abundance. However, *N. bruchi* is reported to survive better at lower temperatures when compared to *N. eichhorniae*, which prefers warmer temperature[6,7]. Therefore release of both species of weevils is recommended in parts of the country where low winter and high summer temperature, prevail.

The author is grateful to Mr N. Chandrasekhar for technical assistance.

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CELL CYCLE AND SISTER CHROMATID EXCHANGES IN VIRUS-INFECTED CHINESE HAMSTER OVERY CELLS

J. M. CHIPLONKAR, R. P. DEOLANKAR* and S. V. GANGODKAR*

National Tissue Culture Facility, Department of Zoology, University of Poona, Pune 411 007, India.
**Division of Tissue Culture and Cell Biology, National Institute of Virology, 20-A Dr Ambedkar Road, Pune 411 007, India.*

It was shown earlier[1] that herpes simplex virus types 1 and 2 (HSV–1 and HSV–2), vaccinia virus and simian virus–40 (SV–40) induce sister chromatid exchanges (SCEs) in Chinese hamster ovary (CHO) cells. With a view to studying the events which can potentially induce SCEs in virus-infected cells, CHO cells were infected with these viruses and the frequencies of SCEs were monitored at various times thereafter. This allowed virus-specific events to be sequentially and selectively superimposed on the cellular S phase. Results of these studies are described here.

CHO cells were grown in the presence of 5-bromodeoxyuridine (BUdR, 1 μg ml) for two cell cycles during logarithmic phase. The cultures were inoculated with 2 logs each of HSV–1 (753166), HSV–2 (753167), vaccinia (671061) and SV–40 (776), and incubated at 37°C for various periods from 1 to 15 h. c-Metaphase chromosome preparations were made with a 2 h colchicine treatment (0.05 μg/ml). Sister chromatid differential staining was carried out by the fluorescence-plus-Giemsa technique[2], with minor modifications. Thirty well-spread metaphases, each containing 21 chromosomes (stem-line population), were scored from every sample.

The results obtained from three identical experiments are summarized in figure 1. SCE frequencies

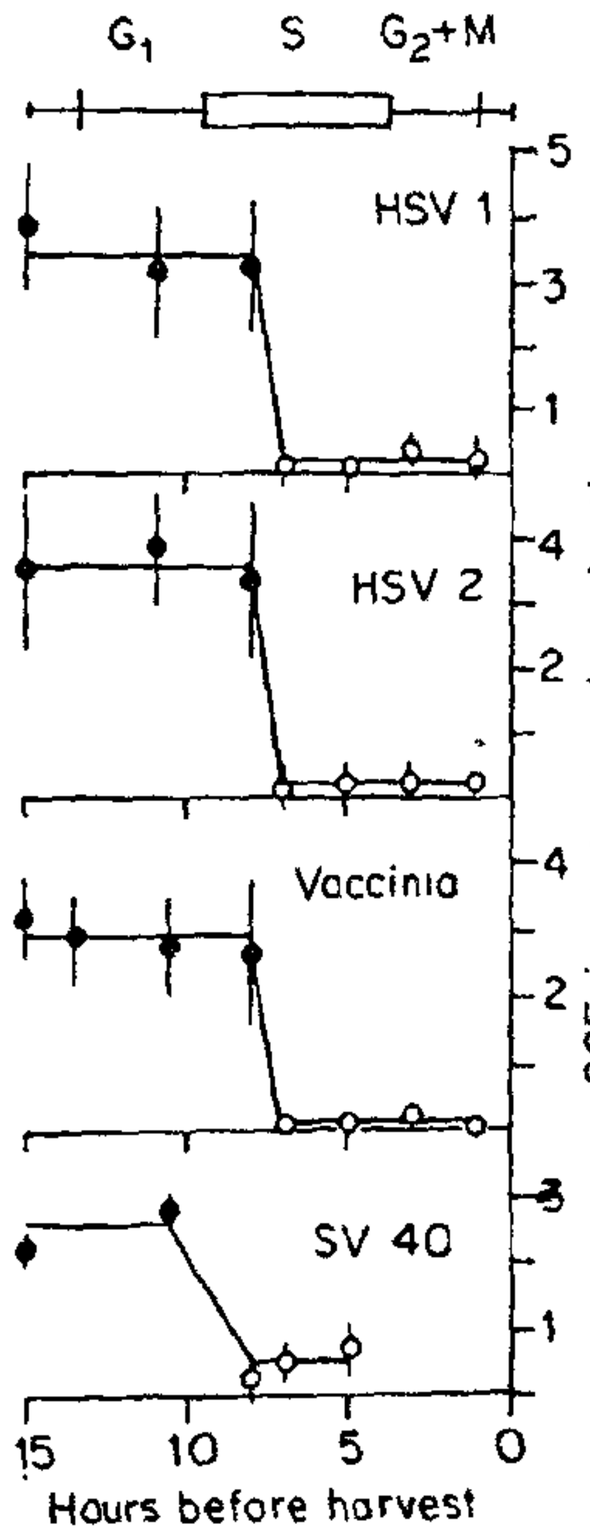

Figure 1. Frequencies of SCEs in virus-infected CHO cells in relation to the time of infection during the cell cycle. Each point represents average of 90 observations from 3 identical experiments. Control SCE frequencies are normalized to zero. SCE frequencies denoted by symbols ○ do not differ significantly while those denoted by symbol ● differ significantly from the base level ($P < 0.01$)

in virus-infected cultures were plotted as increase over the control frequencies. It can be seen that in cultures infected with HSV-1, HSV-2 and vaccinia virus, the SCE frequencies showed no change up to 7 h before harvest (hbh). At 8 hbh, there was a sudden rise over the base level SCE frequencies ($P<0.01$). These elevated SCE frequencies were thereafter maintained up to 15 hbh. SV–40-infected cultures exhibited a similar profile although the rise appeared slightly later, around 10 hbh.

The experimental protocol included a set time frame of 25 h for BUdR labelling. During this period, metaphases with a history of BUdR incorporation in two consecutive rounds of DNA replication could be obtained from uninfected control as well as virus-infected cultures, as evidenced by differential staining of sister chromatids. This indicated that delays of significant consequence to the cell cycle may not have been introduced as a result of virus infection. Therefore, superimposition of virus infection time points directly over cell cycle phases (expressed as hbh), would provide useful temporal correlation.

The above results therefore indicated that when the cells were infected in G2 or late S, there was no increase in the SCE level. On the other hand, when the infection occurred in early S or G1, or in G2 of the previous cycle, there was a significant increase in the SCE level. It appears from these observations that for increase in SCEs in virus-infected cells, a major part of S phase must follow virus inoculation. The observations further indicate that the event(s) responsible for SCE induction must appear relatively early after virus infection.

It is interesting to note that strikingly similar profiles for SCE increase were observed with all the viruses used, irrespective of differences in their nature, replication mechanisms and lytic/transformation cycles. Vaccinia virus multiplies in cytoplasm, while HSV–1 and HSV–2 multiplication involves the nucleus; CHO cells are non-permissive to SV–40 virus multiplication, but can be transformed by this virus. It is, therefore, apparent that some event(s) common to or similar in the infection processes of all these viruses may play an important role in the induction of SCEs in CHO cells.

A comparison of SCE profiles for ultraviolet- and mitomycin–C-treated cells has been made by Shafer[3]. In the former case, the profile appears to closely follow the pattern of cellular DNA synthesis, while in the latter case, it does not. From these studies a distinction has been drawn between the longevities of lesions introduced in the host DNA and their contribution to the formation of SCEs. Lesions caused by mitomycin–C treatment during G1 phase appear to remain unrepaired for longer periods, and the damaged DNA is carried into the subsequent S phase. Such lesions may lead to SCEs employing 'bypass' mechanisms. Therefore, mitomycin–C treatment leads to elevated levels of SCE in G1 phase. On the other hand, lesions induced by UV appear to be short-lived. Therefore such lesions inflicted on G1 DNA could be repaired during the same phase, and the possibility of damaged DNA being carried into the S phase is eliminated. Thus such lesions do not lead to SCEs, and, consequently, SCE profiles during G1 phase do not show elevations. For both mitomycin–C and UV, treatment in the later part of S and in G2 phase is not expected to generate SCEs because lesions remain unexpressed in the absence of a following synthetic phase. Consequently no rise in SCE frequencies can be expected during late S or G2 of the last cycle. However, long-lived lesions inflicted during this part of the penultimate cell cycle may be carried over to the S phase of the last cell cycle. In this event, it may be expected that treatment during G2 phase of the penultimate cycle would be expressed as elevated SCE level.

Since the elevated SCE frequencies were maintained in G1 phase in the infected cells, it was apparent that these profiles did not follow the pattern of cellular DNA synthesis. In this respect, these profiles were comparable to those obtained after mitomycin–C treatment[3]. By analogy, therefore, DNA damages introduced in the virus-infected cells can be considered to be long-lived lesions; any short-lived lesions, of the type induced by ultraviolet irradiation probably do not play any role in the formation of SCEs. Furthermore, the SCE profiles also suggest that processes modulating the unreplicated host DNA[3,4] are more likely to occur during the production of virus-induced SCEs.

The specific early molecular event(s) in virus-infected cells which would introduce long-lived lesions in the unreplicated host DNA and thereby induce SCEs is still to be identified. Nichols *et al*[5] have shown the coincident appearance of T-antigen-positive cells and higher SCE frequencies in four SV–40-transformed human diploid fibroblast cultures a few passages after transformation. This may be a pointer. However, the present studies indicate that the cause of SCE induction may well be restricted to the very early stages of virus infection.

15 December 1987; Revised 14 April 1988

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RESIDUES OF GENTAMICIN IN BUFFALO CALVES (*BUBALUS BUBALIS*) AND DOMESTIC FOWL (*GALLUS DOMESTICUS*)

SATISH K. GARG* and B. D. GARG
Department of Pharmacology, Haryana Agricultural University, Hissar 125 004, India
** Department of Physiology and Pharmacology, College of Veterinary Science, H. P. Krishi Vishva Vidyalaya, Palampur 176 062, India.*

GENTAMICIN is one of the broad spectrum antibiotics and has gained considerable importance in veterinary medicine during the last decade. Detailed pharmacokinetic studies of gentamicin have been conducted in various mammals, some of the avian and lower vertebrate species. However, the data on the tissue persistence of gentamicin following multiple parenteral administration is only limited to human beings[1], guinea pig foetuses[2], sheep[3] and cows[4]. Inspite of the availability of a good deal of data on the therapeutic efficacy of gentamicin in many diseases, this antibiotic has not yet been approved by the Food and Drug Administration (FDA) for clinical use in food animals. Knowledge

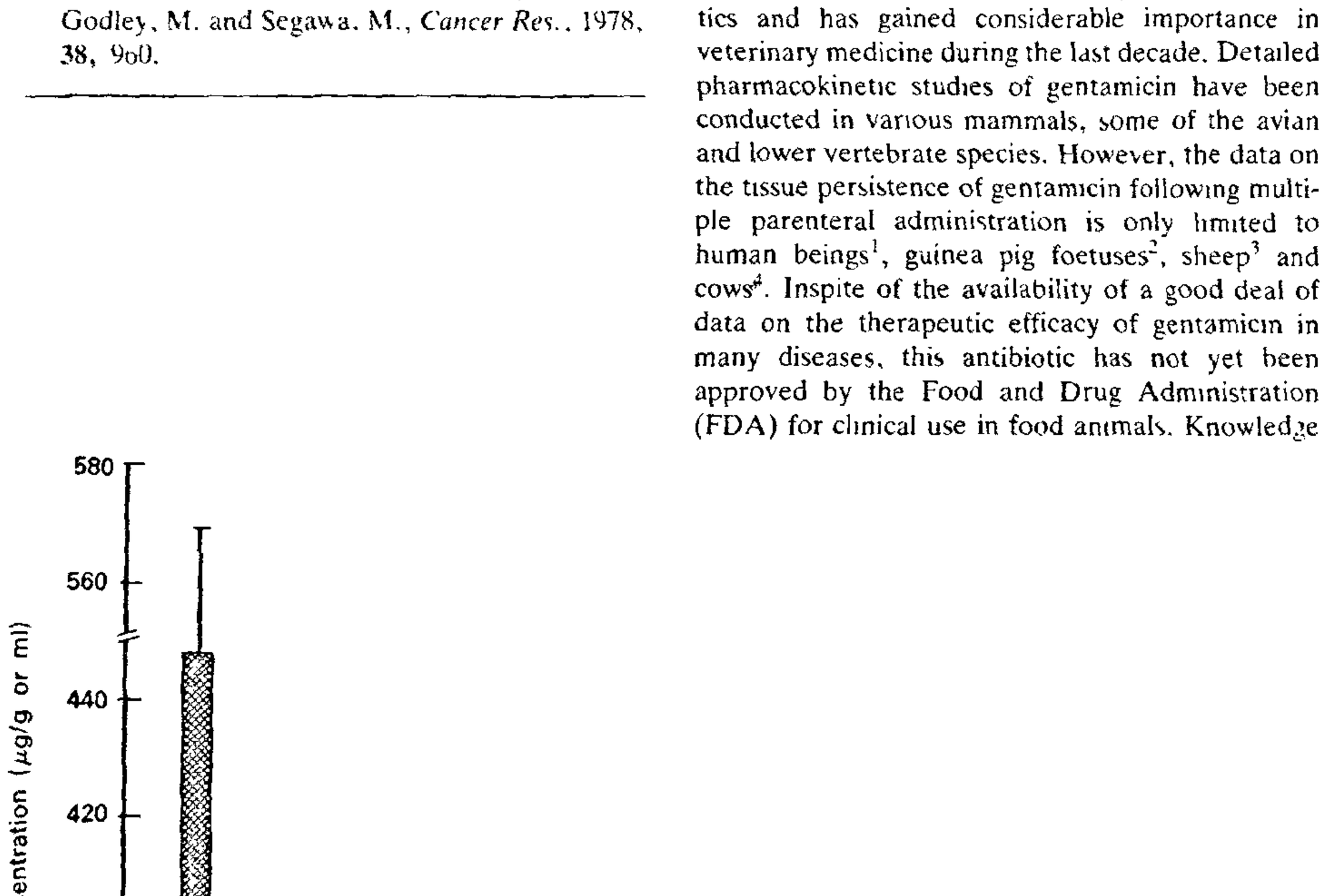

Figure 1. Concentration of gentamicin in body tissues and fluids of buffalo calves administered with a loading dose (6.22 mg/kg, im) and maintained with 5.06 mg/kg (im), every 8 h daily, for seven days.