elaborated by these cells. Whether or not polymorphonuclear cells also produce interferon cannot be determined from the data now available.

The number of variables in the experimental system precluded quantitative comparison of interferon titers. It should be entirely feasible, however, to control these variables and thus derive significant information on rate and degree of interferon production by leucocytes from patients with a variety of diseases.

Whether interferon-like substances are actually produced by leucocytes *in vivo* remains uncertain though there appears to be no reason to consider this unlikely. In this regard it is of interest that leucocytes from patients with classical congenital agammaglobulinemia produce interferon *in vitro*. It is well known that this group encounters relatively little difficulty with naturally acquired viral diseases and it has been suspected that their recovery may depend on factors other than conventional antibody.

Though we have been unable to demonstrate production of interferon by uninoculated leucocytes taken from patients with acute viral diseases, it should be emphasized that in these cases no virus was isolated from peripheral blood. If leucocytes were taken during the viremic phase of a disease such as measles it is entirely possible that interferon might be produced by these cells when subsequently maintained *in vitro*. This supposition would seem to find support from the following considerations: 1) measles virus has been isolated from the buffy coat of leucocytes from patients with measles(9). 2) measles virus has been propagated in suspensions of human and monkey leucocytes(1). 3) measles virus, as we have shown on 2 occasions, does induce production of interferon by leucocytes *in vitro*.

We were unable to detect interferon in the medium of normal leucocytes inoculated with material from patients with rubella. However, this approach to the detection of certain non-cytopathogenic agents might prove to be of considerable value, since it is possible that such agents adapted to the human host might multiply in human leucocytes maintained *in vitro* and reveal their presence by the production of interferon.

Summary. Suspensions of leucocytes from 2 normal donors and 18 patients with various diseases, including 3 patients with congenital agammaglobulinemia were exposed to Sendai or measles virus and maintained *in vitro* for several days. Production of interferon occurred in 21 of 22 preparations. It is suggested that interferon-like substances may be liberated by leucocytes *in vivo*, after infection with some viruses and that production of interferon by leucocytes may provide a criterion for presence of non-cytopathic viruses.

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Fetal Plasma Potassium.* (27073)

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Christianson and Jones(1) have summarized evidence that fetal plasma potassium concentration is far higher than the normal adult range. Reported values are as high as 17 meq/l(2), while others are around 9 meq/l.

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Since these levels are in the lethal zone for the adult, and since some of our analyses and those of others(3-6) were far below this level, our data were analyzed for the cause of the variation. The elevated K concentrations appear to be associated with conditions of fetal hypoxia, and there is no evidence in our series that fetuses normally exist *in utero* with such high concentrations.

Procedure. On days 19-21 of gestation, blood of fetal rats has been collected in hematocrit capillaries from the punctured chin sinus and centrifuged immediately. Hemolysis was not present. In some experiments, the samples were collected in less than one minute after delivery by Caesarean section. In others, the whole uterus had been removed, the fetuses delivered from their membranes in less than 14 minutes, and the blood sampled in less than 30 minutes. In still others, the dissections required 20-40 minutes, with sampling completed before the end of an hour. Some fetuses survived each procedure. Potassium was determined by Beckman B flame photometry.

Results. Table 1 summarizes the evidence that fetal plasma K is elevated in proportion to the duration of conditions resulting in hypoxia. When fetuses were sampled before hypoxia could become severe, plasma K concentration was in the upper half of the range for normal adults. Only in moribund or dead fetuses were concentrations of 9 meq/l and above observed. In littermates that were able to establish rhythmic respiration, concentration seldom exceeded 7.5 meq/l.

Discussion. The discrepancies between values in the literature may be explained similarly, as also indicated by Brandt, Harned, and Cooke(4), who have reported fetal values in the normal adult K range for normal fetuses, followed by significant increases after hypoxia was induced. Samples were obtained through indwelling catheters. Other studies in which the fetus was in good viable condition report fetal plasma K concentrations in the normal adult range for several species(4,5,6). All of these laboratories have been concerned with fetal respiratory problems as well as electrolytes and are perhaps more aware of the necessity for obtaining samples before hypoxia develops. Others apparently have been less cautious in avoiding sampling under conditions in which hypoxia was probable, e.g., tissues brought from the slaughterhouse or drainage of blood after decapitation. High values comparable to our delayed dissections are reported in these studies.

It is well known that plasma K rises rapidly as death approaches in adults, and there is reason to believe that fetal plasma K behaves similarly. Battaglia, *et al.*(7) have shown that fetal osmotic pressure rises rapidly on brief exposure to hypoxia. At least a part of the increase is undoubtedly due to increased K. Whether the K elevation is quantitatively different from that of adults is uncertain. Nevertheless, plasma K concentrations of fetuses measured after hypoxia of only a few minutes' duration should not be designated "normal".

Conclusion. Fetal plasma potassium concentration is normally within the range reported for adults. Markedly elevated K concentrations are observed after brief periods of hypoxia. Concentrations up to 17 meq/l may be anticipated if the fetus dies.

TABLE I. The Effect of Hypoxia on Fetal Plasma Potassium.

Duration of stay within membranes after removal of uterus from mother, min.	Respiratory responsiveness at delivery	Mean plasma K, meq/l ± S.D.	Maximum plasma K, meq/l	Number of fetuses	Number of litters
<1	Good	5.6 ± 0.6	6.4	13	3
<14	Present or rhythmic	6.2 ± 1.1	7.5	17	5
	Absent	9.3 ± 1.3	12.1	7	7
	All fetuses	7.1 ± 1.8	—	24	8
20-40	Present but not rhythmic	9.8 ± 1.2	12.6	11	5
	Absent	12.8 ± 2.1	16.6	31	9
	All fetuses	12.0 ± 2.3	—	42	13

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Western Encephalitis Virus in Massachusetts.*† (27074)

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This report describes the first recorded recovery of western encephalitis (WE) virus from mosquitoes and birds in Massachusetts and serological evidence indicating that a considerable amount of enzootic activity of the virus has occurred there.

The initial isolation of WE virus east of the Appalachian Mountains was obtained from an immature house sparrow *(Passer domesticus domesticus)* collected in New Jersey during Sept. 1953(1). In 1952 the virus was isolated in Louisiana(2) from a loggerhead shrike *(Lanius ludovicianus)*, a Carolina chickadee *(Parus carolinensis)*, a cardinal *(Richmondea cardinalis)*, and from 2 mosquito species *(Culiseta melanura* and *Aedes infirmatus)*. Subsequent isolations of WE virus from mosquitoes, *Culex pipiens* in North Carolina and *C. melanura* in New Jersey, during 1955 and 1956 led to the hypothesis that "———— *C. melanura* may be an endemic vector of WE in the eastern part of the country"(3).

Materials and Methods. In southeastern Massachusetts, mosquitoes were collected alive in New Jersey-type light traps and bird-baited traps, anesthetized with chloroform and identified, grouped by species in lots numbering up to 50, and stored in an electric freezer at −65°C in corked Wassermann tubes until tested. Samples of avian blood were obtained by cardiac or wing vein puncture, diluted 1:5 in a physiological saline solution containing 0.01% heparin, and placed on wet ice for transport to the virus laboratory for testing. Pheasant brain specimens were obtained through the courtesy of Dr. George P. Faddoul‡. Chick embryo tissue cultures(4) were used in testing the mosquitoes and avian specimens for virus.

Suspensions of the mosquitoes were prepared for inoculation into the tissue cultures. To prepare the suspensions, a few drops of diluent were added to each pool of mosquitoes in its Wassermann storage tube. The diluent used was Hanks' balanced salt solution buf-

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