Danian coccoliths in the alleged 'mainly redeposited Maastrichtian sediments' at Ekofisk. They also do not recognize that nuances of the North Sea Central Graben stratigraphy are not germane to the Arctic Ocean spillover model except to the extent that they triggered the thinking process that ultimately led to the formulation of that model.

With regard to the passage between the Arctic Ocean and the Pacific, Clark and Kitchell impute temporal and spatial precision to reconstructions that are not warranted. If the size of the gap between Alaska and Siberia was significant prior to 60 to 63 million years ago (*4*), then presumably, the gap became insignificant at that time. Tarling and Mitchell (*5*) assign an age of 64 million years to the Cretaceous-Tertiary boundary; McDougall (*6*) has determined an age of 62.9 to 64.9 million years for basalts overlain by more than 100 m of late Maastrichtian sediment on Ninetyeast Ridge. If we allow for a possible error of a few percent in the above dates and estimates, a sufficient time overlap exists between the various events to accommodate the needs of the Arctic spillover model.

The silicoflagellates from core 437 have been restudied by Bukry (*7*) who concludes that the age of the assemblage cannot be fixed more closely than Campanian or Maastrichtian, that " . . . the assemblages of core 437 result from abnormal marine conditions or/and different age than the other known assemblages", and that " . . . this and other marine silicoflagellate assemblages in the Arctic area does not preclude a brief terminal Cretaceous freshening event." These are substantially the same conclusions advanced earlier (*8*). Another conclusion that is still valid is that data from core 422 are irrelevant, other than to underscore the prediction of the Arctic spillover model that the Arctic Ocean was indeed a normal marine body in early Paleocene time; this, incidentally, would be unlikely if Clark and Kitchell's reasoning is followed to a logical conclusion (that is, the Bering Strait closed 60 to 63 million years ago and the passage to the North Atlantic did not open until 58 million years ago). Turtelot and Rye's (*9*) data are of consequence in the Arctic spillover model primarily because the data suggest the capacity of the latest Cretaceous precipitation and runoff pattern to achieve a flushing of the Arctic Ocean.

With regard to the timing of the opening between the Arctic Ocean and the North Atlantic, Clark and Kitchell do not distinguish between rifting and crustal accretion at spreading ridges. It is the latter that is recorded by magnetic anomalies; the former must precede the latter. In the Greenland-Norwegian Sea, as in the Eurasian Basin of the Arctic Ocean, there exist strips of "deep water" crust landward of magnetic anomaly 24; these strips are of sufficient width to accommodate anomalies 25 through 28, although in both cases these anomalies cannot (as yet) be recognized (*10, 11*). If these strips of "deep water" crust do represent initial stages of spreading, then crustal accretion began only a scant million years or so after the terminal Cretaceous catastrophe. Rifting, of course, must have come even earlier. To attempt placing tighter time constraints on rifting between Greenland and Norway is, at this time, unrealistic.

As to the volume of the latest Cretaceous Arctic Ocean, most reconstructions require the Amerasian Basin to be underlain by very old crust, crust that probably was already in thermal or isostatic equilibrium in latest Cretaceous time (*11, 12*). The very large amount of post-Cretaceous sediment fill, therefore, probably has reduced the volume of the basin. Similarly, nearly all reconstructions require some post-Cretaceous compression of the Amerasian Basin. A best estimate for the volume of the latest Cretaceous Arctic Ocean is that it may have been less than the volume of the present-day Arctic Ocean but probably was substantially greater than the present volume of the Amerasian Basin alone. Three-fourths of the present volume does not seem excessive; but even half the volume of the present Arctic Ocean, given a favorable mixing model with normal seawater, would be more than adequate to achieve the kill of the stenohaline surface plankton.

Although none of the arguments advanced by Clark and Kitchell (*1*) points to a fatal flaw in the Arctic spillover model, it remains a model, nevertheless, yet to be tested.

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Food Colors and Behavior

It would be unfortunate if the data presented by Weiss *et al.* (*1*) encouraged professional nonspecialists and parents to believe that there is a strong association between food colorings in the diet and what Weiss terms "problem behaviors." The danger of misinterpretation of the results of this study derives in part from the authors' interpretation that the data "further strengthen the accumulating evidence . . . that modest doses of synthetic colors . . . can provoke disturbed behavior in children" (*1*, p. 1488). One clearly responsive child out of the 22 studied represents no more than a rare case of food-color sensitivity. The rarity of this single responder is far greater than 1 out of 22, since the children studied were preselected as "responders" on the basis of open trials with the Feingold diet. If the 22 "responders" represented 50 percent of the subject population in the open trial (a figure frequently mentioned by Feingold) and, further, if this subject population of children exhibiting "problem behaviors" represents roughly 5 percent of the general childhood population (*2*), the finding of one responsive child indicates that about 0.114 percent of the preschool and elementary school children in the United States may be sensitive to food coloring. This is not meant to imply that a disorder occurring at a rate of about 1/1000 is unimportant: on the contrary, if it were not for the concern for such rare disorders, diseases such as phenylketonuria would not today be a manageable disease. However, this still leaves nearly 98 percent of the disturbed children exhibiting "problem behaviors" of unidentified origin, and indicates that the "Feingold hypothesis" has received undue attention.

The study of Weiss *et al.* (*1*) also raises methodological issues. For example, if the children in the study had not been diagnosed as hyperkinetic, why had they been on the Feingold diet? If they were not hyperkinetic nor had any diagnos-

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able psychiatric problem, what was meant by the term "problem behaviors." Nowhere was the subject population clearly described.

The strategy of allowing each parent to pick items from five different behavioral checklists leads irrevocably to a case study approach; that is, 22 "studies," each with an N of 1. Such an approach precludes the possibility of deriving scientific generalizations. Moreover, each of the behavioral rating scales alone, administered according to standardized procedures, has known reliability, validity, and factor loadings. What was the validity of using these diverse items in nonstandardized combinations? And what kinds of items did the parents select as being "aversive" or "positive"? Such information was lacking.

That teachers' observations were discarded when they proved "undependable" was an unfortunate design decision: teachers would probably have been less biased than the parents who had already put their children on the "Feingold diet."

It is also disturbing that in the one child who responded "dramatically" to the color challenges, the mother as data collector knew when her child had received the challenge, ostensibly because of the child's behavior, but other hypotheses cannot be ruled out, for example, that the mother discovered some physical symptom in the child or some telltale sign in the beverages that provided clues to the presence of the colorings. In addition, the time between ingestion of the drink and parental ratings was not rigidly controlled, but allowed to vary randomly over a period of up to 3.5 hours after consumption of the test beverage. Thus, potentially valuable data on the time course of the responses under investigation were, apparently, not collected.

At best, the data of Weiss *et al.* suggest that sensitivity to food coloring is a rare phenomenon: at worst, the methodological problems prevent them from adding to our understanding of the issue of food colors and behavior.

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Brunner *et al.* (*1*) ascribe to us (*2*) a position we did not adopt, then attack it. We never claimed to be testing the therapeutic efficacy of the Feingold diet. Nor did we claim to be estimating population parameters. We wrote:

> A clinical trial like ours is not a group experiment, but 22 separate experiments. Our aim was not to estimate population prevalence or sensitivity, but simply to determine if behavioral sensitivity to color additives could be demonstrated in a controlled trial.

Our statement that "these data further strengthen the accumulating evidence . . . that modest doses of synthetic colors . . . can provoke disturbed behavior in children," is hardly radical. Two severe critics of Feingold have issued statements not that disparate. Lipton *et al.* (*3*), although disputing the breadth of Feingold's claims, also note that "on the other hand, the available evidence suggests that some hyperactive children benefit from the additive-free diet and react adversely to the color challenge." Stare *et al.* (*4*) observed that "the diet crossover data appear to indicate that the Feingold regimen does have a positive impact on behavior of some hyperactive children." They also wrote that "specific challenge experiments indicate that the symptoms of a small subgroup of all hyperactive children appear to be sensitive to the artificial food colors in their diet."

The prevalence calculations by Brunner *et al.* are a bizarre montage of assumptions. Prevalence is in the eye of the beholder. Behold: Swanson and Kinsbourne (*5*), in the same issue of *Science*, reported adverse effects of food dyes in 17 of 20 hyperactive children (85 percent). And the largest study of the Feingold elimination diet (*6*) found that all ten boys in a preschool sample improved on the diet (100 percent). Animal studies also reveal behavioral toxicity from low doses of food dyes, especially in neonates (*7*).

Table 1. Comparison of daily intake of food dyes (in milligrams).

Color	Weiss *et al.* (*2*)	ADI*
Blue 1	0.80	300
Blue 2	0.15	37
Green 3	0.11	150
Red 3	0.57	150
Red 40	13.80	420
Yellow 5	9.07	300
Yellow 6	10.70	300

* Allowable daily intake of food dyes person (data from the Food and Drug Administration).

Some criticisms by Brunner *et al.* are due to a hasty reading of the report. For example, we did include a standard measure: Conners Scale ratings [see figure 2 and reference 9 in (*2*)]. Others arose because complicated studies cannot wholly be compressed into a *Science* report. But Butcher attended a meeting at which I discussed these issues (*8*).

Brunner *et al.* refuse to grasp the toxicologic implications of the Feingold literature. Behavioral testing is not a standard element in food additive safety evaluation, and this neglect has led to the situation depicted in Table 1. The amount of colors permitted in the diet, according to conventional toxicity assessment, is about 50 times the amount eliciting behavioral reactions in some children. Not only has the current safety margin evaporated, but, as noted by Stare *et al.* (*4*), sensitivity "appears to be concentrated among younger participants." Can alleged specialists in behavioral teratology remain smug about such an inference?

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