

numbers $k < (l_0)^{-1}$, and is proportional to $k^{-\alpha}$ for larger wave numbers. Jokipii and Lerche⁷ give $l_0 = 150$ pc, and find an average interstellar field of 3×10^{-6} gauss, so for ~ 5 GeV per nucleon particles, $r_g/l_0 \sim 1.4 \times 10^{-8}$. Because this ratio is so small, K_1 as determined from equation

Table 1 Adopted parameters

Parameter	Nominal value	Range
n	1 cm^{-3}	0.2–2
L	$1.5 \times 10^3 \text{ pc}$	1.0–3.0
λ	5 g cm^{-2}	3–8
l_0	$1.5 \times 10^2 \text{ pc}$	0.5–2.0
B_0	$3 \times 10^{-6} \text{ gauss}$	2–5

(2) is very sensitive to the exponent α of the magnetic-field power spectrum. The values calculated from equation (2) are given in Table 2 for several values of α . The uncertainties given in Table 2 are obtained by varying the parameters through the range shown in Table 1. Comparing the ‘observed’ result from equation (1) with the entries in the table, one concludes that

$$\alpha = 1.5 \pm 0.2 \quad (3)$$

if the simple diffusive model of cosmic-ray transport is appropriate. The uncertainty quoted in equation (3) is the worst-case limit; the statistical uncertainty would be much less.

This value of the power spectral index ($\alpha \sim 1.5$) of the galactic magnetic

Table 2 Calculated diffusion coefficients

α	$K_1(\text{cm}^2 \text{ s}^{-1})$
1.2	$(5.3 \pm 3.0) \times 10^{24}$
1.4	$(1.5 \pm 0.8) \times 10^{26}$
1.5	$(1.0 \pm 0.5) \times 10^{27}$
1.6	$(6.9 \pm 4.0) \times 10^{27}$
1.8	$(4.6 \pm 3.0) \times 10^{29}$

field is the same as the exponent of the interplanetary field and close to the Kolmogorov value of 5/3. The argument here includes the assumption of one continuous power law from the correlation scale of the fluctuations (150 pc) all the way down to the resonant wavelength for 5 GeV per nucleon particles (2×10^{-6} pc). For comparison, Somogyi obtains $\alpha = 1.7 \pm 0.1$ in the simple diffusion model for cosmic-ray transport with the distance to the boundary of the diffusing region independent of energy. His result is based on the resonant wavelengths for 10^{12} – 10^{15} GeV per nucleon particles, or scales of 4×10^{-4} – 0.4 pc.

Somogyi’s result and that of equation (3) can be shown to agree even more

closely if one adopts his model and changes his parameters slightly. If the energy dependence of the cosmic-ray pathlength $\lambda(E) \sim E^{-\beta}$, with $\beta = 0.50 \pm 0.1$, as recent calculations indicate⁸, rather than $\beta = 0.2 \pm 0.1$ as adopted by Somogyi, then his results indicate that the size of the confinement volume does not depend on energy, and that $\alpha = 1.55 \pm 0.1$, in agreement with equation (3). Since equations (1) to (3) yield $\beta = 2 - \alpha$, the calculations in this letter predict $\beta \sim 0.5$, in agreement with the conclusions of Juliusson *et al.*⁸.

Thus the method presented here gives the result $\alpha = 1.5 \pm 0.2$, and predicts that the cosmic-ray pathlength varies as E^{-1} . Adopting this dependence of pathlength on energy, as supported by recent observations, one can use Somogyi’s results to obtain $\alpha = 1.55 \pm 0.1$ and to show that the size of the diffusing region is independent of energy. The similarity of these two results, based on quite different assumptions, suggests that cosmic rays may be a useful tool for probing the interstellar magnetic field.

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A. J. OWENS

*Department of Physics,
Lake Forest College,
Lake Forest, Illinois 60075*

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SOMOGYI REPLIES—The value of α as derived by Owens¹ is based on a single point of the function expressed by his equation (2), and Owens assumed that the magnetic power spectrum had the form of a power function with a constant exponent in the range $2 \times 10^{-6} < k^{-1} < 150$ pc. In my paper², indications are given that the anisotropy and lifetime are power functions of energy, and it is proved that in this case the magnetic power spectrum is a power function at least in the range $10^{-3} \text{ pc} < k^{-1} < 1 \text{ pc}$ corresponding to the energy range $10^{12} \text{ eV} < E < 10^{15} \text{ eV}$ of anisotropy measurements with large statistical accuracies. The range of k as given in my paper² is thus an experimentally established one.

It would be difficult to argue which of the two α values is better established. Both are rather uncertain. I agree with

Owens that their agreement is remarkable, especially if recognising that they are based on different experimental evidences independent of each other.

*Central Research Institute of Physics,
Budapest, Hungary*

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How specific are nuclear ‘receptors’ for thyroid hormones ?

TATA has questioned¹ the biological relevance of the binding of T_3 to the nucleus. He presents four main arguments against a physiological role for the T_3 binding components in the nucleus. First, the presence of high-affinity, saturable binding sites for thyroid hormones of similar characteristics in a number of other subcellular fractions. Second, the lack of analogy with steroid hormone receptors. Third, the absence of parallelism between the binding of thyroid hormone analogues to the nucleus and the biological activity. Fourth, the fact that only 15–20% of the intracellular triiodothyronine is located in the nucleus.

We believe that several of Tata’s conclusions are invalid.

On the first point. The K_a of the binding of T_3 by the nucleus has been incorrectly cited from the work of Samuels and Tsai² (Tata’s ref. 19). The correct value is $3.3 \times 10^{10} \text{ mol}^{-1}$ ($K_d = 29 \text{ pmol}$) for the nuclei of GH₁ cells. The same high K_a has been found³ for the binding of T_3 to the nucleus of human lymphocytes and rat liver and kidney nuclei⁴. The fact that these values are much higher than those reported by Tata for the other subcellular components favours the possibility of a physiological role for this type of binding site within the nucleus.

With the technique used by Tata to measure specific binding (his Fig. 2) it is impossible to show high affinity binding sites in the nucleus, because the tracer concentration used is 60 times the maximal binding capacity in the incubation mixtures (binding capacity $2.9 \text{ fmol } T_3 \text{ per } 100 \mu\text{g DNA}$; see ref. 4).

Concerning the second point, the lack of analogy with steroid hormone receptor binding has been reported by Surks *et al.*³ (Tata’s ref. 17) and Visser *et al.*⁴. Why does this lack of analogy imply that thyroid hormone binding in the nucleus is without physiological relevance? Why should the nuclear binding of chemically different substances proceed along identical lines?