Twelve-hour-old eggs of an improved multivoltine strain MY2 (which spins dark greenish yellow cocoons, with plain larvae) were kept at 18°C for 72 hr, then they were transferred to another chamber at 20°C for 48 hr and finally at 22°C for a period of 48 hr. The eggs were then incubated at 25°C till hatching. Hatched worms were brushed in four cellular (eggs laid by single moth) replications and reared during October 1985. The larval abnormalities were clear only after the *II* moult. Abnormal larvae were separated from each replication and observed through a binocular-stereozoom microscope.

The percentage of abnormal worms seen in each replication is given in table 1. Various kinds of induced abnormalities are shown in figure 1. In some malformed larvae, an additional spiracle was observed in the V abdominal segment (figure 1a). Some of the abnormal larvae were devoid of the first pair of legs in the VI abdominal segment (figure 1b). A dermal protuberance was seen at the junction of VII and VIII abdominal segments (figure 1c). Another interesting feature of E-group genes is the torsion of segments resulting in asymmetrical fusion of segments particularly on the dorsal side (figure 1d). In some larvae, bifurcation of the thoracic leg was observed (figure 1e). The most striking feature is that most of the larvae had additional caudal horns (figure 1f) and sometimes up to four caudal horns were seen. In some larvae, the first pair of abdominal legs was markedly reduced (figure 1g). Another peculiarity was the appearance of an extra leg on the VII abdominal segment (figure 1h). Asymmetrical fusion of caudal segments caused twisting of caudal region in most of the larvae (figure 1i, j) as well as irregular position of the abdominal legs (figure 1k). Up to 48.9% abnormal worms appeared in one replication. The moths which emerged out of the surviving abnormal larvae laid fewer eggs and of low hatchability (table 1).

Kawaguchi *et al*[6] reported several kinds of similar malformed larvae of *B. mori* when 0.5 hr to 24 hr old eggs were soaked in N-methyl-N. nitrosourea, a mutagenic and carcinogenic agent.

In another study[5], the number of lunar-shaped paired markings has been found to vary in the mutant called *multilunar* (which normally possesses six pairs of markings on the dorsal side of all abdominal segments of the larva) by low temperature of the eggs during the early embryonic development.

In the present study, seven multivoltine strains were exposed to low temperature during early embroynic stage but only one strain i.e. MY2 showed larval abnormalities. This indicates that the induction of E-alleles abnormalities by low temperature incubation appears to be strain-specific. Hence, the present study facilitates identification of the strains which are susceptible to the induction of the aforesaid abnormalities to retain the healthy pure strains for commercial silk production.

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ANTITUMOR EFFECT OF CISPLATIN AND GLUCOSE IN MICE BEARING DALTON'S LYMPHOMA

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CISPLATIN has been used successfully as a potent antitumor compound against a variety of experimental tumors in mice[1–4]. It has been suggested that tumor regression by cisplatin is a result of enhancement of the host's immune system[5–11]. Although, this compound results in an increase in survival time of animals when administered at therapeutical dose, a carcinogenic risk always remains in the treated animals[12,13]. Some anticancer drugs are much more effective when used along with glucose[14–16]. Several antitumor drugs were tried in acidic conditions induced by tumor cell glycolysis and among these, carbazilquinone was most cytocidal when coupled with Ehrlich ascites tumor cell glycolysis[16]. Recently, benzylidine D-glucose was found to be effective in causing tumor necrosis in rats bearing hepatocellular carcinomas[17]. Chemotherapeutical studies were done by injecting tumor cells preincubated *in vitro* with different drugs and glucose[16]. The present study involves the direct treatment of tumor *in vivo* with subtherapeutical dose of cisplatin along with glucose to

evolve a nontoxic chemotherapy for the selective destruction of tumor cells.

Transplantable Dalton's lymphoma in C_3H/He strain of mice was used. The day of tumor transplantation was taken as day 0. On the 6th day animals bearing tumor were given a single i.p. injection of 3 mg/kg cisplatin alone or in combination with five repeated (alternate days or weekly) injections of glucose. Controls were run for each group. Increase in survival time of the tumor-bearing mice and the per cent of tumor-free survivals were studied in experimental and control groups.

Tumor cells have a higher rate of glycolysis[18]. This vigorous glycolytic activity of tumor cells leads to the production of greater amount of lactic acid when incubated with glucose resulting in decrease in the local extracellular pH of tumor cells[15]. Release of lactic acid by tumor cells treated with glucose *in vitro* has already been reported[14,16]. When 75 mg/kg glucose was injected i.p. in tumored mice, this amount was utilized by the tumor cells causing a decrease in pH in their vicinity. This decrease in pH has some effect on tumor growth resulting in an increase in survival time of the tumor-bearing mice (figure 1). Injection of subtherapeutical dose of cisplatin (3 mg/kg) in tumored mice resulted in an increase in their mean survival time (figure 2). Animals receiving subtherapeutical dose of cisplatin along with single injection of glucose (75 mg/kg)

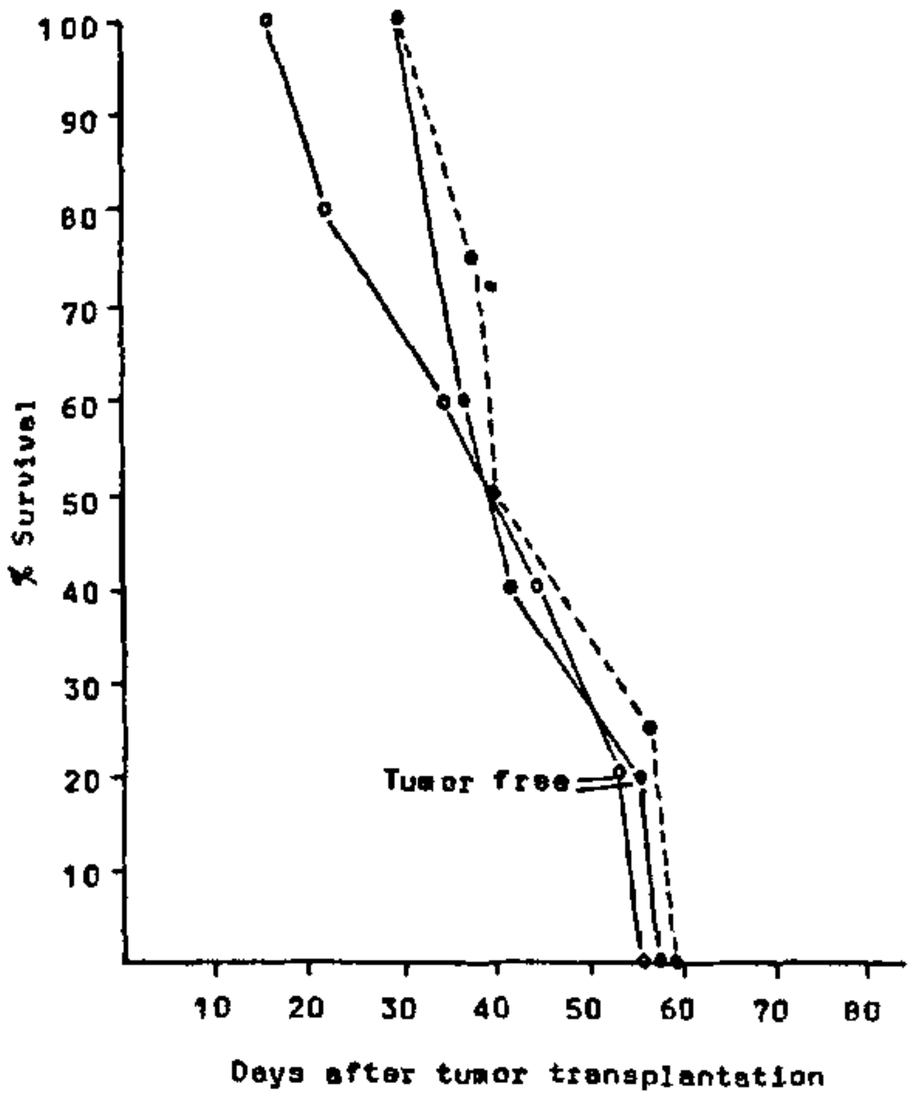

Figure 1. Effect of single and five repeated injections of glucose (75 mg/kg). [●---●, single injection; ●—●, alternate injection, and ○—○, weekly injection.]

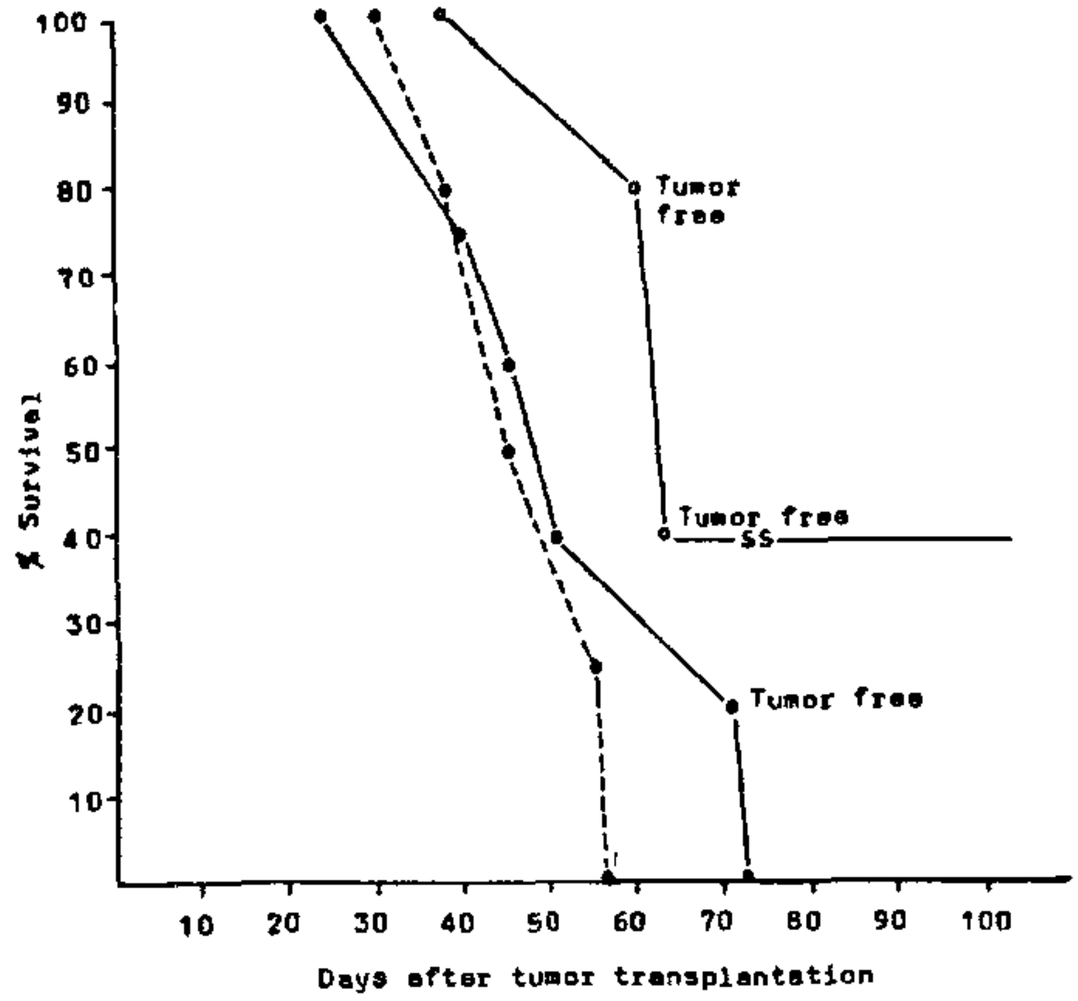

Figure 2. Subtherapeutical dose of cisplatin (3 mg/kg) combined with repeated injections of glucose. [●---●, 3 mg/kg cisplatin; ●—●, 3 mg/kg cisplatin + alternate day glucose injection, and ○—○, 3 mg/kg cisplatin + weekly glucose injection.]

i.p., survived with tumor growth. Despite giving antitumor compound cisplatin, in all the animals tumor increases gradually in size without any sign of regression but repeated injections of glucose (75 mg/kg, i.p.) slowed down the growth rate causing an increase in the survival time of the tumored mice. In almost 80% of mice there was complete regression of tumor after 5 weekly injections of glucose and they appeared as tumor-free survivals (figure 2). Controls receiving repeated injections of glucose without cisplatin either on alternate days or weekly, showed regression in a few animals (figure 1) suggesting that low pH or acidic conditions around the tumor cells may itself be a hazard for their multiplication as reported earlier[14]. Recently, similar results involving tumor necrosis after injecting repeated injections of benzylidine D-glucose in rats bearing hepatocellular carcinomas have been reported[17]. Subtherapeutical dose of cisplatin with single injection of glucose was found to be ineffective in causing regression in tumored mice; however, repeated injections (weekly) of glucose resulted in 80% tumor-free survivals at a later stage. Even repeated injections of glucose on alternate days along with single subtherapeutical dose of cisplatin showed therapeutical effect to some extent but were not as effective as injections every week (figure 2). This suggests that cisplatin gets potentiated at a particular pH which is to be maintained by

weekly injections of glucose. Alternate day glucose injections may fail to maintain that required pH which helps to accelerate the antitumor effect of this compound. These results coincide with earlier findings where anticancer drugs are reported to be more effective when coupled with tumor cell glycolysis[14–16]. It has been reported that although this compound has significant antitumor effect when injected at therapeutical dose (9 mg/kg), a carcinogenic risk always remains in the treated animals at a later stage[12,13]. Hence, to avoid this risk in the present system, subtherapeutical dose of cisplatin with repeated injections of glucose is utilized to evolve a nontoxic therapy of this tumor. Such a low dose of cisplatin in combination with glucose is proved to be effective in causing antitumor effect in tumored mice by giving tumor-free survivals as the result of tumor regression.

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NEWS

THE AIDS VIRUS–WELL KNOWN BUT A MYSTERY

The natural history of the AIDS virus makes it extremely well suited for evading the immune system–and a hard target for vaccine development. If knowledge is power, then the research community should be gaining a firm ascendancy over the virus that causes AIDS. In the 4 years since that virus now called human immunodeficiency virus 1 (HIV-1), was discovered, its genome has been cloned and sequenced, and the various genes have been identified, including at least five "accessory" genes that are not found in more ordinary viruses. A great deal has also been learned about the transmission of HIV-1 and the body's responses to it. "Never has so much knowledge been generated in such a short time as in the case of the human retroviruses and AIDS". (Science, 24 April 1987). *News Monitor*, Vol. 2, No. 3, July 1987, p. 3 (Published by: Centre for Advancement of Biotechnology–Prof. K. S. Gopalakrishnan, 1276, 32 G Cross, IV Block (T), Jayanagar, Bangalore 560 011)