

This object should be warmly welcomed by advocates of the cold dark matter theory. A dark halo, an apparently young, chemically unevolved stellar component and a plentiful supply of gas are the ingredients expected for a young galaxy. Cold dark matter theory<sup>5</sup> prescribes the continuous rapid formation of many dwarf galaxies from the typical density fluctuations; larger fluctuations collapse later. A few of these dwarfs are incorporated into luminous galaxies. It is necessary to introduce the idea of biasing into this theory to suppress the formation of an excessive number of young galaxies. According to our version<sup>6</sup> of this idea, dwarf galaxy formation is interrupted by the onset of supernova-driven winds that strip out the gas. Perhaps during the fiery vigour of massive star formation, these objects would be recognizable as blue compact dwarfs. By the present epoch, however, one expects the Universe to be full of dwarf galaxies, pale remnants of blue, compact galaxies.

Occasionally, one might come across a rare, delayed fluctuation that recently collapsed. So Bergvall and Jörsäter<sup>2</sup> prefer

to attribute the blue and compact nature of ESO400-G43 to such an event, so that no underlying component of older stars can be expected. An alternative explanation is that a dwarf galaxy has encountered a cloud of weakly enriched gas that fuels the formation of new stars as it falls inwards. The massive halo provides a gravitational well capable of accreting intergalactic gas. The density of such gas can be estimated from observations of enriched gas in rich clusters, and is probably adequate in less dense regions of the Universe to fuel accretion. Future observations, especially in the infrared, will refine the search for an older stellar population, and so help discriminate between the two options of virgin birth or resurrection for the origin of such remarkable blue compact dwarfs. □

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## Virology

# A case of mistaken non-identity

Carel Mulder

IN 1985, when Daniel *et al.* isolated<sup>1</sup> a virus that resembled human immunodeficiency virus (HIV) from Rhesus macaques afflicted with simian AIDS — the monkey equivalent of human AIDS — the stage was set for new developments in understanding the range and relationships of immunodeficiency viruses. Unfortunately, the plot of the events that followed served largely to confuse spectators. The denouement appears in the exchange between Kestler, Daniel and collaborators and Essex and Kanki on pages 619 and 621 of this issue<sup>2,3</sup>, respectively.

The Daniel *et al.* virus, now called SIV<sub>MAC</sub> (but originally STLV-III<sub>MAC</sub>), answered all of Koch's postulates as the causative agent of simian AIDS, a rare disease found in macaque colonies at the New England and Californian regional primate centres. (The more common immunodeficiency found in these macaque colonies, SAIDS, is caused by a non-lentivirus: Mason Pfizer monkey virus-like D-type retrovirus, also first isolated by Daniel, a virologist who discovered at least ten new virus groups.)

Thereafter, SIV<sub>MAC</sub> was found by Essex and Kanki to react with sera from a sizeable proportion of African green monkeys as well as sera from healthy prostitutes in Senegal that did not react with HIV-1, a virus not known in West Africa at that time. Kanki and collabora-

tors subsequently reported<sup>4</sup> the isolation from these Senegalese women of a virus they named HTLV-IV, and the isolation from captive and wild African green monkeys<sup>5</sup>, of a virus they called STLV-III<sub>AGM</sub>. Shortly after, Clavel, Montagnier and co-workers reported the isolation of HIV-2 (originally named LAV-2) from HIV-1-negative West Africans with an AIDS-like disease. Nucleotide-sequence analysis<sup>6</sup> of these new viruses showed that HIV-2 is much more closely related to SIV<sub>MAC</sub> than to HIV-1.

For a time, there was no reason to be concerned about the identity of either STLV-III<sub>AGM</sub> or HTLV-IV, but doubts began to arise, first as other groups were unable to reproduce the isolation of a virus from African green monkeys by the procedure published for STLV-III<sub>AGM</sub>, and then as sequence data emerged.

With a change in procedure, Hayami's group in Tokyo has recently been able to isolate 19 SIV<sub>AGM</sub> viruses (ref. 6; M. Hayami, personal communication) from captive and wild Ethiopian and Kenyan African green monkeys. Using this procedure, at least two other groups have now also obtained SIV<sub>AGM</sub> isolates (R. Kurth *et al.* and M. Daniel, personal communications). The sequences of these SIV<sub>AGM</sub> isolates appear to be very different from those of STLV-III<sub>AGM</sub> but similar to each other with a variability comparable

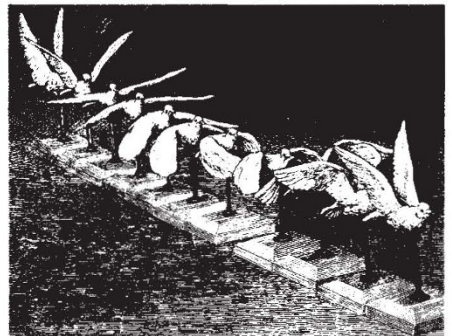
to that found among HIV-1, or HIV-2 isolates (M. Hayami and K. Ishikawa, personal communication).

Given the sequence variation among HIV-1 isolates that was already known at the time, it was very surprising when first Mullins's group<sup>7,8</sup> and then Hahn's<sup>9</sup> reported that six out of six STLV-III<sub>AGM</sub> and three out of three HTLV-IV isolates had identical or nearly identical restriction-endonuclease cleavage patterns and nucleotide sequences. This is most unusual, both for lentiviruses in general and for HIV-like viruses in particular. Now, Daniel and collaborators, in their paper in this issue<sup>2</sup>, find the explanation for this curious finding and solve the controversy on the origin of the nearly identical virus isolates. Whereas SIV<sub>MAC</sub> isolated from different macaques have variable endonuclease patterns and sequences even when epidemiologically related, the restriction pattern of one of their isolates, SIV<sub>MAC251</sub>, is identical to those of HTLV-IV and STLV-III<sub>AGM</sub>; SIV<sub>MAC251</sub> is also one of the two SIV<sub>MAC</sub> strains provided to Kanki by Daniel<sup>10</sup> for her seroepidemiological studies. It honours Essex and Kanki that they readily admit in their reply to Daniel's group on page 621 that all these questionable isolates should now be considered laboratory contaminations of their African green monkey and Senegalese human cells with SIV<sub>MAC</sub>. Too seldom do researchers in this field retract data found to be erroneous.

The misidentification of the STLV-III<sub>AGM</sub> and HTLV-IV isolates will not alter the results of their seroepidemiological surveys except that their Senegalese cohort should now be labelled HIV-2 seropositive. Both B. Hahn (personal communication) and Essex's group have now isolated genuine HIV-2 from the cohort of healthy Senegalese prostitutes, and Essex's group has also isolated HIV-2 from HIV-1-negative Senegalese AIDS patients (Marlink, personal communication). To minimize the confusion raised by

## 100 years ago

### MECHANISM OF THE FLIGHT OF BIRDS



Bronze figures representing eleven successive positions in the stroke of a pigeon's wing achieved by the use of photochronography. From *Nature* **37**, 372; 16 February 1888.