marcescens) did contain thioUMP, and their ribosomes were sensitive to the thiol reagent; whereas the rRNA of two (*Pseudomonas aeruginosa* and *Bacillus subtilis*) contained no detectable amounts of thioUMP, nor were the ribosomes of these two organisms sensitive to the thiol reagent.

We therefore conclude that in those bacteria whose rRNA contains thioUMP, the thionucleotide contributes to the stability of the 70S ribosomes.

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The Histones of Some Human Tissues

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Considerable attention has been given recently to the role of the histones, the basic proteins of the nucleoprotein complex. Despite suggestions that these proteins might control gene expression (De Reuck & Knight, 1966), the histones of many tissues appear remarkably similar (Hnilica, 1967). Since little information is available concerning the histones of human tissues, the present report describes the isolation and partial characterization of histones from human thymus, liver, placenta, bronchial tumour and leucocytes.

Preliminary experiments showed that extracts of whole histones from such tissues contained electrophoretic components similar to those of calf thymus whole histone. Histone fractions were then prepared by the methods of Johns (1964). In general, method 1 was used with nucleoprotein obtained from nuclei prepared by conventional sucrose methods. With leucocytes, method 2 was used with nuclei prepared by lysis with acetic acid (MacGillivray & Monjardino, 1968) and treated with acidic ethanol (Pogo, Allfrey & Mirsky, 1966). Total and N-terminal amino acid analysis together with electrophoretic evidence showed that each of the human tissues studied contained the same four main histone components and that these were similar to the histone fractions of calf thymus. The yields of fractions F1 and F2(b) were found to be high in human thymus, in tumour and in some preparations of leucocytes.

These results suggest that the general pattern of histones seen in lower animals is conserved during the evolutionary process that led to man. Similar types of histones have also been described in trout (Palau & Butler, 1966) and in molluscs (Cozcolluela & Subirana, 1968). Peptide analysis

(Hnilica, 1966; Laurence, Phillips & Butler, 1966) and partial sequences (Smith, 1968) of some histones have shown the similarity of the primary structure of these proteins in different species. In addition, histones appear to be poor antigens (Hnilica, 1966; MacGillivray & Monjardino, 1968). These observations indicate that the genes coding for histones may be of primitive origin.

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Protein Components of Fowl-Plague Virus

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Fowl-plague virus is an influenza A virus, consisting of a helical nucleoprotein core surrounded by a lipoprotein envelope. Apart from the type-specific complement-fixing antigen of the nucleoprotein, the virus envelope contains two subtype viral antigens, neuraminidase and haemagglutinin.

The virus was purified by adsorption-elution from chick erythrocytes, centrifugation to a sucrose-D₂O interface and successive centrifugation on gradients of sucrose and potassium tartrate. The virus was free of obvious contamination, as judged by the electron microscope, and when [³H]valine-labelled virus was centrifuged in a caesium chloride density gradient, all the radioactivity was associated with virus haemagglutinin at a density of 1.250g./ml. Extraction of the proteins from the purified virus (both Dutch and Rostock strains) by the method of Summers, Maizel and Darnell (1967), followed by polyacrylamide-gel electrophoresis showed that the virus contained at least four major components and probably two minor components, as judged by staining with Amido Black or by counting of [³H]valine-labelled virus. The slowest of the major components was rather diffuse. No difference was

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observed between the two strains of virus used. When the subcellular fractions of the infected cells were examined by the double-labelling method of Hay, Skehel & Burke (1968), structural virus proteins could be identified as early as 3 hr. after infection, and in addition, some virus-directed non-structural proteins were also evident. Identification of the components of the virus has been commenced by polyacrylamide-gel electrophoresis of the virus haemagglutinin, obtained by ether-splitting of the virus, followed by absorption-elution from chick erythrocytes.

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The Effect of Infection with Fowl-Plague Virus on Protein Synthesis in Chick-Embryo Cells

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When chick-embryo cells were infected with fowl-plague virus the rate of protein synthesis fell to 30–40% of control by 7 hr. after infection (Scholtissek, 1965). This phenomenon was investigated in an attempt to elucidate the mechanism of the inhibitory effect (cf. Martin & Kerr, 1968).

Multiplication of fowl-plague virus can be inhibited either by inactivation of the infecting virus particle or by treatment of the host cell with various inhibitory agents. When virus that had been irradiated with increasing doses of ultraviolet light was used for infection, and the rate of protein synthesis measured 7–8 hr. after infection, it was found that the ability to depress protein synthesis was lost with first-order kinetics but more slowly than the ability of the virus to produce infective virus. Virus multiplication was, therefore, not necessary in order to depress protein synthesis, and the virus component responsible for this effect was susceptible to ultraviolet irradiation. Infective virus, in contrast with ultraviolet-inactivated virus, did not induce interferon production, probably because of the inhibition of cellular protein synthesis.

Virus multiplication was also blocked by treatment of the cells with low doses of *p*-fluorophenylalanine, either actinomycin or ultraviolet light, or high doses of *p*-fluorophenylalanine, agents which appear to act at progressively earlier points in the viral-multiplication process (White & Cheyne, 1966). In every case the inhibitory effect was unaffected, showing that if it is due to a virus-coded product then this product must be formed very early in the multiplication process, i.e. before the

formation of the polymerase, which is actinomycin-sensitive (Ho & Walters, 1966). The inhibitory effect was not a consequence of an inhibition of RNA synthesis, since the latter was depressed at about the same rate as protein synthesis. Moreover, since the inhibitory effect was unaffected by actinomycin treatment of the cells, it is unlikely that it is mediated by the formation of a new cellular messenger RNA. The simplest explanation is that the inhibitory effect is due to a component of the virus acting at the translational level, but how virus protein synthesis escapes the inhibitory effect is unknown.

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Ribonucleic Acid Synthesis in Fowl-Plague-Virus-Infected Chick-Embryo Cells

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In an investigation of the replication of fowl-plague-virus RNA, the properties of RNA polymerase fractions in virus-infected cells were determined. Increased polymerase activity was detected in both nuclear and ribosomal fractions of infected cells. Both activities increased in the cells up to 6 hr. after infection, after which time the ribosomal activity continued to increase. The nuclear activity was optimal at pH 8.0 and increased linearly with the time of incubation up to 25 min., and with protein concentration up to 1 mg./assay. This fraction was optimally active in the presence of either 1.4 mM-Mg²⁺ (twofold stimulation over the uninfected-cell fraction) or 3.3 mM-Mn²⁺ (sixfold stimulation), and the stimulations were completely dependent on the presence of all four nucleoside triphosphates. Activity was not changed in the presence of actinomycin D at 20 µg./assay, and was not detected in the presence of ammonium sulphate.

The activity associated with the ribosomal fraction showed a similar time-course and pH-, Mg²⁺-, and nucleoside triphosphate-dependence characteristics, except that 15-fold stimulations, compared with the uninfected-cell fraction, were usually observed. Mn²⁺ did not activate more than Mg²⁺, 10% satd. ammonium sulphate inhibited the reaction, and actinomycin D had no effect. The activity was half-maximal at 15 µmoles of UTP/assay.