PERSPECTIVES: CLIMATE CHANGE

Lessons for a New Millennium

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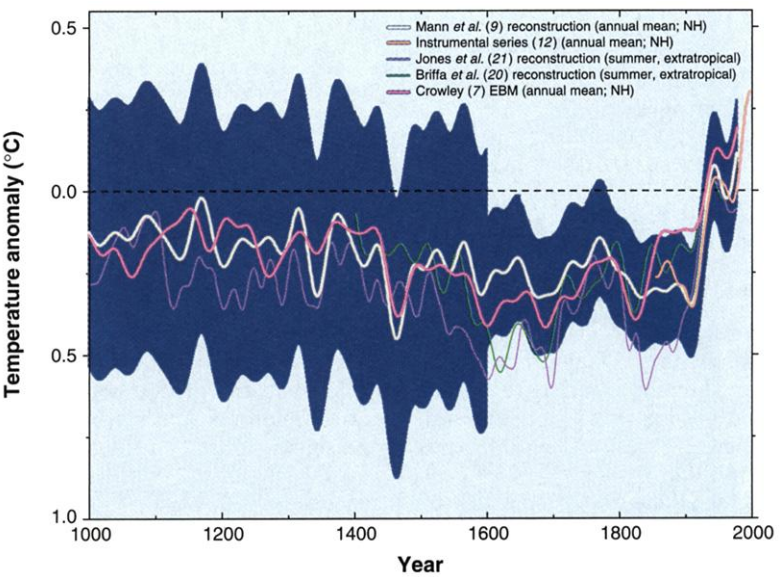
A key factor hampering our ability to confidently assess the human influence on the warming of the past century is our limited understanding of the climate changes believed to have occurred in previous centuries. What caused the "Little Ice Age" of the 15th to 19th centuries or the putative "Medieval Warm Period" of earlier centuries (*1*, *2*)? Might not the same, presumably natural, factors bear some responsibility for the dramatic warming of the 20th century (*3*–*6*)? On page 270 of this issue, Crowley (*7*) provides some convincing answers to these questions and makes a compelling case for the assertion that anthropogenic greenhouse gas increases are behind the continued warming of the globe.

Conventional approaches to understanding the factors underlying the recent warming have involved complex numerical models of the combined ocean-atmosphere system. Although highly suggestive of a detectable human influence on climate, these studies have been limited by intrinsic uncertainties in comparing model-predicted climate change patterns with the instrumental climate record. At roughly one century, the latter is too short to allow unambiguous attribution of changes to human influences (*8*).

Crowley's study circumvents this limitation by making use of empirical information about longer term climate variability. The author uses an Energy Balance Model (EBM), calibrated to exhibit a similar response to external radiative influences as more elaborate coupled ocean-atmosphere models. This allows an efficient investigation of forced changes in annual mean temperatures in the Northern Hemisphere over the past millennium. The model is driven with (admittedly uncertain) empirical estimates of the time histories of the most relevant factors affecting the atmosphere's radiative balance (solar radiative output, volcanic aerosol loading, anthropogenic greenhouse gas concentrations, and industrial aerosols). Comparison of the predicted response with independent (although also uncertain) estimates of Northern Hemisphere annual temperature variations over the past millennium based on proxies such as tree rings, ice cores, and corals, which naturally record climate variations (*9*, *10*) (see the figure), yields fairly close agreement (*11*). Of equal interest, however, is the level of disagreement: Within estimated uncertainties, the amplitude of the residual temperature variations not explained by the model agrees precisely with the typical amplitude of purely random or "stochastic" climate variability observed in coupled ocean-atmosphere models.



Temperature histories explained? Comparison of proxy reconstructions of annual mean Northern Hemisphere (NH) temperature change (*9*) with the EBM results described by Crowley (*7*). The blue-shaded region represents the approximate uncertainty range in the empirical temperature estimates of (*9*). Two extratropical warm-season Northern Hemisphere temperature reconstructions (*20*, *21*) are shown for comparison.

Crowley's report thus strengthens the case for a detectable human influence on 20th century global warming by establishing that (i) much of the climate history of the past millennium can be explained in terms of a few well-established, physically well-constrained radiative forcings, (ii) the dramatic warming of the 20th century can almost certainly not be explained by the natural forcings, but instead requires the emergent anthropogenic forcings of the 20th century, and (iii) more detailed climate models used to detect and attribute observed patterns of climate change to anthropogenic factors (*8*) appear to capture the unforced component of climate variability with sufficient accuracy. The last conclusion strengthens the independent conclusion drawn from simulations using more complex models that human-induced climate change is now detectable.

Nonetheless, Crowley's study does not explain the entire climate history of the past millennium. The model does not, for example, reproduce the cooling of the late 19th century that is seen both in proxy-based climate reconstructions (*9*, *10*) and the early instrumental record (*12*); the warming, in essence, begins too soon in the model. One possible explanation offered by Crowley is that both the reconstructions and the instrumental record may independently underestimate the hemispheric temperatures during this period, for example, because of sparse spatial sampling. A better explanation, however, also noted by Crowley, is that a potentially important surface radiative forcing not included in his simulations—land usage changes, which affect Earth's surface albedo—may be responsible for the observed cooling. A recent study (*13*) indicates that anthropogenic large-scale land usage changes should have culminated in an annual mean cooling of more than 0.3°C in the 19th century. This additional anthropogenic forcing is not only large enough to explain the discrepancy between observation and Crowley's EBM results, it has also been implicated (*14*) in another residual discrepancy, namely the observed differences between conventional proxy-based estimates of past hemispheric temperature changes (*9*, *10*) and ground surface temperature estimates from borehole profiles (*15*).

Crowley's study also does not explain the regional complexity of surface temperature trends during the past millennium. There is little doubt that the temperature anomalies associated with the Little Ice Age and the Medieval Warm Period were far more prominent in some regions (such as Europe) than in others. These large regional anomalies vary in amplitude, timing, and

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sign and thus average out to yield more modest variations for the Northern Hemisphere on the whole (*9*, *10*). In recent decades, Europe has warmed faster than the Northern Hemisphere on the whole, whereas certain regions in the North Atlantic have actually cooled in the face of widespread warming. This is a result of a combination of regional temperature overprints by the North Atlantic Oscillation (NAO) and related, but distinct, patterns of multidecadal variability associated with the thermohaline circulation of the North Atlantic (*16*, *17*).

It is quite reasonable to assume that similar factors were associated with the pronounced temperature changes in Europe in past centuries that accompanied more modest hemispheric-wide temperature changes. Keigwin and Pickart (*18*) have shown evidence that a heterogeneous temperature pattern in the North Atlantic region consistent with the NAO coincided with the European Medieval Warm Period and Little Ice Age. There is evidence that the aforementioned multidecadal variations in the North Atlantic can couple to variations in solar radiative output that occur on similar time scales (*19*).

Could a similar mode of North Atlantic variability resonate with solar radiative variations at millennial time scales, imprinting a regional pattern of enhanced anomalies on top of the more modest hemispheric-scale warming that Crowley's study attributes in part to solar forcing at these time scales? Only further, more detailed modeling studies and expanded networks of paleoclimate indicators will further elucidate the spatial and temporal patterns of climate change in past centuries.

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PERSPECTIVES: MOLECULAR BIOLOGY

The Mad Ways of Meiosis

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Many organisms, including ourselves, are diploid, that is, they have paired homologous chromosomes (1, 2, 3, and so on) and two sex chromosomes (XX in females and XY in males). Meiosis is the cellular process by which diploid reproductive cells shed one whole set of chromosomes before they differentiate into haploid gametes (sperm or egg). This remarkable process involves two steps, each accompanied by a reduction in chromosome number. First, all the chromosomes replicate to form joined pairs of chromatids; then, homologous chromatid pairs bind (synapse) to each other (see the figure). The homologs separate and are pulled to opposite spindle poles and the cell divides into two daughters (meiosis I). Immediately thereafter (without an intervening interphase) a second spindle is assembled in each daughter cell, and the sister chromatids of each homolog are segregated equally to opposite spindle poles (meiosis II).

A low incidence of unequal chromosome segregation during meiosis seems to be no big deal—right? Wrong. For humans the gain or loss of just a single chromosome during meiosis in either egg or sperm can have devastating consequences. On page 300 of this issue, Shonn *et al.* (*1*) suggest a possible cause of chromosome missegregation during meiosis and propose that the fidelity of chromosome distribution depends on the signaling protein Mad2.

About 20% of all conceptions have major chromosomal abnormalities caused by missegregation of chromosomes during meiosis (*2*). Most fetuses with autosomal trisomy (three copies of a chromosome) and all of those with autosomal monosomy (one copy of a chromosome) are spontaneously aborted. Fetuses with autosomal trisomy of chromosomes 21 (Down syndrome), 13 (Patau syndrome), or 18 (Edwards syndrome) survive until birth but have severe physical and mental abnormalities. Trisomies of the sex chromosomes include XXY (Klinefelter syndrome), which results in mental retardation and sterility, and XYY, which may be associated with a predisposition toward antisocial behavior.

At the onset of meiosis I or II, a specialized complex of proteins on each chromatid, called the kinetochore, captures microtubules coming from one of the two spindle poles (see the figure). For chromosome segregation to be equal, each homolog in meiosis I and each sister chromatid in meiosis II must become attached to microtubules coming from opposite spindle poles. Sister kinetochores, however, do not capture microtubules simultaneously; as a consequence, the chromosome pairs begin to move toward the pole belonging to whichever microtubules they attach to first. The problem is that the amount of time required for the unattached kinetochores to capture microtubules emanating from the other (now distant) pole is highly variable. This difficulty is compounded by the small number of microtubules that extend far enough to reach the unattached kinetochores. If the cell initiates anaphase and starts to segregate chromosomes before all the homolog pairs have established connections with both spindle poles, some gametes will inherit two copies of a missegregated chromosome, and others none (see the figure). Two copies of any chromosome in one of the gametes causes trisomy in the embryo; no copy of any chromosome (except X or Y) gives rise to monosomy, an embryonic lethal abnormality. The loss of the X or Y chromosome in sperm produces the XO genotype (Turner syndrome).

Somatic cells in mitosis (normal cell division) ensure the correct attachment of daughter chromosomes to opposite spindle poles by a molecular safeguard called the spindle checkpoint. This checkpoint detects the presence of even a single unattached kinetochore and arrests the progress of mitosis until the unattached kinetochore captures microtubules from the distant spindle pole. Mutations that compromise the spindle checkpoint contribute to chromosome instability, a hallmark of many human cancers (*3*). Although the activity of this checkpoint in meiosis has been documented in insect spermatocytes (*4*), most analyses have been conducted in mitotic cells, with scant attention devoted to the checkpoint's importance in meiosis.

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