Evidence from Base-Pair Kinetics for Two Types of Adenine Tract Structures in Solution: Their Relation to DNA Curvature†

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ABSTRACT: We have measured the base-pair lifetimes in oligodeoxynucleotides containing tracts of A·T base pairs using imino proton magnetic resonance. When the tract contains *more than four consecutive A·T base pairs, possibly including a 5′-AT step but not a 5′-TA step*, anomalously long lifetimes are observed. For example, the lifetimes of the central A·T base pairs of the dodecamer 5′-d-CGCAAAAAAGCG are 122 and 91 ms at 15 °C whereas, in the same conditions, the lifetime of the central A·T pair of the decamer 5′-d-CGCGATCGCG is only 4 ms, a value similar to those measured in several other B-DNA oligoduplexes [Leroy et al. (1988) *J. Mol. Biol. 200*, 223–238]. This strongly suggests that, in tracts of four A·T pairs or more, a conformation distinct from standard B-DNA is formed cooperatively. All sequences known to generate curved DNA exhibit anomalously long base-pair lifetimes. This is the first local and physical property shown to correlate with DNA curvature. Our observations suggest that the structure responsible for the long lifetimes is involved in the curvature of DNA.

The curvature of DNA-carrying adenine tracts ($dA_n \cdot dT_n$) has been demonstrated by the modification of electrophoretic mobility (Wu & Crothers, 1984). The models proposed to explain it fall into two classes.

In the first class, a single conformation describes all adenine tracts. This may be a specific assumption, as in the junction model (Koo et al., 1986; Koo & Crothers, 1988), or it may follow from the assumption that the geometric relation between two successive base pairs (helix screw, tilt and roll angles) depends on the two base pairs only, irrespective of the upstream or downstream sequence (Ulanovsky & Trifonov, 1987; Calladine et al., 1988). In the second class, adenine tracts may adopt different structures depending on their length and environment (Burkhoff & Tullius, 1988; Diekmann & von Kitzing, 1988).

Consider, for example, the two decamers 5′-CGTTTTAAAA and 5′-CGAAAATTTT, which give rise by repetition to straight and curved polymers, respectively (Hagerman, 1986). According to the first description, the difference results from the geometrical combination of dinucleotide steps among which GT, TA, and AC in the first polymer differ from GA, AT, and TC in the second, whereas all the AA steps (and TT steps) are the same in both sequences. In the second description, the structures of the A_4 (and/or T_4) stretches could differ in the two sequences, a possibility supported by the recent observation of differential sensitivity to cleavage by the hydroxyl radical (Burkhoff & Tullius, 1988).

Local structural information on sequences that do, or do not, induce the curvature of DNA is required for advancing this problem. Crystallographic studies of two DNA-curving sequences (Nelson et al., 1987; Coll et al., 1987) demonstrate an anomalous, so-called B′ structure in which the A·T base pairs have a large propeller twist, the successive base pairs are connected by a bifurcated hydrogen bond, and the minor groove is narrow. The latter feature has also been proposed for DNA-curving adenine tracts on the basis of the cleavage experiments (Burkhoff & Tullius, 1987).

Nuclear magnetic resonance of imino protons provides a simple and efficient method for measuring the lifetime and for evaluating the dissociation constant of individual base pairs. Although these properties do not translate directly into structural terms, they may help in their investigation. At the very least, they have the quality of being *local*. We have investigated base-pair kinetics of adenine tracts and find large anomalies, which correlate strikingly with DNA curving, as characterized by electrophoretic migration. This suggests that DNA-curving adenine tracts have an anomalous structure that is lacking in noncurving tracts. A tentative rule is proposed for its appearance.

Methods

The methods for assigning spectra and measuring base-pair lifetimes in oligoduplexes have been described (Leroy et al., 1988). The imino proton NMR spectrum of the duplex is first assigned as usual. The exchange time of each imino proton is measured as a function of the concentration of added proton-exchange catalyst (ammonia) by using broadening, longitudinal relaxation, or magnetization transfer from water (Figure 1). Extrapolation to infinite catalyst concentration yields the base-pair lifetime (Figure 2).

Oligonucleotides were synthesized with a solid-phase Pharmacia gene assembler by the β-cyanoethyl phosphoramidite method and purified by high-pressure gel filtration on a poly(vinylimidazole) column (PVDI 300-10, Société française Chromato Colonne). The NMR samples contained 50 A_{260} units in 200 μL of a solution adjusted to pH 8.8.

All nucleic acids in this paper are deoxyoligomers in duplex form, whether self-complementary or not. They are designated by the base sequence of one strand, starting from the 5′ end of this strand. Three families of oligoduplexes were investigated. The first one is 5′-d-$CGCA_nGCG$, together with the complementary strand. It includes, for $n = 6$, the duplex for which a structure has been obtained by crystallography (Nelson et al., 1987). The oligoduplexes built on 5′-d-$CGCA_2XA_2GCG$ (where X is I or G and is paired with C)

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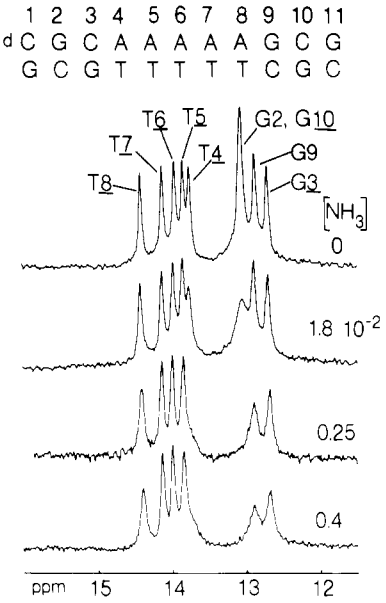

FIGURE 1: Effect of ammonia base, a proton-exchange catalyst, on the imino proton spectrum of the oligoduplex formed by 5′-d-CGCA$_5$GCG and its complementary strand. Assignments were obtained by inter imino proton NOEs. Exchange broadening is large for the imino protons of all Gs and of T4 (T4 designates the base paired to A4). It is clear for T8 but barely visible for T5, T6, and T7. In such cases, proton-exchange times are measured by longitudinal relaxation or magnetization transfer from water. The rest of the spectrum is not affected by the catalyst (not shown). T = 15 °C, pH 8.8.

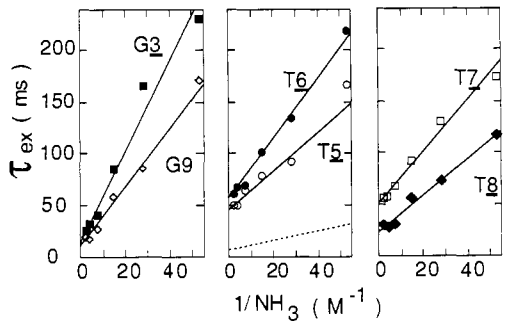

FIGURE 2: Extrapolation of imino proton exchange time to infinite catalyst concentration yields base-pair lifetime. The error is estimated at less than 15%. For comparison, the lower line in the middle panel represents the exchange times of the alternating sequence $(AT)_7$ whose lifetime (6 ms) and dissociation constant (proportional to the slope) are representative of normal A·T base pairs. Same duplex and same conditions as in Figure 1.

are considered part of the same family, and 5′-d-A_{14} as well.

The second and third families consist of duplexes built on the self-complementary sequences 5′-d-GGA$_n$T$_n$CC and 5′-d-CCT$_n$A$_n$GG, respectively. Base-pair lifetimes were measured at 15 °C and are given in the tables and figures.

RESULTS

In all sequences, the lifetimes of the flanking G·C base pairs are normal, the first two being controlled by end effects and the third (where appropriate) ranging from 4 to 14 ms, values that are comparable to the corresponding ones in numerous B-DNA sequences (Leroy et al., 1988).

Table I gives the base-pair lifetimes in sequences carrying an A$_n$ tract with n = 2–6 and n = 14, duplexed with the complementary strand. For n = 2 and 3, the A·T base-pair lifetimes are comparable to those previously observed in numerous duplexes (Leroy et al., 1988), which are always less than 7 ms and more than 0.8 ms except for end effects.

Table I[a]

													sequence
5′-d-	C	G	C	A	A	G	C	G					1
	*[b]	*	4	≤1	3	7	*	*					
	C	G	C	A	A	A	G	C	G				2
	*	*	8	≤3	6	7	6	*	*				
	C	G	C	A	A	A	A	G	C	G			3
	*	*	5	≤3	22	23	18	4	*	*			
	C	G	C	A	A	A	A	A	G	C	G		4
	*	*	14	≤3	47	57	51	25	11	*	*		
	C	G	C	A	A	I	A	A	G	C	G		5
	*	*	14	≤3	24	33	17	17	9	*	*		
	C	G	C	A	A	G	A	A	G	C	G		6
	*	*	4	≤3	≤3	23	4	5	4	*	*		
	C	G	C	A	A	A	A	A	A	G	C	G	7
	*	*	12	≤3	54	122	91	84	28	11	*	*	
				A_{14}									8
				>60[c]									

[a] Base-pair lifetimes (milliseconds) at 15 °C, pH 8.8. Each deoxy duplex is made up of the sequence indicated, combined with its complementary strand. [b] Asterisks indicate less than 1 ms. [c] Unresolved peaks.

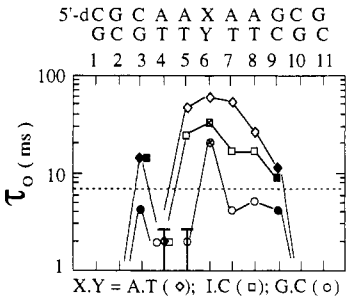

FIGURE 3: Base-pair lifetimes of CGCA$_2$XA$_2$GCG, where X stands for A, I, or G paired with T, C, and C, respectively. Data are those of Table I. The dotted line signals the maximum A·T base-pair lifetime observed in any duplex outside A tracts.

For n = 4, the lifetimes are distinctly longer, and they increase even more for n = 5, 6 (the "Nelson" sequence), and 14, for which the lifetimes are *an order of magnitude* larger than standard values.

In these tracts, the lifetime follows a distinct pattern (Figure 3). It is normal for the first A·T pair, or even shorter than the average of standard values. It is anomalously long, starting from the second pair, increasing to the third pair, and then decreasing. The last A·T base pair of the tract has the shortest of all the anomalously long lifetimes.

The lifetimes in the A$_2$IA$_2$ tract are also anomalously long, even though the I·C base pair cannot accept or donate bifurcated hydrogen bonds such as those observed in the crystal structure of the CGCA$_6$GCG duplex (Nelson et al., 1987). The lifetimes are reduced about 2-fold with respect to the A$_5$ tract (Figure 3). In contrast, the lifetimes in the A$_2$GA$_2$ tract are normal.

The lifetime anomaly is less conspicuous at higher temperatures. The base-pair opening activation energy has been measured in the duplex dA$_{14}$ (96 kJ/M) and for the long-lived base pairs of GGAAATTTCC (75 and 100 kJ/M for AT$_4$ and AT$_5$, respectively). These values are larger than those, ca. 50 kJ/M, for normal A·T base pairs (Leroy et al., 1988).

Table II gives lifetime values for self-complementary sequences incorporating A$_n$T$_n$ tracts. As a first approximation, an A$_n$T$_n$ tract behaves as an A$_{2n}$ tract, indicating that *an AT step does not disrupt the structure responsible for the long lifetime*. Thus the AT tract is normal, like A$_2$, but A$_2$T$_2$ is

Table II[a]

																			sequence
5′-d-	C	G	C	G	A:	T	C	G	C	G									9
	*[b]	*	9	16	4														
			G	G	A	A:	T	T	C	C									10
			*	*	9	21													
			G	G	A	A	A:	T	T	T	C	C							11
			*	*	≤1	86	86												
			G	G	A	A	A	A	A	A:	T	T	T	T	T	T	C	C	12
			*	*	≤1	60	60	100	100	65									

[a] Base-pair lifetimes (milliseconds) of self-complementary duplexes at 15 °C, pH 8.8. There is structural and kinetic symmetry with respect to the middle of the sequence. [b] Asterisks indicate less than 1 ms.

Table III[a]

																			sequence
5′-d-				C	C	T	T	T:	A	A	A	G	G						13
				*	*				≤1	5	1								
			C	C	T	T	T	T:	A	A	A	A	G	G					14
			*	*					4	19	17	1							
	C	C	T	T	T	T	T	T:	A	A	A	A	A	A	G	G			15
	*	*							6	26	34	36	36	≤3					

[a] Base-pair lifetimes (milliseconds) of self-complementary duplexes at 15 °C, pH 8.8. The 5′ end of the A tract is located in the middle of the sequence, which is a center of structural and kinetic symmetry. [b] Asterisks indicate less than 1 ms.

anomalous, like A_4. Note, however, that in A_6T_6 the lifetime of the two (identical) base pairs at the AT step is slightly shorter than that of their neighbor.

Table III gives lifetime values for T_nA_n tracts. Long lifetimes are found for $n \geq 4$, like for the A_n tracts of Table I. Also, the lifetime at the TA step is always short. These two properties indicate that, contrary to the AT step, *the TA step disrupts the structure responsible for long lifetimes.* One notes that the anomalous lifetimes are shorter in a T_nA_n tract than in the related A_n tract (for example, see $n = 6$).

We have also measured the base-pair dissociation constant on the basis of the efficiency of the proton-exchange catalyst (Kochoyan et al., 1987; Leroy et al., in preparation). For long-lived base pairs, it is about 10^{-6}, ten times smaller than for standard A·T base pairs.

Discussion

Tentative Rule for the Occurrence of the Kinetic Anomaly. The long lifetimes observed here are uniquely associated with the adenine/thymine tracts of DNA. They are not found in $CGCG(AT)_6CGCG$ or GGA_3GCT_3CC [see Leroy et al. (1988) for these and other sequences] or in alternating oligo[d(AT)]·oligo[d(AT)] (Figure 2) or poly(rA)·poly(rU) (Leroy et al., 1985a).

On the basis of the results reported here, we attempt to formulate a rule for the occurrence of the lifetime anomaly, as follows. Anomalously long base-pair lifetimes are found in DNA tracts of contiguous A·T base pairs. The tract must have a length of at least four (four is marginal), which may include a 5′-AT-3′ step but not a TA step. The A·T base pair at the 5′-A end of the tract is not anomalously long. No G·C pair is part of the tract.

This simple rule is followed without exception by all the sequences whose lifetimes have been measured. In Table I, the A_n tracts display the anomaly for $n \geq 4$. In Table II, the A_nT_n tracts include an AT step and their length is $2n$; as expected from the rule, lifetimes are short for $2n = 2$, and long lifetimes are found for $2n = 4$, 6, and 12. We are confident that the lifetimes are long for all values of $2n > 2$, in particular for $2n = 8$, i.e., A_4T_4.

In Table III, the T_nA_n tracts include a TA step, and they therefore carry two identical adenine tracts of length n, starting, on the 5′ side, with the A that is in the middle of the sequence. Lifetimes are short for $n = 3$ and long for $n = 4$ and 6 as predicted.

More data are needed for ascertaining and improving the tentative rule. End effects might be significant when an A tract starts or ends as close as two base pairs from the end of the duplex, as in Tables II and III. Distinctions may also be required regarding the flanking sequences. The effect of temperature must be explored. We note that there are other instances of long base-pair lifetimes, for example, in the case of Z-DNA (Leroy et al., in preparation) and for certain base pairs of transfer RNA (Leroy et al., 1985b).

Structural Implications. The kinetic anomaly is strong evidence for a structure which deviates from B-DNA. The structure forms cooperatively, requiring at least four A·T base pairs, as described by the tentative rule. The increased lifetimes for longer A·T sequences (Table I) indicate an enhancement of the structural anomaly; i.e., cooperativity extends beyond the minimal four base pairs. We expect the structure to be rather stable, given the small base-pair dissociation constant.

An obvious candidate is the B′ structure of adenine tracts observed in crystals of $CGCA_6GCG$ (sequence 7) and of $CGCA_3T_3GCG$, which is similar to sequence 11. The B′ structure combines a large propeller twist of base pairs, good stacking, bifurcated hydrogen bonds, and a narrow minor groove.

Good stacking could promote base-pair stability. A narrow minor groove is compatible with an AT step but not with a TA step (Burkhoff & Tullius, 1988), in agreement with the short lifetime and with the disruptive effect observed in the sequences of Table III and embodied in the proposed rule. The G amino group is also incompatible with a narrow minor groove.

We saw that the A_2IA_2 tract displays anomalous kinetics, even though the I·C base pair cannot connect to either of its neighbors by a bifurcated hydrogen bond. However, this does not rule out the B′ form, since the bifurcated bond's energetic contribution may be small and dispensable (Nelson et al., 1987). If the link between the B′ structure and long lifetimes

is correct, the minimum tract length for the formation of the B′ structure would be expected to be 4, a proposition that could be tested with crystals of appropriate duplexes or in solution by structural NMR methods.

The biochemical significance is suggested by two observations. First, the disruptive effect of a TA step on lifetimes may be considered in relation to the observation that such steps respond differently from other sequences of A·T pairs to attack by nucleases (Drew & Travers, 1984). Second, the minimum tract length required for the kinetic anomaly coincides with the minimum tract length (four or five), which displays preferential settings on the nucleosome supercoil (Nelson et al., 1987; Satchwell et al., 1986).

Independent evidence for a specific structure of adenine tracts is provided by the differential sensitivity to cleavage by the hydroxyl radical. Burkhoff and Tullius found progressive protection from cleavage in the 5′ to 3′ direction of adenine tracts of kinetoplast DNA, and they interpreted this as indicating a narrowing of the minor groove (Burkhoff & Tullius, 1987). The length of the tracts was five or more, if one includes AT steps. More recently, they investigated sequences incorporating A_4T_4 or T_4A_4 tracts (Burkhoff & Tullius, 1988). The former tract was protected from cleavage, the latter not. The interpretation was that the former tract has a specific structure, whereas the latter is in the B form.

These findings are strikingly similar to ours regarding A_3T_3 and T_3A_3, with anomalously long lifetimes in the former and short ones in the latter. The situation is less clear regarding A_4T_4 vs T_4A_4. Interpolation of Table II indicates that the lifetimes in A_4T_4 are long, in the range of 60–100 ms. The lifetimes in T_4A_4 are in the range of 20 ms, like A_4 or A_2T_2, in agreement with our proposed rule. They are also anomalous, even if they are much shorter than in A_4T_4. This suggests that in T_4A_4 also the non-B structure is formed, either incompletely or only part of the time, whereas the cleavage experiments indicate a B structure.

A possible explanation would be that hydroxyl cleavage does not detect the partially formed structure. An alternative explanation may lie in the difference in experimental conditions. The base-pair lifetimes were measured at 15 °C, in 0.2 M NaCl, pH 8.8, whereas the hydroxyl cleavage was carried out at room temperature, in low salt, pH 7.

Connection with DNA Curvature. We have examined (and in some cases designed) the sequences of Tables I–III with reference to the DNA-curving capacity of their adenine/thymine tract upon incorporation in repeating 10-mers. Figure 4 correlates the electrophoretic mobility anomaly R of the repeated 10-mer with the longest A·T base-pair lifetime of the tract in all 12 cases where the data are available. The correspondence is striking.

First, all the tracts with short ($\leq$7 ms) lifetimes, namely, A_3, A_2GA_2, AT, and T_3A_3, correlate with normal mobility (k ln $R \leq 0.18$). The corresponding points fall in the box at the lower left of the plot.

Second, most of the tracts with long lifetimes correlate with a large mobility anomaly (k ln $R \geq 0.39$), indicative of curvature. This is the case for A_4, A_2IA_2, A_5, A_6, and A_3T_3; the corresponding points fall in the box at the upper right of the plot.

Third, the lower right-hand area of Figure 4 is empty: all polydecamers known to be curved are based on sequences displaying long lifetimes.

The correlation suggests that the structure causing anomalous base-pair lifetimes is involved in DNA curving. If so, one understands why T_3A_3 is noncurving whereas A_3T_3 and

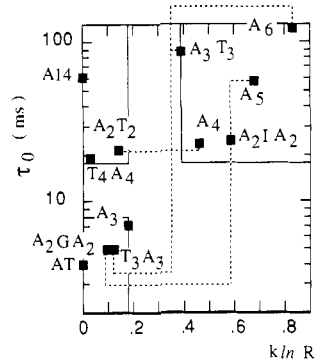

FIGURE 4: Correlation plot of the electrophoretic mobility anomaly, at room temperature, with the longest base-pair lifetime in sequences of Tables I–III. The electrophoretic mobility anomaly is characterized by k ln R, where R is the ratio of the length of the repeated sequence to the length of a random sequence having the same mobility and k is a correction factor for gel composition (Calladine et al., 1988). The repeated polymer related to A_{14} is poly(dA)·poly(dT). The R values are those measured for repeated sequences of 150 base pairs. They are taken feom Hagerman (1986) (T_4A_4), Hagerman (1988) (AT, A_2T_2, A_3T_3, and T_3A_3), Koo et al. (1986) (A_2GA_2, A_3, and A_4), and Koo and Crothers (1988) (A_5, A_6, and A_{14}). For A_2IA_2, the mobility anomaly was computed by comparison with A_5 (Koo & Crothers, 1987). The gap in electrophoretic mobility anomaly between 0.18 and 0.39 provides the distinction between straight and bent DNA. Points corresponding to adenine/thymine tracts of identical length are connected by a dashed line.

A_6 do induce curvature and why A_3 is noncurving, in contrast to A_4, since both T_3A_3 and A_3 have base-pair lifetimes indicative of B-DNA.

The comparison of A_3T_3 and T_3A_3 shows that base-pair lifetimes and, by extension, DNA conformation are dependent on a neighborhood extending beyond first neighbors. This indicates that DNA structure cannot be described solely by a universal set of dinucleotide geometries.

The correlation fails for the three sequences that fall in the left-hand corner of Figure 4, corresponding to long lifetimes but no curvature. The lack of curvature of polydecamers based on T_4A_4 (Hagerman, 1986) may be due to curving compensation, the geometry of the T_4 and A_4 tracts being the same in both duplexes. This is the assumption made in determinations of universal dinucleotide roll and tilt angles (Koo & Crothers, 1988; Ulanovsky & Trifonov, 1987; Calladine et al., 1988).

Another possibility is that T_4A_4 is noncurving because it is B-DNA in the conditions of the mobility measurement (Hagerman, 1986), which was done at room temperature, like the hydroxyl cleavage experiments, but in contrast to the conditions of the lifetime measurements (see above). If this is the correct explanation, the roll and tilt angles of recent models would have to be revised, as already suggested (Burkhoff & Tullius, 1988). This interpretation is reinforced by the previous discussion of A_3T_3 and T_3A_3.

Like T_4A_4, A_2T_2 is noncurving despite long lifetimes, and the same explanations may be considered. In both cases, the lifetimes, although long, are never more than 21 ms.

The long lifetime of A·T base pairs in A_{14}, combined with the lack of curvature of its polymeric form, poly(dA)·poly(dT) (Koo & Crothers, 1988), indicates that a continuous adenine tract is either straight or shaped so that there is no net curvature over long lengths. The situation for the $A_{10}T_{10}$ and $T_{10}A_{10}$ tracts is similar: lifetimes are long except for the TA step (not shown), but the corresponding polymer has only a small mobility anomaly (Koo & Crothers, 1988). Long lifetimes are also expected for the A_5 and A_4T_4 tracts of OHA, a 31-mer whose normal mobility has been ascribed to com-

pensation between the curvatures of different segments (Koo & Crothers, 1988).

Conclusion

Our measurements show that tracts of four or more A·T base pairs containing no 5′-TA junction display a characteristic physical property: anomalously long base-pair lifetimes. This indicates that the tract is a cooperative unit whose structure deviates from B-DNA. The structure, which is observed in solution, could be identical with the B′ form recently characterized in crystals, even though a bifurcated hydrogen bond is not required for the anomalous kinetics.

The conditions for anomalous kinetics correlate very well with those for DNA curvature. In particular, the base-pair lifetimes in A_3T_3 are anomalous but those of T_3A_3 are not. The lifetime anomaly is the first physical and local property of DNA in solution that correlates with DNA curvature. We propose that the cooperative structure which generates the lifetime anomaly is involved in the curvature of DNA by tracts of A·T base pairs.

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