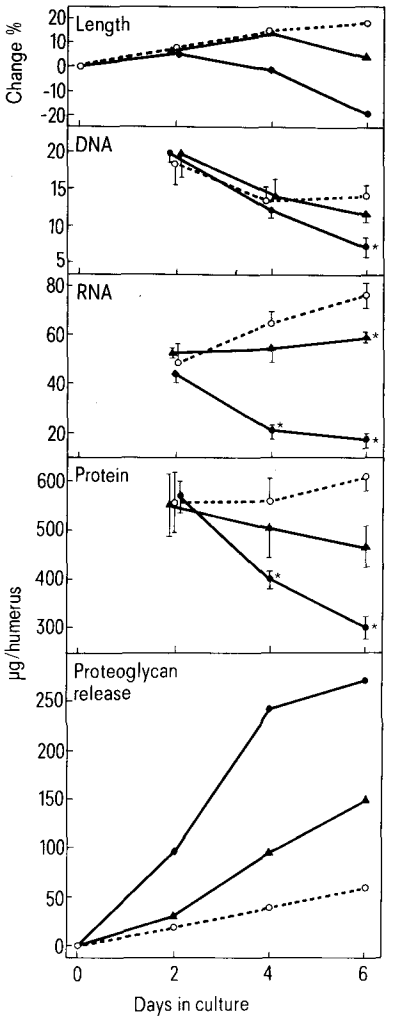


Fig. 2

Fig. 2. Time course of retinoic acid-induced changes in length, DNA, RNA and protein content and in the release of proteoglycan in limb bones of late fetal rats. Humeri (4 per dish) were incubated in supplemented culture medium F-10 (final volume 5 ml) at 36 °C. Before incubation, and then every 2 days, the contour of the magnified bones (× 14.1, using a reversed microscope and a projecting prisma) was drawn and the length determined. At the time indicated, the humeri, after washing in Ringer's salt solution, were extracted in 1 ml of 10% (w/v) trichloroacetic acid and the extract discarded. Nucleic acids were extracted with 10% (w/w) perchloric acid at 70 °C for 30 min and the DNA and RNA determined[23,24]. The remaining tissue was dissolved in 1N NaOH and the protein measured[25]. Aliquots of the culture medium were collected and the amount of proteoglycan released into the medium was measured by the Alcian blue assay[22], using chondroitinsulfate as standard. Since the humeri were distributed randomly, it was assumed that the values at day 0 were similar for all groups. Except for the proteoglycan release, the results are the mean of 4 determinations, ± SD. Treatments were: ○, controls; ▲, 0.6 µM retinoic acid; ●, 20 µM retinoic acid, *$p < 0.01$.

that hormone or vitamin. Since the effects of the hormonal form of vitamin $D_3$, 1,25-dihydroxycholecalciferol[20,21], are also known to depend upon continous RNA and protein synthesis, it is most likely that cellular effects of certain vitamins, like those of some hormones, are mediated by regulation of protein synthesis, presumably at the transcriptional level. However, further work is needed to ascertain whether the vitamin A effect only represents a quantitative or rather a qualitative change in protein synthesis.

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## Effect of infection of mice with Friend leukemia complex viruses on background antibody-forming cell production in vitro[1]

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*Summary.* Friend leukemia complex (FLC) and Rowson-Parr virus (RPV) infections of donor mice depress the production of background antibody-forming cells by splenocytes cultured in the absence of specific antigenic stimulation.

During investigations on the immunodepressive properties of Friend leukemia complex (FLC), it was shown that adult mice infected with viruses belonging to this complex exhibit increased numbers of splenic background antibody-forming cells to various antigens[2,3]. Obviously, this is a paradoxical effect, since in the same mice artifically stimulated antibody responses are suppressed[4]. Here it is shown that after infection of donor mice with FLC viruses the

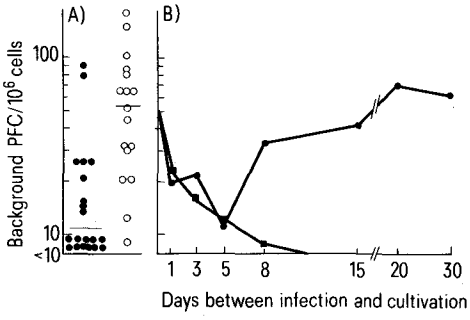
production in vitro of background plaque forming cells (BPFC) against sheep erythrocytes (SE) by spleen cells is depressed.

Adult BALB/c mice were injected with either Rowson-Parr virus (RPV) or FLC as described[5]. 5 million pooled splenocytes obtained at various times after infection, from 3 or more mice, were cultured in Marbrook chambers[6] exactly as reported[5], but the addition of SE was omitted. Since BPFC development in vitro is influenced by the source of serum used to supplement the medium[6,7], a single batch of fetal calf serum (Flow Lab., Rockville, Md.) was used. After 5 days incubation, the cells were collected, counted, tested for viability and assayed for BPFC with SE as test antigen[2]. The results are expressed as number of BPFC/$10^6$ viable nucleated cells recovered.

The figure A shows the effects of RPV infection performed 5 days before cultivation on BPFC production. Despite the use of spleen pools, individual values were rather variable in both normal and infected cultures. However, in the latter a reduction of BPFC numbers was evident. The mean number of BPFC was 11 in the infected cultures and 54 in the controls ($p < 0.01$). It is noteworthy that cell survival in infected and control cultures was similar: nucleated cells recovered were respectively $1.09 \pm 0.42 \times 10^6$ and $1.17 \pm 0.39 \times 10^6$ (mean±SD) per culture.

The figure B depicts the relation between time of RPV or FLC infection and number of BPFC developed. Both infections depressed PBFC production rapidly. However, the depression caused by RPV had waned by the 2nd week of infection, while that induced by FLC was irreversible and progressive. Thus the effects of RPV and FLC on BPFC parallel those on antibody-forming cells produced by SE-stimulated cultures[5].

These results show that nonspecifically stimulated antibody responses in vitro are depressed by FLC viruses in a manner similar to specifically stimulated antibody responses. It seems reasonable to assume that there is a strict correlation between BPFC produced in vitro by spleen cells and BPFC present in the spleen of unimmunized animals, since both a) are produced without manifest stimulation, b) release IgM antibody, c) are independent from T cell activity[8,9], d) are enhanced by B cell mitogens[10,11], e) are probably triggered by cross-reacting antigens; but proof is lacking because in vivo studies with germ-free animals have been inconclusive[12] and in vitro BPFC arise also in the absence of serum[13], supposedly the main source of cross-reactions[6,7]. If this assumption is correct, the results presented here lend support to the idea that the enhancement of splenic BPFC induced by FLC viruses in vivo is not due to increased generation rate but to changed behavior. An altered circulation pattern appears the most plausible explanation[2,14].




Effects of infection with viruses of the Friend leukemia complex (FLC) on background plaque-forming cells (BPFC) production by spleen cells in culture. *A* Numbers of BPFC produced by individual cultures from uninfected mice (○) and from mice infected with RPV 5 days before cultivation(●). *B* Mean number of BPFC produced by cultures from mice at various times from RPV (●) or FLC infection (■) (each point represents the average of at least 5 cultures).

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## Screening of newly synthesized non-alkylating sterilant compounds against the adult *Chrotogonus trachypterus* Blanch. (Orthoptera: Acrididae)[1]

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*Summary.* Out of the 9 new non-alkylating compounds screened against *C. trachypterus*, the compounds 62206, 24220 and 51160 have been found most effective, 51007 and 50882 exhibit intermediate effectiveness, 61914 only slightly effective and 50994, 2406 and 51240 almost non-effective.

9 newly synthesized non-alkylating compounds[2] were screened against the adult *Chrotogonus trachypterus* Blanch., an acridid pest, to determine their effect on the reproductive capacity when applied by dipping method.

*Materials and methods.* The compounds employed in the present investigation were dissolved in glass-distilled water (GDW) to the required concentration, except 3-amidino dithiocarbazic acid (61914) which was dissolved in solvent