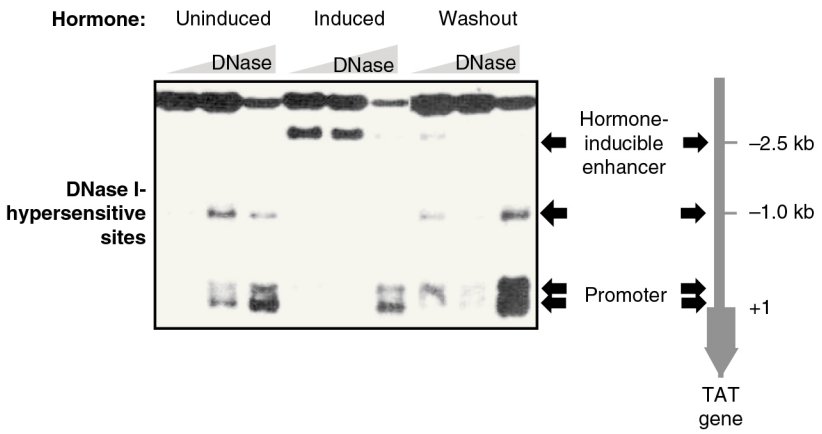

Figure 1. DNase I-hypersensitivity (DH) analysis reveals rapid and reversible local nucleosome remodeling in vivo. The figure shows primary data from a classical DH analysis (Reik et al. 1991). The chromatin organization at the glucocorticoid-responsive enhancer element, 2.5 kb upstream of the promoter of the tyrosine aminotransferase gene, was probed in rat liver cells. Isolated nuclei of cells are digested with increasing amounts of DNase I. Digested genomic DNA is purified, cleaved with a restriction enzyme, resolved by agarose gel electrophoresis, and subjected to Southern blotting. The DH sites are revealed by indirect end-labeling of restriction fragments through hybridization of a small radioactive probe). They are marked with arrows. In the silent, uninduced state, there are two DH sites at the promoter and one at −1 kb upstream. When the gene is activated on hormone induction with corticosterone, nucleosomes are remodeled at the enhancer within 15 min. A new DH site appears 2.5 kb upstream of the transcriptional start site, caused by chromatin remodeling (see "induced" columns). This correlates with the binding of glucocorticoid receptors and a complex set of remodeling factors. On removal of the hormone ("washout"), the factors dissociate and canonical nucleosomes reform within 15 min and the −2.5-kb enhancer DH disappears. The enhanced cleavage at the promoter reflects the transcriptional status of the gene.