rat do not show a rise in alanine transaminase activity after cortisol treatment. The factors which control the response of this enzyme in tumor and liver in partially hepatectomized and pregnant rats and the significance of enzyme induction in relation to the responsiveness of certain tissues to corticosteroids remain to be determined.

Summary. Consequences related to the metabolic requirements for growth of new tissues during fetal development and liver regeneration were studied with regard to: 1) activity of a transaminase enzyme, alanine-α-ketoglutarate transaminase; 2) growth of Walker carcinoma 256; and 3) response of the enzyme and the tumor to treatment with cortisol. The livers of pregnant or partially hepatectomized rats did not show an increase in alanine transaminase activity after administration of cortisol at doses which produced a significant response of this enzyme in normal control animals. The growth of the Walker carcinoma 256 in partially hepatectomized rats and pregnant animals was inhibited by 40% and 80%, respectively. While liver regeneration in tumor-bearing animals was retarded by 50%, the growing tumor had no observable effect on fetal development. Alanine transaminase activity was below normal endogenous levels in both regenerating liver and liver from pregnant rats.

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Mycoplasma granularum of Swine Origin as a Tissue Culture Contaminant. (31192)

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The problems of tissue culture contamination with mycoplasma, together with identification of species and source of isolates, have been reviewed recently(1,2). Most strains recovered have been of human origin, particularly *Mycoplasma hominis* type 1 and *M. orale* 1. Saprophytic *M. laidlawii* has been described as a contaminant on one occasion (3), as well as an avian strain, *M. gallisepticum*(4). The first suggestion that animal mycoplasma might be more extensively involved in tissue culture contamination occurred when agents recovered from cell cultures inoculated with human leukemic bone marrow(5) and spleen(6) were later identified as mycoplasma (strains Negroni(7) and 880 (8)) and subsequently typed as *M. pulmonis* (9,10), a serotype originally found in rats and mice. However, the recovery of *M. pulmonis* from uninoculated tissue cultures has not been reported and the impression that rodent strains are present in human tissues

TABLE I. Mycoplasma Strains Employed in Indirect Fluorescent Antibody Tests.

Animal host	Serotype	Strain designation
Human	*M. hominis* type 1	PG-25
	M. hominis type 2	Campo
	M. fermentans	PG-18
	M. salivarium	PG-20
	M. pneumoniae	Eaton-FH
	M. orale 1	CH 19299
	M. orale 2	CH 20247
	Unclassified	Navel
Bovine	*M. bovigenitalium*	PG-11
Canine	*M. spumans*	PG-13
	M. canis	PG-14
	M. maculosum	PG-15
Avian	*M. gallisepticum*	S6
	M. gallinarium	PG-16
Murine	*M. neurolyticum*	Type A
	M. histotropicus	Type C
	M. arthritidis	PG- 6
	M. pulmonis	PG-34
Porcine	*M. hyorhinis*	7
	M. granularum	31B and 39
Tissue culture	Unclassified	GDL
	Unclassified	Wistar 3
Saprophytic	*M. laidlawii* A	PG-8
	M. laidlawii B	Smith B

requires further study. There is evidence that a rat strain, *M. arthritidis,* is serologically identical to *M. hominis* type 2 strains recovered from the human genital tract(3).

Friend and coworkers(11) isolated a mycoplasma from murine leukemia cells in culture. Attempts to identify the agent in this laboratory indicated a close relationship to a swine mycoplasma strain originally thought to be *M. hyorhinis.* Further serological study showed that both the tissue culture contaminant and the swine mycoplasma being carried as *M. hyorhinis* were actually *M. granularum* strains, another serotype common in swine. Purcell and associates(12) also recently identified as *M. hyorhinis* a large group of heretofore unclassified mycoplasma strains recovered from normal tissue cultures and cell cultures used for passage of human tumor material(13-15). The results of studies relating a tissue culture isolate to *M. granularum* are reported here.

Materials and methods. Hyperimmune rabbit antisera to the mycoplasma strains listed in Table I were employed in an indirect fluorescent antibody (FA) test(16) against washed, broth mycoplasma antigens grown in 20% horse serum broth(1), or Difco serum fraction medium (9 parts PPLO broth, 1 part 25% yeast extract, 500 units/ml penicillin, and 1% Difco serum fraction). All mycoplasma strains were cloned for single colony isolates prior to preparation of antigen and antibody.

Results. Growth characteristics of Friend mycoplasma strain. The mycoplasma isolated from murine leukemia cells grew rapidly in both 20% horse serum broth or the serum fraction medium. Cultures in serum fraction broth were visibly turbid within 24 hours and sediment accumulated in the bottom of tubes after 48 hours. Cultures in liquid media were smooth and gave little evidence of a granular or flocculent type of growth. When plated to either 20% horse serum or serum fraction agar, classical "fried egg" colonies were observed after 48 hours. The Friend strain and other swine mycoplasma fermented glucose with production of acid within 24-48 hours.

Serological tests. When antigen prepared from broth cultures of the Friend mycoplasma strain was tested in the indirect FA test against antisera to prototype mycoplasma strains, only antisera to the 31 B swine strain showed evidence of a serological relationship. This strain was recovered in 1962 from a direct broth culture of a nasal swab obtained from a boar and was received from Dr. W. P. Switzer shortly thereafter. On the basis of its growth characteristics and recovery site it was thought to be a strain of *M. hyorhinis,* but its serological properties were not determined. In an attempt to confirm the identification of the Friend mycoplasma as *M. hyorhinis* another culture (strain 7) of this serotype was obtained with its appropriate antisera. The results of FA tests (Table II) indicated that neither the 31 B nor Friend mycoplasma strain was related to *M. hyorhinis.*

From evidence that the murine tissue culture isolate was related to swine mycoplasma, serological comparisons against *M. granularum* (strain 39) were carried out. These tests showed (Table II) that the tissue culture isolate and 31 B strains were *M. granularum* serotypes and that all 3 *M. granularum* strains were distinct from *M. hyorhinis.*

TABLE II. Indirect Fluorescent Antibody Studies with Mycoplasma of Tissue Culture and Swine Origin.

	Reciprocal of FA titer for antisera to:					
	M. granularum		*M. hyorhinis*	GDL group		Friend
Antigen	Strain 31B	Strain 39	Strain 7	GDL	Wistar 3	Mycoplasma
M. granularum						
Strain 31B	512	>512	16	<16	<16	256
Strain 39	256	>512	16	<16	<16	128
M. hyorhinis						
Strain 7	16	<16	128	64	256	<16
Tissue culture						
GDL	<16	<16	128	128	512	<16
Wistar 3	16	<16	256	256	>512	<16
Friend strain	512	256	<16	<16	<16	1024

Further studies with the swine mycoplasma and other tissue culture isolates confirmed the work of Purcell and associates(12) that members of the GDL or F group(13-15) of mycoplasma are related to *M. hyorhinis* (Table II).

Pathogenicity tests. Through the courtesy of Dr. W. P. Switzer, broth cultures of the Friend mycoplasma were inoculated intraperitoneally into three 3-4-week-old pigs. The swine polyserositis characteristic of *M. hyorhinis* was not observed.

Discussion. M. granularum was first described by Switzer(17) when mycoplasma recovered from the nasal cavity of swine showed several characteristics distinct from those previously described for *M. hyorhinis*(18). The granular strains were found to occur in 5-20% of swine herds and to be associated with an uncomplicated arthritis, without polyserositis, in older pigs. The classification of swine mycoplasma has been complicated by a serological heterogeneity in many of the strains, particularly among *M. hyorhinis* and those strains involved in swine enzootic pneumonia (19,20). Recent work of Dinter *et al*(20), has, however, shown that *M. granularum* is antigenically unrelated to other swine strains in both growth-inhibition and gel-diffusion tests, and the results presented here indicate that the FA test may also be useful in distinguishing swine mycoplasma strains.

The recovery of swine, and possibly murine mycoplasma, from cell cultures doubtless reemphasizes the need for control of such contamination but may, indeed, also provide some suggestions as to the possible sources of such contamination. There is no definitive evidence that reagents employed in tissue culture media are the usual sources of mycoplasma contamination, although the possimility that filtrable mycoplasma are present in certain animal sera and are not recoverable in culture until after tissue culture passage is still an open question. The close proximity of tissue culture laboratories to animal colonies also offers a possible source, particularly where the animal hosts are latently or normally infected with pulmonary mycoplasma capable of being discharged into the atmosphere as droplet aerosols. Another likely origin of animal mycoplasma in cell cultures is the contamination of cell lines with mycoplasma-infected tissue cells of murine or swine tissue cultures being carried in the same laboratory. Hayflick(1) recently discussed this possibility in regard to tissue cultures infected with mycoplasma from the human oropharynx. However, in the laboratory where *M. granularum* was isolated, only murine cells were in culture and swine tissues had not been introduced at any time. Until additional information is available concerning the origin of tissue culture contamination with swine and murine mycoplasma, cell culturists must be cognizant of the possible contamination with other animal mycoplasma.

Summary. A mycoplasma isolated from murine leukemia cells in culture was found to be serologically similar to a mycoplasma strain recovered from the nasal cavity of a pig. Additional serological tests revealed both strains to be *Mycoplasma granularum,* a species previously identified only in swine hosts.

Tissue culture contamination with swine mycoplasma reemphasizes control problems in maintaining uncontaminated cell lines and poses major questions about sources of these strains in cell cultures.

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Effects of Hypophysectomy and Growth Hormone on Renal Compensatory Hypertrophy in Rats.* (31193)

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For many years there has been disagreement as to whether renal compensatory hypertrophy (RCH) can occur in hypophysectomized rats. McQueen-Williams and Thompson(1) and Cologne(2) reported that in the absence of the anterior hypophysis compensatory renal hypertrophy does not occur. Fontaine and Veil(3), Braun-Menendez and Houssay(4), Rolf and White(5), and Astarabadi(6) concluded that renal compensatory hypertrophy could still occur after hypophysectomy. However, Astarabadi, in more recent investigations(7-9), has found that renal compensatory hypertrophy does not occur in the absence of the pituitary.

In all these experiments the remaining kidney was examined 6-86 days after the nephrectomy, and most commonly 10-25 days after the nephrectomy. Yet Addis(10) has reported that following uniliateral nephrectomy in normal rats the rate of renal compensatory hypertrophy (or restoration of renal tissue as he preferred to call it) is most rapid in the first 48 hours, continuing thereafter at a slower rate. In this study we have observed the response to unilateral nephrectomy at 2, 5 and 10 days after nephrectomy in normal rats, hypophysectomized rats, and hypophysectomized rats receiving hormone replacement.

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