identified. The surgery was done by an experienced veterinarian (*4*), who stated that he had never seen an animal without at least vestigial organs. White blood cells from the two chromatin-positive cats were established in tissue culture (*5*) by a modification of the method published by Mellman *et al.* (*6*). Growth was obtained in culture from the first cat, but technical problems limited the number of countable cells to three. These showed 39 chromosomes rather than the normal number of 38 reported by various workers (*7*) and confirmed by our procedures with blood from normal cats. Better preparations were obtained from the second cat, the typical calico, and the chromosome number in nine cells was also 39. Further work is being done to establish which chromosomes are involved in the abnormality, and we are preparing idiograms for chromosomes in tissue culture from normal and abnormal cats (*8*). It should be emphasized that this chromosome abnormality would not be limited to the male with calico markings. The rare coat color merely served as a convenient primary screening device.

We feel that this demonstration of a spontaneously occurring chromosome abnormality in which the chromosome number is $2n + 1$ is an encouragement to seek associations of sex-linked characteristics with such chromosome abnormalities in mammals more suitable than the cat to experimental laboratory study of factors involved in the etiology of such disorders. Russell and his associates (*9*) demonstrated the occurrence of fertile XO ($2n - 1$) females in a population of laboratory mice. This may or may not be proved etiologically analogous to gonadal dysgenesis in human females, an abnormality in which the sex chromosomes are XO ($2n - 1$). Thus both a decreased and an increased number has now been found in mammals other than humans. The similarity with respect to testicular development between the effect of an extra X chromosome in humans and these cats in which the additional chromosome is presumed to be an X chromosome is also of marked interest in its implication that specific genetic mechanisms may be located on the analogous chromosomes in two differing species. Finally, the utility of the buccal mucosal smear technique in screening individuals for this chromosomal abnormality appears to be valid in cats as well as humans (*10*).

Note added in proof: Since this report was submitted, L. B. Russell has published a review of the genetics of mammalian sex chromosomes as studied in mice at the Oak Ridge National Laboratory, Oak Ridge, Tenn. [*Science* **133**, 1795 (1961)]. She cites the finding of an XXY male mouse, apparently sterile, among 6368 animals observed. The apparent sterility of this mouse extends to three the number of species in which abnormality of gametogenesis is associated with an additional X chromosome in the male.

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Simian Malaria in the Philippines

Abstract. The first field study of simian malaria in the Philippines found that malaria occurred in 8.6 percent of the animals tested. Although based on a very limited study, this report suggests that the simian reservoir of malaria is probably of limited significance for the human population in the Philippines.

The first field study of simian malaria in the Philippines has been conducted in view of the recent report of malaria transmission between lower monkeys and humans (*1*) and because of the possible significance of this finding on current methods for eradicating malaria.

In the western Pacific region, simian malaria has been reported in Taiwan, Borneo, Java, Sumatra, Malaya, and Indochina (*2*). There are no published accounts of related studies having been made in the Philippines, but *Plasmodium inui* has been isolated from Philippine monkeys sent to the United States and England (*3*).

In a preliminary blood survey within the Philippines, 16 positives (8.6 percent) were found among 186 *Macaca irus* (*M. cynomolgus*). From the northern provinces of Cagayan, Nueva Vizcaya, and Bulacan, the sample (24 animals) was negative. From the southern province of Palawan and the island of Mindanao, 16 positives were found in 162 animals.

The infected animals did not appear to be seriously ill. The parasites contained pigment during the trophozoite stage. No enlargement of red cells or Schuffner's stippling was found. The close morphological resemblance to *Plasmodium malariae* suggests the identification of *P. inui*. Halberstadter and Prowazek (*4*) first described this species in *Macaca irus* and *M. nemestrina* from the island of Borneo, which lies adjacent to the southern boundary of the Philippine archipelago. Since the flora and fauna of the southern islands are similar in many respects to neighboring Borneo, the presence of *Plasmodium inui* in both areas is not an unexpected finding.

For the past several years, the distribution of the Philippine human population has been undergoing a major shift away from the overpopulated coasts and valleys toward newly opened public lands which characteristically lie in hilly uplands bordering forested mountain ranges. Many variables contribute to the high potential for the transmission of malaria along these population frontiers, not the least of which are the many open, sunlit upland streams that constitute a highly favorable breeding habitat for the common Philippine vector, *Anopheles minimus flavirostris*.

It is an associated factor that monkeys are present in large numbers in the forests which lie adjacent to the advancing edge of new settlements. As a general pattern, it is often necessary for the new settler to spend some time within the forest in order to clear and burn his land before he can plant the usual upland crops of rice or corn. Careful assessment of the many epidemiological variables which apply at this geographical location indicates that the human malaria reservoir accounts for the source of *all* infections which have been investigated up to the present time. While the present report is based

on a very limited study, it does suggest that the simian reservoir is probably of limited significance in the Philippines for the following reasons: (i) *Plasmodium malariae*, which bears the closest resemblance to *P. inui*, was identified in only 26 out of a total of 41,945 positive human blood smears between 1 July 1959 and 30 June 1960. (ii) In frontier settlements, the species of malaria are divided almost equally between *P. falciparum* and *P. vivax*, which are both morphologically dissimilar to *P. inui*. (iii) In the northern Philippines (Luzon), the pattern of malaria parallels the pattern in the southern islands without evidence, so far, that simian malaria is present.

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Effect of Actinomycin D on Cellular Nucleic Acid Synthesis and Virus Production

Abstract. Actinomycin D inhibits the synthesis of ribonucleic acid in L cells and the yield of vaccinia virus containing deoxyribonucleic acid, but it does not inhibit cellular deoxyribonucleic acid synthesis or the multiplication of Mengo virus containing ribonucleic acid. These observations serve to distinguish the replication of viral ribonucleic from ribonucleic acid synthesis which is controlled by viral or cellular deoxyribonucleic acid.

Actinomycin D is a bright red antibiotic containing two peptides, which was first reported by Vining and Waksman (*1*). It possesses strong antibacterial activity against gram-positive organisms (*2*) and, on a weight basis, is the most potent chemotherapeutic antitumor agent known (*3*). It has been reported to be antimutagenic (*4*).

Mammalian cells grown in the presence of actinomycin D lose their nucleoli and much of their histochemically demonstrable ribonucleic acid (RNA) (*5*, *6*). In the present study we report that actinomycin selectively and irreversibly suppresses mammalian cellular RNA biosynthesis, at least up to 48 hours after exposure—the period during which affected cells have been observed.

Strain 929 L-cells monolayers were maintained and propagated as previously described (*6*). In addition, spinner cultures (*7*) were employed. Cell monolayers, seeded on cover slips and exposed to suitable radioactive nucleic acid precursors, were examined autoradiographically by the method of Doniach and Pelc (*8*).

When normal L cells are incubated with H^3-cytidine (0.5 μc, 0.4μg/ml, 3 to 6 hours) and inspected after radioautography, a portion of the radioactivity incorporated into acid-insoluble material may be solubilized by deoxyribonuclease. The remainder can be rendered acid-soluble by digestion with ribonuclease.

After exposure to actinomycin D (1.0 μg/ml for 8 hours) and subsequent incubation for 16 hours, L cells continue to incorporate H^3-cytidine into acid-insoluble material. In this case, however, none of the incorporated radioactivity becomes acid-soluble after ribonuclease treatment; all of it is solubilized by deoxyribonuclease. We conclude that cellular RNA synthesis, but not DNA synthesis, has been completely arrested by antecedent incubation with actinomycin.

Similar findings are shown in Table 1, in which pairs of spinner cultures are compared with respect to the incorporation of H^3-leucine into protein, H -uridine into RNA, and H^3-thymidine into DNA 4 hours after initial exposure to actinomycin. One pair of cultures contained actinomycin D (0.2 μg/ml), the other served as control. While the incorporation of leucine into protein and of thymidine into DNA were not affected by the antibiotic, uridine incorporation into RNA was depressed. After 24 hours, uridine uptake into RNA of the cells growing in the presence of actinomycin was still further decreased relative to the control.

The effect of actinomycin on virus growth has also been investigated. In some experiments cells had been treated previously with appropriate concentrations of the antibiotic, while in others actinomycin was present throughout the period of virus absorption and growth. The results were independent of the type of exposure.

Table 1. Incorporaton of precursors into protein, RNA, and DNA*

Compound	Control	Actinomycin treated
Leucine-H^3 into protein	50,980	49,620
Uridine-H^3 into RNA	160,500	66,500
Thymidine-H^3 into DNA	194,000	181,000

* Values expressed as total counts per minute for infinitely thin platings of equal aliquots obtained as follows: one pair of replicate cultures incubated in Eagle's medium containing 0.2 mmole of L-leucine received DL-leucine-H^3 (1 μc/ml) and thymidine-H^3 (0.5 μc, 0.4 μg/ml) and the second pair uridine-H^3 (0.5 μc, 0.8 μg/ml). One culture of each pair was exposed to actinomycin (0.2 μg/ml) for 30 min before and 3½ hours after addition of labeled compounds. Samples of 25 ml each were centrifuged and carriers added; they were then washed with phosphate-buffered saline, extracted with cold 0.25*N* $HClO_4$ for 1 hour, and washed with ethanol and ethanol-ether. For leucine and thymidine determinations the washed pellets were hydrolyzed in 0.5*N* $HClO_4$, washed with ethanol and ethanol-ether and the pellets and hydrolysates were counted. For uridine measurement the pellets were incubated with ribonuclease (150 μg/ml) for 2 hours at 37°C in .005*M* tris buffer, *p*H 8, made 0.2*N* with $HClO_4$, centrifuged, and the supernatant was counted.

The multiplication of vaccinia, a DNA virus, is sensitive to actinomycin, but somewhat less so than division of the host cell: 0.1 μg/ml inhibited vaccinia growth by 99 percent, whereas 0.005 μg/ml suppressed host cell division. On the other hand, concentrations of actinomycin in as high as 10 μg/ml did not inhibit the growth or affect the yield of Mengo virus, a ribonucleic acid virus.

Parallel findings have been obtained with mitomycin, high concentrations of which inhibit cellular but not viral RNA synthesis (*9*). Whereas mitomycin appears to affect RNA synthesis by destroying the genes under whose control the various cellular RNA species are synthesized, actinomycin would seem to leave the genetic apparatus intact since DNA replication is not abolished. This is supported by studies on the effects of actinomycin S on phage synthesis (*10*). The inhibition of T2-phage reproduction by actinomycin does not result in inhibition of DNA synthesis in the phage-infected cell, whereas no phage protein appears to be made. Actinomycin thus appears to block the expression of genetic potentialities by interfering with that portion of RNA synthesis which is dependent on or governed by cellular or viral DNA. This is, therefore, a second line of evidence serving to differentiate replication of viral RNA from that of