ABSTRACT

We describe the derivation of three human ovarian carcinoma cell lines and the comparison of their properties with two previously described cell lines of like histology (SKOV-3 and CAOV-3). Two of the new lines (HOC-1 and HOC-7) were derived from separate ascites tumors (at 9-month intervals) of a patient with well-differentiated serous adenocarcinoma of the ovary. The third new line, HEY, was derived from a human ovarian cancer xenograft (HX-62) originally grown from a peritoneal deposit of a patient with moderately differentiated papillary cystadenocarcinoma of the ovary. The cell lines demonstrated differential ability to grow in semisolid culture and as xenografts in immunologically deprived CBA/CJ mice. Dose-response curves were generated for clonogenic cell survival of cells exposed to common chemotherapeutic agents; one of the lines (HEY) shows a degree of resistance to the alkylating agent cis-diaminedichloroplatinum(ll) (cis-platinum). Common karyological features included structural abnormalities of chromosomes 3 and 11. Heterogeneity of expression of ovarian tumor-associated antigens was documented.

INTRODUCTION

A limited number of established cell lines from human ovarian carcinoma cells have been described previously (8, 10, 12, 22, 29). To varying degrees, these cell lines have been shown to retain tissue-specific characteristics enabling their use in studies of tumor-associated antigenicity (18), hormone production and dependency (14), and growth control (28).

A number of recent studies have shown that primary ovarian tumor cells are also able to be grown in short-term semisolid cultures and that assessments can be made of the properties of clonogenic cell survival of cells exposed to common chemotherapeutic agents; one of the lines (HEY) shows a degree of resistance to the alkylating agent cis-diaminedichloroplatinum(ll) (cis-platinum). Common karyological features included structural abnormalities of chromosomes 3 and 11. Heterogeneity of expression of ovarian tumor-associated antigens was documented.

MATERIALS AND METHODS

Patient Material

Case A. Cell lines HOC-1 and HOC-7 were initiated from separate malignant ascites (April 29, 1980 and December 16, 1980) of a patient with a serous well-differentiated Stage III adenocarcinoma of the ovary. Her treatment history was as follows. Diagnosis was by exploratory laparotomy 2 years prior to first ascites sample. For 7 months prior to first paracentesis, she had no treatment; for 9 months between initiation of HOC-1 and HOC-7, she had paracentesis at approximately monthly intervals. The characteristics of the cells obtained by paracentesis are described elsewhere (17).

Case B. Cell line HEY was initiated from a disaggregated xenografted ovarian tumor. The derivation of the xenografted tumor (HX-62) (obtained from Dr. A. Jones, Institute for Cancer Research, Sutton, England) has been described (21). The initial biopsy was a peritoneal deposit of a moderately differentiated papillary cystadenocarcinoma of the ovary. The cell line was initiated after 3 passages of the xenograft in immunologically deprived mice.

Initiation and Maintenance of Cell Lines HOC-1, HOC-7, and HEY

Case A. HOC-1 and HOC-7 were initiated from separate ascites samples in identical fashion. Ascites cells were harvested by centrifugation at 600 x g for 10 min and resuspended in α-MEM medium plus FCS (10%, v/v). Mononuclear cells were prepared by Ficoll-Hypaque, and the washed cells were frozen in liquid nitrogen in dimethyl sulfoxide (10%, v/v) and FCS (20% v/v). Thawed cells were subsequently seeded in MEM plus 10% FCS at a concentration of approximately 10⁴ cells/flask, and culture was initiated. Passaging by trypsinization (0.025%) has continued for approximately 24 months on a weekly basis (1:10 in α-MEM:10% FCS), and cells at various passages have been maintained frozen.

Case B. An ovarian tumor xenograft HX-62 (21), passage 3, was grown in the flank of an immunologically deprived CBA/CJ mouse (15). Tumor pieces were aseptically removed and washed in α-MEM:10% FCS. They were minced with crossed scalpels, forced through a fine copper screen, and passed through needles of decreasing size to 23 gauge. The resulting cell suspension was seeded and passaged as described for Case A. The line has been maintained in this manner and by freezing at various passages for 18 months.

Maintenance of Established Cell Lines

Cell lines CAOV-3 and SKOV-3 were obtained from the American Type Culture Collection, Rockville, MD. No banded karyotypes have been published previously on these lines. The cell lines were obtained at passages 30 and 19, respectively. Both have been maintained by passage continuously on a weekly basis (SKOV-3: 1:8 in α-MEM containing 15% FCS; CAOV-3: 1:4 in α-MEM containing 10% FCS) and by cryopreservation.

Measurement of Clonogenicity

Assessments of clonogenic cell frequency in plastic-adherent cultures were performed by seeding an appropriate cell number in triplicate T-25 flasks in α-MEM containing 10% FCS, staining with methylene blue after 1 to 2 weeks of culture, and counting colony number (>50 cells) under a microscope.

1 Supported by grants from the National Cancer Institute of Canada (R. N. B.) and by Department of Health and Human Services Grants CA 29476 and CA17094 from the National Cancer Institute (J. M. T.).
2 To whom requests for reprints should be addressed.
3 Scholar of the Leukemia Society of America.
4 The abbreviations used are: MEM, minimal essential medium; FCS, fetal calf serum; CEA, carcinoembryonic antigen; PBS, phosphate-buffered saline; cis-platinum, cis-diaminedichloroplatinum(ll).
low-power inverted microscopy.

Semisolid cultures were initiated in similar fashion in 35-mm tissue culture dishes in 2-layer systems of agar (0.3%, w/v) on agar (0.5%, v/v) or methylcellulose (0.8%, v/v) on agar (0.5%, v/v). Colony counts were performed at an appropriate time of incubation determined by serial observation, using a criterion of ≥50 cells as designation of a clonal unit.

Growth as Xenografts

Cell lines (2 x 10⁶ cells) were injected into the right thigh muscle of immunologically deprived CBA/CJ mice (15). Following transplantation, all animals were observed weekly until their tumors grew to a mean diameter of 1.0 cm or for at least 4 months in the absence of tumors. Excised tumors were fixed in formalin, and thin paraffin sections were cut and stained with hematoxylin and eosin for histological analysis.

 Cytofluorometric Analysis of Antigen Presentation

The antibodies used detected NB/70K (4), OC-125 (3), Ca antigen (2), CEA (4), and BA-2. A rabbit antiserum directed against NB/70K and the murine monoclonal antibodies detecting OC-125 and Ba-2 were generously provided by Dr. G. I. Urbach, Wellesley Hospital, Toronto, Ontario, Canada; Dr. R. Bast, Sydney Farber Cancer Center, Boston, MA; and Dr. T. Lebien, University of Minnesota, Minneapolis, MN, respectively.

Murine monoclonal antibodies directed against the antigens CA and CEA were purchased from Wellcome Diagnostics, England, and Hybritech, La Jolla, CA, respectively. Either fluorescent goat anti-rabbit or goat anti-mouse IgG (Cappel) was used as second antibody as appropriate.

Quantitation of the levels of ovarian tumor-associated surface antigens on cells was performed on a Coulter Epics flow cytometer. Cells (1.5 x 10⁶) suspended in PBS were reacted with the first antibody for 30 min at 4°C. The cells were then washed twice and resuspended in PBS containing an appropriate fluorescein-conjugated second antibody. After 30 min incubation at 4°C, cells were washed 3 times in PBS and analyzed at 488 nm. Antibodies were always used at saturating concentrations.

Karyotyping

Harvesting, slide preparation, and chromosome banding analysis of all cell lines were performed as described previously (26).

Electron Microscopy

Cell layers were fixed in situ with 3.5% glutaraldehyde in cacodylate buffer, postfixed for 1 h in 1% buffered osmium tetroxide, and stained for 30 min with 20% aqueous uranyl acetate. Following dehydration in parallel to the monolayer and stained with uranyl acetate and lead citrate before being examined in an Elmiskop I electron microscope. All cell lines demonstrated tight junctions and surface microvilli; Fig. 1B shows typical cell junctions from the cell line HOC-7.

Cell Growth Characteristics. The doubling times for plastic-adherent growth of the lines under the growth conditions described in "Materials and Methods" are: HOC-1, 36 h; HOC-7, 20.5 ± 3.0 h; HEY, 17.0 ± 3.0 h; SKOV-3, 17.5 ± 2.0 h; CAOV-3, 6.1 ± 1.5 h.

16.4 h; HEY, 30 h; SKOV-3, 28.8 h; CAOV-3, 78 h. All cell lines will form recognizable colonies when grown in low cell density on plastic substrate (Fig. 1A). Colony growth efficiency is linearly related to plated cell number for all lines. The plating efficiencies and gross colony descriptions are given in detail in Table 1A. In addition, clonal growth was initiated in culture rendered semisolid by agar or methylcellulose (Table 1B). In all cases of positive clonogenicity, frequency of colony growth could be linearly related to the number of cells plated. Gross colony morphology was similar for all positive lines, tightly packed spherical groups of cells allowing easy quantitation.

Aliquots (2 x 10⁶) of cells from all lines were also injected into the flanks of immunologically deprived CBA/CJ mice, and subsequent tumor development was monitored. The HEY and SKOV-3 cell lines produced continuously growing tumors in 100% of mice. Tumors reached 1 cm diameter after approximately 5 weeks. No tumors developed within 3 months after injection of HOC-1, HOC-7, or CAOV-3. Resulting tumors from HEY and SKOV-3 were assessed pathologically and had a history consistent with that of adenocarcinoma. A representative example of a tumor formed from the HEY cell line is shown in hematoxylin-eosin-stained section in Fig. 1C.

Presentation of Tumor-associated Antigens. Expression of antigens NB/70K, OC-125, CA-1, CEA, and BA-2 were determined by flow cytometry on cells harvested by gentle trypsinization and exposed to appropriate antibody combinations. The result of a typical experiment with the expression of CEA, OC-125, and BA-2 on cell line HOC-7 is shown in Chart 1. Expression of antigens was classified as positive (strong to weak) or negative, and the accumulated results are presented in Table 1A. Trypsinized cells resuspended in α-MEM:10% FCS and incubated in roller tubes for 24 h showed the same patterns of antibody recognition. Parallel assessment of antigen presentation by fluorescence microscopy yielded qualitatively similar data (results not shown).

Karyology. A comparison of numerical and structural chromosome alterations between all 5 cell lines is presented in Table 3, with documentation of chromosome alterations presented in Figs. 2 to 5. Results from these cell lines are in good general agreement with chromosome alterations commonly observed in direct specimens from human ovarian adenocarcinomas (Ref. 25; for review, see Ref. 22). Those chromosomes most consistently involved in either structural or numeric alterations were chromosomes 3, 6, and 11. Structural alterations of the short arm of chromosome 3 involving bands p12-13 were found in 4 of 5 cell lines. One example of an altered chromosome is shown in Table 3A.

Table 1

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Assessment of clonogenic growth of ovarian cancer cell lines</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Growth on plastic substrate</td>
<td>B. Growth in semisolid medium</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Plating efficiency (%)</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>HOC-1</td>
<td>11.5 ± 1.0</td>
</tr>
<tr>
<td>HOC-7</td>
<td>20.5 ± 3.0</td>
</tr>
<tr>
<td>HEY</td>
<td>17.0 ± 3.0</td>
</tr>
<tr>
<td>SKOV-3</td>
<td>17.5 ± 2.0</td>
</tr>
<tr>
<td>CAOV-3</td>
<td>6.1 ± 1.5</td>
</tr>
</tbody>
</table>

*I. Royston, personal communication.
HUMAN OVARIAN CANCER CELL LINES

Table 2

<table>
<thead>
<tr>
<th>Tumor-associated antigen</th>
<th>Cell line</th>
<th>NB70K</th>
<th>OC-125</th>
<th>Ca-1</th>
<th>CEA</th>
<th>Ba-2</th>
</tr>
</thead>
<tbody>
<tr>
<td>HOC-1</td>
<td>+*</td>
<td>+</td>
<td>Not tested</td>
<td>Not tested</td>
<td>Not tested</td>
<td>+</td>
</tr>
<tr>
<td>HOC-7</td>
<td>+</td>
<td>+</td>
<td></td>
<td></td>
<td>-</td>
<td>++</td>
</tr>
<tr>
<td>HEY</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>++</td>
</tr>
<tr>
<td>SKOV-3</td>
<td>±</td>
<td>+</td>
<td>++</td>
<td>++</td>
<td>++</td>
<td></td>
</tr>
<tr>
<td>CAOV-3</td>
<td>±</td>
<td>+</td>
<td>++</td>
<td>++</td>
<td>++</td>
<td></td>
</tr>
</tbody>
</table>

*++, strongly positive; +, positive; ±, weakly positive; −, negative.

Chart 1. Flow cytometric analysis of binding of antibodies to CEA, OC-125, and Ba-2 to the HOC-7 cell line. Control represents HOC-7 cells exposed to nonspecific mouse IgG.

DISCUSSION

We describe the establishment of 3 new ovarian carcinoma cell lines. Two of these lines (HOC-1 and HOC-7) were established from the ascites tumor of an individual patient at different stages of her disease. This patient has been the subject of previous reports concerning clonal evolution and disease progression (17, 26). The third line (HEY) was established from a human ovarian tumor which had been passaged previously as a xenograft in immunologically deprived mice (21).

All 3 lines display epithelial morphology under light and electron microscopy (Fig. 1). Growth on plastic displayed similar large cuboidal cells forming a whorling cobblestone appearance without obvious fibroblastic contaminants. When plated at low cell numbers for colony formation, the cell line HEY was noted to form clonal units which were much more loosely packed than the other cell lines (Table 1). This would make quantitation of clonogenicity more difficult at high cell inocula.

The proliferative features of the cell lines are characteristic. To summarize the data of Table 1 and xenografting experiments,

displayed an apparently bimodal distribution around 49 and 94 chromosomes (with a wide variation in chromosome number observed between these 'modes'). The remaining tumors displayed a mode near-triploid for SKOV and CAOV and pseudo-diploid for HOC-1 and HOC-7.

Drug Sensitivity. We have assessed the drug sensitivity of the lines HOC-7, HEY, SKOV-3, and CAOV-3 to 3 agents commonly applied to the chemotherapy of ovarian cancer, Adriamycin, vinblastine, and cis-platinum. Response of the lines to the drugs was assessed by measurement of fractional survival of clonogenic cells (plastic adherent) in response to continuous contact with the drugs during growth of the cells. An example of the dose-response curves obtained is shown in Chart 2 for HEY and SKOV-3 cells exposed to cis-platinum. The combined data are summarized in Table 4 as the doses of drug required to reduce survival to 10% of control.

[del(3)(p13:)]. Consistent with earlier reports of ovarian cancers (16, 23, 27), alteration of the long arm of chromosome 6 was a common finding in these cell lines. In all lines except HOC-1 and HOC-7, a simple deletion involving band regions 6q21-23 was observed to be a clonal feature, present in approximately 100% of all cells examined. Finally, structural alterations of chromosome 11 were found in 4 of 5 tumor lines, with the most frequently altered band region being 11q13-21. In 3 cases, a simple deletion of 11q was observed; while in a single case (HOC-7) (Fig. 5), a translocation involving 11q13 was observed.

In addition to these identifiable chromosome alterations, all tumor lines with the exception of HOC-1 and HOC-7 contained numerous unidentified marker chromosomes (umars) (Figs. 2 to 4).

Unfortunately, karyotypic information on the primary tumor specimens from which these lines were derived was available only for the HOC-1 and HOC-7 cell lines. In these lines, results comparing the in vivo karyotype with the karyotype of cells grown in vitro in monolayer culture revealed conservation of most structural and numeric clonal alterations. Finally, the HOC-7 cell line demonstrated significant karyotypic "evolution" (as defined as increasing karyotypic progression) based upon results of serial harvests over a 1-year period in vitro (26).

The HEY cell line demonstrated a highly aneuploid karyotype with a high percentage of polyploid cells (>50%) and numerous clonal marker chromosomes. The chromosome number of the 75 cells examined varied within a range of 43 to 100 and...
### Table 3

Cytogenetic features of 5 established ovarian carcinoma cell lines

<table>
<thead>
<tr>
<th>Cell line</th>
<th>Banding</th>
<th>No. of cells counted</th>
<th>Range Mode</th>
<th>Clonal structural abnormalities</th>
</tr>
</thead>
<tbody>
<tr>
<td>SKOV</td>
<td>G-,Q-</td>
<td>25</td>
<td>32-88</td>
<td>74</td>
</tr>
<tr>
<td>CAOV</td>
<td>G-,Q-,C-</td>
<td>25</td>
<td>43-4N</td>
<td>69</td>
</tr>
<tr>
<td>HEY</td>
<td>G-</td>
<td>75</td>
<td>45-113</td>
<td>ND</td>
</tr>
<tr>
<td>HOC-1</td>
<td>G-,Q-</td>
<td>100</td>
<td>34-4N</td>
<td>49</td>
</tr>
<tr>
<td>HOC-7</td>
<td>G-,Q-</td>
<td>100</td>
<td>42-4N</td>
<td>50</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>del(1;13)(p22;q13q34)</th>
<th>del(3)(p13)</th>
<th>del(6)(q23)</th>
<th>del(11)(q21)</th>
<th>del(12)(q22)</th>
<th>±15-19 umars*</th>
</tr>
</thead>
<tbody>
<tr>
<td>SKOV</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAOV</td>
<td>del(1;13)(p22)</td>
<td>del(3)(q21)</td>
<td>del(6)(q23)</td>
<td>t(7;13)</td>
<td>del(3)(q21)</td>
<td></td>
</tr>
<tr>
<td>HEY</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HOC-1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HOC-7</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

*umars, unidentified marker chromosome; ND, not determined.

---

Cis-platinum (µg/ml)

**Chart 2.** Dose-response curves for cytotoxicity of cis-platinum to ovarian cancer cell lines; drug was included in the culture medium at the concentrations indicated. ○, HEY; ●, SKOV-3. Clonogenic cells on plastic were assessed, and fractional survival was calculated. Points, means for triplicate flasks; bars, SE.

### Table 4

Drug sensitivity of 4 ovarian cancer cell lines

<table>
<thead>
<tr>
<th>Cell line</th>
<th>Adriamycin (µg/ml)</th>
<th>Vinblastine (µg/ml)</th>
<th>cis-Platinum (µg/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HOC-7</td>
<td>4 x 10^{-3}</td>
<td>2 x 10^{-4}</td>
<td>5 x 10^{-2}</td>
</tr>
<tr>
<td>HEY</td>
<td>4 x 10^{-3}</td>
<td>4 x 10^{-4}</td>
<td>1.0</td>
</tr>
<tr>
<td>SKOV-3</td>
<td>8 x 10^{-4}</td>
<td>6 x 10^{-5}</td>
<td>7.5 x 10^{-2}</td>
</tr>
<tr>
<td>CAOV-3</td>
<td>2.5 x 10^{-4}</td>
<td>3 x 10^{-5}</td>
<td>&lt;2.5 x 10^{-1}</td>
</tr>
</tbody>
</table>

HOC-1 is clonogenic on plastic but will not grow in semisolid medium or in immunologically deprived mice. HOC-7, however, will grow as colonies on plastic and in semisolid medium but also is not tumorigenic in the xenogeneic host. HEY grows under all 3 circumstances. Of the previously established cell lines, SKOV-3 likewise grows under all 3 circumstances, while CAOV-3 fails to grow in semisolid medium but is tumorigenic in immunologically deprived mice (9). Appropriate selection of cell line can therefore be made to study therapeutic effects in culture and in vivo.

Analysis of the cell surface display of ovarian cancer-associated antigens revealed an individual pattern of expression. HOC-1 and HOC-7 were NB/70K, OC-125, and BA-2 positive. HEY was negative for all antigens except BA-2. Of the 2 established lines, SKOV-3 was negative for all markers, and CAOV-3 was positive for all antigens except CEA. The tumors of origin of the lines HOC-1, HOC-7, and HEY contained some cells positive for all 5 tumor-associated antigens (4). However, it is also clear that many of the original tumor cells were "null" with respect to these markers. It is possible, therefore, that the lack of expression of some of the ovarian tumor-associated antigens in these cell lines reflects the origin of the lines from non-antigen-expressing tumor cells. Alternatively, the expression of these antigens may be related to a process of cell differentiation rather than to the transformed state (4). Nevertheless, the antigen expression pattern clearly is a characteristic of the individual line; as such, these cells may prove valuable in assessing the biological function of these tumor-associated antigens.

Considerable heterogeneity is seen in the ability of the cell lines to form colonies in semisolid or viscous cultures. HOC-7 demonstrates a high plating efficiency (10%) in agar but grows poorly in methylcellulose (0.5% plating efficiency). SKOV-3, however, shows higher plating efficiency in methylcellulose. Such individual preferences for semisolid support have also been noted among directly cultured ovarian carcinoma ascites samples (5).

There is no concordance between features of tissue culture...
growth and tumorigenicity in immunologically deprived mice.

HOC-1 and HOC-7 were derived from biopsies from an individual patient at different stages of disease progression. The only characteristic of the cell lines which point to a clear divergence of properties is that of growth in semisolid medium. HOC-1 is unable to proliferate in agar or methylcellulose while HOC-7 grows well in agar and to a lesser degree in methylcellulose. All other measured characteristics are indistinguishable, including their inability to form tumors in immunologically deprived mice.

Bandaging analysis of all 5 ovarian tumor lines revealed an aneuploid karyotype with numerous numeric and structural abnormalities. Chromosomes 3, 6, and 11 appeared to represent the most frequently altered chromosomes in these lines, a finding consistent with earlier reports (16, 23, 24, 27). Banding analysis of 3 cell lines (SKOV, CAOV, and HEY) demonstrated a simple deletion of chromosome 6q21-23, a region of the long arm of chromosome 6 which has been suggested by several investigators to represent a nonrandom site of chromosome change in ovarian neoplasia (16, 23, 27). It has been reported recently that translocations or deletions involving this same region of chromosome 6 have also been shown to occur nonrandomly in human malignant melanoma (25). The molecular basis which may explain the similarity in chromosome change between melanoma and ovarian cancer may have been identified recently when Harper et al. (13) mapped the c-myb oncogene to chromosome 6q22-24. Currently, it is unknown whether the alterations of chromosome 6 in melanoma or ovarian cancer are directly related to the location of the c-myb oncogene. Investigations are under way in our laboratories to elucidate whether the c-myb oncogene is located at or near the breakpoint of 6q demonstrated in these ovarian cell lines (or whether it is translocated elsewhere within the genome). However, preliminary experiments indicate that the c-myb gene is not expressed at abnormal high levels in the HEY cell line.*

The range of drug sensitivity seen in 4 lines to Adriamycin, vinblastine, and cis-platinum is shown in Table 4. In general, Adriamycin and vinblastine sensitivity falls within a narrow range, but considerably more variability is seen in the sensitivity to cis-platinum. Of particular interest is the apparent resistance of the HEY cell line to cis-platinum. This line may therefore prove to be valuable in assessing mechanisms of cis-platinum resistance in culture and in vivo since it also can be grown as a xenografted tumor.

In summary, we believe that it will be possible, on the basis of these data, to select a model ovarian carcinoma cell line with the combination of characteristics necessary to answer many questions relating to ovarian tumor biology and therapeutic effects.

ACKNOWLEDGMENTS

We gratefully acknowledge the assistance of Dr. S. Fine in providing the tumor material used to derive cell lines HOC-1 and HOC-7 and Dr. P. Selby for initiating the xenografted tumor HK-82 (the source of HEY cell line) in our laboratory. Our thanks also to Dr. I. Tannock for providing the facilities of his laboratory in the xenografting studies, to the late Dr. A. Howatson for the electron microscopic studies, and to F. Thompson, J. Coxhead, and K. Massey for excellent technical assistance in the karyotyping studies.

REFERENCES

Fig. 1. A, representative Wright's-stained colony of HOC-7 cells adherent to the culture flask. × 100. B, high-power electron micrograph of HOC-7 cell line in monolayer culture. Thick arrows, macula adherens; thin arrow, puncta (zonula) adherens. × 3200. C, thin section of tumor formed by HEY cell line in immunologically deprived mice. H & E, × 350.
Fig. 2. Q-banded karyotype of SKOV cell line. Identifiable clonal structural markers (arrows) are described in more detail in Table 3. Deletion 10p chromosome seen in this karyotype was not a clonal change.

Fig. 3. Q-banded metaphase karyotype of the CAOV cell line. Arrows, identifiable clonal structural markers. A detailed description of these markers is given in Table 3.
Fig. 4. G-banded karyotype of the HEY cell line. Arrow, del(6)(q21-23:) chromosome. Other clonal structural markers observed in this karyotype include: m1 = t(5;7)(p15.2;?), m2 = i(8q), m3 = del(3)(p12:); m4 = a putative del(9)(p13:) chromosome; and a number of unidentified marker chromosomes (umar). Inset, additional examples of clonal markers from another cell, including a del(11)(q21:) chromosome which was a clonal marker that was not present in the karyotype. Clonal markers m5 and m6 could not be positively identified.
Fig. 5. A, G-banded partial metaphase spread from the HOC-1 cell line showing the only clonal structural abnormality detected, a pericentric inversion of chromosome 3. A G-banded partial metaphase from a late passage of the HOC-7 cell line in B displays the same inv(3) marker in addition to other clonal structural rearrangements which include a t(11;13) chromosome. A nucleolar organizing region (NOR)-banded partial spread of late-passage HOC-7 in C demonstrates that the t(11;13) marker has retained the “stalk” region of chromosome 13, thereby serving to confirm the proposed breakpoints at 11q13 and 13p13. Open arrows in C, the NOR bands on another D-group chromosome and 2 G-group chromosomes which are in satellite association. Further description of clonal markers is given in Table 3.
Comparative Properties of Five Human Ovarian Adenocarcinoma Cell Lines

R. N. Buick, R. Pullano and J. M. Trent


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