COMPARISON OF RED RECTANGLE EMISSION LINES WITH LABORATORY WAVELENGTHS FOR CH^+

Observed wavelength (Å)	Laboratory wavelength (Å)	Assignment	Difference (Å)
4,225.82	4,225.70	$R(3)$	0.12
4,227.14	4,227.06	$R(2)$	0.08
4,229.45	4,229.35	$R(1)$	0.10
4,232.70	4,232.55	$R(0)$	0.15
4,237.67	4,237.56	$Q(1)$	0.11
4,239.49	4,239.38	$Q(2)$	0.11

Observed data from ref. 3; laboratory data from ref. 4.

In studies of CH^+ in diffuse interstellar clouds, the $R(0)$ absorption line at 4,232.55 Å alone is detected because the temperature is sufficiently low for only the lowest rotational energy level to be populated. The observation of six lines of CH^+ from the Red Rectangle shows that the molecule is warmer than in diffuse clouds.

Using published quantum mechanical line strengths[5] we have determined the rotational temperature of CH^+ to be 120 ± 50 K, using a least-squares fitting routine. The observed (calculated) intensities of the lines relative to the $Q(1)$ line are: $Q(1)$ 1.0 (1.0), $Q(2)$ 0.9 (1.0), $R(0)$ 0.6 (0.7), $R(1)$ 0.6 (0.6) and $R(2)$ 0.5 (0.4). The value of about 120 K is very low compared with the temperature of the star at the centre of the Red Rectangle nebula; moreover, the CH^+ linewidths of about 0.25 Å are two to three times narrower than those of the stellar absorption lines of atomic carbon. These data confirm that the molecule is in the surrounding nebula rather than the star.

Although CH^+ has been known in interstellar clouds for many years, it appears that the Red Rectangle is the only nebula in which CH^+ has so far been detected. The identification of CH^+ in the Red Rectangle, coupled with the recent recognition of some of the unassigned 'diffuse interstellar bands' in emission from the same nebula[6,7], leads to the interesting question of whether the high abundance of CH^+ in diffuse clouds might be related to the presence of diffuse band carriers in these clouds.

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Coley's vaccine and TNF therapy

SIR — C. O. Starnes in his Commentary[1] drew attention to the remarkable success that Coley achieved around the turn of this century in the treatment of cancer patients with his 'mixed toxins'. On the basis of an analysis of the historical records, Starnes proposed that bacterial toxin-related and tumour necrosis factor (TNF)-related treatments should be restricted to those patients suffering from sarcoma or lymphoma, and that further efforts should be concentrated on a search for so far unidentified factors made in response to Coley's vaccine. We agree with him and with Rook[2] that TNF does not account for all the effects observed after administration of Coley's toxins, but we nevertheless also believe that Starnes' conclusions may in some respects have been misleading.

First, despite the relatively disappointing results obtained during earlier clinical trials in which TNF was used as an ordinary chemotherapeutic agent, the often dramatic beneficial results obtained in recent clinical trials with high-dose TNF in isolated limb perfusion[3], in the treatment of ascites[4], or other forms of loco-regional treatment[5], leave little room for doubt about the clinical relevance of TNF (preferably in conjunction with other cytokines) as an anticancer treatment.

It is to be hoped that we will learn how to control the shock-inducing effect in a selective way so that these results may be extended to treatments involving the administration of TNF in the general circulation.

Second, in addition to the animal studies showing beneficial effects in sarcoma models, other animal studies in which TNF was used in conjunction with interferons showed a similar beneficial effect for melanomas[6,7] and for carcinomas[8]. More important, isolated limb perfusion of human patients shows that not only sarcomas, but also melanomas and squamous cell carcinomas[9], respond to the antitumour activity of TNF (plus interferons). In an isolated limb perfusion trial involving 52 melanoma patients, 90% complete remission and 10% partial remission were obtained. The antitumour effect involves activation of polymorphonuclear leukocytes and of the endothelial cell bed, with overexpression of adhesion molecules, which is much stronger in the tumour vasculature than in the normal one. This results in coagulative and haemorrhagic necrosis exclusively limited to the tumour tissue.

Although it is correct that a better patient selection might be important in improving the results obtained with TNF-related treatment, we nevertheless believe that such a selection should not be based primarily on the tumour histology, but rather on (as yet insufficiently identifiable) parameters predicting the susceptibility towards the systemic toxicity of TNF, properties of the tumour and its microvascular system, and perhaps on previous treatment. Extensive research on the mechanism of action of TNF (alone or combined with interferons) at the cellular level has provided no evidence for a correlation of the susceptibility with the embryological origin of the cell, but rather with the degree of malignancy[10].

In our view, it would be unfortunate to deny this treatment to those melanoma, sarcoma and carcinoma patients who do qualify for treatment; protocols and conditions need to be further explored.

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