19. Slices were made from 9- to 13-day-old rats, and the CA1 region was injected with either BGVV or CaMKII(1–290)VV. The slices were incubated for 6 hours at 33° to 35°C. After incubation, the CA1 region was dissected and homogenized in buffer on ice. For the CaMKII assay, homogenization buffer contained 10 mM tris-HCl (pH 7.4), 1 mM EGTA, 0.5 mM dithiothreitol (DTT), 0.1 mM phenylmethanesulfonyl fluoride (PMSF) (20 μg/ml), leupeptin (5 μg/ml), and soybean trypsin inhibitor (20 μg/ml). Assays for CaMKII activity were carried out by adding an equal volume of assay buffer [100 μM syntide-2, 50 mM Hepes (pH 7.4), 20 mM MgCl, 1 mM DTT, 200 μM adenosine triphosphate (ATP) ([γ-^{32}P]ATP, 50 μCi/ml), 10 μM PKI(6–22)-amide, and 4 μM PKC(19–36)] to the homogenate and incubating at 30°C for 1 min. To determine the percent of Ca^{2+}-dependent phosphorylation, we performed the reaction in the presence of 2 mM Ca^{2+} and 3 μM calmodulin. The Ca^{2+}-independent phosphorylation was determined by adding 5 mM EGTA to the assay buffer. The homogenization buffer for PKC assays contained 20 mM tris-HCl (pH 7.5), 0.5 mM EGTA, 0.5 mM EDTA, 0.5% Triton X-100, aprotinin (25 μg/ml), and leupeptin (25 μg/ml). Assays were carried out by adding an equal volume of assay buffer [25 μM Ac-MBP(4–14), 20 mM tris (pH 7.5), 20 mM MgCl, 10 mM EGTA, 100 μM ATP ([γ-^{32}P]ATP, 50 μCi/ml), ± 20 μM PKC(19–36)] to the homogenate. For the PKA assay tissue was homogenized in buffer containing 50 mM tris-HCl (pH 7.5), 5 mM EDTA, and 0.5% Triton X-100. PKA assay buffer was 50 μM Kemptide, 50 mM tris (pH 7.5), 10 mM MgCl, bovine serum albumin (1 mg/ml), 100 μM ATP ([γ-^{32}P]ATP, 50 μCi/ml), ± 1 μM PKI(6–22)-amide). For PKC and PKA assays, tubes containing the inhibitor peptide were incubated at room temperature for 15 to 20 min to allow time for the peptide to bind. For PKC and PKA assays, reaction tubes were incubated at 30°C for 12 min. For all assays, the reactions were stopped by spotting half of the reaction mixture onto phosphocellulose disks and washing them in 1% phosphoric acid to remove unincorporated [γ-^{32}P]ATP. Initial experiments showed that the amount of radiolabeled phosphate incorporation was linear between 30 s and 16 min, and rate of phosphorylation was linear with protein concentrations of 0.29 to 12 μg/ml. Two to 9 μg were used for all assays. All measurements were made in triplicate. Basal PKC and PKA activities were calculated as the difference between phosphorylation of the substrate peptide in the presence and absence of inhibitor peptide. Average Ca^{2+}-independent activity in the absence of infection was 22 ± 1.8%. Slices, as well as cultured cells infected with BGVV, often showed a slightly increased level of kinase activity compared with uninfected tissue. This increase was not statistically significant and did not affect any monitored electrophysiological measure.
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23. The lowest stimulus used for each recording was the maximal stimulus that elicited an average transmission of less than 5 pA. This stimulus value varied for different experiments (Fig. 2, B and C) but was not significantly different in the groups infected with CaMKII(1–290)VV or BGVV (BGVV, 16 ± 2 V, n = 14 compared with CaMKII(1–290)VV, 17 ± 2.5 V, n = 16, $P > 0.75$; this includes data with and without APV). From this subthreshold value, stimulus strength was increased in 1- to 2-V increments. We attempted extracellular field recording experiments, but we were unable to get reliable synaptic potentials possibly because of the young age of these animals and the injection and incubation protocols.
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28. A short baseline period was used to prevent washout of LTP (*22*), which could theoretically occur at shorter times in CaMKII(1–290)VV-infected slices. To ensure stable transmission, we stimulated afferents for ~40 trials after gaining cell-attached patch configuration before gaining whole-cell access. The stability of transmission in the test and control slices after the pairing protocol attests to the generally stable conditions. Furthermore, the blind manner of conducting these experiments prevents bias possibly introduced by deciding when transmission is stable.
29. We cannot rule out the possibility that the CaMKII activity introduced by the virus is having effects on targets not accessible by endogenous CaMKII. However, presence of the much larger protein β-galactosidase in dendrites indicates that CaMKII(1–290) should readily reach synaptic targets.
30. Although comparisons among groups of slices is less reliable than within-slice comparison, the experiments were conducted in a blind fashion. This approach prevents any bias possibly introduced by handling of the slices, the relative positioning of recording and stimulating electrodes, or determination of subthreshold and minimal stimulus levels.
31. Although similar arguments have been put forth to support such a role for PKC, we feel the case is weaker for this enzyme because (i) postsynaptic injection of PKC was not shown to prevent further LTP (*7*) and (ii) experiments in which phorbol esters occluded LTP (*7*) a high drug concentration was used; lower concentrations appear not to occlude LTP [(*27*); D. Muller, J. Turnbull, M. Baudry, G. Lynch, *Proc. Natl. Acad. Sci. U.S.A.* **85**, 6997 (1988)].
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34. We thank J. Lisman, H. Cline, and the members of the Malinow and Perlman laboratories for helpful comments and technical assistance; A. Silva for help with protein kinase assays; E. Kandel for communication of unpublished data; R. Mauer for providing GH3 cells and constructs necessary for luciferase-based assays of CaMKII activity; and B. Moss for vaccinia viruses and vectors.

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TECHNICAL COMMENTS

Seasonal Precipitation Timing and Ice Core Records

C. D. Charles *et al.* performed global circulation model experiments of moisture source changes in Greenland (*1*). Their results speak to the risk of interpreting records of isotopic shifts strictly as "temperature," because changes in the location of dominant moisture sources for Greenland precipitation probably changed the effective $\delta^{18}O$-temperature relationship temporally and spatially between the last glacial maximum (LGM) and the present. In light of our preliminary findings from the GISP2 core, we would like to comment on the interpretation of the results of Charles *et al.* and urge caution in how they are applied because they have important implications for paleoclimate reconstruction from ice cores.

Local temperature does play an important role in $\delta^{18}O$ variations in polar ice cores (*2*). Such changes are usually interpreted as indicative of global climate change. The validity of this approach is borne out by the good agreement among paleotemperature records derived from ice cores, ocean sediments, terrestrial pollen records, and so forth. Yet, local isotope values may change as a result of factors other than temperature. Moisture source variability is undoubtedly one of these factors, but of potentially comparable importance is the seasonal variation in the timing of precipitation events, regardless of the source region. Unfortunately, the necessary sensitivity tests have not been run on the global circulation model to examine the importance of this factor (*3*).

Any atmospheric constituent that exhibits large seasonal changes and relatively small long-term changes will be sensitive to the seasonal timing of precipitation (*4*).

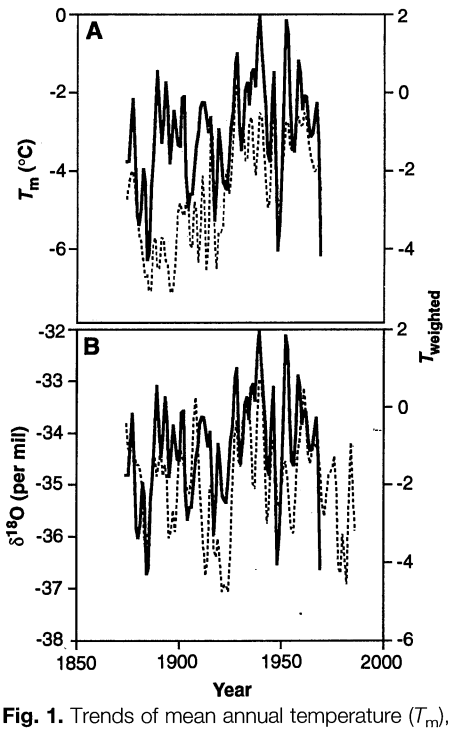

Fig. 1. Trends of mean annual temperature (T_m), accumulation-weighted temperature ($T_{weighted}$), at Jakobshavn, and $\delta^{18}O$ at Summit, Greenland, 1874 to 1970. (**A**) T_m (dashed line) compared with $T_{weighted}$ (bold line). (**B**) $\delta^{18}O$ (dashed line) compared with $T_{weighted}$ (bold line).

This certainly applies to $\delta^{18}O$ in Greenland, which exhibits a 20 per mil seasonality but a maximum long-term change on the order of 8 per mil. The magnitude of this effect can be seen if one compares the mean annual temperature (T_m) at Jakobshavn, Greenland, with the "precipitation-weighted" temperature (Fig. 1A):

$$T_{weighted} = \int_0^{1\ year} T_t \dot{C}_t dt \Big/ C_{annual}$$

where T_t and $\dot{C}_t$ are the temperature and accumulation rate, respectively, at time t. Even small changes in precipitation lead to a difference of several degrees Celsius between T_m and $T_{weighted}$. At Summit, Greenland, $\delta^{18}O$ more closely tracks $T_{weighted}$ than T_m.

A potential problem with attributing $\delta^{18}O$ shifts to changes in moisture source distribution is that the mechanisms required (for example, orographic steering by the Laurentide ice sheet) do not occur as rapidly as some observed $\delta^{18}O$ anomalies. On the other hand, plausible rapid changes in North Atlantic sea surface conditions (such as extent of sea ice) could cause significant changes in the subannual distribution of precipitation events.

Many of the artifacts that would be introduced into the ice core record because of changes in the location of moisture source regions would also result from changes in precipitation seasonality. For example, an increase in deuterium excess values (d), which could be interpreted as indicative of a change in source region sea-surface temperature or humidity, would also result from an increase in the ratio of summer as opposed to winter accumulation, because d exhibits a marked seasonal cycle with a late summer peak.

At present, anomalies in Greenland ice core records should not be interpreted solely in terms of source region variations. On the other hand, the emphasis by Charles *et al.* on the need for a multidimensional perspective in the interpretation of ice cores is important. The combined use of $\delta^{18}O$, δD, and ionic species in the new Summit, Greenland, cores, should make it possible to answer empirically some of the questions raised by GCM experiments as to the interpretation of $\delta^{18}O$ records in terms of temperature.

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Maximum Parasitism Rates and Successful Biological Control

Recently, B. A. Hawkins *et al.* (*1*) tested the refuge hypothesis (*2*) and found that maximum parasitism rates following release of parasitoids in exotic locations (an estimate of the host's refuge) were positively associated with successful control of insect pests. In a reply to criticisms by Myers *et al.* (*3*) and Williams and Hails (*4*), Hawkins *et al.* (*5*) further suggested that according to the hypothesis, maximum parasitism in a host's native region should also be associated with the ability of introduced parasitoids to depress host densities in exotic locations.

We tested this by documenting maximum parasitism rates within the native ranges of 58 species that were pests in exotic locations where they were subjected to biological control by means of parasitoid introductions (*6*). We found that the probability of successful control significantly increases with the maximum parasitism rate within the host's native range (Fig. 1). The substantial scatter around the regression line indicates that the probability of successful control is affected by other factors as well, including climatic and ecological differences between native and exotic locations and idiosyncrasies in the protocols followed for each parasitoid introduction (*2–4*). Despite these complications, maximum parasitism rate in native locations still provides a significant measure of the ability of parasitoids to depress host densities below economic injury levels when both hosts and parasitoids are exotic. We also note that there is a threshold for success; no control was achieved for any pest suffering less than 32% maximum parasitism in its native range (Fig. 1). A similar threshold between 33 and 36% was found for maximum parasitism rates in exotic locations (*1*). Consequently, successful biological control can be predicted in part by a relatively simple measure of a host's susceptibility to attack in either native or exotic locations.

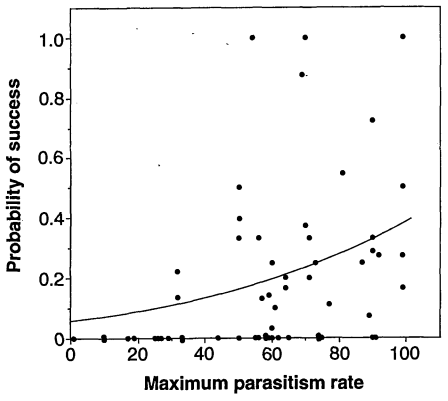

Fig. 1. Relation between maximum percentage parasitism within a host's native range and the probability of success for biological control in exotic locations. Maximum percent parasitism was tested against the number of successes (partial, substantial and complete pooled), weighted by the total number of attempts that have been made against each pest (failures + successes; 787 total introductions against all pest species). Analysis followed the protocols in the GLIM statistical package and was conducted by M. J. Crawley, Silwood Park. The line is described by $\text{logit}(y) = \ln(p/q) = -2.737 + 0.023x$, $\chi^2 = 12.11$, $df = 1$, $P < 0.001$.

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6. Maximum parasitism rates included attack by the entire parasitoid fauna and were obtained from primary literature sources and compilations of biological control projects. Outcomes of parasitoid introductions were obtained from the BIOCAT database, compiled by the CAB International Institute of Biological Control, Silwood Park, United Kingdom. The data and sources are available upon request.

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