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DNA Looping, Supercoiling and Tension

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In complex organisms, activation or repression of gene expression by proteins bound to enhancer or silencer elements located several kilobases away from the promoter is a well recognized phenomenon. However, a mechanistic understanding of any of these multiprotein interactions is still incomplete. Part of the difficulty in characterizing long-range interactions is the complexity of the regulatory systems and also an underestimation of the effect of DNA supercoiling and tension. Supercoiling is expected to promote interactions between DNA sites because it winds the DNA into compact plectonemes in which distant DNA segments more frequently draw close. The idea that DNA is also under various levels of tension is becoming more widely accepted. Forces that stretch the double helix in vivo are the electrostatic repulsion among the negatively charged phosphate groups along the DNA backbone, the action of motor enzymes perhaps acting upon a topologically constrained sequence of DNA or chromosome segregation during cell mitosis following DNA replication. Presently, little is known about the tension acting on DNA in vivo, but characterization of how physiological regulatory processes, such as loop formation, depend on DNA tension in vitro will indicate the stretching force regimes likely to exist in vivo. In this light, the well studied CI protein of bacteriophage l, which was recently found to cause a of 3.8 kbp loop in DNA, is an ideal system in which to characterize long-range gene regulation. The large size of the loop lends itself to single-molecule techniques, which allow characterization of the dynamics of CI-mediated l DNA looping under controlled levels of supercoiling and tension. Such experiments are being used to discover the principles of long-range interactions in l and in more complex systems.