radio frequencies[3] the spectral index is $-0{\cdot}25$ and the flux at 960 Mc/s is $5{\cdot}0 \times 10^{-25}$ W m^{-2} (c/s)$^{-1}$.

Between 6000 Å and 10,250 Å, eleven 120-Å bands were measured with an accuracy of 10 per cent. These measures indicate that the relatively flat energy distribution given in the equation here applies as far as 8400 Å. Beyond 8400 Å the flux may increase significantly. Between 3300 Å and 8400 Å the energy distribution cannot be represented, even approximately, by the flux from a black-body or a normal star. At least part of the optical continuum radiation must be synchrotron radiation.

During the course of the infra-red observations a strong emission feature was found near 7600 Å. Further observations with a 50-Å band-width placed the emission line at 7590 Å with a possible error of about 10 Å. The emission profile was found to be similar to that of the emission line at 5632 Å. Using this line and others in the visual spectrum Schmidt has shown that the most prominent emission features are Balmer lines and that the line at 7590 Å is $H\alpha$. Using Schmidt's red-shift $\Delta\lambda/\lambda_0$ of 0·158, $H\alpha$ should appear at 7599 Å; this is in satisfactory agreement with observation, when it is recalled that the atmospheric A band absorbs strongly beyond 7594 Å. It is possible that the [NII] lines which have unshifted wave-lengths of 6548 Å and 6583 Å can contribute to the emission feature identified as $H\alpha$. A large contribution, however, would shift the line significantly towards the red. The relative positions of $H\alpha$, $H\beta$, $H\gamma$, and $H\delta$ cannot be produced by applying a red-shift to any other hydrogen-like ion spectrum.

Further observations, particularly in the infra-red, will be made in the near future.

[1] Oke, J. B., *Astrophys. J.*, **131**, 358 (1960).
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RED-SHIFT OF THE UNUSUAL RADIO SOURCE: 3C 48

By Dr. JESSE L. GREENSTEIN
Mount Wilson and Palomar Observatories, Carnegie Institution of Washington, California Institute of Technology
AND
Dr. THOMAS A. MATTHEWS
Owens Valley Radio Observatory, California Institute of Technology

THE radio source 3C 48 was announced to be a star[1] in our Galaxy on the basis of its extremely small radio diameter[2], stellar appearance on direct photographs and unusual spectrum. Detailed spectroscopic study at Palomar by Greenstein during the past year gave only partially successful identifications of its weak, broad emission lines; the possibility that they might be permitted transitions in high stages of ionization could not be proved or disproved. Hydrogen was absent but several approximate coincidences with He II and O VI were suggested.

The discovery by Schmidt (a preceding article) of much broader emission lines in the apparently stellar radio source, 3C 273, suggested a red-shift of 0·16 for 3C 273 if the lines were interpreted as the Balmer series. In 3C 48 no such series was apparent; measurable lines still do not coincide with the hydrogen series. However, the possibility of a very large red-shift, which had been considered many times, was re-explored successfully. 3C 48 has a spectrum containing one very strong emission feature near λ3832 which is 35 Å wide and about 10 other weaker features near 23 Å in width. The sharper lines are listed in Table 1 in order of decreasing intensity. Some broad lines or groups of lines between 50 and 100 Å width may be red-shifted hydrogen lines.

Table 1. Identifications and Observed Red-shifts

Wave-length λ*	Wave-length λ lab.		Source	λ*/λ lab.
3832·3	2796 } 2803 }	2798	Mg II	1·3697:
4685·0	3426		[Ne V]	1·3676
5098	3729 } 3726 }	3727	[O II]	1·3679
4575	3346		[Ne V]	1·3673
5289	3868		[Ne III]	1·3671
4065·7	2975:		[Ne V]	1·3667:

The weighted mean red-shift $d\lambda/\lambda_0$ is $0{\cdot}3675 \pm 0{\cdot}0003$, an apparent velocity of +110,200 km/sec. The slightly discrepant value for λ2975 of [Ne V] is compatible with the uncertainty of ± 3 Å in the wave-length predicted by Bowen[3]. The Mg II permitted resonance doublet has a small additional displacement to longer wave-lengths, possibly caused by self-absorption in an expanding shell; it is the strongest emission line in the rocket-ultra-violet spectrum of the Sun. The forbidden lines are similar to those in other intense extragalactic radio sources.

So large a red-shift, second only to that of the intense radio source 3C 295, will have important implications in cosmological speculation. A very interesting alternative, that the source is a nearby ultra-dense star of radius near 10 km containing neutrons, hyperons, etc., has been explored and seems to meet insuperable objections from the spectroscopic point of view. The small volume for the shell required by the observed small gradient of the gravitational potential is incompatible with the strength of the forbidden lines.

The distance of 3C 48, interpreted as the central core of an explosion in a very abnormal galaxy, may be estimated as $1{\cdot}10 \times 10^9$ parsecs; the visual absolute magnitude is then −24·0, or −24·5 corrected for interstellar absorption. The minimum correction for the effect of red-shift is of the order of 2 v/c and a value between 4 and 5 times v/c is probable for a normal galaxy. The absolute visual magnitude of 3C 48 is then brighter than −25·2 and possibly as bright as −26·3, 10–30 times greater than that of the brightest giant ellipticals[4] hitherto recognized, which are near −22·7 and another factor of five brighter than our own Galaxy, near −21·0.

As a radio source at a distance of $1{\cdot}1 \times 10^9$ parsecs 3C 48 is not markedly different from other known strong radio sources like 3C 295 or Cygnus A. The one feature in which it does differ from most sources is in its high surface brightness. This is partially due to its extremely small radio size of ⩽ 1 sec of arc[2]. The optical size is comparable, being also ⩽ 1 sec of arc[5]. At the assumed distance such angular sizes indicate that both the optical and radio emission arise within a diameter of ⩽ 5500 parsecs. The radio diameter might even be comparable with or less than that of 3C 71 (*NGC* 1068) the diameter of which is about 700 parsecs. However, 3C 71 has 5 orders of magnitude less radio emission.

If we determine the integrated radio emission of 3C 48 from the observed spectral index of the radio spectrum, and correct for the red-shift, we find that 3C 48 is comparable with 3C 295, emitting 4×10^{44} erg/sec of radio-frequency power. The cut-off frequencies were 7×10^7 c/s and 10^{11} c/s. The lower limit is indicated by the observed radio spectrum and the upper limit is an assumed one.

The absolute magnitudes of the galaxies connected with 3C 295 and Cygnus A, corrected for interstellar absorption, are $M_v = -21{\cdot}0$ and $-21{\cdot}6$ (using a red-shift correction of 2 v/c) or $M_v = -22{\cdot}4$ and $-21{\cdot}8$ (using a correction

of 5 v/c) respectively. Thus 3*C* 48 radiates about 50 times more powerfully in the optical region than other more normal but intense radio galaxies. In contrast, the absolute radio luminosity of 3*C* 48 is the same as that of Cygnus *A* and 3*C* 295. The unusually strong optical radiation may be synchrotron radiation as suggested (for other reasons) by Matthews and Sandage[5].

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HUMAN CANCER: MENDELIAN INHERITANCE OR VERTICAL TRANSMISSION?

By Dr. P. R. J. BURCH

Medical Research Council, Environmental Radiation Research Unit, Department of Medical Physics, University of Leeds, The General Infirmary, Leeds 1

THE purpose of this article is to consider whether the familial tendency to malignant disease should be attributed to genetic inheritance or to the transmission of an oncogenic virus from either parent to offspring ('vertical' transmission[1]). It will be shown that this is one of those situations, rare in cancer research, where the available evidence is adequate to allow almost unambiguous conclusions to be drawn about an issue of fundamental importance.

Following many demonstrations of the vertical transmission of leukæmogenic viruses in mice, Gross[2] has recently stated: " . . . one could therefore regard the development of cancer, leukæmia, or that of any other [the printed form is not 'any other' but 'another': it is presumed that 'any other' is intended] malignant tumour, as the result of an activation, frequently merely accidental of an oncogenic agent, hitherto masked, and carried by the host since birth. . . . Oncogenic agents carried in descendants of certain families, in humans as well as in other species, may have a higher pathogenic potential and be more readily activated than those carried in other families. . . . Thus cancer, leukæmia, or other malignant tumours would develop more frequently in members of certain families than in others". Elsewhere it is made clear that the 'oncogenic agent' is a virus.

This authoritative and influential working hypothesis inspires much contemporary cancer research, and is supported by an enormous volume of experimental evidence obtained from chickens, mice and rats.

Nevertheless, it was claimed recently[3], in harmony with previous conclusions[4-6], that the 'inheritance factor' in human leukæmias usually takes the form of an apparent dominant 'predisposing' mutation of incomplete penetrance; furthermore, the several ætiologies of the various forms of this neoplastic disease are all polygenic in nature[3]. Haldane[7] in 1938 arrived at a similar conclusion with respect to cancer in general on the basis of Little's[8] statistical investigations of cancer inheritance. In rare families, but especially in those where the parents are consanguineous, childhood leukæmias and lymphomas can exhibit apparent recessive inheritance[9,10].

I have deduced[3,11] that malignancies in adult human beings generally develop from one or more cells containing a total of four specific (nuclear) gene mutations, one of which is often inherited, the remainder originating in one or more somatic cell lines.

The incompleteness of the penetrance of the inherited mutation arises because the probability of a somatic cell accumulating the fourth and final 'carcinogenic mutation' within the normal life-span is less than unity.

The evidence from families where the parents are consanguineous[9,10] indicates that two out of the four 'carcinogenic' mutations affect both homologous genes at a particular locus. More indirect evidence and arguments[12] are consistent with the view that the remaining two mutations affect both homologous genes at another locus. The ætiology of many childhood and adolescence malignancies[3] should involve a primarily non-genetic 'stress' factor such as (1) a hormonal stimulus; (2) a pyogenic infection (notably pneumonia[6,13]); or (3) infection, by 'horizontal' transmission, with an oncogenic virus. Direct epidemiological support for (3) has lately been obtained in the ætiology of a malignant lymphoma in children living in certain regions of Africa[14].

Familial Leukæmia and Mutation Frequency

By calculating (1) the size of (genetic) carrier subpopulations; (2) the 'penetrance' of an inherited mutation in the phenotypic form of a childhood leukæmia, the approximate frequency of childhood leukæmia in multiple sibs[6] can be accounted for[3]. Using Haldane's[15] "indirect method" for the calculation of mutation frequencies (where approximate genetic equilibrium is assumed) the derived values for the mutation frequencies of 'leukæmogenic' genes are closely comparable with known frequencies at other loci[3,11]. They lie at the upper end of the normal range[16] for dominant and sex-linked loci; but they include some effects attributable to gross chromosomal changes. Thus, trisomy for chromosome 21 (mongolism, or Down's syndrome) is associated with a very high leukæmia incidence in childhood[6,17,18], and the discovery of the *Ch*[1] chromosome by Gunz *et al.*[19] shows that the inheritance of a chromosome 21 from which a portion of the short arm has been either deleted or translocated, predisposes to chronic lymphatic leukæmia.

There is another source of uncertainty that could introduce an overestimation of the mutation frequency. It is assumed that only two major 'leukæmogenic' loci are implicated with respect to each of the two groups of genetically related leukæmias[3]. Although immunological evidence indicates that one specific structural locus may be involved in the ætiology of many carcinomas[12], and although the phenomena of the *Ph*[1] and *Ch*[1] chromosomes[19,20] are consistent with specific loci being associated with leukæmogenesis, the possibility cannot be dismissed that there may be several loci of similar phenotypic expression. It is evident from the calculated mutation frequency that the number of major 'leukæmogenic' loci cannot be high, but it could be greater than two. (Other loci will undoubtedly affect the phenotypic expression of the major loci through their influence on factors such as growth, or growth-rate, but their net effect on the calculated mutation frequency should in general be small.)

The measure of agreement between established mutation frequencies and those calculated for the hypothetical major 'leukæmogenic' genes is obviously of considerable theoretical significance; it is rather unlikely that it represents a mere chance coincidence.

Direct Cytogenetic Evidence for an Inheritance Factor

With regard to the theoretical ætiology of chronic lymphatic leukæmia it was stated[3]: "Chronic lymphatic