much safer place to live in than the woods of temperate regions. Crises are rare; day after day passes in a peaceful, almost monotonous routine". Is it not possible that a similar pattern of life history mortality, high when very young, low thereafter, and a lack of seasonal changes, have enabled the evolution of high diversity both in the tropical forest and in the deep sea? If this is so, the critical specialisations would presumably be in minimising the early mortality in various ways, and small brood sizes may be a necessary part of these specialisations, by making detection by predators more difficult[4]. There would then be much less pressure for niche specialisation among the adults.

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Contemporaneity of late Cretaceous extinctions

THE dramatic extinctions at the end of the Cretaceous, both on land and in the sea, have long provoked speculation, but there is little direct evidence. Their existence is why the Cainozoic Era is distinguished from the Mesozoic, and in practice they are used in stratigraphy to make the Cretaceous–Palaeocene boundary. If the extinctions occurred at different times in different places, this practice would have to be revised.

Butler *et al.*[1] presented magnetostratigraphic evidence that dinosaur extinction in northern New Mexico occurred slightly later than the marine extinctions in central Italy. They seem to generalise this difference to all marine and terrestrial rock sequences, but this may be incorrect.

On the basis of extensive evidence of diverse kinds from Montana, we have been able to reconstruct ecologically the process of terrestrial extinctions there (refs 2–4 and unpublished). The time scale was ecologically very long but geologically very short (10^4–10^5 yr), just as in marine sequences[5,6]. The process, which affected marsupials almost as severely as dinosaurs but left the freshwater community entirely unaffected, was a protracted ecological succession which seems to have operated mainly by diffuse competition with placental and multituberculate mammals. This invading mammalian community[7,8] soon expanded worldwide to give rise to most of our familiar Cainozoic mammals.

We have predicted[2,3] that terrestrial extinctions farther south occurred later than those in Montana, but we were able to find[2] only two flimsy data relevant to

this prediction. Both were from South America and both supported the prediction. The extinctions in Montana were probably not the earliest, as dinosaurs occurred at least to about 75°N palaeolatitude[2,9] and we expect that dinosaurs disappeared here a little before they did near 50°N in Montana.

It would be a remarkable coincidence if the great marine and terrestrial extinctions did not have causal elements in common. We believe that these extinctions were in part synchronous but that the terrestrial extinctions moved south as the rapidly evolving placental mammals expanded out from their ancestral temperate community. Dinosaurs may well have survived into the Eocene in India[2], which was an island during the critical interval.

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Do fatty acids inhibit gibberellin-induced amylolysis?

BULLER *et al.*[1] reported that fatty acids inhibit gibberellin-induced amylolysis in barley endosperm. Their experiments as described do not show if the effect is on induction of amylase by gibberellin, on amylase activity, or on resistance of starch to amylase attack, although they discuss results as "the exceptional efficacy of pentanoic (C_5) and nonanoic (C_9) acids in overcoming the effect of GA . . . ".

Mitchell and Zillmann[2] showed that fatty acids increased flour and starch pasting ability, and some years ago I measured similar effects. In the Amylograph[3], with the naturally occurring enzymes inactivated[3], a wheat flour had maximum paste viscosity 1,205 Brabender Units (BU). Fatty acid additions (500 mg each to the mixture of 60 g flour and 440 ml water) gave the follow-

ing increases of maximum viscosity, confirming Mitchell and Zillmann's observations: C_6, 45 BU; C_7, 85 BU; C_8, 160 BU; C_9, 125 BU; C_{10}, 10 BU and C_{11}, 5 BU.

If the flour enzymes (including amylases) were not inactivated, then C_8 fatty acid had remarkably greater effect. Adding 500 mg (58 μmol per g flour, or 7.9 mM) increased viscosity from 495 BU to 975 BU. This effect for very low amylase activity suggests that fatty acid altered starch properties and made it more resistant to enzymatic breakdown. Commercial glyceryl monostearate (GMS) has a similar effect but GMS plus C_8 acid was no more effective than one component, nor did glyceryl monocaprylate strengthen starch more markedly than equimolar amounts of C_8 acid or GMS.

I suggest that fatty acids about C_8 can make starch more resistant to amylase degradation. Thus the effect demonstrated by Buller *et al.* could be of fatty acid making starch more resistant to amylolysis rather than an effect associated with gibberellin.

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BULLER AND REID REPLY—We have reported that saturated fatty acids of chain length around C_9 are potent inhibitors of gibberellin-triggered amylolysis in embryo-free barley halfgrains[1]. We are aware that the efficacy of these acids "in overcoming the effect of GA" might be due either to their interfering with some aspect of the production, activation or secretion of amylolytic enzymes or to their inhibition of the amylolytic enzymes themselves. We are now investigating the biochemical mechanism(s) of fatty acid action.

In experiments carefully controlled to eliminate fatty acid effects on amylase activity in our assay systems we have observed that the fatty acids of chain lengths C_7, C_8 and C_9 at 5×10^{-4} M completely inhibit the release of amylase activity from gibberellin-treated barley aleurone layers. This suggests that the fatty acids can interfere, directly or indirectly, with amylase 'induction' or release.

Yet fatty acids can also inhibit the activity of amylolytic enzymes. Nonanoate (C_9) at 10^{-3} M reduces by about 60% (relative to a citrate control) the