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Sunlight Ultraviolet and Bacterial DNA Base Ratios

Natural exposure to ultraviolet may be an evolutionary pressure toward high guanine plus cytosine in DNA.

C. E. Singer and Bruce N. Ames

The base composition of DNA is a constant characteristic of a given species. The percentage of guanine (G) plus cytosine (C) in the DNA (G + C content) varies widely among species and ranges from about 23 percent to 74 percent. Although about 1000 G + C contents have been measured, mostly in bacteria, no satisfactory explanation has been advanced for why a particular species has a high or low G + C content. We propose that bacterial species exposed to sunlight evolve high G + C contents to avoid thymine specific damage from the ultraviolet radiation in sunlight. Although there may be similar effects in some fungi (*1*) and higher algae, we restrict our discussion primarily to bacteria, because in higher organisms screening due to the larger cell size and cell mass appears to be a major factor. In addition, higher organisms are diploid or have multiple copies of the genetic material, and this complicates any analysis of ultraviolet sensitivity.

Environment and G + C Content

We have found a strong correlation between the amount of sunlight to which a bacterium is normally exposed and its G + C content. Because of uncertainties concerning many bacterial habitats we have restricted ourselves to those habitats which clearly receive a high ultraviolet exposure or a low ultraviolet exposure. Bacteria exposed to considerable sunlight (bacteria with aerial reproduction, aquatic bacteria, and carotenoid-containing bacteria) almost universally have high

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G + C contents (and therefore a low thymine content). Bacteria with no exposure to sunlight (nonphotosynthetic obligate anaerobes and obligate parasites) all have low G + C contents. Several environments of high or low ultraviolet exposure and several bacterial genera which inhabit each environment are mentioned below; G + C contents are given as a percentage after each genus (see also Table 1).

Bacteria which reproduce aerially would be expected to have a large amount of sunlight exposure because their reproductive cells are usually suspended above the solid substrate (*2*). These are the soil actinomycetes which reproduce by means of aerial or surface conidia (for example, *Micromonospora*, 72 percent, and *Streptomyces*, 71 percent) and the fruiting myxobacteria (for example, *Polyangium*, 69 percent; *Myxococcus*, 68 percent; and *Chondromyces*, 65 percent).

Most organisms that live near the surface of the water in aquatic environments should be exposed to large amounts of ultraviolet. The intensity of 300 nanometer radiation is reduced by only 50 percent every 10 meters in water and every 3 meters in seawater (*3*). The aquatic aerobes (*2, 4*) *Actinoplanes*, 74 percent; *Sphaerotilus*, 70 percent; *Prosthecomicrobium*, 68 percent; *Halobacterium*, 67 percent; and *Caulobacter*, 64 percent; and the photosynthetic anaerobes (*2*) *Chromatium*, 65 percent; *Rhodospirillum*, 61 percent; and *Chlorobium*, 57 percent, all have the predicted high G + C contents.

Carotenoids protect bacteria from photooxidations mediated by visible light (*5*). No other function has been reported for carotenoids in most nonphotosynthetic bacteria, and we therefore conclude that most bacteria which contain carotenoids have these pigments because they are often exposed to sunlight in the environment. Some such genera have already been mentioned (such as *Halobacterium*). Others (*6*) are *Cellulomonas*, 74 percent; *Mycobacterium*, 68 percent; *Xanthomonas*, 67 percent; *Sarcina*, 67 percent; *Flavobacterium*, 66 percent; and *Micrococcus*, 65 percent. There are some apparent exceptions to the correlation between sunlight exposure and G + C content when carotenoids are used as an indicator of sunlight exposure, although there may be alternate explanations in some of these cases. These are *Mycoplasma*, 29 percent; *Saprospira*, 44 percent; *Cytophaga*, 39 percent. The few *Mycoplasma* (primarily internal parasites) that contain carotenoids use carotenoids in a novel way as a substitute for sterol (*7*) and presumably not for protection from sunlight. The flexibacteria (*8*) (for example, *Saprospira*, 44 percent, and *Cytophaga*, 39 percent) constitute the only anomaly which is difficult to explain.

Strict anaerobes which come from nonaquatic environments would not be expected to be exposed to any sunlight. It is hard to imagine how such organisms could receive sunlight exposure without also receiving a fatal oxygen exposure. *Ramibacterium*, 30 percent; *Clostridium*, 31 percent; *Treponema*, 36 percent; *Catenabacterium*, 39 percent; *Bacteroides*, 42 percent; and *Fusobacterium*, 45 percent, are all obligate anaerobes (*2*).

Strictly internal parasites (such as most species of *Mycoplasma*, 29 percent) should have no sunlight exposure (*2*). The same is true for the intracellular parasites *Wolbachia*, 31 percent, and *Chlamydia*, 43 percent (*2*).

Thus there is indeed a very strong positive correlation of G + C content with sunlight exposure. Over two dozen bacterial genera listed above fit the correlation while there are only one or two possible exceptions.

We have briefly investigated the habitats of the remaining bacteria for which we have nucleotide compositions. We believe that the correlation may hold for most if not all of these remaining genera; but their ecology is complicated, and we will not discuss this group. For example, the habitat of the well-studied species *Escherichia coli*, 50 percent, and *Salmonella typhimurium*, 50 percent, is usually thought of as animal intestines, a gloomy environment. However, they both have a strong photorepair system that requires visible light to repair thymine dimers; this suggests that their nonenteric habitats are also important. According to our theory, their intermedate G + C

Table 1. The G + C contents were obtained for a large number of bacterial species (*29, 30*). The species names from the reviews (*29*) were checked against *Bergey's Manual of Determinative Bacteriology* (*2*). Unlisted species were discarded and misnamed species were reassigned to the appropriate genera. This left too many species to analyze conveniently. We therefore assigned to each genus the median G + C content of the species in that genus. Using the median is convenient, accurate enough for our purposes, and avoids skewing of the G + C content of a genus greatly by one or two misclassified species. Sunlight exposures are listed as they are estimated in our text (High or Low), or no estimate is made (*). The numbers in parentheses represent the number of species examined.

G + C (%)	Genus	Ultraviolet exposure	G + C (%)	Genus	Ultraviolet exposure
74	Actinoplanes (1)	High	53	Corynebacterium (18)	High
74	Cellulomonas (1)	High	53	Aerobacter (2)	*
72	Micromonospora (1)	High	53	Erwinia (8)	*
71	Streptomyces (11)	High	53	Vibrio (8)	*
70	Nocardia (2)	High	52	Spirillum (2)	*
70	Sphaerotilus (2)	High	51	Escherichia (4)	*
69	Polyangium (1)	High	51	Neisseria (8)	*
68	Myxococcus (3)	High	51	Salmonella (7)	*
68	Prosthecomicrobium (2)	High	51	Shigella (4)	*
68	Mycobacterium (6)	High	50	Bdellovibrio (1)	*
67	Halobacterium (2)	High	45	Fusobacterium (2)	Low
67	Sarcina (1)	High	45	Lactobacillus (7)	*
67	Propionibacterium (7)	*	44	Saprospira (1)	High
67	Xanthomonas (7)	High	43	Coxiella (1)	*
66	Flavobacterium (3)	High	43	Chlamydia (2)	Low
65	Chromatium (1)	High	42	Bacteroides (1)	Low
65	Chromobacterium (1)	*	41	Moraxella (2)	*
65	Chondromyces (1)	High	41	Pasteurella (2)	*
65	Micrococcus (5)	High	40	Bacillus (16)	*
64	Thiobacillus (2)	*	40	Leuconostoc (1)	*
64	Caulobacter (1)	High	39	Catenabacterium (1)	Low
63	Pseudomonas (15)	*	39	Proteus (4)	*
63	Rhizobium (2)	High	39	Haemophilus (4)	*
62	Arthrobacter (1)	*	39	Cytophaga (3)	High
61	Rhodospirillum (1)	High	38	Listeria (1)	*
60	Gluconobacter (1)	*	38	Rickettsia (1)	*
59	Agrobacterium (3)	*	38	Streptococcus (9)	*
59	Acetobacter (4)	*	37	Leptospira (1)	*
58	Brucella (1)	*	36	Treponema (1)	Low
57	Chlorobium (1)	High	36	Veillonella (1)	*
57	Azotobacter (2)	*	35	Sporocytophaga (1)	*
57	Aeromonas (2)	*	34	Staphylococcus (2)	*
57	Alcaligenes (1)	*	31	Wolbachia (1)	Low
55	Desulfovibrio (3)	*	31	Clostridium (14)	Low
55	Serratia (2)	*	30	Ramibacterium (1)	Low
55	Paracolobactrum (1)	*	29	Mycoplasma (9)	Low
55	Klebsiella (2)	*			

content is consistent with an intermittent exposure to ultraviolet.

The blue-green algae have a wide range (*9*) of G + C contents (35 percent to 71 percent) but are a particularly difficult class to assign to an effective ultraviolet exposure. They are, in most cases, both highly colonial and highly pigmented, and this raises a screening problem. The organization of the genetic material is not known, but some blue-green algae are unusually resistant to ultraviolet and ionizing radiation.

Ultraviolet Damage and Protection

The ultraviolet radiation in sunlight is sufficient to cause considerable damage to unprotected DNA (*10*). In the range of 290 to 320 nanometers there is a significant spectral overlap between the ultraviolet transmitted by the atmosphere (*11*) and ultraviolet absorption by DNA (Fig. 1). Pyrimidine dimers are thought to be the major product of ultraviolet damage (*12*). We have calculated that about ten dimers per minute would be formed in an *Escherichia coli* chromosome by the ultraviolet in direct overhead sunlight at sea level (Fig. 1). Because a single unrepaired dimer may be lethal (*13, 14*), all organisms exposed to sunlight must have some protection against the ultraviolet in sunlight.

We will discuss three protective mechanisms against damage by ultraviolet: screening of the ultraviolet by cytoplasmic material, repair of the DNA, and the evolution of the base ratio of the DNA.

Screening of sunlight ultraviolet by two major cellular constituents, tryptophan and RNA, is important for cellular protection because both absorb light near 300 nanometers. A 50-micrometer length of cellular mass would screen out about 50 percent of the ultraviolet radiation (*15*); thus an *E. coli* cell with a diameter of 0.5 micrometer transmits almost all of the ultraviolet, whereas a *Paramecium* with a diameter of 50 micrometers absorbs part of this radiation in its cytoplasm. Human sweat contains urocanic acid (whose molecular weight is 138 and whose molar extinction coefficient, ε, at 300 nanometers is 7000) which absorbs ultraviolet from sunlight (*16*). In order to screen themselves with a pigment, bacteria would need one (for example, with a molecular weight of 200, $\varepsilon_{300\ \mathrm{nm}} = 10{,}000$) that accounted for 10 percent of their dry weight to absorb half of the incident ultraviolet radiation. We have found no report of such a pigment at this concentration in bacteria.

Three modes of repair of damaged DNA are known, and they have been discussed extensively (*14*). These are photorepair, excision repair, and recombination repair. One would like to know exactly how much damage by ultraviolet escapes these repair systems, but this determination cannot be made with the data available (*17*). Repair systems seem to be present in all organisms, but there is insufficient data to compare them.

Even a small amount of killing after ultraviolet damage repair should be a significant selective disadvantage. For example, a killing of 10^{-10} per generation (one ten-billionth of the organisms) with one generation per day, should be of evolutionary significance in 30 million years (10^{10} days). We believe it highly unlikely that any repair system is efficient enough to reduce the ultraviolet radiation damage to an evolutionarily insignificant value. In addition, some forms of ultraviolet damage such as DNA interchain cross-links and DNA-protein cross-links are not known to be repairable. Interchain cross-links are thymine specific (*12*), and DNA-protein cross-links may be as well (*18*).

Base Ratio Changes

An organism which could further reduce ultraviolet killing by reducing the amount of thymine in its DNA, without sacrificing anything else, should gain a significant selective advantage (*19*). The calculated number of ultraviolet photodimerization targets de-

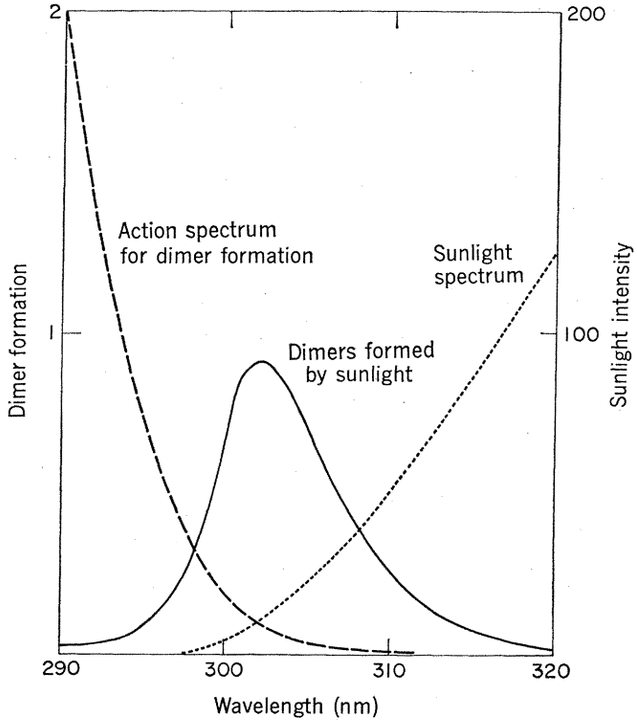

Fig. 1. Pyrimidine dimer formation as a function of wavelength. The dashed line indicates the number of dimers formed per erg per square millimeter in an *E. coli* chromosome and is plotted relative to the known rate of dimer formation of 6.5 dimers per chromosome per erg per square millimeter at a wavelength of 253.7 nanometers (*31*). The points are plotted on the assumption that the action spectrum for dimer formation is approximately the same as the thymidine (or the cytidine) absorption spectrum. The action spectrum for dimer formation in the *E. coli* chromosome is known to follow thymine (and thymidine) absorption from 240 to 290 nanometers (*32*). The dotted line shows overhead sunlight received at the earth's surface (*11*) in ergs per square millimeter per minute per nanometer. The data on the amount of ultraviolet light at about 300 nanometers which reaches the earth are sparse, and this line is therefore only approximate. The solid line shows the number of dimers per minute per nanometer formed in sunlight in an *E. coli* chromosome. This curve is obtained by multiplying the last two curves described. Integrating this curve from 290 to 320 nanometers gives the value of ten dimers per minute formed in an *E. coli* chromosome.

creases continuously to 40 percent of the initial value as the G + C content of randomly ordered double stranded DNA increases from 25 percent to 75 percent. This is because thymine dimers constitute 50 percent of the ultraviolet photoproducts, cytosine-thymine dimers 40 percent, and cytosine dimers only 10 percent (*13*) (in the 50 percent G + C *E. coli*). Haynes (*20*) has shown experimentally that ultraviolet sensitivity at high doses decreases with increasing G + C content in bacteria. Thus, an increase in G + C content should lead to a selective advantage for small organisms exposed to strong sunlight.

We propose that the nature of the genetic code permits organisms to change their base ratio with little or no cost. Changes could occur either within the set of codons for the same amino acid (synonymous codons) or between codons for similar amino acids.

The base ratio of DNA can be changed without changing the amino acid sequences of the proteins which the DNA codes for. Conversions between synonymous codons recognized by the same transfer RNA (tRNA) should not be detrimental. According to the Crick wobble theory (*21*), while the messenger RNA is being translated, any tRNA which recognizes adenine (A) in the third position of the codon also recognizes G and any tRNA which recognizes C in the third position also recognizes uracil. Since most tRNA species have been shown to have this property (*22*), the G +C content of the DNA could change by at least ± 10 percent due to the substitutions between codons recognized by the same tRNA without any change in protein sequences.

Some conversions between synonymous codons involve changes between tRNA species. If there are no special functions for such codons, then the DNA of *E. coli* (50 percent G+C), for example, could vary from 31 percent G + C content to 68 percent G + C content without any change in the amino acids of any protein.

In addition to changes within a set of synonymous codons, it is possible to change from a codon for one amino acid to another codon for a similar amino acid by the occurrence of a functionally neutral mutation (*23*). Evidence for the occurrence of these mutations is given by the different amino acid composition of proteins in bacteria with high and low G + C contents; this difference reflects the availability of various codons in DNA of a specific G +C content (*24*).

The above mechanisms for changing G + C content only operate for that (large) portion of the genome which codes for protein. There may be more or less severe restrictions on the G + C content of the remainder. In any case, it is clear that large changes in G + C content can occur with little or no change in the properties of the cell.

A simple mechanism would allow rapid shifts in G + C content as a result of different ultraviolet exposure in a new ecological niche. Cox and Yanofsky (*25*) analyzed a "mutator" gene in *E. coli* which increased the G + C content by 0.025 percent in 82 generations, a rate of one percent G + C per 3×10^3 generations. We visualize a mechanism whereby such a "mutator" gene appears in a population and gradually changes the G + C content of the progeny. This mutator gene then reverts in a descendant in which only neutral mutations had occurred. The descendant then has a selective advantage, and its progeny slowly take over the population. Although this mechanism is not essential to our theory, it is an attractive mechanism for response to ultraviolet exposure.

Selective Pressure toward Low G + C Content

Although evolutionary pressure from ultraviolet exposure explains why organisms exposed to sunlight have high G + C contents, it does not explain why some have G + C contents below 50 percent. It can be calculated from the binomial distribution that, if the G + C contents of species not exposed to sunlight were random, then there would be a negligible chance of a species with a G + C content falling outside the range 49 percent to 51 percent (*26*). Thus, there must be some evolutionary pressure toward low G + C content. This could be either a pressure which operates only in the absence of sunlight, or more likely, a lower level pressure which is overwhelmed when the organism occupies a niche with high ultraviolet exposure.

Kaplan and Zavarine (*27*) have reported a correlation between high G + C contents and ionizing radiation sensitivity. Thus, cosmic rays and natural background radioactivity could produce a universal pressure toward low G + C content. However, the generality of Kaplan and Zavarine's observation is in doubt (*28*).

A possible source of pressure toward low G + C content could be the presence of naturally occurring alkylating chemicals that are known to attack the guanine in DNA.

Although we are confident that thymine-specific damage from sunlight ultraviolet accounts for high G + C contents, there is insufficient evidence to decide among the above and other possible explanations for the very low G + C contents of DNA of bacteria not exposed to sunlight.

Conclusions and Outlook

Our proposal that in certain bacteria high G + C content has evolved as a defense against ultraviolet radiation should be tempered by the inherent lack of definitiveness of evolutionary arguments. There is also a need for more knowledge about bacterial habitats and repair systems. The recognition of the importance of ultraviolet light as a powerful force in the life of bacteria should contribute to the understanding of, and interest in, these areas.

Bacterial taxonomists in recent years have relied heavily on G + C contents for determining bacterial relationships. Our article points out the importance of the bacterial habitat in the interpretation of relationships based on G + C content. It will be of interest to explore the possibility of convergent evolution to high G + C content of bacterial groups that are not closely related, and of divergent evolution to different G + C contents of bacterial groups that are closely related but live in habitats with different ultraviolet exposure.

Thus, ultraviolet damage of DNA apparently has been of tremendous importance in the origin and evolution of life. As has been pointed out (*11*), until the development of photosynthesis produced oxygen there was no ozone to filter out the ultraviolet, and the ultraviolet flux was enormously higher than it is now. This decrease in the ultraviolet intensity must have enabled life to take evolutionary paths which would be unavailable to organisms subjected to a high ultraviolet flux. The influence of ultraviolet on the evolution of microorganisms other than bacteria and on certain key developments in evolution such as diploidy and the development of eukaryotic animals and plants remains to be investigated.

We postulate an evolutionary pres-

sure toward low G + C content in environments not subjected to ultraviolet radiation, but the nature of this pressure remains an open question.

Note added in proof: A recent report on the sunlight sensitivity of the yeast *Saccharomyces cerevisiae* (*33*) adds support to our argument. Yeast is killed by sunlight, but is enormously more sensitive if a mutant lacking the excision repair system is used: thus DNA is the target. The bulk of the damage is pyrimidine dimers as shown by the fact that a double mutant lacking both excision repair and photorepair is much more sensitive to sunlight than the excision repair mutant alone.

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Attention and Psychological Change in the Young Child

Analysis of early determinants of attention provides insights into the nature of psychological growth.

Jerome Kagan

One of the great unanswered psychological questions concerns the mechanisms responsible for the transformations in organization of behavior and cognitive structure that define growth and differentiation. Until recently most of these changes were viewed as the product of learning. The child was presumably born unmarked, and the imposing hand of experience taught him the structures that defined him. Hence, many behavioral scientists agreed that learning was the central mystery to unravel, and conditioning was the fundamental mechanism of learning. There is a growing consensus, however, that conditioning may be too limited a process to explain the breadth and variety of change characteristic of behavioral and psychological structures. What was once a unitary problem has become a set of more manageable and theoretically sounder themes.

Category of Change

It is always desirable to categorize phenomena according to the hypothetical processes that produced them. But since psychology has not discovered these primary mechanisms, it is often limited to descriptive classifications. One category includes alterations in the probability that a stimulus will evoke a given response, which is a brief operation-

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