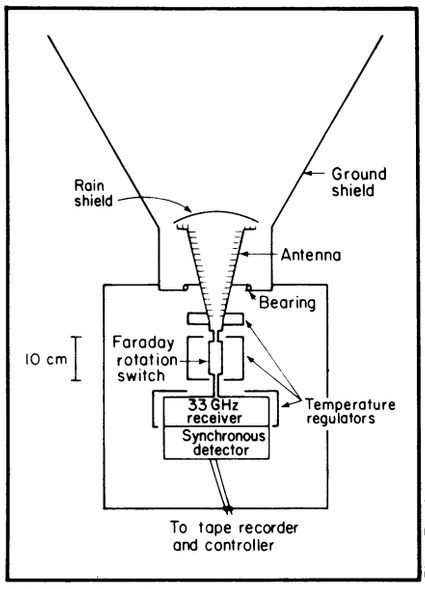
An isotropic cosmic past, maybe

Since its discovery in 1965 the universal cosmic radio background, the radiation that has the spectrum of a blackbody at 3° K and comes from every quarter of the universe, has more or less dominated cosmology. It has set the limits within which cosmological models must stay, and it offers the opportunity to learn about the earliest times of the universe.

The blackbody is believed (or hoped) to represent a kind of universal fundament, smooth and evenly moving, expanding with the expansion of the universe. So if anyone discovers the slightest irregularity in the blackbody, it becomes a matter of widespread comment and cogitation. Among those that have called up a widespread reaction was the report about two years ago by George F. Smoot and collaborators of the Lawrence Berkeley Laboratory in California that there is a slight temperature distinction. Looking in one particular direction, the spectrum seems 3.5 millidegrees different from other directions. This has been interpreted as indicating a motion of the earth with respect to the blackbody, a motion of the earth superimposed on the expansion of the universe (SN: 7/16/77, p. 36).

The question then arises: How evenly is the expansion proceeding? Putting the question another way: How isotropic is the universe? Assuming that the temperature anomaly already measured is entirely due to the earth's motion gives a figure for the intensity of the radiation that would indicate an intrinsically isotropic blackbody. But are the investigators subtracting too much as being due to terrestrial motion? An independent check on the intrinsic isotropy of the blackbody would come from the polarization of the radiation.

An antenna to study polarization of the blackbody was set up on the roof of the building in Berkeley where the astrophysics group's offices are. So far it has observed three circles around the celestial north pole, at declinations 38°, 53° and 63°. So far no polarization anisotropies have been found, Philip M. Lubin and Smoot report in the Jan. 8 PHYSICAL REVIEW LETTERS. But this is not their last word. "We're still looking," says Lubin.



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Blackbody polarization antenna.

The polarization of the blackbody comes from a time when the universe consisted mostly of hot electrons, protons and the photons or light particles that are the blackbody. As the photons roamed the universe, they would collide with the electrons, and be scattered by them. Each scattering changed the polarization of a photon. Gradually, as the universe expanded and cooled, the electrons joined the protons to form hydrogen atoms, and scattering of the photons ceased. The photons retained whatever polarization they had, and those that have survived retain it still. Any excess of motion of the universe in one direction or the other that may have been present when the scattering was going on will show up in the polarization of a large number of photons as a departure from a net zero polarization as the antenna looks the right way.

Isotropy or anisotropy is the difference between a universe that expands in the multidimensional equivalent of a sphere and one that expands with twists, pancake shapes or alternating ins and outs like kneading dough. The last have been called mixmaster universes; the present generation might understand better if the term were Cuisinart universes. □

Dietary cholesterol and colon cancer

First dietary cholesterol was identified as a heart attack risk factor. Then cholesterol endogenous to the body was suggested as a protection against cancer (SN: 12/30/78, p. 439). And now, even more provocative and baffling, at least in view of the above finding, dietary cholesterol may play a role in colon cancer.

Although colon-rectal cancer is the most common internal cancer in the United States, its cause or causes are unknown. Western dietary factors, however, appear to be implicated since high-fat diets correlate with the geographic incidence of colon cancer. What's more, patients with colon cancer have high levels of fecal cholesterol and fecal bile acids (which derive from fecal cholesterol), and there is evidence suggesting that fecal cholesterol and bile acids might be culprits in colon cancer. Now J. P. Cruse and colleagues at University College Hospital Medical School in London have explored dietary cholesterol's possible ability to cause or help colon cancer. Specifically, they tested dietary cholesterol's ability to enhance the carcinogenicity of a chemical known to induce colon cancer experimentally. This was DMH (dimethylhydrazine).

The researchers gave one group of rats a cholesterol-free, chemically defined liquid diet, another group of rats the same diet with cholesterol and still a third group of rats a standard formula diet (in order to serve as a control to the other two groups). By design all three diets were fiber-free, since dietary fiber may protect against colon cancer (SN: 2/18/78, p. 104), and provided equivalent caloric intakes. After two weeks on their respective diets, half the animals in each group were injected with the carcinogen DMH weekly for 13 weeks. The other half in each group served as controls and received equivalent amounts of saline. All of the animals were kept on their particular diets for the rest of their lives.

The results were analyzed after 56 weeks of observation. DMH-treated animals fed on the cholesterol-free liquid diet showed a highly significant delay before developing tumors and a highly significant increase in survival compared with DMH-treated animals fed the solid diet. This finding suggested that the liquid diet protected against DMH-induced cancer. On the other hand, when both groups of rats on the liquid diet were compared, the group receiving cholesterol developed tumors much faster and had shorter survival times than did the group not getting cholesterol. This result implied that the addition of cholesterol to the liquid diet not only abolished the ability of the diet to protect against DMH-induced colon cancer but enhanced the development and spread of DMH-induced colon cancer. Still further evidence that cholesterol acted as an accomplice to DMH rather than inducing colon cancer alone lies in the fact that rats receiving cholesterol and a liquid diet but not DMH remained tumor free and alive. The researchers conclude on the basis of the above findings that "dietary cholesterol is a factor determining the rate of development of experimental colon cancer. If this applied to man, as the metabolic evidence would suggest, then human colon cancer might be a preventable disease."

However, Cruse and his co-workers do not elaborate on how human colon cancer might be prevented. Could a diet low in cholesterol be the answer? Or would a diet low in cholesterol but high in dietary fibers be even more protective, since lack of fiber in the diet has also been linked with colon cancer (SN: 2/18/78, p. 104)? The Finns eat a diet as high in cholesterol as that of Americans, yet they have a much lower incidence of colon cancer, apparently because they consume a lot of rye bread (a type of fiber) with their meals (SN: 7/15/78, p. 40). □