In the coming atomic age nuclear energy will be the driving power of the social order.

The foregoing are not the views of a scientist; they are those of a layman who has taken a keen interest in certain branches of science for many years. Though he has worked as a machinist in a war plant until recently, and spent his life prior to that in the mines in South Wales and America as a miner, he has reflected a good deal on some of the major problems of life.

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The Atomic Bomb and the Anthropologists

In his admirable address, "Science and Our Future" (*Science*, 1946, **103**, 415–417), Dr. E. U. Condon very properly criticizes the viewpoint of anthropologists who "fatalistically await death, reading papers to an academic society meeting in a museum in Philadelphia." I think it should be placed on record that almost all the anthropologists present at that meeting rejected that viewpoint, and that upon my moving and Dr. Margaret Mead seconding, the following resolution was unanimously adopted by the American Anthropological Association:

RESOLVED: That the American Anthropological Association, constituted of scientists interested in the study of human nature and society, recognizes the responsibility of anthropologists to study the effects of the discovery of the use of atomic energy, and to participate actively with other scientists in efforts to make appropriate social inventions to guard against the dangers, and utilize the promise, inherent in atomic use.

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Pathogenesis of Kernicterus

The purpose of this communication is to present our concept of the pathogenesis of kernicterus (jaundice of the nuclear masses of the brain), based on recent clinical, serological, and pathological observations. Until recently there was no explanation for the post-mortem finding of kernicterus only in cases of icterus gravis neonatorum (a form of congenital hemolytic disease or erythroblastosis) and not following other types of jaundice, even of severe degree.

As is well known, the great majority of cases of congenital hemolytic disease occur when an Rh− mother, already sensitized to the Rh factor by a previous pregnancy or transfusion, bears an Rh+ fetus. The antibodies in the sensitized mother's serum may be of two principal varieties, namely, bivalent antibodies (agglutinins) and/or univalent antibodies (glutinins or blockers). If the maternal serum contains a high titer of univalent antibodies, the Rh+ fetus will almost surely be stillborn. With low-titered univalent antibodies, viable infants who recover after suitable transfusion therapy are the rule.

In our experience, when the complication of kernicterus supervenes, the antibodies in the maternal serum are almost always of the bivalent variety (agglutinins). This suggests the following mechanism for the development of kernicterus. Maternal Rh agglutinins in the infant's circulation combine with its Rh+ erythrocytes, bringing about the formation of small clumps (agglutinates of red cells) which plug the smaller arterioles with the formation of agglutination thrombi. In an organ like the liver, the resulting damage may be one factor in the production of jaundice, thus explaining its lack of correlation with the degree of anemia. In the bone-marrow, the resulting irritation may cause a pouring forth of nucleated red cells into the circulation. In the brain, plugging of terminal vessels can produce areas of ischemic infarcts; the nuclear masses would be most vulnerable, since ganglion cells are particularly susceptible to anoxia. Due to the concomitant presence of deep jaundice, the damaged ganglion cells take up bilirubin—a sort of *in vivo* staining reaction. If the infant dies at this point, post-mortem examination will show the presence of kernicterus. Infants surviving the immediate neonatal period and dying after the jaundice has disappeared will show at post-mortem examination evidence of cerebral damage but without kernicterus. In a recent case studied by us, where death occurred 48 hours after birth, in addition to the post-mortem finding of kernicterus, histologic study showed the blood vessels of the brain to be packed full by agglutinated masses of erythrocytes, as required by our theory.

Infants with kernicterus occasionally survive, in which case they develop signs of a diffuse neurologic disorder of varying severity, usually accompanied by mental deficiency. Such cases are rare and would be expected to make up only a very small fraction of the group of so-called nonspecific mental deficiency cases. Therefore it could be predicted a priori that attempts such as have recently been made to show a statistical difference in the distribution of the Rh factor among mothers of mentally deficient children in comparison with the normal population would almost surely yield insignificant results unless the series is large enough to detect a small percentage difference.

Attempts have been made to show a connection between Wilson's disease and kernicterus. In three cases of Wilson's disease studied by us we found no evidence of isoimmunization of pregnancy, indicating a dissimilarity in the pathogenesis of the two conditions.

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Genetics and Biological Theory

E. B. Wilson, in his classical work (*The cell in development and heredity*. New York: Macmillan, 1928), states: "In practice all purposes of experimental analysis are sufficiently met if the hereditary 'units,' 'genes' or 'pangens' be thought of merely as modifiers which call forth responses, this way or that, according to their

specific nature. To speak of them as 'determiners' is to make use of a convenient figure of speech; but this need imply no more than that they are differentials by the use of which we are enabled accurately to analyze the observed results'' (p. 1113). To take this concept to its logical conclusion has a radical effect upon biological theory, as the writer has shown in a series of papers on the causes of color patterns. It is preferable to use the term ''inhibitor'' rather than ''modifier,'' since the latter has a more restricted meaning in genetics, and extend to the whole of genetics the concept of inhibiting factors which is used to explain melanin pigment inhibition in animals. The concept of hereditary factors as inhibitors is not generally used by plant geneticists, though the evidence is in its favor. Thus, the chromosome-borne inhibitors in albino plants are quite obviously responsible for the inhibition of plastid development, which may be complete as in white plants, or incomplete inhibition as in yellow plants, with still less inhibition for red-colored plastids (chromoplasts). Since flower color is caused by sap pigments produced by plastids, or chromoplasts, or both, then the inhibiting factor hypothesis has only to be extended to include the selective inhibition of one or more of the three primary pigments produced by the plastids, namely, red, yellow, and blue, or incomplete inhibition of the plastids themselves, together with a time variable when the inhibitors become effective, to account for color patterns. In animals there is no evidence that chromosome-borne factors are responsible for melanin pigment synthesis but only its partial or complete inhibition, the variegated patterns being caused by the time when the inhibiting substances appear in early or fetal life. The evidence points to black color as being the uninhibited expression of melanin pigmentation, and hence the conventional multiple allelic series in rodents is each reduced by one, namely, normal black color. Thus, the agouti series in rabbits has only two members, agouti or wild-type, and black-and-tan, normal black color occurring in the absence of these inhibitors provided no others are present.

The inhibitors arise spontaneously, as evidenced by mutations, and range from the dominant, gross type to those of the modifying or regulatory class, the latter being responsible for the nongenetically fixable grades of white spotting and size. The breeding methods of inbreeding, outcrossing, and selection are used for the purpose of attempting to fix the desired types of inhibitors and purge the organism of unnecessary ones. Thus, inbreeding maize purges the plant of some inhibitors but renders others homozygous. The crossing of two inbred strains which are homozygous for a maximum number of different inhibitors will yield the most vigorous hybrids. If the inhibitors could be destroyed, especially selectively, the breeding methods would be simplified. Submitting organisms to X-rays merely increases the number of harmful inhibitors or produces lethal effects through chromosome breakages. However, the inhibiting substances produced by the inhibitors can be neutralized in many cases. An extreme example is seen in the use of plant hormones as herbicides which seem to neutralize cellular inhibiting substances to the extent that the plant grows itself out.

The implication of the inhibiting factor hypothesis is that growth is an inherent characteristic and the chromosome-borne factors merely inhibit metabolism in varying degrees. In other words, under optimum conditions of temperature, light, and nutrition and with a minimum of internal inhibitions caused by chromosome-borne inhibitors, together with an absence of toxic products of metabolism or the neutralization of such, cell growth would be at its maximum rate, and differentiation would not occur. This view has some support from tissue-culture experiments. It is also supported on evolutionary grounds. Thus, the first spontaneously originated compound to form organic matter must have had the constitutional characteristic for its own duplication and hence growth; it could not have acquired this characteristic, as it could not have survived to do so. What would be necessary would be a breaking up of the organic mass to prevent it from becoming a victim of its own toxic products of metabolism. The spontaneous origin of inhibitors would provide the necessary mechanism, since inhibitors have the characteristic of repulsion after duplication, as is evident in mitosis and by inference also in single-celled organisms. Evidence that complex molecular entities duplicate themselves is seen, in addition to the chromosome-borne inhibitors, in plastids and virus molecules, all of which require the specific organization of the cell to do so.

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Are Scientists Irresponsibles?

The leading article, ''Can we curb the irresponsibles?,'' by Lawrence K. Frank (*Science*, 1946, **103**, 349–351), deals with a most important matter on which clear thinking is eminently desirable, but after reading it three times, I find myself much confused. Perhaps I am unduly obtuse, but I need clarification of Dr. Frank's position.

I wholly agree that responsibility for aggression must be brought home to the guilty individuals and adequate punishment inflicted on them, and that the Nürnberg trial is, therefore, fully justified and is based on principles which must be maintained if we are to hope for lasting peace.

But Dr. Frank seems to proceed from this sound basis to the propositions that scientists, by working on atomic bombs are guilty of aggression and may be held liable to punishment, and that an association of scientists may be able by their own action to relegate the atomic bomb to extinction. I cannot follow him on either of these points.

Surely there is a clear distinction between contributing to our military power and using that power for aggression. The scientific work which produced the atomic bomb differed not in character but only in effectiveness from other scientific work which contributed to the development of faster fighting planes, more