BIOGRAPHICAL INFORMATION

Max R. Zelle

BORN: Polk County, Iowa, October 11, 1915

EDUCATION: Diploma, Alleman High School, Alleman, Iowa 1932, Valedictorian.
B.S. degree in Animal Husbandry, Iowa State College, March, 1937.
Ph.D. degree in Genetics, Iowa State College, December, 1940.

POSITIONS AND EXPERIENCE:

1935-36	Asst. County Agricultural Agent, Iowa State Extension Service, Audubon, Iowa
1937-38	Fellowship in Genetics, Iowa State College
1938-40	Graduate Assistant In Genetics, Iowa State College
1940-44	Assistant Professor of Animal Husbandry (Genetics), Purdue University
1944-46	Ensign and Lt.(j.g.) USNR, Classified Research on Biological Warfare, Camp Detrick, Frederick, Maryland
1946-48	Biologist, Laboratory of Physical Biology, National Institutes of Health, Bethesda, Maryland
1948-58	Professor of Bacteriology, Cornell University, Ithaca, New York
1950-51	Geneticist, Biology Branch, Division of Biology and Medicine, Atomic Energy Commission, Washington, D. C. (On leave from Cornell Univ.)
1951-54	Consultant, Division of Biology and Medicine, Atomic Energy Commission
1947-49 & 1952-57	Consultant, Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee
1952, '54 and '56	Summer Guest Investigator, Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee
1957-58	Geneticist, Biology Branch, Division of Biology and Medicine, Atomic Energy Commission, Washington, D. C. (On leave from Cornell Univ.)
1958-60	Chief, Biology Branch, Division of Biology and Medicine, Atomic Energy Commission, Washington, D. C.
1960-61	Assistant Director for Biological Sciences, Division of Biology and Medicine, Atomic Energy Commission, Washington, D. C.
1961-62	Professor of Genetics and Director, Center for Radiological Sciences, University of Washington, Seattle, Washington
1962-69	Director, Division of Biological and Medical Research, Argonne National Laboratory, Argonne, Illinois

POSITIONS AND EXPERIENCE: (Con't)

1958-65	Advisor to United States delegation to the United Nations Scientific Committee on the Effects on Atomic Radiation
1963-69	Lecturer in Zoology, University of Chicago, Chicago, Illinois
1963-72	Member of Editorial Board, Mutation Research
1965-74	Co-editor, Advances in Radiation Biology series: Academic Press
1969-73	Member, Radiological Health Study Section, Bureau of Radiological Health, Food and Drug Administration, Department of Health, Education and Welfare
1969 +	Chairman, Department of Radiology and Radiation Biology, Colorado State University, Fort Collins, Colorado
1970	Member, Search Committee for Dean of College of Veterinary Medicine and Biomedical Sciences
1970-73	Member, Editorial Board, Applied Microbiology
1970-73	Member, Steering Committee, Western Interstate Nuclear Board, Denver, Colorado
1971-74	Faculty Council, CSU, Representative at large
1971-75	Member, Cultural Programs Advisory Committee, CSU
1971-73	Member of Steering Committee, Faculty Council, CSU
1972 +	Radiation Control Officer and Member, Radiation Safety Committee, CSU
1972	Consultant, U. S. Atomic Energy Commission, Plowshare Programs Office
1974	Chairman, Search Committee for Associate Professor of Radiology
1975 +	Member, Intersociety Liason Committee, Health Physics Society
1974-76	Member, Faculty Benefits Committee, CSU
1976-77	Chairman, Faculty Benefits Committee, CSU
1976-77	Chairman, Search Committee for Associate Professor of Radiological Physics
1977	Chairman, Search Committee for Director of Collaborative Radiological Health Laboratory
1977	Chairman, Search Committee for Head of Microbiology
1978	Chairman, Search Committee for Associate Dean, College of Veterinary Medicine and Biomedical Sciences
1978	Consultant, University of Tennessee Institute of Radiation Biology
1979	Consultant, American Pharmaceutic Association
1979+	Member, CSU Committee on Responsibilities and Standing of the Academic Faculty
1980+	Member, CSU Advisory Committee for Adult Students

HONORS AND PROFESSIONAL SOCIETIES:

Honor Student, College of Agriculture, Iowa State University
Scabbard and Blade, Military Honorary
Alpha Zeta, Agricultural Honorary
Sigma Xi
Phi Kappa Phi
Who's Who in the World (4th Edition, 1978)
Who's Who in America
Who's Who in the West
Who's Who in Atoms
Listed in the Blue Book; Leaders of the English Speaking World
Dictionary of International Biography
Honorary Editorial Board, Photochemistry and Photobiology 1962-1970
Cosmos Club, Washington, D. C.
American Association for Advancement of Science, Fellow 1955
American Institute of Biological Sciences
American Society for Microbiology
American Society of Naturalists
American Society for Photobiology
Genetics Society of America
Radiation Research Society: Councilor in Biology, 1960-63; Secretary-Treasurer Elect 1971; Secretary-Treasurer 1972-75; Member, Committee for Advancement of Radiation Research, 1974-76; Chairman, Education Committee 1974-77
Health Physics Society: President, Central Rocky Mountain Chapter 1976-1977

RESEARCH INTERESTS: Radiation Biology, Genetics, Microbiology

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Zelle, M. R. 1942. Genetic constitutions of host and pathogen in mouse typhoid. J. Infectious Diseases 71: 131-152.

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Shrewsbury, C. L., C. Harper, F. N. Andrews, and M. R. Zelle. 1942. The limitations of oat straw as a roughage for maintenance, lactation and growth in sheep. J. Animal Sci. 1: 126-130.

Shrewsbury, C. L., F. N. Andrews, C. Harper, and M. R. Zelle. 1943. The value of alfalfa and certain of its fractions in the nutrition of breeding ewes. J. Animal Sci. 2: 209-220.

Gowen, J. W., and M. R. Zelle. 1945. Irradiation effects on genetic resistance of mice to mouse typhoid. J. Infectious Diseases 77: 85-91.

Young, G. A., Jr., M. R. Zelle, and R. E. Lincoln. 1946. Respiratory pathogenicity of Bacillus anthracis spores. I. Methods of study and observations on pathogenesis. J. Infectious Diseases 79: 233-246.

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Young, G. A., Jr., and M. R. Zelle. 1946. Respiratory pathogenicity of Bacillus anthracis spores. IV. Chemical-biological synergisms. J. Infectious Diseases 79: 266-271.

Jarrett, E. T., Lt. (HC) USN, M. R. Zelle, and A. Hollaender. 1948. Studies of the control of acute respiratory disease among naval recruits. II. Limitations of ultraviolet irradiation in reducing air-borne bacteria in barracks with low ceilings. Am. J. Hyg. 48: 233-239.

Buck, J. B., M. L. Keister, and M. R. Zelle. 1950. An areal method for calibrating microburettes. Anal. Chim. Acta 4: 130-134.

Zelle, M. R. 1951. A simple single-cell technique for genetic studies of bacteria. J. Bacteriol. 61: 345-349.

Open Literature Publications, con't:

Zelle, M. R., and J. Lederberg. 1951. Single-cell isolations of diploid heterozygous Escherichia coli. J. Bacteriol. 61: 351-355.

Dondero, N. C., and M. R. Zelle. 1953. Observations on the formation and behavior of "conjugation" cells and large bodies in Azotobacter agile. Science 118: 34-36.

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Zelle, M. R. 1955. Genetics of microorganisms. In: Annual Review of Microbiology, Vol. 9. Ed.: C. E. Clifton. Stanford, Calif.: Annual Reviews, Inc., pp. 45-96.

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Zelle, M. R. 1958. Diagnostic X-rays from the viewpoint of a geneticist. Med. Ann. of the District of Columbia, 27: 455-459.

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Kvetkas, Marilyn J., and M. R. Zelle. 1967. Genetic analysis of a large-cell, radiation-resistant strain of Escherichia coli. Microbial Genetics Bulletin, Oak Ridge National Laboratory 27: 7-8.

Kvetkas, Marilyn J., and Max R. Zelle. 1968. Genetic analysis of a large-cell, radiation-resistant strain of Escherichia coli. Bacteriol. Proc. 68: 62.

Kvetkas, Marilyn J., Robert E. Krisch, and Max R. Zelle. 1970. Physiological studies of a large-cell, radiation-resistant, mutant strain of Escherichia coli. Bacteriol. Proc. 70: 38.

Zelle, M. R. 1969. Dr. Douglas E. Smith - Physiologist and photobiologist. In: NATO International Advanced Study Institute on Biological Effects of Visible Light. Photochemistry and Photobiology of "Photodynamic Action." Univ. of Sassari, Sardinia, Italy, Sept. 3-17, 1969, pp. 106-107.

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Kvetkas, M., and M. R. Zelle. 1966. Genetic analysis of a large cell strain of Escherichia coli. ANL-7278, pp. 162-164.

Rehfeld, C. E., M. R. Zelle, D. E. Doyle, J. A. Pagels, and M. H. Dipert. 1967. Analysis of familial influence on mean erythrocyte volume. ANL-7409, pp. 117-119.

Kvetkas, M. J., and M. R. Zelle. 1967. Genetic analysis of a large cell strain of Escherichia coli. ANL-7409, pp. 142-143.

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Kvetkas, M. J., R. E. Krisch, and M. R. Zelle. 1968. Genetical and physiological studies of a large-cell, radiation-resistant strain of Escherichia coli. Part II. Physiological studies. ANL-7535, pp. 3-5.

Fritz, T. E., R. C. Zeman, and M. R. Zelle. 1969. Studies of the physiology of the Beagle thyroid gland. II. Pathology and familial incidence of thyroiditis in a closed Beagle colony. ANL-7635, pp. 107-108.

ABSTRACTS OF PAPERS PRESENTED AT THE 1939 MEETINGS OF THE GENETICS SOCIETY OF AMERICA

COLUMBUS, OHIO, DECEMBER 28–30, 1939

E. W. LINDSTROM, *Secretary*
Department of Genetics
Iowa State College, Ames, Iowa

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ZELLE, M. R., and GOWEN, JOHN W., Iowa State College, Ames, Iowa: *On the origin of epidemic virulence.*——When an epidemic starts, a concatenation of events occurs which enables a particular pathogen to sweep through a species. To particularize these events constitutes a most significant problem of disease resistance. If host and pathogen are closely integrated, one must change in susceptibility or the other in invasive power if epidemic disease proportions are reached. Following SCHOTT's experiments, our own have shown that susceptibility or resistance in different host populations may be markedly increased by 25 generations of controlled breeding. But these changes are too slow to explain the shift from a stable population to the unbalance of epidemics. The immediate origin seems rather in mutation of the pathogen's virulence. The selective force isolating and purifying the population to the invasive type would lie in the hosts.——Four experiments with mouse typhoid, *S. aetrycke*, support this analytical view. In experiments 1, 2 and 4 the host type did not materially influence virulence. The third experiment was markedly different. Bacteria, passed through resistant mice, suddenly became the most virulent experienced; those through the susceptible mice showed no such change. Reversal of hosts caused the very slightly virulent strain, previously inhabiting the susceptible mice, to gain virulence explosively in the third passage through resistant mice. The virulent strain from resistant mice, passed through the susceptibles, remained stable. The results thus far indicate infrequent pathogenic mutation with host selection as a cause of epidemic virulence, agreeing with work of WELLHAUSEN (1937) and LINCOLN (1939) on the corn bacterial wilt relationship.

ABSTRACTS OF PAPERS PRESENTED AT THE 1940 MEETINGS OF THE GENETICS SOCIETY OF AMERICA

PHILADELPHIA, PA., DECEMBER 30–JAN. 1, 1941

E. W. LINDSTROM, *Secretary*
Department of Genetics
Iowa State College, Ames, Iowa

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ZELLE, MAX R., and GOWEN, JOHN W., Iowa State College, Ames, Iowa: *Interaction of the genetic constitutions of host and pathogen in mouse typhoid.*—— Among the factors responsible for the inception of epidemic disease, the sudden appearance of a highly virulent pathogen in a suceptible community is of first importance. The findings of these investigations indicate that high virulence results from the rare occurrence of a more pathogenic mutant within the bacterial population. Selection of the virulent form in the host results in the virulent form replacing the avirulent. The environments within genetically resistant and susceptible mice are equally favorable to the virulent bacteria. Four experiments, in each of which the same initial culture of *Salmonella typhimurium* was passed through both resistant and susceptible mice, uphold this viewpoint. In the first two experiments the original culture was a mixture of bacterial types differing in virulence. Increases in virulence occurred in both resistant and susceptible mice. The initial culture for the third experiment was a single cell culture of low virulence and slightly rough colony type. Ten passages through susceptible hosts resulted in no change in virulence. After one passage through resistant mice, virulence changed suddenly to significantly higher virulence. Examination of the resulting bacterial population showed it of predominately smoother colony type than its parent. Particular strains of virulent and avirulent bacteria may be classified by their phenotypic appearance. The differential replacement of the avirulent by the virulent bacteria has been checked by introducing known mixtures of the two types into susceptible and resistant hosts.

ABSTRACTS OF PAPERS PRESENTED AT THE 1940 MEETINGS OF THE GENETICS SOCIETY OF AMERICA

PHILADELPHIA, PA., DECEMBER 30–JAN. 1, 1941

E. W. LINDSTROM, *Secretary*
Department of Genetics
Iowa State College, Ames, Iowa

174

ZELLE, MAX R., Iowa State College, Ames, Iowa: *On the mechanism of variegation in bacterial colonies.*——An unstable smooth colony culture of *Salmonella typhimurium* exhibiting a high mutation rate to a stable rough type has been studied by a technique designed to demonstrate that, at least in this instance, the S→R transformation is a discontinuous change. The parent strain is a single-celled, smooth organism whose progeny are invariably mixtures of smooth and rough types. Rough colonies from this mixture give only rough. Employing a modified micromanipulator technique, single cells from a smooth colony are seeded upon a thin film of agar mounted on a cover slip on a moist chamber. After division the two daughter cells are separated by a microneedle and their location marked. Upon further division the separation is repeated until ultimately the cells are allowed to develop microcolonies which are transferred to broth, incubated, and plated. If the original single cell was smooth, the ultimate single cell progeny cultures derived from it exhibit both colony types. If the ultimate single cell progeny isolated is a rough mutation then only rough colonies develop. Two mutations have occurred in 302 single-cell isolations of this type giving a mutation rate of 0.0066 per cell generation. In one of these isolates its sib-cell was inviable; in the other its sib-cell was smooth. Considering only the manner in which the changes occur, the simplest hypothesis is that the bacteria are haploid with a high mutation rate for the particular locus. However, the significantly higher frequency of inviable cells among the smooth bacteria indicate a more complex mechanism.