We have studied the conformation of the peptide Ac-EPKRSVAFKKTKKEVKKATPKK (CH-1), free in solution and bound to the DNA, by Fourier-transform infrared spectroscopy. The peptide belongs to the COOH-terminal domain of histone H1\(^6\) (residues 99–121) and is adjacent to the central globular domain of the protein. In aqueous (D\(_2\)O) solution the amide I\(^*\) is dominated by component bands at 1643 cm\(^{-1}\) and 1662 cm\(^{-1}\), which have been assigned to random coil conformations and turns, respectively. In accordance with previous NMR results, the latter component has been interpreted as arising in turn-like conformations in rapid equilib-rium with unfolded states. The peptide becomes fully structured either in 90% trifluoroethanol (TFE) solution or upon interaction with the DNA. In these conditions, the contributions of turn (1662 cm\(^{-1}\)) and random coil components virtually disappear. In TFE, the spectrum is dominated by the \(\alpha\)-helical component (1654 cm\(^{-1}\)). The band at 1662 cm\(^{-1}\) shifts to 1670 cm\(^{-1}\), and has been assigned to the COOH-terminal TPKK turn motif in a more stable turn conformation. A band at 1637 cm\(^{-1}\), also present in TFE, has been assigned to the \(3_{10}\) helical structure. The amide I\(^*\) band of the complexes with the DNA retains the components that were attributed to \(3_{10}\), \(\alpha\)-helix and the TPKK turn. In the complexes with the DNA, the \(\alpha\)-helical component observed in TFE splits into two components at 1657 cm\(^{-1}\) and 1647 cm\(^{-1}\). Both components are inside the spectral region of \(\alpha\)-helical structures. Our results support the presence of inducible helical and turn elements, both sharing the character of DNA-binding motifs.

Histone H1 has a role in the stabilization of both the nucleosome and chromatin higher order structure. H1 linker histones have a characteristic three-domain structure (1). The central globular domain consists of a three-helix bundle with a \(\beta\)-hairpin at the C terminus (2–4), that is similar to the winged-helix motif found in some sequence-specific DNA-binding proteins. The amino-terminal and carboxyl-terminal tail-like domains are highly basic. H1 plays a key role in the folding of the nucleosomal arrays into the 30-nm chromatin fiber. Participation in nucleosome positioning has been proposed as a possible mechanism by which H1 could contribute to transcriptional regulation (5, 6). Experiments in vivo indicate that H1 does not function as a global transcriptional repressor, but instead participates in complexes that either activate or repress specific genes (7–11). Previous work has established that the globular domain of H1 is sufficient to direct specific gene repression in early Xenopus embryos (12). Other gene-specific effects, such as gene activation of the mouse mammary tumor virus (13) or the activation or repression of specific genes in Tetrahymena (14) are regulated by phosphorylation localized to the tail-like domains.

The study of the structure of the terminal domains when bound to the DNA may contribute to the understanding of H1 function. H1 terminal domains have little or no structure in solution. The COOH-terminal domain, however, acquires a substantial proportion of \(\alpha\)-helical structure in the presence of helical inducers such as TFE\(^1\) and HClO\(_4\) (15–17). The COOH-terminal domain is rich in lysine, alanine, and proline and binds to the linker DNA. It is required for chromatin condensation and is responsible for the ordered aggregation of DNA giving rise to the “\(\psi\)-DNA” spectrum in circular dichroism (18).

We previously studied the conformational properties of a peptide (CH-1) belonging to the COOH-terminal domain of the H1 subtype H1\(^{10}\) by high resolution \(^1\)H NMR (17). The peptide is adjacent to the globular domain and contains the longest proline-free fragment in the COOH-terminal domain. In aqueous solution, CH-1 behaves as a mainly unstructured peptide, although turn-like conformations in rapid equilibrium with the unfolded state may be present. Addition of TFE resulted in a substantial increase of the helical content. The helical region presents a marked amphipathic character, with all positively charged residues concentrated on one face of the helix and all the hydrophobic residues on the opposite face. This kind of arrangement could affect the conformation of the initial part of the linker DNA at the entry/exit point of the nucleosome. The last four residues of the peptide, TPKK, adopt a turn conformation in TFE. Sequences of this kind have been proposed as DNA binding motifs (19, 20).

We have now studied the interaction of the CH-1 peptide with the DNA by IR spectroscopy to identify features of the low-resolution structure of the bound peptide. IR spectroscopy is particularly well suited to the study of the complexes of DNA.
with basic peptides since it is not affected by turbidity. The important questions are whether the interaction with the DNA induces the folding of the peptide and, if so, whether the structure of the bound peptide is similar to the structure in TFE solution. Our results indicate that on binding to nonspecific DNA targets the peptide acquires a substantial proportion of helical structure, while the TPKKK motif at the COOH-terminal end of the peptide folds in a stable turn conformation.

**EXPERIMENTAL PROCEDURES**

**Peptide Synthesis**—The peptide Ac-EPKRSVAFKKTKKEVKATÒPKK (CH-1) was synthesized by standard methods (Neosystem Laboratoire, Strasbourg, France). Peptide homogeneity was determined by high-performance liquid chromatography on Nucleosil C18. The peptide composition was confirmed by amino acid analysis and the molecular mass was checked by mass spectrometry. The sequence of the peptide corresponds to residues 99 to 121 at the COOH terminus of histone H1. The sequence is common to the mouse and the rat and presents two conservative substitutions in humans, at positions 102 (Arg→Lys) and 113 (Val→Ile). The peptide was acetylated during the synthesis process to remove the dipole destabilization effect.

**Infrared Measurements**—Peptide samples were measured at 4.6 mg/ml. Peptide-DNA complexes contained 6.7 mg/ml DNA and the appropriate amount of peptide. Samples were measured in 10 mM HEPES, pH 7.0, plus either 10 or 70 mM NaCl. Prior to sample preparation, any residual trifluoroacetate counterions, used both in peptide synthesis and in the high performance liquid chromatography mobile phase, were replaced with chloride ions by five lyophilizations in 10 mM HCl. The trifluoroacetate ion has a strong infrared absorbance at 1670 cm⁻¹, which interferes with the characterization of the amide I band (21). A volume of 300 μl of HCl solution per 60 μg of peptide was used in each lyophilization. To transfer the samples to D₂O buffer, the aqueous solution was evaporated in a Speed-Vac (Savant) evaporator and then reconstituted in D₂O.

The spectra were recorded in a Nicolet Magna II 550 spectrometer equipped with a MCT detector using a demountable liquid cell (Harrick Scientific, Ossining, NY) with calcium fluoride windows and 50-μm spacers. Typically, 1000 scans for each spectrum, background and sample, were collected and the spectra were obtained with a nominal resolution of 2 cm⁻¹, at 22 °C.

Data treatment and band decomposition of the original amide I spectra have been described previously (22). Briefly, for each component, four parameters were considered, band position, band height, band width, and band shape. The number and position of component bands was obtained through deconvolution. Initial heights were set at 90% of those of the original spectrum for the bands in the wings and at the most intense component and at 70% of the original intensity for the other bands (23).

**FIG. 1.** The amide I band of the IR spectra of the CH-1 peptide at 22 °C in 10 mM HEPES, 10 mM NaCl, and D₂O, pD 7, and 4.6 mg/ml peptide concentration. A, free peptide in aqueous solution; B, free peptide in 90% TFE solution; C, spectrum of the peptide bound to mouse DNA, r (peptide/DNA ratio) = 0.7 (w/w); D, spectrum of the peptide bound to poly(dA-dT)poly(dA-dT), r = 0.7; E, spectrum of the complex of the peptide bound to poly(dA-dT)poly(dT), r = 0.7. The DNA contribution to the spectra of the complexes was subtracted as described under "Experimental Procedures."

In decomposing the amide I band, gaussian components were used. The baseline was removed before fitting. The curve-fitting procedure was accomplished in two steps. (i) The band position was fixed, allowing width and heights to approach final values, and (ii) band positions were left to change. Band decomposition was performed using CURVEFIT running under SpectraCalc (Galactic Inc., Salem, NH). The fitting result was evaluated visually by overlapping the reconstituted overall curve on the original spectrum and by examining the residual obtained by subtracting the fitting from the original curve. The procedure gave differences of less than 1% in band areas after the artificial spectra were submitted to the curve fitting procedure. The frequency positions of the band centers were independently evaluated by second derivative procedures; they were always very close to the positions found by deconvolution.

**Spectra of Peptide-DNA Complexes**—Complexes of the CH-1 peptide with mouse DNA, alternating poly(dA-dT)poly(dA-dT), and homopolymeric poly(dA)poly(dT) were prepared. The DNA contribution to the spectra was subtracted using a DNA sample of the same concentration. The symmetric component of the phosphate vibration at 1087 cm⁻¹ was used to check the accuracy of the subtraction, for this band shows no overlap with other vibrations, it is not substantially affected by the interaction of the peptide and its intensity is proportional to the DNA concentration. The DNA spectrum was weighted so as to cancel the absorption at 1087 cm⁻¹ in the difference spectra.
RESULTS

IR Spectroscopy in Aqueous Solution and TFE—The amide I band of the CH-1 peptide in aqueous (D$_2$O) and TFE solutions and in complexes with DNA are shown in Fig. 1. The number and position of the component bands were obtained by Fourier deconvolution and used for the curve-fitting of the original envelope by an iterative process as described previously (22) (Figs. 2-4). Values corresponding to band position and percentage area are given in Table I.

In aqueous solution, the amide I shows a major band (representing 38% of the total amide I intensity) at 1643 cm$^{-1}$, which is typical of the random coil (24). The other main component (25% of the total amide I intensity), which appears at 1662 cm$^{-1}$, was assigned to turns. In aqueous solution, the NMR did not show a significant helical population (17). However, the presence of abundant medium intensity NOE correlations between sequential amide protons, which are only close enough in folded structures, suggested that turn-like conformations in rapid equilibrium with the unfolded state could be present. The turns at 1662 cm$^{-1}$ observed in IR should, therefore, be short-lived and in rapid exchange with open states. Minor components at 1652 cm$^{-1}$ (5%) and 1637 cm$^{-1}$ (6%) were attributed to $\alpha$-helix and $\beta$-sheet helix, respectively, as discussed below (Fig. 2A). A band is present near 1625 cm$^{-1}$ (16%). This component also contributed to the spectra in TFE solution (7%) and in the complexes with the DNA (6–14%). This component was first observed in homopolyptides (25). It has been assigned to peptides in extended conformations involving in intermolecular hydrogen bonding, such as in monomer-monomer interactions. It is also seen in irreversibly aggregated proteins (22, 26–28). In human low density lipoproteins it was assigned to “low frequency” $\beta$-sheets (29) or to $\beta$-structure less accessible to the external solution (30). In this context, this component could be tentatively assigned to a fraction of aggregated peptide.

In 90% TFE solution (Fig. 2B), the random coil vibration at 1643 cm$^{-1}$ was only a minor component (3%), while the $\alpha$-helix band at 1654 cm$^{-1}$ became the main component, with 51% of the total amide I band. In TFE, the band at 1637 cm$^{-1}$, that was present as a minor component in water solution, was observed with increased intensity (14% of the amide I intensity). Bands at this wavenumber have been attributed to 3$\overline{10}$ helix by several authors (31–34). This band position is also found in $\beta$-sheet structures (24), but in this structure must be accompanied by a high frequency component at 1675 cm$^{-1}$ (22), that was not observed in our case. Therefore, in CH-1, we assigned the band at 1637 cm$^{-1}$ to 3$\overline{10}$ helical structure. The assignment is supported by structure calculations based on NOE constraints observed in 90% TFE, conditions where NMR shows no sign of $\beta$-structure (17).

In TFE, a component at 1670 cm$^{-1}$, representing about 20% of the amide I band, substitutes the component at 1662 cm$^{-1}$. Bands around 1670 cm$^{-1}$ are also attributed to turns. The main contribution to this band probably arises from the TPKK motif at the COOH-terminal end of the peptide. The motif has no defined structure in aqueous solution, but in 90% TFE solution it adopts a type (I) $\beta$-turn, an $\alpha$-turn, or a combination of both. In these conditions, the rest of the peptide is basically helical as shown both by IR and NMR. The band at 1662 cm$^{-1}$, found in aqueous solution, would thus correspond to less stable turn-like conformations in rapid equilibrium with disordered states, while the band at 1670 cm$^{-1}$ would arise from the more stable TPKK motif.

IR Spectroscopy of the Peptide Bound to the DNA—We have studied the structure of the peptide bound to several types of DNA: mouse DNA, alternating poly(dA-dT)-poly(dA-dT) and homopolymeric poly(dA)-poly(dT) (Fig. 3). The DNA contribution to the spectra of peptide-DNA complexes was subtracted using a DNA sample of the same concentration. The asymmetric component of the phosphate vibration at 1087 cm$^{-1}$ was used to check the accuracy of the subtraction as described under “Experimental Procedures.” Spectra of complexes of different peptide/DNA ratio ($r$) were also recorded (Fig. 4). The spectra were always basically identical regardless of the $r$ value of the complexes, indicating that the amide I region was not significantly affected by DNA spectral changes.

The band at 1670 cm$^{-1}$, which in TFE solution was attributed to the COOH-terminal TPKK motif in a turn conformation, was still observed in the complexes (Fig. 3). It amounts to about 23% of the amide I band. As the turn is not stable in aqueous solution, its presence in the peptide-DNA complexes indicates that the turn structure is induced by the interaction with the DNA.

In complexes with the DNA, the $\alpha$-helix band splits into two components at 1657 cm$^{-1}$ and 1647 cm$^{-1}$, each representing about 20% of the total amide I intensity. Both components are in the spectral region of $\alpha$-helical structures. The possibility that the faces of the amphipathic helical region of the peptide are exposed to different environments in the complexes, giving rise to different vibrations, will be discussed. A band at 1637 cm$^{-1}$, representing about 20% of the amide I intensity, was also present. It has been attributed to 3$\overline{10}$ helix, in accordance with the assignment in the peptide in TFE solution. This component was present with low intensity (6%) in the free peptide in water solution. Its significant increase in the complexes indicates that this kind of helical structure is induced upon interaction with the DNA.

The percentages and positions of the different components of the amide I band in the spectra of the complexes with alternating poly(dA-dT)-poly(dA-dT) and homopolymeric poly(dA)-poly(dT) are very similar to those obtained with mouse DNA (Fig. 3).

| Band position (cm$^{-1}$) | Band area (%) |
|--------------------------|---------------|
|                          | D$_2$O | 90% TFE | CH1/mouse DNA | CH1/p[dA-dT] | p[dA-dT] | CH1/p(dA) | p(dT) |
| 1624–26                  | 16    | 7      | 0            | 14           | 0        | 29           | 24 |
| 1637–38                  | 6     | 14     | 20           | 0            | 0        | 0            | 0  |
| 1642–43                  | 32    | 3      | 0            | 0            | 0        | 0            | 0  |
| 1647                     | 0     | 0      | 0            | 19           | 0        | 0            | 0  |
| 1652                     | 5     | 5      | 0            | 0            | 0        | 0            | 0  |
| 1654                     | 0     | 51     | 23           | 20           | 0        | 0            | 0  |
| 1657                     | 0     | 0      | 0            | 20           | 0        | 0            | 0  |
| 1662                     | 25    | 0      | 0            | 0            | 0        | 0            | 0  |
| 1670                     | 0     | 20     | 24           | 29           | 0        | 0            | 26 |

TABLE I

Band position (cm$^{-1}$) and percentage area (%) corresponding to the components obtained after curve fitting of the amide I band of the CH-1 peptide in aqueous (D$_2$O) and TFE solution and in the complexes with the DNA.
DISCUSSION

We have shown that double-stranded DNA can promote helix and turn formation in a peptide belonging to the COOH-terminal domain of histone H1 that has a very low amount of secondary structure in solution. The COOH-terminal domain is presumably involved in the organization of the linker DNA in chromatin. The CH-1 peptide is adjacent to the globular domain and could affect the conformation of the initial part of the linker DNA at the entry/exit of the nucleosome. This could have important consequences in those cases where gene regulation is mediated by the tail domains (13, 14).

In aqueous solution, the IR spectrum of the free peptide is dominated by the random coil band at 1643 cm$^{-1}$ and by a band at 1662 cm$^{-1}$, which is usually attributed to turns. Previous NMR studies of the peptide in water solution showed abundant NN($i$, $i+1$) NOE connectivities of medium intensity, indicating that in these conditions the conformational ensemble of the
peptide included a substantial population of folded conformations. Medium and strong βSi/i, i+1) connectivities, which are commonly observed in type I (or type III) turns and in helical structures (35) were also present (17). Long range order was, however, absent. We conclude that the band at 1662 cm⁻¹ originates in short-lived turn-like conformations, that interconvert rapidly with the extended chain random coil form. Such an ensemble of secondary structures of low stability was referred to by Dyson et al. (36) as nascent helix, since they are readily stabilized in an ordered helical conformation by long range interactions or in the presence of TFE. The assignment of the 1662 cm⁻¹ band is further supported by the disappearance of this component in TFE solution and in the complexes with the DNA, conditions that both stabilize a significant amount of helical structure.

In TFE solution and in the DNA complexes, the 1662 cm⁻¹ band shifts to 1670 cm⁻¹, indicating a change in the turn pattern (22). We attribute this band to the TPKK motif at the COOH-terminal end of the peptide. In the presence of secondary structure inducers (such as TFE and dimethyl sulfoxide), (T/S/i)PK/R/K/R sequences adopt turn conformations, that could be a type (I) β-turn, a σ-turn, or a βr-turn with two hydrogen bonds (17, 19, 20). Although it is not easy to attribute a particular component of the IR spectrum to a given region of a peptide; in this case, the intense band at 1670 cm⁻¹ can hardly arise from sequences other than the TPKK motif since in 90% TFE the rest of the peptide predominantly adopts a helical structure (Table I). The important observation is that the band at 1670 cm⁻¹ is present in the complexes with the DNA, and with a similar intensity as in the free peptide in TFE solution, indicating that the interaction with the DNA stabilizes the turn conformation of the TPKK motif. In summary, the band at 1662 cm⁻¹ observed in water solution would arise from transient turn structures that would be stabilized into helical structure with long-range order in water/TFE mixtures or by interaction with the DNA. The band at 1670 cm⁻¹ would arise from the well defined, and presumably more stable, TPKK motif. The folding of the TPKK motif upon interaction with the DNA confirms that this sequence indeed behaves in H1 as a DNA binding motif as previously proposed (19, 20).

In 90% TFE solution, the CH-1 peptide becomes helical between Pro¹⁰⁰ and Val¹¹⁶ (17). In these conditions, the amide I band is dominated by the 3₁₀ helix in the complexes with the DNA, and with a similar intensity as in the free peptide in TFE solution, indicating that the interaction with the DNA stabilizes the turn conformation of the TPKK motif. In summary, the band at 1662 cm⁻¹ observed in water solution would arise from transient turn structures that would be stabilized into helical structure with long-range order in water/TFE mixtures or by interaction with the DNA. The band at 1670 cm⁻¹ would arise from the well defined, and presumably more stable, TPKK motif. The folding of the TPKK motif upon interaction with the DNA confirms that this sequence indeed behaves in H1 as a DNA binding motif as previously proposed (19, 20).

The component at 1637 cm⁻¹ is present in the complexes, with an intensity (17-20%) slightly higher than in TFE, suggesting that the 3₁₀ helical structure is also induced upon interaction with the DNA. In the complexes, the contribution of the α-helix is split into two components at 1657 cm⁻¹ and 1647 cm⁻¹, which together represent about 40% of the total amide I intensity. The interaction with the DNA would thus induce a considerable amount of α-helical structure as compared with the small amount that may be present in aqueous solution. The presence of two components in the spectral region of the α-helix may be caused by a distortion of the helical region upon interaction with the DNA, which could be favored by the extreme amphipathic character of the helical region of the peptide, with all the basic residues on one side of the helix and all the hydrophobic residues on the other (17). It should also be noted that the vibrations of α-helical structures can be sensitive to their environment (37). The turbidity of the solutions of the peptide-DNA complexes, at the concentrations used in the IR experiments, suggests that the hydrophobic faces of the peptide bound to different DNA molecules could be in contact. In the complexes, the charged face of the helix bound to the DNA and the hydrophobic face may, therefore, be exposed to very different environments. Bands around 1647 cm⁻¹ have been assigned to α-helical structures. For instance, in magainins, a family of positively charged antimicrobial amphipathic peptides, which are reminiscent of the CH-1 peptide, an amide I band centered at 1647 cm⁻¹ was assigned to α-helix in the presence of negatively charged liposomes (38). Bands at 1656 cm⁻¹ and 1646 cm⁻¹ were also reported for the chaperone GroEL and assigned to different types of α-helix (39).

Taken together, the contributions of the peptide at 1657 cm⁻¹, 1647 cm⁻¹, and 1637 cm⁻¹ in the complexes with the DNA, which have been attributed to helical structure, are equivalent to the amount of helical structure of the peptide in TFE (Table I). In addition, the turn conformation of the TPKK motif is also induced upon interaction with the DNA. It thus appears that in this peptide, TFE reveals the conformational propensities of the DNA-bound peptide with high fidelity. Our results, showing the presence of inducible helical and turn elements in CH-1, support the view that both kinds of structural elements behave as DNA binding motifs in the COOH-terminal domain of histone H1.

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REFERENCES
1. Hartman, P. G., Chapman, G. E., Moss, T., and Bradbury, E. M. (1977) Eur. J. Biochem. 77, 45-51
2. Clere, G. M., Gronenborn, A. M., Nilges, M., Sukumaran, D. K., and Zarbock, J. (1987) EMBO J. 6, 1833-1842
3. Cerf, C., Lippens, G., Muyldermans, S., Segers, A., Ramakrishnan, V., Wodak, S. J., Hallenga, K., and Wyns, L. (1993) Biochemistry 32, 11345-11351
4. Ramakrishnan, V., Finch, J. T., Graziano, V., Lee, P. L., and Sweet, R. M. (1993) Nature 362, 219-223
5. Crane-Robinson, C. (1999) BioEssays 21, 367-371
6. Howe, L., Itoh, T., Katagiri, C., and Assís, J. (1998) Biochemistry 37, 7077-7082
7. Zlatanova, J., and Van Holde, K. (1992) J. Cell Sci. 103, 889-895
8. Khochbin, S., and Wolffe, A. P. (1994) Eur. J. Biochem. 225, 501-510
9. Bouvet, P., Dimitrov, S., and Wolffe, A. P. (1994) Genes Dev. 8, 1147-1159
10. Shen, X., and Gorovsky, M. A. (1996) Cell 86, 475-483
11. Wolffe, A. P., Khochbin, S. P., and Dimitrov, S. (1997) BioEssays 19, 249-255
12. Vermaak, D., Steinbach, O. C., Dimitrov, S., Rupp, R. A. W., and Wolffe, A. P. (1998) Curr. Biol. 8, 533-536
13. Lee, H. H., and Archer, T. K. (1998) EMBO J. 17, 1454-1466
14. Dou, Y., Miizen, C. A., Abrams, M., Allis, C. D., and Gorovsky, M. A. (1999) Mol. Cell 4, 641-647
15. Clark, D. J., Hill, C. S., Martin, S. R., and Thomas, J. O. (1968) EMBO J. 7, 69-75
16. Hill, C. S., Martin, S. R., and Thomas, J. O. (1989) EMBO J. 8, 2591-2599
17. Vila, R., Ponte, I., Jiménez, M. A., Ries, M., and Suaa, P. (2000) Protein Sci. 9, 627-636
18. Moran, F., Montero, F., Azorín, F., and Suaa, P. (1985) Biophys. Chem. 22, 125-129
19. Suzuoki, M. (1989) EMBO J. 8, 797-804
20. Suzuoki, M., Gerstein, M., and Johnson, T. (1993) Protein Eng. 6, 565-574
21. Susiewicz, W. K., Mantsch, H. H., and Chapman, D. (1993) Biochemistry 32, 389-394
22. Arrondo, J. L. R., and González, M. (1999) Prog. Biophys. Mol. Biol. 72, 367-405
23. Abbott, T. P., Wolf, W. J., Wu, Y. V., Butterfield, R. O., and Kleiman, R. (1990) Appl. Spectrosoc. 45, 1665-1673
24. Butler, D. M., and Susi, H. (1986) Biochemistry 25, 4589-4597
25. Suzi, H. (1969) in Structure and Stability of Biological Macromolecules (Timasheff, S. N., and Stevens, L., eds) pp. 575-663, Marcel Dekker Inc., New York
26. Alvaraz, J., Haris, P. I., Lee, D. C., and Chapman, D. (1987) Biochim. Biophys.
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27. Jackson, M., Haris, P. I., and Chapman, D. (1991) Biochemistry 30, 9681–9686
28. Surewicz, W. K., Lødd, J. J., and Mantsch, H. H. (1996) Biochemistry 29, 8106–8111
29. Goormaghtigh, E., De Meutter, J., Vanloo, B., Brasseur, R., Rosseneu, M., and Ruysschaert, J. M. (1989) Biochem. Biophys. Acta 1006, 147–150
30. Herzyk, E., Lee, D. C., Dunn, R. C., Bruckdorfer, K. R., and Chapman, D. (1987) Biochim. Biophys. Acta 922, 145–154
31. Dwivedi, A., Krimm, S., and Malcolm, B. R. (1984) Biopolymers 23, 2025–2065
32. Halloway, P. W., and Mantsch, H. H. (1989) Biochemistry 28, 931–935
33. Prestrelski, S. J., Byler, D. M., and Thompson, M. P. (1991) Int. J. Peptide Protein Res. 37, 508–512
34. Mück, S. M., Martinez, G. V., Fiori, W. R., Todd, A. P., and Milhauser, G. L. (1992) Nature 359, 653–655
35. Wüthrich, K., Billeter, M., and Braun, W. (1984) J. Mol. Biol. 180, 715–740
36. Dyson, J., Rance, M., Houghten, R. A., Wright, P. E., and Lerner, R. A. (1988) J. Mol. Biol. 201, 201–217
37. Haris, P. I., and Capman, D. (1995) Biopolymers 37, 251–263
38. Jackson, M., Mantsch, H. H., and Spencer, J. H. (1992) Biochemistry 31, 7289–7293
39. Galán, A., Sot, B., Llorca, O., Carrascosa, J. L., Valpuesta, J. M., and Muga, A. (2001) J. Biol. Chem. 276, 957–964
Induction of Secondary Structure in a COOH-terminal Peptide of Histone H1 by Interaction with the DNA: AN INFRARED SPECTROSCOPY STUDY
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