cated by the notations (*a*) and (*b*) in Table 1, provide an index of the reproducibility of the method. The data for hafnium are not in agreement with those obtained recently by Merz (*5*), who reported an average hafnium abundance of 1.4 ppm for five chondrites. The chemical procedures used by Merz did not specifically separate zirconium from hafnium. Chemical yields for both elements were calculated only on the basis of the amount of zirconium carrier added. Fractionation of zirconium and hafnium in the procedures used by Merz may account for the differing results.

Pinson, Ahrens, and Franck (*6*) have analyzed 26 chondrites for zirconium by spectrography. The average zirconium abundance for their samples was 33 ppm, a value which is in good agreement with ours. They reported a zirconium abundance of 30 ppm for the Johnstown achondrite, a value close to ours.

Data obtained by Merz (*5*), Pinson, Ahrens, and Franck (*6*), and by us, for four meteorites where direct comparisons are possible, are given in Table 2. Although there is good agreement among the analyses for zirconium, our values for hafnium are nearly an order of magnitude smaller than the corresponding values of Merz (*5*). While it is possible that the average hafnium abundance for a meteorite may change slightly as replicate samples are processed and chemical yields are improved, there appears to be no reason to doubt that the hafnium abundances in our samples are near the 0.2 ppm level.

Our results are consistent with abundances of 0.21 ppm (*7*) and 0.13 ppm (*8*) predicted by current theories of nucleosynthesis. An average Zr/Hf ratio of 190 for stone meteorites is obtained from our data. Suess and Urey (*9*) used a value of 110 in establishing their abundance curves. Ahrens (*10*) has suggested a possible fractionation of zirconium and hafnium between the earth and meteorites on the basis of Merz's data. On the basis of our data, such a fractionation may still be indicated. However, the new data are in the direction of a depletion of zirconium in the crustal rocks of the earth with respect to the stone meteorites. Much additional data will be required to establish firmly the relationship of zirconium and hafnium in meteoritic and terrestrial materials.

W. D. EHMANN
J. L. SETSER

Department of Chemistry, University of Kentucky, Lexington

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Cytochemical Assay of Interferon Produced by Duck Hepatitis Virus

Abstract. *A microscopic, cytochemical technique for assay of interferon is described in which psittacosis virus is the indicator agent. Interferon, produced in tissue culture cells in response to duck hepatitis virus, is used to illustrate the procedure.*

The properties of the challenge virus have influenced the methods chosen for assay of interferon (*1*). A change of hemagglutin production, of cytopathogenic effect, or of plaque formation has been useful for detection and analysis of interferon action. Assay techniques, however, have not been standardized. Since, for example, one plaque may reflect the activity of an aggregate of virus particles, quantitative determinations remain equivocal. Furthermore, the mechanism by which virus replication is altered by interferon has not yet been clearly defined: an assay procedure which may be of value in the analysis of interferon action utilizes the psittacosis virus particle as the indicator of inhibition. This agent replicates as a visible intracellular chemical indicator system. The replication pattern of psittacosis virus in cells is completed within 48 hours (*2*, *3*). When a series of infected cell cultures are stained by acridine orange and examined by fluorescence microscopy (*4*), the initial infective DNA-staining virus particle which enters the cytoplasm becomes coated with RNA-staining material ("red ball") within 20 hours. The resulting inclusion enlarges and undergoes a sequence of fluorescent color changes from red to orange to yellow to green. The virus induces a dynamic sequence of cytochemical changes coincident with maturation, and substances which interfere with this maturation sequence can be readily detected (*5*). As employed in this study, tissue cells were rendered resistant to psittacosis virus by exposure to interferon induced in tissue cultures of homologous cells by duck hepatitis virus (DHV) (*6*). The virus particle entered the cytoplasm, but the maturation sequence was stopped at the noninfective "red ball" stage.

Chick embryos infected with DHV were emulsified in Mixture 199 (10 percent wt./vol.). This was clarified by centrifugation and stored as stock virus at −40°C. Chorioallantoic fluid from chick embryos infected with TT

Table 1. Effect of DHV-interferon on replication of psittacosis virus in chick embryo cells, as determined by virus assay in McCoy cell by infected cell count method (*8*). Values are particles per milliliter.

Interferon preparation	Time interval (hours) between absorption of virus and addition of interferon					
	0	6	12	20	24	28
109-uv*	0	0	0	0	7.5×10^3	7.5×10^3
110-h†	0	0	7×10^2	3×10^3	8.3×10^3	6.8×10^3
Control	2.02×10^5					

* Interferon prepared from ultraviolet-inactivated DHV. † Interferon prepared from heat-inactivated DHV.

Table 2. Effect of delayed challenge of psittacosis virus on chick embryo cells treated with DHV-interferon. Values are the percentages by which the virus was reduced, as compared with controls.

Interferon preparation	No. of hours of absorption of interferon by cells before addition of psittacosis virus			
	0	3	6	21
109-uv*	100	100	85	73
150-DHV TC†	100	100	100	89

*Interferon prepared with ultraviolet-inactivated DHV inoculum. †Interferon harvested from DHV-infected chick embryo tissue cultures.

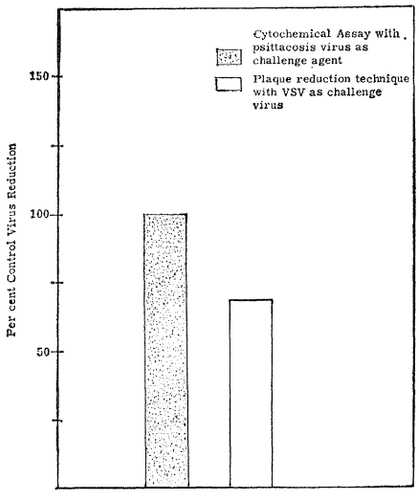

Fig. 1. Comparison of methods showing effect of DHV-interferon on challenge viruses.

strain psittacosis virus (*7*) was pooled, ampouled, and stored as stock virus at −40°C. Duck hepatitis virus was assayed by methods described previously (*6*).

Primary cell cultures were prepared from 11-day-old chicken embryo tissues either by the trypsinization procedure or in the form of explants on cover slips in Leighton tubes. Psittacosis virus was assayed on McCoy cell monolayer preparations on cover slips (*8*). Nutrient fluids for the tissue cultures consisted of Mixture 199 supplemented with 0.5 percent lactalbumin hydrolysate, 10 percent heat-inactivated calf serum, and 100 μg streptomycin per milliliter, adjusted to *p*H 7 with sodium bicarbonate.

Fluids with interferon activity were prepared in cultures of chicken embryo tissues which had been inoculated with viable DHV, and in other tissues which were inoculated with heat- or ultraviolet-inactivated DHV, plus viable DHV. After fluids from the tissue culture preparations had been rendered noninfectious by heat, they were examined for interferon by plaque assay with vesicular stomatitis (VSV), with Western equine encephalomyelitis (WEE), and with Eastern equine encephalomyelitis (EEE) viruses. Control cell cultures were treated with fluids prepared in normal chick embryo cells.

Interferon produced in response to DHV caused a significant reduction of VSV, WEE, and EEE virus activity as determined by the plaque assay technique. All interferon preparations caused more than 50 percent reduction of virus. The interferon was destroyed by trypsin but not by ribonuclease and deoxyribonuclease. Interferon activity was not significantly affected either by DHV antiserums prepared from chickens and rabbits, by serum from normal chickens, or by psittacosis antiserum prepared in rabbits. Interferon activity was destroyed by heating at 100°C for 15 minutes and at 80°C for 1 hour, but not at 56°C for 6 hours. Treatment of fluids by freezing, ultrasonic vibration, or ether reduced the interference effect. Centrifugation at 90,000*g* for 2 hours or dialysis at *p*H 2 resulted in no appreciable change of activity. Interferon was stable when stored at 4°C for approximately 4 months. To test for a direct extracellular virus-inactivating effect of the inhibitory agent, DHV-interferon was incubated with vesicular stomatitis virus and with psittacosis virus for ½ hour at 37°C and tested by the plaque reduction method and by the cytochemical assay procedure. Neither of the two viruses was thereby inactivated.

After it was proved that the virus-inhibiting agent is an interferon-like substance, its effect on psittacosis virus was demonstrated in two steps. First, interferon and virus were added to cultures of chick embryo tissue cells and 40 hours later the cells were examined for mature virus by fluorescence microscopy (*3*). Second, duplicate tissue cultures were frozen once and assayed for viable psittacosis virus on monolayer cultures of McCoy cells (*8*).

Interferon interrupted psittacosis, virus for ½ hour at 37°C and stage of replication. Examination of tissues after 40 hours of incubation revealed cells with virus at "red ball" stage. However, some mature virus was found if inhibition was not complete. Since the "red balls" are noninfective, only mature virus was detected in the subsequent assay. It is probable that the cells with "red balls" had absorbed interferon. Quantitative interference was demonstrable when the inhibitory agent was added to cells simultaneously with or 3 hours prior to the addition of virus (Tables 1 and 2). When the addition of virus to interferon-treated cells was delayed, there was no significant difference in virus production. Each of five DHV-interferon preparations significantly inhibited the maturation of psittacosis virus in the challenge inoculum. Figure 1 shows a comparison of interferon effect as determined by the plaque reduction and by the cytochemical assay techniques. The microscopic assay method here described provides a sensitive, quantitative, visible cytochemical indicator system, which reflects the stage at which interferon interrupts replication of the virus (*9*).

E. A. SUELTENFUSS
MORRIS POLLARD
Lobund Laboratory, University of Notre Dame, Notre Dame, Indiana

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Binasal Hemianopia as an Early Stage in Binocular Color Rivalry

Abstract. *Fifty years ago Köllner reported that the initial fleeting sensation in binocular color rivalry is a bipartite color field such that the color presented to the left eye appears to the left of the color presented to the right eye. A method is described for maintaining such quasi hemianopia for long periods of time.*

What will be seen when discrepant stimulation is applied to corresponding parts of the two retinas? Woodworth addressed this question and outlined the responses he felt the brain makes to such a conflict of cues: "It might disregard one retina and respond only to the other; or it might respond to the combination in several ways: by fusion of the monocular fields, by seeing one through the other, or where possible, by getting a depth effect." (*1*). Generally, the well-accepted notion of the binocular relation is that when the monocular stimuli cannot merge to