inhalation of carbon dioxide and panic attacks. Psychiatry Res 1995; 57:267–273

GIAMPAOLO PERNA, M.D., PH.D.
SILVIA COCCHI, M.D.
ERNESTINA POLITI, M.D.
LAURA BELLODI, M.D.
Milan, Italy

Triplet Repeat Diseases in Man, Microbes, and Molecules

TO THE EDITOR: Dramatic progress has been made in the past 5 years in our understanding of the etiology of more than nine human genetic neuromuscular and neurodegenerative diseases (1). The clinical observation of anticipation can be ascribed to the expansion of triplet repeat sequences. These characteristics were suggested to be involved also in neuropsychiatric syndromes, including forms of dementia, hereditary ataxia, parkinsonism, bipolar affective disorder, schizophrenia, and autism (2–4). The non-Mendelian trait of anticipation may be due to the slippage of the complementary DNA strands during replication. The establishment of a genetically and biochemically tractable system for elucidating the molecular mechanisms that are responsible for expansion, and thus anticipation, would be a significant advance.

It is of interest that *Escherichia coli* shows several remarkable molecular similarities to humans, including 1) genetic instability (expansions and deletions) of triplet repeat sequences (CTG·CAG, CGG·CCG, or AAG·CTT) (1, 5–8), 2) longer repeats that are more unstable than shorter sequences (1, 5–11), 3) preferential expansion of CTG·CAG (9) (this repeat sequence was found in six of the nine triplet repeat diseases), 4) repeat sequence imperfections (polymorphisms) that stabilize long tracts of triplet repeat sequences (1, 5–11), 5) similar types of imperfections (polymorphisms), such as the poly purinepoly pyrimidine motif in the Friedreich's ataxia AAG repeat sequence [7, 8]), 6) approximately similar lengths of the smallest deletion products (10–20 triplet repeats) (1, 5), and 7) DNA polymerases that pause in long CTG·CAG, CGG·CCG, and AAG·CTT sequences (5, 8), which render them susceptible to mutations.

In summary, I submit that certain features of the molecular processes related to the involvement of triplet repeat sequences in human hereditary diseases may be elucidated effectively in simple cellular systems. Obviously, a number of other developmental and neurological questions can only be solved in higher eucaryotic cells. Thus, some features of the "unstable genes—unstable mind" concept (4) may be tractable in genetically defined systems in mice, microbes, and molecules.

REFERENCES

1. Davies K, Warren S (eds): Genome Analysis, vol 7: Genome Rearrangement and Stability. Cold Spring Harbor, NY, Cold Spring Harbor Laboratory Press, 1993
2. Morris AG, Gaitonde E, McKenna PJ, Mollon JD, Hunt DM: CAG repeat expansions and schizophrenia: association with disease in females and with early age-at-onset. Hum Mol Genet 1995; 4:1957–1961
3. Margolis RL, Stine OC, McInnis MG, Ranen NG, Rubinsztein DC, Leggo J, Jones-Brando LV, Kidwal AS, Loev SJ, Breschel TS, Callahan C, Simpson SG, DePaulo JR, McMahon FJ, Jain S, Paykel ES, Walsh C, DeLisi LE, Crow TJ, Torrey EF, Ashworth RG, Macke JP, Nathans J, Ross CA: cDNA cloning of a human homologue of the *Caenorhabditis elegans* cell fate-determining gene *mab*-21: expression, chromosomal localization and analysis of a highly polymorphic $(CAG)_n$ trinucleotide repeat. Hum Mol Genet 1996; 5:607–616
4. Petronis A, Kennedy JL: Unstable genes—unstable mind? Am J Psychiatry 1995; 152:164–172
5. Wells RD: Molecular basis of genetic instability of triplet repeats. J Biol Chem 1996; 271:2875–2878
6. Kang S, Jaworski A, Ohshima K, Wells RD: Expansion and deletion of CTG repeats from human disease genes are determined by the direction of replication in *E coli*. Nat Genet 1995; 10:213–218
7. Campuzano V, Montermini L, Molto MD, Pianese L, Cossee M, Cavalcanti F, Monros E, Rodius F, Duclos F, Monticelli A, Zara F, Canizares J, Koutnikova H, Bidichandani SI, Gellera C, Brice A, Trouillas P, De Michele G, Filla A, De Frutos R, Palau F, Patel PI, Di Donato S, Mandel J-L, Cocozza S, Koenig M, Pandolfo M: Friedreich's ataxia: autosomal recessive disease caused by an intronic GAA triplet repeat expansion. Science 1996; 271:1423–1427
8. Ohshima K, Kang S, Larson JE, Wells RD: Cloning, characterization, and properties of seven triplet repeat DNA sequences. J Biol Chem 1996; 271:16773–16783
9. Ohshima K, Kang S, Wells RD: CTG triplet repeats from human hereditary disease are dominant genetic expansion products in *Escherichia coli*. J Biol Chem 1996; 271:1853–1856
10. Shimizu M, Gellibolian R, Oostra BA, Wells RD: Cloning, characterization, and properties of plasmids containing CGG triplet repeats from the FMR-1 gene. J Mol Biol 1996; 258:614–626
11. Kang S, Ohshima K, Jaworski A, Wells RD: CTG triplet repeats from the myotonic dystrophy gene are expanded in *Escherichia coli* distal to the replication origin as a single large event. J Mol Biol 1996; 258:543–547

ROBERT D. WELLS, PH.D.
Houston, Tex.

Debating Dissociative Diagnoses

TO THE EDITOR: The diagnosis of dissociative identity disorder, formerly multiple personality disorder, has generated considerable debate. Some argue that the disorder is common and underdiagnosed (1), while others claim that dissociative identity disorder is a rare or frankly artifactual diagnosis (2). Our impression is that dissociative identity disorder is frequently accepted as a valid diagnostic entity in the United States but regarded with greater skepticism in other English-speaking countries.

To test this impression, we collected all articles and letters regarding dissociative identity disorder that appeared between 1976 and 1995 in *The American Journal of Psychiatry*, *The British Journal of Psychiatry*, *The Canadian Journal of Psychiatry*, and *The Australian and New Zealand Journal of Psychiatry*. We rated each article or letter as "skeptical" if it argued that dissociative identity disorder was 1) vastly overdiagnosed or 2) an artifact promoted by suggestive influences. We considered all other articles "nonskeptical," including those that merely acknowledged the existence of literature that was skeptical of dissociative identity disorder. In *The American Journal of Psychiatry*, we found 37 articles and letters, of which five (14%) were rated as skeptical and 32 (86%) nonskeptical. By contrast, in the combined journals from Great Britain and its largest English-speaking commonwealth countries, we found 45 articles and letters, of which 24 (53%) were skeptical, and only 21 (47%) were nonskeptical. This difference in rates of skeptical papers is highly unlikely to be due to chance ($p=0.0003$, Fisher's exact test, two-tailed).

We next examined circulation figures. In the United States, combined individual and institutional subscriptions to *The American Journal of Psychiatry* (46,457) dwarf those of the

journals from Great Britain (1,054), Canada (503), and Australia/New Zealand (57). Thus, it appears that in America, psychiatrists receive an overwhelmingly more positive view of dissociative identity disorder than their English-speaking counterparts elsewhere.

How can these differences be explained? Some have suggested that they may be due to unfamiliarity with dissociative identity disorder among non-American clinicians (3) or that the problem reflects an error on the part of Britain's influential academic establishment (4). An alternative, provocative hypothesis is that dissociative identity disorder be designated a culture-bound syndrome (5), on the theory that many American therapists and their patients are involved in a subcultural mutual belief system about the existence of dissociative identity disorder, abetted perhaps by dramatized American media presentations of people diagnosed with the disorder. Although the validity of these competing hypotheses remains to be tested, we are clearly obliged to explain these remarkable national differences in the acceptance of the dissociative identity disorder diagnosis.

REFERENCES

1. Ross CA: Multiple Personality Disorder: Diagnosis, Clinical Features, and Treatment. New York, John Wiley and Sons, 1989
2. Merskey H: Multiple personality disorder and false memory syndrome (editorial). Br J Psychiatry 1995; 166:281–283
3. Chande A: Manufacture of multiple personality disorder (letter). Br J Psychiatry 1992; 161:269
4. Putnam FW: Multiple personality disorder (letter). Br J Psychiatry 1992; 161:415–416
5. Kenny M: The Passion of Ansel Bourne: Multiple Personality in American Culture. Washington, DC, Smithsonian Institution Press, 1986

JOHN HOCHMAN, M.D.
Encino, Calif.
HARRISON G. POPE, JR., M.D.
Belmont, Mass.

Biased Book Review

TO THE EDITOR: In his recent review of *Psychodynamic Concepts in General Psychiatry* (1), Dr. Hartmann is critical of the editorial integration of the text, even though he recommends as useful over 50% of the 24 chapters. While I respect his prerogative as a reviewer to find fault with the book, I do not accept his presumption that my intent as editor was to be engaged in simply a "hopeful collecting" of the authors and their chapters. My and the coeditors' efforts were to gather together in one book clinicians of considerable expertise in their subspecialty areas—a "roster of excellent authors"—and invite them to present in clinical detail their understanding of how the psychodynamic method informs their work and heals their patients.

Dr. Hartmann's presumptiveness approaches the heart of the question about his ability to fairly review this work. This issue is acknowledged in his off-handed declaration of having "not a conflict of interest" in reviewing the book. He claims to having had a mere "previous look" at the text before publication. In fact, Dr. Hartmann had, or at least attempted to have, a great deal more involvement in the production of this text. Before publication Dr. Hartmann contacted me by phone and fax numerous times and strongly suggested that I make changes in the completed manuscript, which was shown to him by the publisher. After considering a number of his suggestions and reviewing them with the authors, I did agree with some of them. I did not agree with others. Dr. Hartmann persisted. He called me again at home and was quite adamant that if I failed to make the changes he was "suggesting" to me, then perhaps it would be best that the book not be published. Most of his anger, which by now was not even barely concealed, was directed at the chapter "The Depressed Male Homosexual Patient," which fills 16 of the 497 pages of the book. He insisted that this chapter should be omitted or "entirely rewritten." In fact, the chapter was significantly edited, in part because of some of Dr. Hartmann's helpful suggestions. Still, that did not suffice, and he faxed me yet again a line-by-line critique of much of the chapter with recommendations for how those lines should be further rewritten to satisfy him. Dr. Hartmann's feelings about that one chapter, his frustration that it was not changed according to his demands, and his overall involvement with the book were so considerable that on the very day it was published he faxed me an angry letter declaring that that one chapter had ruined the entire book. I will respectfully leave it to the readers of the book to evaluate this and the other chapters and their conclusions that are derived from extensive clinical data.

I believe that the *Journal* is ill-served by Dr. Hartmann's failure to fully acknowledge his extensive attempts to influence the preparation of this book and, accordingly, to have recused himself from reviewing it.

REFERENCE

1. Hartmann L: Book review, Schwartz HJ, Bleiberg E, Weissman SH (eds): Psychodynamic Concepts in General Psychiatry. Am J Psychiatry 1996; 153:1102–1103

HARVEY J. SCHWARTZ, M.D.
Philadelphia, Pa.

Dr. Hartmann Replies

TO THE EDITOR: I think it is not a conflict of interest to dislike subsequent versions of the same book twice and to say so. I would have liked a book on psychodynamic concepts to be a better book. Although I was far from the only critic (and the history of criticisms is not precisely as Dr. Schwartz recalls), and my finding some partial values in the book is not quite as he states, to reply at length to his complaints would be to give further and undeserved publicity to an inadequately edited and overall not very good book. One could recommend a few of the chapters, but the book remains a missed opportunity.

LAWRENCE HARTMANN, M.D.
Cambridge, Mass.

Homicidal Behavior in Drug Abusing Psychiatric Patients

TO THE EDITOR: The recent report of Ihsan M. Salloum, M.D., M.P.H., and colleagues (1) of greater homicidal behavior in psychiatric patients with concurrent alcohol and cocaine abuse is very interesting, particularly because combined use has not been previously linked to greater violence. On the contrary, controlled laboratory studies have suggested that alcohol-induced behavioral impairment is attenuated by stimulants (unpublished 1990 study of D.A. Beezley-Smith) and cocaine (2). Furthermore, in a study of psychiatric emer-