Letters

Women in Science

As a woman scientist, as well as a longtime supporter of women in science, I am pleased to see *Science* devote space to a review of *Hypatia's Heritage: A History of Women in Science from Antiquity Through the Nineteenth Century* (27 Feb., p. 1092). But I am nonplussed by Ann H. Koblitz's reference, simultaneously gratuitous and erroneous, to my own book on a rather different subject, *Reflections on Gender and Science* (*1*).

Most readers of *Science* are well aware of the demographic predominance of men in scientific professions; they are probably also aware of the enormous obstacles (both cultural and institutional) that women in science have historically had to contend with. But they may be less aware of the role that gender stereotypes (in particular, the conventional definition of science as "masculine") have played in the exclusion of women from science.

In *Reflections on Gender and Science*, I do indeed, as Koblitz remarks, invoke the masculine pronoun to refer to the architects of modern science, and I do so to underline two facts—not simply that the "founding fathers of modern science" were in fact male, but also that they conceptualized science, explicitly and self-consciously, as a specifically "masculine" endeavor: better, and purer, than other philosophies precisely to the extent that it excluded attributes or values that were regarded as "feminine." In other words, the conception of science as "masculine" began, not with contemporary feminists, but with the original makers of science.

To acknowledge this history is not in any way to "aver" that there have been no women scientists. Indeed, my earlier book on Barbara McClintock (*2*) begins with a lengthy discussion of the impressive number and caliber of women at the beginning of this century struggling to pursue scientific careers. And Koblitz is quite right that they were not the first. One would hope that calling attention to the deep cultural obstacles women in science have faced would not be read as implying that women scientists neither existed nor are in any sense less capable than their male colleagues.

But there is another point here as well. The focus of *Reflections on Gender and Science* is not, finally, on cultural obstacles facing women in science, but rather on the role that gender stereotypes have played within the actual workings of science. The exclusion of values culturally relegated to the female domain has led to an effective "masculinization" of science—to an unwitting alliance between scientific values and ideals of masculinity embraced by our particular culture. All of our best hopes for science—our very aspirations to objectivity and universality—would argue that such alliance (and exclusion) would be to the detriment not only of women scientists but of all scientists and indeed to the detriment of science itself.

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Adolescence and Mental Illness

Deborah M. Barnes, in her summary of biological issues in schizophrenia (Research News, 23 Jan., p. 430), mentions in passing that "some researchers are beginning to ask . . . whether certain developmental changes in the brain that take place around puberty may be related to schizophrenia." These changes should be of special interest to clinicians as well as to basic scientists because the onset of schizophrenia in the peripubertal period is one of the most reliable characteristics of this subtle and baffling disorder. Despite disturbances of behavior so extreme as to render patients nonfunctional for decades, there has not yet been demonstrated a single brain abnormality specific to schizophrenia.

Evidence converging from several disciplines indicates that the frequent onset of schizophrenia during or shortly after adolescence may be related to a profound reorganization of the brain during the second decade of life (*1*). Among the most striking phenomena are a massive reduction in the amplitude and duration of the delta electroencephalogram (EEG) of deep (stage 4) sleep (*2*) and a decline in cerebral oxygen consumption ($CMRO_2$). An early study with the Kety-Schmidt nitrous oxide method, which gives a weighted average of the metabolic rate of gray and white matter, suggested a $CMRO_2$ decline of about 25% during the second decade (*3*). An effect of this magnitude is not trivial, being equal to the average difference between senile and normal elderly individuals (*4*). Recent investigations by Chugani and his colleagues (*5*) indicate that the developmental decline in cortical $CMRO_2$ is even greater: using positron emission tomography, they found that cortical brain metabolism was reduced during the second decade by as much as 50%. A structural basis for this change is provided by Huttenlocher's discovery (*6*) of a sharp decline in the density of synapses in human frontal cortex during the second decade. The available data suggest that the curves for these three variables—cerebral metabolic rate, amplitude and duration of stage 4 EEG waves, and cortical synaptic density—are approximately parallel over the first 20 years of life: each rises steeply after birth to a maximum (about twice the adult level) at 2 to 5 years, maintains the high level to the end of the first decade, and then declines until, at the end of the second decade, the adult level has been reached. The falloffs are steepest between ages 10 and 15.

Huttenlocher suggested that the decline in synaptic density during adolescence is a final manifestation of a mechanism used repeatedly in earlier brain development: a programmed elimination of excess neural elements to achieve a fine-tuning of the nervous system (*7*). This same change could account for the decline in the delta EEG of deep sleep and in $CMRO_2$ and for certain other brain and behavioral changes of adolescence (*1*). A defect in this (presumably) genetically controlled process might impair mechanisms of neural integration and thereby produce the illness in at least a subgroup of patients with the schizophrenic syndrome. Other psychiatric illnesses that appear after adolescence—such as classical mania and depression—may involve different faults in the same process.

Whether or not the brain changes of adolescence prove causal to schizophrenia or to other psychiatric disorders, they are of considerable basic importance to those concerned with the ontogeny of the human brain. Thus, to the intense psychosocial, sexual-endocrine, and cognitive changes of adolescence, we can add modifications in brain physiology and structure. Investigation of the interaction of these phenomena, and of their bearing on mental illness, could be mutually beneficial for neuroscience and behavioral research.

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