from the primary process of the collapse and fragmentation of interstellar clouds, producing objects no less massive than about 10 to 20 Jupiter masses (*6*). Planets form as a result of secondary processes in the flattened accretion disks surrounding newly formed stars. Because accretion disk masses are typically a fraction of the mass of the central protostar and a number of planets may form, planetary masses should only be a small fraction of their star's mass.

Unfortunately, brown dwarf stars probably occur primarily in isolation as single stars, or as members of brown dwarf binary systems. Because of the absence of a more massive luminous companion, these stars may well elude discovery until more powerful infrared telescopes (for example, the Space Infrared Telescope Facility) are employed in the uncertain future.

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Blocked Ontogeny

I would like to comment on the statement given prominence in an article by Jean L. Marx (Research News, 15 May, p. 778): "The view that cancer results from a block in differentiation is naïve at best." No one can quarrel with the efforts by many investigators to overcome blocks in differentiation in tumor cells by using natural factors or chemotherapeutic agents. It should be made clear, however, that the concept of "oncogeny as blocked ontogeny" (*1*) was from the outset understood as "partially blocked ontogeny" (*2*). Differentiation need not be completely blocked, only blocked *enough*. The blocked ontogeny hypothesis is not naïve; it provides the only framework today for integrating ongoing experiments in developmental biology and carcinogenesis at the molecular level.

Further development of the hypothesis calls for experiments that examine the interaction of three kinds of regulatory genes (*3*). Arbitrary designations for these genes are here given as (i) the *gf*, expressed as a growth factor GF; (ii) the *sr*, expressed as a suppressor receptor SR; and (iii) the *s*, expressed as a growth inhibitor or suppressor S. Moore and his collaborators (*4*) mention a growth factor that can stimulate the cell cycle at low concentrations and stimulate differentiation at higher concentrations, and they refer to qualitative concentration effects observed by Metcalf.

The blocked ontogeny hypothesis suggests that stem cells express *gf* (or receptors for GF from other cells), but not the genes for SR or S. Differentiation leads to expression of *sr*, and *s* and may be promoted by increased levels of GF over and above those needed for growth. Partially blocked ontogeny could result from mutations in *sr* that lower affinity of SR for S or even that lead to total loss of SR. Closure of a feedback loop that would promote normal homeostatic balance between cell reproduction and differentiation might be effected by the production of second signals when S combines with SR, which would lead to decreased expression of *gf*. Recent work by Trosko and his colleagues on the role of gap junctions in intercellular communication (*5*) suggests that the appearance and function of gap junction may be a step in a homeostatic feedback loop that leads from GF to SR to S and back to *gf*.

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Erratum: In the letter "Cancinogenicity and allergenicity" by Merril Eisenbud (26 June, p. 1613), the first sentence of the second paragraph should have read, "Two metals (Ba and Bi) are reported to be neither allergenic nor carcinogenic."

Erratum: In the article "Splicing of messenger RNA precursors" by Phillip A. Sharp (13 Feb., p. 766), the results of C. Weissmann (*53*) were incorrectly described. As H. Hornig *et al.* show [*Nature (London)* **324**, 589 (1987)], a C at the branch site of a precursor RNA does not arrest splicing at the intermediate stage, while either a U or a G at this position does arrest splicing at this point.

Erratum: The caption for the map accompanying the article "Bolivia swaps debt for conservation" by John Walsh (News & Comment, 7 Aug., p. 596) does not make clear that the land indicated remains in Bolivian ownership and will be a conservation area.