanism by which dust could produce nonthermal radio noise. If a dust grain residing in an interstellar cloud is bombarded with moderately fast atoms or ions, these collisions transfer angular momentum to the grains. Their rotational frequency may then reach values comparable with radio frequencies. Grains are usually charged and can behave as rotating electric dipoles if their centers of mass and of charge do not coincide—either because their shapes are irregular or as a result of statistical fluctuations in the charge distribution on the grain. Erickson postulated that under these conditions, spinning grains would emit observable radio waves.

Hoyle and Wickramasinge (*3*) revisited the problem in the context of galactic HII regions, that is, regions in the Milky Way where interstellar hydrogen has been ionized by photons with energies >13.6 eV emitted by massive stars. An ionized, magnetized gas as found in HII regions usually emits predominantly through synchrotron processes. But the authors speculated that nonthermal radio emission might be as important as synchrotron emissions for explaining the observed flux at gigahertz frequencies from HII regions.

After these early studies, the field remained essentially dormant, probably because the predictions could not be tested with existing observational tools. In the meantime, Purcell and Spitzer (*4*, *5*) set the theoretical basis for a better understanding of grain rotation. They showed that grains can rotate suprathermally, that is, with an energy much greater than kT (where T is the temperature of the system and k is the Boltzmann constant) as a result of torques acting on grain shape irreg-

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