LIMITS ON ASYMMETRIC ORTHOPOSITRONIUM FORMATION IN HIGH Z OPTICALLY ACTIVE MOLECULES

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Abstract. The proposed connection between the parity violating handedness of beta particles in radioactive decay and the sign (L) of biological chirality (the Vester–Ulbricht [V–U] hypothesis) is experimentally tested. The theoretically predicted asymmetry in triplet positronium formation (A_{Ps}) is measured in several high Z optically active molecules using low energy positrons with a net helicity. We find $A_{Ps} < 3 \times 10^{-4}$ in selenocystine ($Z = 34$) and thyroxine ($Z = 53$), excluding part of the theoretically predicted range of $4 \times 10^{-3} > A_{Ps} > 2 \times 10^{-6}$ in these molecules. The connection between these limits and limits on asymmetric radiolysis (A_R) is made, with a new limit of $A_R > 10^{-9}$ being placed. This limit on A_R, which is thirty times lower than a previous measurement in the amino acid leucine ($Z = 6$), is still not small enough to rule out the V–U hypothesis.

1. Introduction

We report new experimental results concerning the Vester–Ulbricht hypothesis which states that asymmetric radiolysis by electrons from beta decay caused the observed sign of biological chirality (L amino acids, D sugars). If the chirality of biological molecules has a causal origin, it must be a result of the weak interaction, the only parity-violating fundamental interaction (see reference [1] for a complete discussion of this point). Two manifestations of the weak interaction, asymmetric radiolysis from longitudinally polarized electrons produced in beta decay [2, 3] and molecular energy differences due to weak neutral currents [4] have been suggested as sources of a chiral polarization η induced in a near equilibrium system. Here η is defined as $\eta \equiv (\eta_L - \eta_D)/(\eta_L + \eta_D)$, where η_L and η_D are the numbers of L and D molecules present and a system is defined to be the population of $N = \eta_L + \eta_D$ chiral molecules subject to the effects of the weak interaction prior to subsequent processing by any amplification mechanism.

Stochastic fluctuations in the number of molecules present in a system also produce a chiral polarization, η_F. In systems near equilibrium or in static systems, η_F must be less than η to produce a causal origin for the present biological chirality. Since $\eta_F \propto N^{-\frac{1}{2}}$, the parameters of a system where the condition $\eta_F < \eta$ obtains will be determined by the magnitude of η. In the subsequent discussion we ignore the effects of random causal mechanisms such as circularly polarized light. The chiral polarization due to weak neutral currents, η_{nc}, has a value $|\eta_{nc}| \sim 10^{-17}$, and in fact η_{nc} has been calculated to be positive for L alanine and the L peptides in the α-helix and β sheet conformation [4]. By comparison the chiral polarization due to asymmetric radiolysis, η_R, can range (at

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$T = 20\,°C$) from $\eta_R \sim 10^{-17}$ for the level of β radiolysis typical of the naturally occurring isotopes ^{40}K and ^{14}C which existed in the early earth's crust and oceans, to values as large as $\eta_R = 3 \times 10^{-12}$ for strong β sources such as ^{26}Al or natural nuclear reactors which could well have been present in the prebiotic environments [5]. The question of which isomer is favored by radiolysis has not, as yet, been answered either theoretically or experimentally. For the largest value of η_R, the number of molecules required for $\eta_R \sim 3\eta_F = 3N^{-\frac{1}{2}}$ in an equilibrium system of monomers prior to amplification is roughly [5] $N \sim 9\eta_R{}^{-2} = 10^{24}$ (about 150 g of alanine).

The value of $\eta_R = 3 \times 10^{-12}$ is based on a calculated asymmetry in beta radiolysis, $A_R = 10^{-11}$. Here $A_R \equiv (\sigma_L - \sigma_D)/(\sigma_L + \sigma_D)$ with $\sigma_{L,D}$ the cross sections for ionization of L, D molecules by longitudinally polarized electrons (electron spin preferentially aligned with respect to momentum). However, the theoretical value of A_R can lie in the range 10^{-10}–10^{-12} yielding $3 \times 10^{-11} < \eta_R < 3 \times 10^{-13}$. Thus, assuming the theoretical range of η_R to be correct, the value of N must lie between $10^{22} < N < 10^{26}$, corresponding to 1.5 g to 15 kg of alanine. The theoretical uncertainty in A_R, therefore, leads to a large uncertainty in the size required of a system for $\eta_R > \eta_F$.

The near equilibrium systems referred to above must be operated on by some type of amplification mechanism in order to produce the present biological homochirality. Recently, a simple model of one such amplification mechanism, spontaneous symmetry breaking in autocatalytic systems due to the energy difference between enantiomers caused by weak neutral currents, has been considered [6, 7]. In this model, two systems of achiral reactants combine to form a chiral product, and it has been shown that, under certain plausible prebiotic conditions, when the initial system passes through a nonequilibrium state, a homochiral system, whose sign is determined by the sign of η_{nc}, can result even for η_{nc} as small as 10^{-17}. More recently it has been shown that two *chiral* systems with an initial chiral polarization η_R or η_{nc} can serve as the starting systems for the spontaneous symmetry breaking mechanism [8]. Because the effects of asymmetric radiolysis can be as much as six orders of magnitude larger than η_{nc}, η_R can supplant weak neutral currents as the generator of this spontaneous symmetry breaking. Consequently, if autocatalytic systems played an important role in determining the homochirality of life, asymmetric radiolysis is likely to have been the dominant weak interaction effect.

As a result of the above recent theoretical progress in the study of η_R and η_{nc} in systems of monomers under various prebiotic scenarios and of the subsequent amplification, it has become of immediate importance to perform experimental tests of the theories from which A_R is calculated. These tests would help in (i) the determination of the relative importance of η_R and η_{nc}, (ii) the determination of whether the D or L isomer is favored by radiolysis, and (iii) the determination of the value of A_R, or limits on its size, which would in turn establish limits on the smallest number of chiral molecules needed in a static system for the condition $\eta_R > \eta_F$ to obtain. We discuss in the succeeding sections the results of such an experiment which has established a new upper limit on A_R.

Although the magnitude of A_R is too small to be directly observed experimentally,

theory shows that a related effect should occur which is observable and which can be used to obtain A_R, albeit indirectly. This effect is asymmetric formation of triplet (ortho) positronium (oPs) by longitudially polarized positrons. Specifically, we may define an experimentally observable oPs formation asymmetry A_{Ps} as:

$$A_{Ps} \equiv \frac{f^+(L) - f^-(L)}{f^+(L) + f^-(L)} = \frac{f^+(L) - f^+(D)}{f^+(L) + f^+(D)}. \tag{1}$$

Here $f^+(L)$, $f^-(L)$ refers to the fraction of oPs formed (with respect to total Ps formation – triplet plus singlet) for positrons of (+ or −) longitudinal polarization (positron spin parallel or anti-parallel to positron momentum) incident on L molecules. Analogous definitions hold for $f^{\pm}(D)$ and, by symmetry $f^{\pm}(L) = f^{\mp}(D)$. The magnitude of A_R is small because the exchange interaction required to produce A_R is small at β electron energies of 100 keV. However, Ps formation occurs at energies of 2–10 electron volts which causes $A_{Ps} \sim (10^4\text{–}10^5)A_R$. The value of A_R can be related to that of A_{Ps} by the theoretical expression which will be derived below.

The asymmetry in the interaction of longitudinally polarized β electrons with chiral molecules which is predicted by the bound helical electron model [2] is given by

$$A_R = \eta_{e^-}(\alpha Z)^2 \frac{h(e^-)}{2E_p \ln(E_p)}. \tag{2}$$

Here $\alpha = \frac{1}{137}$ is the fine structure constant, Z is the atomic number of a dominant heavy atom in an asymmetric environment of the molecule, η_{e^-} is a molecular asymmetry factor, estimated to be 10^{-3} to 10^{-1}, E_p is the energy of the primary β electron (typically 100 keV), and $h(e^-)$ is the degree of helicity (longitudinal polarization) of this particle at the time of ionization ($h(e^-) \sim 0.5$). We note that a direct calculation of η_{e^-} has not been completed for even the simplest chiral molecule. However, an explicit calculation of an analogous quantity related to *Ps* formation (η_{Ps}), discussed below, has been completed [2]. In reference [2] η_{e^-} was estimated to be within an order of magnitude of η_{Ps}, and in view of the discussion in the introduction, a direct calculation of η_{e^-} should now be of great value. For typical biological molecules, $Z = 6$, and for $h(e^-) = 0.5$, A_{e^-} is expected to be of order 10^{-10}–10^{-12} as mentioned earlier, where the range is due to the uncertainty in η_{e^-}.

The bound helical electron model also gives

$$A_{Ps} = h_f(e^+)\eta_{Ps}(\alpha Z_T)^2, \tag{3}$$

where Z_T is the atomic number of a dominant heavy atom in an asymmetric environment in the target molecule, η_{Ps} is an asymmetry factor analogous to η_{e^-}, and $h_f(e^+)$ is the helicity of the positron at the time of oPs formation. In the model molecule (twisted ethylene) η_{Ps} has been calculated to be 10^{-2} [2]. Taking $h(e^-) = 0.5$, $E_P = 100$ keV, and $Z = 6$ in Equation (1), and assuming $|\eta_{e^-}| \approx |\eta_{Ps}|$ (the relative signs

have not yet been determined), Equations (2) and (3) can be combined to give

$$A_R = C\frac{A_{Ps}}{h_f(e^+)Z_T^2}, \tag{4}$$

where $C = 3 \times 10^{-4}$. Implicit in the application of Equation (4) to prebiotic conditions are the assumptions that the population of chiral molecules which was drawn upon in the synthesis of life had an average atomic number $Z = 6$, and that no high Z chemical reactions participated in this synthesis. Since the accuracy of measuring A_{Ps} is essentially independent of Z_T and, from Equation (3), $A_{Ps} \propto Z_T^2$, measurement of A_{Ps} in molecules of high Z_T allows the inferred limits on A_R to be reduced accordingly. The experiment discussed below uses two such high Z molecules to set limits a factor of 100 lower than a recent measurement of similar sensitivity in the biological amino acid leucine [9].

2. Experimental technique

The experimental apparatus and techniques have been discussed in detail elsewhere [9, 10]. Briefly, a beam of positrons of controlled initial helicity, measured to be $h_0 = 0.48 \pm 0.02$, [11] is directed onto an amino acid sample that has been sublimated or deposited on a metal plate. The positrons are implanted into the sample with an initial energy of 200 eV and lose energy by collisions until they reach energies of 2–10 eV, where approximately 10% of the positrons form oPs. The initial helicity, h_o, is reduced by these collisions (which cause velocity randomization) to a final value which is calculated [10, 12] to be $h_f(e^+) = 0.04 \pm 0.01$. The oPs diffuses to the surface and escapes into the vacuum where it lives with the vacuum lifetime of 142 ns. The positrons which do not form vacuum oPs either annihilate directly with an electon, form the short-lived (0.13 ns lifetime) singlet positronium, or form oPs which annihilates within the sample. All of these latter possibilities have lifetimes of less than 2 ns. Thus, by measuring the time between positron arrival and emission of the annihilation gamma rays, we can separate long-lived oPs from other species. To measure the fraction of events in which long-lived oPs is formed, we count events in a time interval from 25 to 500 ns between positron entrance and gamma emission. From this count we subtract the random background and normalize it to the total number of recorded positrons. This ratio, r, is then found for positive and negative beam helicity and the asymmetry, $A_{Ps} \equiv (r^+ - r^-)/(r^+ + r^-)$ is calculated. Asymmetries are measured for several D, DL, and L samples. We expect for any true isomeric asymmetry that $A_{Ps}(L) = -A_{Ps}(D)$ and that $A_{Ps}(DL) = 0$.

3. Results

The asymmetries measured in L, D, and DL isomers of two high Z_T targets are shown in Table I. The quoted errors include both systematic and statistical uncertainty added in quadrature. The results of reference [9] for leucine are also shown in Table I. For each of the three isomers the statistical error (based on Gaussian $\sqrt{N}$ statistics) is

TABLE I

Target	$A_{Ps}(DL) \times 10^{-4}$	$A_{Ps}(L) \times 10^{-4}$	$A_{Ps}(D) \times 10^{-4}$
Leucine	-0.5 ± 1.5	-1.2 ± 1.5	-2.7 ± 1.5
Selenocystine	$+0.1 \pm 4.1$	-9.0 ± 4.6	$+1.4 \pm 4.6$
Thyroxine	-5.9 ± 4.5	$+5.0 \pm 5.1$	$+1.3 \pm 5.0$

TABLE II

The experimental values of A_{Ps}, the theoretical range of A_{Ps} based on two models of asymmetric Ps formation, and the experimental limit on A_R derived from the experimental values of A_{Ps} using Equation (4). The uncertainty in A_R is due to the uncertainty in $h_f(e^+)$ used in Equation (4).

Target	Z_T	A_{Ps} (experimental limit)	A_{Ps} (theoretical range)	A_R (experimental limit)
Leucine	6	$(-1.5 \pm 1.0) \times 10^{-4}$	5×10^{-5}–5×10^{-8}	$(3.0 \pm 1) \times 10^{-8}$
Selenocystine	34	$(+5.2 \pm 3.3) \times 10^{-4}$	2×10^{-3}–2×10^{-6}	$(2.0 \pm 0.5) \times 10^{-9}$
Thyroxine	53	$(+1.9 \pm 3.6) \times 10^{-4}$	4×10^{-3}–4×10^{-6}	$(1.0 \pm 0.3) \times 10^{-9}$

1.5×10^{-4}. The L vs D asymmetry for each of the substances considered in Table I is given by [9] $A_{PS} = [A_{Ps}(L) - A_{Ps}(D)]/2$ and is presented in Table II.

These (experimental) values of A_{Ps} given in Table II should be compared with the predicted ranges of A_{PS}, also shown in Table II. The predicted ranges are obtained from an analysis based in part on a recently completed Monte-Carlo program which tracked the slowing down and subsequent velocity randomization (dehelicitization) of a beam of positrons of 200 eV initial energy injected into water.

Water is a good model of most organic materials as far as electron-molecule interactions are concerned and we assume that this is also true for positron and Ps related processes. Two models predict that the positron can form Ps by different processes: (i) the Ore model which assumes that Ps formation occurs via direct electron capture in the energy range $2\,eV < E < 10\,eV$ (E will be taken subsequently to represent the energy of a positron at the moment of Ps formation), and (ii) the spur model which predicts that Ps formation occurs by the recombination of the e^+ with a secondary electron ejected by the e^+ during its slowing down process. Recent experimental [13] Ps formation data demonstrate that both of these processes occur in single crystal ice. The program we use was checked [14] by successfully modeling this recent experimental data.

The value of A_{Ps} will include contributions from both Ore and spur processes, and since A_{PS} depends on E, i.e., $A_{Ps} = A_{PS}(E)$, we must calculate the average value of $A_{Ps}(E)$ ($\bar{A}_{Ps}$) because this is the quantity actually observed. The value of $A_{Ps}(E)$ for the Ore model was calculated using the bound helical electron formulation for the

prototype molecule twisted ethylene ($Z = 6$) [15]. The spur contribution to $A_{Ps}(E)$ was taken from recent calculations of this process [16] for chiral molecules with carbon as the dominant asymmetric atom. These values of $A_{Ps}(E)$ were combined and inserted into the program in which the e^+ formed Ps at a variety of energies and the weighted average of $A_{Ps}(E)$ over all of the Ps formed ($\bar{A}_{Ps}$) was used to arrive at the ranges of A_{Ps} shown for leucine in Table II. The Z_T^2 dependence of A_{Ps} was then used to scale the $Z = 6$ values to selenocystine ($Z = 34$) and thyroxine ($Z = 53$). Finally, in order to use Equation (4) to derive the limits on A_R, shown in Table II, the weighted average of $h_f(e^+)$ ($\bar{h}_f(e^+)$) was also calculated [10]. We note here that the estimate of $h_f(e^+) = 0.4 \pm 0.2$ used in the results of reference [9] has been revised substantially downward to $\bar{h}_f(e^+) = 0.04 \pm 0.01$, based on this recent Monte-Carlo simulation of e^+ slowing down in insulators. The limit on A_R derived from the leucine result is thus roughly 10 times larger than estimated in reference [9].

In conclusion, we have shown experimentally that $A_{Ps} = (5.2 \pm 3.3) \times 10^{-4}$ in selenocystine and $A_{Ps} = (+1.9 \pm 3.6) \times 10^{-4}$ in thyroxine. We take both results to be consistent with a null result. Both measurements exclude part of the theoretical range predicted for A_{Ps}. The thyroxine ($Z = 53$) result (column 5, Table II) implies that $A_R < 10^{-9}$ in the pool of biological molecules ($Z = 6$) from which life was formed. This new limit represents over an order of magnitude improvement on the result reported in reference [9], revised as discussed above.

We are now constructing a new experimental apparatus in which the sensitivity to A_{Ps} should be increased to $\pm 3 \times 10^{-5}$ from the current $\pm 10^{-4}$. In addition, we will form Ps in a 6 kG magnetic field in our future experiment. This feature will cause the expected value of A_{Ps} to increase by approximately a factor of 3 for conditions which are otherwise identical to those which now obtain, i.e., for the same targets and the same positron beam. Finally the use of a $Z_T = 90$ target will allow a further factor of 3 increase in A_{Ps} over thyroxine ($(90/53)^2$) so that the overall theoretical range for A_{Ps} will be 4×10^{-2}–4×10^{-5}. Thus, with a sensitivity of $\pm 3 \times 10^{-5}$ we will, in all likelihood, be able to observe a positive effect, i.e., a non-zero value of A_{Ps}. If no effect is observed, limits on A_{Ps} of $\pm 3 \times 10^{-5}$ in a $Z = 90$ chiral system will be set, implying a limit on A_R at $Z = 6$ of $A_R < 10^{-11}$.

The significance to the question of the origin of the sign of biological optical activity of the measurement of A_R, or of limits on its value at the 10^{-11} level, has been discussed in the introduction to this article and in previous publications. [5, 8] We emphasize here once more that even if A_R is of order 10^{-12}, as is possible based on current theoretical analysis and experimental limits, the Vester–Ulbricht hypothesis cannot be eliminated as the cause of the specific sign of biological optical activity, given the recent theoretical work on amplification mechanisms [6, 7] which show that values of A_R as small as 10^{-16} can, under certain circumstances, be the cause of the present biological chirality [8]. On the other hand, if A_R is experimentally determined to be of the order 10^{-11} or greater, it may be possible, after appropriate research regarding the conditions that existed on the prebiotic earth, to establish the Vester–Ulbricht hypothesis as the probable cause of the observed sign of biological optical acitivity.

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