

systems could be represented in terms of computer models of their control networks. In that discussion he invoked a 1969 paper in which Stuart Kauffman speculated that different limit cycles of a nonlinear biological cellular network might represent different cell types—a liver cell, the skin on your nose, and so on—so that even though the sequence of nucleotides in the DNA of each cell is identical, different cell types can develop.

However, it's important for readers of *PHYSICS TODAY* to understand that developmental biology has seen tremendous progress in figuring out what machinery drives cellular differentiation. It turns out that Nature developed a fascinating mechanism for differentiating cell types which involves attaching a variety of covalent epigenetic markings such as acetylation of the histone proteins that support the DNA in chromosomes, or methylation of the nucleotides themselves, at specific sites on the cellular DNA of eukaryotes. By that mechanism, the DNA of different cell types becomes modified—without its sequence being changed—in ways that control the expression of the 20 000–30 000 or so genes encoded in human DNA.

An equally significant discovery, which arose out of the human genome project, is that only about 2% of the human genome codes for proteins. Accumulating evidence shows that 50%–80% or maybe more of human DNA, the so-called noncoding type, or ncDNA, is transcribed into RNA molecules of various sizes, many of them quite short (as few as 22 base pairs). And it appears that the whole business of epigenetic marking may be controlled by those various RNAs.¹ Thus the expression of specific genes, which needs to be highly controlled as to timing and position in cells, is probably under the control of the “playbook” represented by the vast amount of genetic information embedded in the ncDNA.²

An even more fascinating possibility is that human memory may also be controlled by epigenetic labeling of DNA.³ Memory-inducing events are known to stimulate protein synthesis in the cell body of neurons, a process associated with synapse formation. It is also conceivable that neuronal gene expression is associated with RNA control of epigenetic markings.⁴ Such a hypothesis would help explain the fact that consolidated memories can last a lifetime in humans, and maybe in tortoises and elephants as well.

Epigenetic labeling could also help account for the huge multiplicity of memory states. If one thinks of marking each of just 25 sites of neuronal DNA with one of three epigenetic markers, one could define a different state of that neuron corresponding to every millisecond of a human lifetime.

So it appears that Nature has come up with a much more robust set of tools for eukaryotes than the simplified nonlinear dynamical networks beloved of physicists. However, bacteria are much simpler systems, having only about 1% of their DNA non-coding. So bacterial metabolism may well be simple enough to be a jumping-off point for understanding complex networks.

References

1. See F. F. Costa, *Gene* **410**, 9 (2008).
2. See J. S. Mattick, *J. Exp. Biol.* **210**, 1526 (2007).
3. See, for instance, Y. I. Arshavsky, *Prog. Neurobiol.* **80**, 99 (2006).
4. S. I. Ashraf, A. L. McLoon, S. M. Sclarsic, S. Kunes, *Cell* **124**, 191 (2006).

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Physics Today in stitches

Thank you for *PHYSICS TODAY*, which consistently contains interesting and important material and performs a role unlike any other journal's.

I noticed the binding of the print edition changed as of the August 2009 issue. I prefer the old binding, which had the volume and issue numbers visible along the spine. I refer to past issues frequently, especially when they are cited in the current issue, and the new binding will make finding old issues much less convenient. As nuisances go, this is a small one, but why change things for the worse? I can't imagine I am the only reader who feels this way.

I hope you will return to a binding that shows the reference data on the spine.

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I'm very grateful for the revised binding on the August 2009 issue of *PHYSICS TODAY*. For many years I have mangled each month's issue by folding it, creasing it, pounding on it and more, in an effort to get the magazine to lie flat on a given page. No more! I can read margin to margin. When I'm on the bus I can fold it back and read only one page, and the binding survives. I can extract book reviews for future reference with a single tug.

Thank you for a change that makes the journal more readable. Now if you could work on the ink formulation so my fingers do not render dark figures uninterpretable in the summer.

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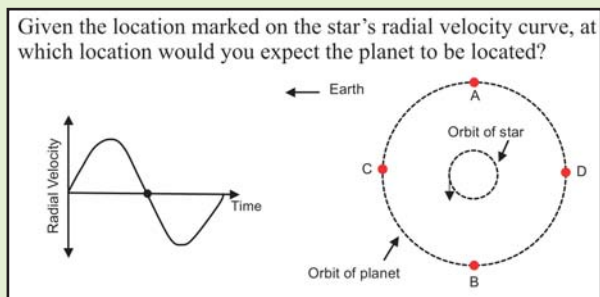
The publisher replies: We appreciate the letter writers' kind words about *PHYSICS TODAY*. Our staff strives to make each issue interesting and useful.

Current economic conditions, especially the decline in advertising revenue, are driving *PHYSICS TODAY*, and many other publications, to look at all expenses. The new binding might be a small nuisance to some and a godsend to others. More important, it saves more than \$30 000 annually in production costs. In addition, the current saddle-stitch binding method is a bit more “green,” and that will influence any decision to return to the previous binding.

Inking is related to characteristics and interactions of paper and ink. It is something we look at periodically with our printer. This, too, is a tradeoff between cost, utility, and environmental concerns.

Randolph Nanna
PHYSICS TODAY
College Park, Maryland ■

Correction



October 2009, page 43—The top figure, which was somehow distorted in the printing process after the proofing stage, is shown correctly at left, with the orbits appearing as concentric circles.