ning stages of active metamorphism has been looked for in oil-test wells that are drilled 20,000 feet and more below the surface where "normal" temperatures are in the order of 200°C; however, such evidence has not yet been found. Recent conclusions (*11*) are that "low-grade" metamorphic rocks, with minerals such as chlorite, epidote, and albite, first form normally at depths in the order of 25,000 to 30,000 feet, where temperatures are probably near 250°C.

Five specimens of drill core have been studied in some detail, and the data are summarized in Table 2. All five specimens were originally shale or siltstone, but they now contain minerals characteristic of the greenschist or chlorite grade of metamorphism. The pre-Tertiary basement of the Salton Sea Basin is considered (*3*, *4*) to be 10,000 to 20,000 feet below the surface throughout most of the structural trench, and relatively young rocks of Pliocene age dominate the upper 5000 to 10000 feet. If this general picture is correct, the low-grade metamorphic rocks of the drill core must be either old basement rocks that have been upfaulted locally as a sliver between branchs of the San Andreas fault, or they are rocks of relatively young Tertiary age that are now being metamorphosed because of high temperatures at these lesser depths.

Some geologists will be of the opinion that mineral changes in this environment should be called "hydrothermal alteration" (commonly produced by the action of ore-depositing waters) rather than "metamorphism." Hydrothermal alteration is normally characterized by new hydrous minerals, and by a decrease in specific gravity of the altered rocks. Metamorphism, on the other hand, normally consists of the formation of new mineral assemblages stable at higher temperatures and pressures than former assemblages; specific gravities usually increase with metamorphism, and water content decreases. In this sense the deep Niland rocks are metamorphic, even though the brines may have brought about some net chemical change.

The age and origin of the metamorphic rocks of the drill core are not determinable with certainty from the present limited data, but several lines of evidence favor the theory of active metamorphism of relatively young rocks.

The data of Table 2 suggest that the grade of metamorphism is increasing as the depth increases within the short interval from 4477 to 4923 feet more rapidly than might be expected for normal old metamorphic rocks. Epidote was found only in veinlets in the two pieces of drill core nearest the surface, but it is abundant in the lower core as a replacement for other silicate minerals. The specific gravity of the drill core in general increases rather rapidly with increased depth, as geologists might expect from a localized rapid downward increase in grade of metamorphism. For comparison elsewhere in the same structural trough (*4*), the drill core from 0 to 4000 feet in depth has an average bulk specific gravity of 2.37; 4000 to 8000 feet, 2.44; and 8000 to 12,000 feet, 2.47. Probably the most significant point in favor of active metamorphism, however, is the fact that wherever bedding is discernible in the five core specimens, it was essentially horizontal, as evidenced by relations to the originally vertical sides of the drill core. In contrast to this, old metamorphic rocks usually have been upfaulted and tilted extensively, and original bedding is only horizontal by coincidence.

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**References and Notes**

1. The well was drilled by Joseph I. O'Neill and associates of Midland, Texas, who have given permission to publish this preliminary report. We are grateful for the courage and skill required to drill so deeply into the fantastic environment of the Niland area.
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8. The 5232-foot well, when standing open, is filled below 58 feet from the land surface with brine that is about 25 percent heavier than pure water at 20°C and is probably similarly heavier at higher temperatures.
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## Low Malignancy of Rous Sarcoma Cells as Evidenced by Poor Transplantability in Turkeys

Abstract. *Homologous and isologous transfers of Rous sarcoma cells in turkeys indicate that growth of the implanted cells contributes little, if at all, to the formation of these neoplasms. Infection of normal host cells by virus appears to be, at least in this species, the major factor in the induction and progressive growth of Rous sarcomas.*

It is well known that Rous sarcoma can be produced in chickens by implantation of tumor cells or injection of cell-free filtrates of the tumor tissue. Tumor production by cell transfer could be the result of either malignant growth of the implanted cells themselves or infection and growth of host cells initiated by virus released from the implanted cells, or a combination of both. There is evidence that in chickens proliferation of the implanted cells does occur in certain instances (*1*). However, preliminary data obtained in our laboratory (*2*) clearly indicated that in turkeys virus was much more important than cells for the induction and progressive growth of these tumors.

In the studies we report now (*3*), large numbers of intact and viable tumor cells, obtained by trypsinization of virus-induced turkey sarcomas, were implanted into the wing web of large numbers of turkeys. These experiments were conducted as follows: 7-day-old Beltsville white turkeys of both sexes were injected subcutaneously in the left wing web with 0.2-ml amounts of standard Rous sarcoma virus that had been prepared from chicken tumor tissue (*4*). Tumors were produced by injection of $10^4$ to $10^5$ pock-forming

Table 1. Tumor production in turkeys by implanted Rous sarcoma (Rs) cells.

| Age (day) | | Cells* implanted | | | Tumor response | |
|---|---|---|---|---|---|---|
| Cell recipient | Cell donor† | Kind | Type of implant | Virus titer‡ | Tu/total§ | Diameter (mm) |
| 7 | 27–29 | Rs | Homologous | 1.9–3.5 | 32/32 | 15–30 |
| 7 | 27–29 | Rs | Homologous | 2.4–3.5 | 26/26 | 15–30 |
| 7¶ | 27–29¶ | Rs¶ | Homologous¶ | 2.4–3.5¶ | 16/26¶ | 3–10¶ |
| 7 | 27–67 | Rs | Homologous | <0.3 | 2/35 | 3 |
| 7 | 27 | Normal‖ | Homologous | <0.3 | 0/15 | 0 |
| 35 | 34–44 | Rs | Homologous | <0.3 | 8/32 | 3–10 |
| 34–44 | 34–44 | Rs | Isologous | <0.3 | 8/26 | 5–10 |
| 35 | | | Cell-free virus | 2.7 | 12/12 | >10 |

* 3 × $10^5$ to 6 × $10^6$ cells per bird. † Tumors produced by inoculation with virus at the age of 7 days. ‡ Log PFU per 0.2 milliliter. § Tu/total = number birds developing tumors / total number birds inoculated. ¶ The same conditions, but antivital antiserum (turkey immune serum diluted 1:10) was also administered. See text. ‖ Control turkey normal wing web cells.

units (PFU) of virus. A few tumors were initiated by inoculation of 2 PFU or less of virus. At various times after infection, birds with well-developed wing tumors were killed, and their tumors were aseptically dissected. The tumor tissue was then processed as previously described (*5*). The number of cells in the resulting cell suspension was determined by staining with trypan blue and counting with a hemocytometer. Only viable cells were counted. In addition, the cells were grown in tissue culture to confirm their viability. A measured sample of the cell suspension was sonically disrupted, and the virus content was determined by assay in embryonated hen eggs (*6*). Another sample of intact cells of the same suspension was used for inoculation into the left wing web of turkeys. Groups of 10 to 15 turkeys were each inoculated with 0.2 ml of a cell suspension. In cases of isologous cell transfer, the bird was placed under light ether anesthesia, a piece of tumor was biopsied and processed by the same procedure, and the resulting cell suspension was reinoculated into the opposite wing web of the same donor bird from which the tumor cells were obtained. After inoculation the birds were examined daily for 35 days, and their tumor sizes were recorded.

Table 1 shows that only those tumor cells that carried appreciable amounts of infectious virus produced tumors in all of the birds inoculated. These tumors grew rapidly and reached a large size; they did not regress within the 35-day period of observation. Exposure of such cells to antiviral antibody prior to implantation into birds resulted in a 38-percent reduction of tumor incidence. Further, the tumors that did appear were small and grew at a slower rate than tumors induced in the absence of antiviral antiserum. The reduction of tumor incidence was even more pronounced if cells that contained less than log 0.3 pock-forming units of virus were implanted. Such cells were obtained from birds with tumors produced with both high and low infecting doses of virus, and all such birds possessed high levels of circulating antiviral antibody (that is, they had a serum neutralization index of $10^3$ to $10^5$ when diluted 1:10). The few tumors that appeared were very small and did not exhibit progressive growth. Some regressed within 5 weeks. Similar results were obtained with isologous transfers of cells. No marked increase in tumor incidence or growth rate was observed and regressions also occurred. The data thus show that Rous sarcoma cells are not readily transplantable and the tumors produced by cell implantation appear to be largely virus-induced. Indeed, even those few tumors that resulted from implantation of cells carrying less than log 0.3 pock-forming units of virus appeared to be virus-induced, since their incidence resembled the incidence encountered when low doses of cell-free virus were used to produce tumors (*7*). The possibility cannot be excluded that a small initial proliferation of the implanted cells did occur. However, it is quite clear that, even if this was the case, it rarely resulted, if at all, in grossly visible tumors. These observations implicate virus and infection of host cells as the major factor in causing malignant, progressive growth of Rous sarcoma transplants in turkeys.

It seems possible, especially in view of recent findings on the pathogenesis of virus-induced Rous sarcoma (*8*), that infectious virus may be essential for maintaining unlimited growth and secondary spread of established tumors as well. If so, immunity to the virus could conceivably limit the growth and systemic dissemination of these neoplasms.

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## Generalized Shwartzman Reaction in the Pregnant Golden Hamster

Abstract. *A single intraperitoneal injection of colchicine regularly elicits the generalized Shwartzman phenomenon in the pregnant golden hamster. Nonpregnant hamsters are refractory both to colchicine and endotoxin. The superiority of colchicine, as opposed to foreign endotoxin, in the pregnant subject, may be attributed to the action of native endotoxin which is allowed parenteral access as a result of colchicine-induced injury to the intestinal mucosa.*

In the course of experiments designed to study the hamster karyotype, it was observed that an intraperitoneal injection of colchicine (150 to 300 mg/kg of body weight) induced a severe reaction in pregnant golden hamsters, although it had relatively little effect in nonpregnant individuals. The pregnant animals became lethargic, exhibited extensor convulsions, and began to abort, with bleeding from the vagina. During the maximum period of observation of 24 hours, the general condition inexorably deteriorated, although death did not ensue. Systematic examination of all animals was performed under Nembutal anesthesia. The renal surface was mottled and finely irregular, with occasional petechial hemorrhages. Accentuation of the lobular pattern of the liver,