A 61,XY Cell Line in a Calf with Extreme Brachygnathia

Abstract
Cells from kidney and adrenal tissues of a male calf with extreme brachygnathia inferior were grown in tissue culture for a chromosome study of this birth defect. Six of 17 metaphases with a 61,XY complement were karyotyped. The extra autosome was a large acrocentric of the A group. This finding provides further evidence that a chromosomal defect is associated with some forms of brachygnathia inferior in cattle.

Introduction
Imperfect development of the mandible in the bovine fetus results in either a lethal or a nonlethal congenital defect (2, 3, 7, 8). The lethal form, commonly called “Andy Gump” calf, is agnathia or complete absence of the lower jaw. The nonlethal form, brachygnathia inferior or “parrot-mouth,” is a slight shortening of the jaw. An intermediate form, extreme brachygnathia, is also lethal.

Gilmore (2) reported agnathia in one Angus and four Jersey calves and that a recessive gene was indicated. Grant (3) reported on 15 cases of underdeveloped mandible in a herd of Dairy Shorthorn cattle. Male calves appeared more susceptible to the abnormality than females and the character was considered a recessive and lethal. The inheritance of “parrot-mouth” is more than just a simple recessive (7, 8).

In Germany and Japan, similar karyotypes of either 61,XY, A+ or 61,XX, A+ were reported in two male and two female calves with brachygnathia (4, 5, 6).

This paper reports a fifth cytogenetic analysis of this congenital defect.

Experimental Methods
A newborn Brown Swiss male calf with extreme brachygnathia was presented to our clinical for examination. Since the calf was having difficulty in breathing, it was euthanized for post mortem examination. Kidney and adrenal tissues were trypsinized and grown in culture according to the method described by Dunn et al. (1). Cells arrested at metaphase were harvested, stained, and photographed (1).

Results and Discussion
The calf’s lower jaw was 4.5 cm shorter than the upper jaw. The palate was cleft along its entire length except for a 1 cm portion posterior to the dental pad. The eyes were subjectively judged to be smaller than normal (microphthalmia). A 1-cm defect was found in the interventricular septum of the heart just below the aortic valve rings. The left kidney was cystic, and both testes were retained near the kidneys.

Thirty metaphases, 13 from kidney cells and 17 from adrenal cells, were photographed for analysis. The distribution of chromosome counts is in Table 1.

The modal number was 61 for each tissue. Six karyotypes were made from metaphases with the 61,XY complement. A typical karyotype is shown in Figure 1. Since the extra chromosome appeared to be larger than any of the autosomal set, the karyotype of these cells is indicated as 61,XY, A+ . A karyotype of a 60,XY cell appeared to be normal.

The degree of brachygnathia and similarity of the karyotypes on five calves of different breeds are in Table 2.

These independent cytogenetic investigations give support to the hypothesis that a chromosomal defect, an autosomal trisomy or increment of one acrocentric chromosome, is a factor in the etiology of the lethal defect, extreme brachygnathia inferior. The 3:2 ratio of males to females does not indicate that males are more susceptible to brachygnathia as previously reported (3).

The possibility of a recessive gene in this case appears remote. A normal calf was born to a second mating of these unrelated Brown

Table 1. Chromosome counts in metaphases from cultured tissues of the calf.

<table>
<thead>
<tr>
<th>Tissue</th>
<th>&lt;56</th>
<th>56</th>
<th>57</th>
<th>58</th>
<th>59</th>
<th>60</th>
<th>61</th>
<th>&gt;61</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kidney</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td>3</td>
<td>7</td>
<td>1</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>Adrenal</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td></td>
<td>4</td>
<td>10</td>
<td>1</td>
<td>17</td>
<td>30</td>
</tr>
<tr>
<td>Total</td>
<td>1</td>
<td>3</td>
<td>1</td>
<td></td>
<td>7</td>
<td>17</td>
<td>1</td>
<td>30</td>
<td></td>
</tr>
</tbody>
</table>
Fig. 1. Typical karyotype.
**Table 2. Similarity of karyotypes of brachygnathia in five calves of different breeds.**

<table>
<thead>
<tr>
<th>Reference</th>
<th>Breed</th>
<th>Shortening of mandible (cm)</th>
<th>Karyotype</th>
</tr>
</thead>
<tbody>
<tr>
<td>Herzog and Höhn (4)</td>
<td>Rotbunt</td>
<td>2.0</td>
<td>61,XX, ? C+</td>
</tr>
<tr>
<td>Mori, et al. (6)</td>
<td>Holstein</td>
<td>3.0</td>
<td>61,XY, ? +</td>
</tr>
<tr>
<td>Höhn and Herzog (5)</td>
<td>Fleckvieh</td>
<td>2.5</td>
<td>61,XY, ? +</td>
</tr>
<tr>
<td></td>
<td>Rotvieh</td>
<td>3.5</td>
<td>61,XX, ? +</td>
</tr>
<tr>
<td>Present case</td>
<td>Brown Swiss</td>
<td>4.5</td>
<td>61,XY, ? A+</td>
</tr>
</tbody>
</table>

Swiss parents, and the sire had over 5,000 calves in artificial breeding with no reports of brachygnathia in his progeny.

Unpublished data by Whitlock (9) raises the possibility of an environmental agent in the etiology of the nonlethal form of brachygnathia. In a herd of Ayrshires with a high incidence of bovine virus diarrhea, eight calves from four artificial inseminating sires were malformed. Three of the four sires produced five calves with "parrot-mouth." One of these was karyotyped and was normal 60,XX.

More cytogenetic studies of all forms of lethal and nonlethal forms of brachygnathia in cattle, together with experimental exposure of pregnant cows to viral or chemical agents, are indicated.

**H. O. DUNN and R. H. JOHNSON, Jr.**
Large Animal Medicine, Obstetrics and Surgery Department, New York State Veterinary College, Ithaca, New York 14850

**Acknowledgment**

The authors gratefully acknowledge Dr. E. A. Holzinger for the pathological examination.

**References**


(9) Whitlock, R. H. Personal communication. 1971.