Slide 1. The linking number for the two boundary curves of the ribbon equals the linking number for any of these curves with the ribbon axis. The proof, letting technicalities aside, is simple. Let the length of \( -v \) continuously shrink to zero, without changing \( +v \). This continuously deforms \( \gamma - v \) into \( \gamma \), without intersection of the two boundaries at any point. \( Lk \) remains constant in the process since \( Lk \) is a topological invariant.

Slide 2. For a chromatin circle, we imagine that the path \( \gamma \) of its DNA axis lies in the surface \( D \) of an appropriately deformed doughnut with axis \( \beta \). At nucleosomal sites, \( \beta \) is congruent with the axis of the DNA superhelix. We may choose \( v \) of the reference ribbon orthogonal to \( D \) to point inward, then \( \gamma + v = \beta \). In addition to the reference ribbon orthogonal to \( D \), whose linking number we call surface linking number, \( SLk \), we consider the ribbon \((\beta, -v)\). Its axis equals one of the two boundary of the reference ribbon orthogonal a to \( D \) and vice versa. According to the previous note, the linking number of \((\beta, -v)\) equals the linking number of \((\gamma, v)\). If \( \beta \) lies in a plane, its writhe is zero and the linking number of \((\beta, -v)\) equals its twist, the number of rotations of \(-v\) about \( \beta \). As long as the linker DNA has no writhed, only the nucleosome core particles contribute to this rotation with \(-1.65\) turns each. The minus sign is due to the left-handed wrapping of the DNA around the histone octamer. Hence, \( SLk = n(-1.65) \), where \( n \) refers to the number of nucleosomes.

Slide 3. The surface linking change per nucleosome \( \Delta SLk = -1.65 \), the helical repeats of naked and nucleosomal DNA are \( h_N = 10.2 \) and \( h_0 = 10.65 \), respectively. The number of base pairs wrapped around the histone octamer is 147.

Slide 4. In deriving \( \Delta Lk = -1 \) per nucleosome, we assumed that the doughnut axis \( \beta \) and the linker DNA is writh-free. This is not true for any of these model of the 30 nm fiber. In the case of the solenoid model the linker DNAs are short helical pieces and are thus writhed and \( \beta \) is helical as well. In the case of the two 2-start models, the linker DNA is straight but \( \beta \) is helical. There are various solutions to this problem. For example, the regularity in nucleosome organization assumed by all three model may not exist. \( \beta \) may randomly change between left-handed (helical ribbon) and right-handed (crossed linker) helical orientations such that its writh remains zero. This may be induced due to varying linker lengths. Note that the helical ribbon model reconciles
close packing with short linkers better than the crossed linker model. Whereas the crossed linker model affords closer packing for fibers with longer linkers (Wu, Bassett and Travers, 2007).

**Slide 6.** Note that there is a yeast homolog in any of the major monophyletic groups highlighted in different colors, indicating that the all the major SNF2 families must have been established early in eukaryotic evolution. Note also that SNF2 is related to prokaryotic DNA helicases. By far not all bacteria possess SNF2 related helicases, and if so they possess just one, whereas eukaryotes possess many. In yeast alone there are 17 SNF2 paralogs, only some of them are shown here.

**Slide 8.** Chromatin remodelers are complexes, very often containing many proteins. Although for SWI/SNF and ISWI it has been demonstrated that the catalytic subunits are sufficient for chromatin remodeling in vitro.

**Slide 11.** From biochemical assays with mononucleosomes it appeared that SWI/SNF generates a persistently altered nucleosome. The altered particle has the sedimentation properties of a dinucleosome.

**Slide 14.** This and the following experiments by Lorch and Kornberg suggest that RSC, starting from the ends, partially unspools the DNA from the histone octamer.

**Slide 16.** An unspooling of DNA from the histone octamer was also supported by topology analysis. An unwrapping of DNA from the nucleosome core particle increase the (negative) surface linking number and thus is thus expected to increase the Lk of the chromatin circle. This was shown by Lorch and Kornberg using two-dimensiona agarose gel electrophoresis. The result of such a separation is shown in this slide. Topoisomers were fractionated by two-dimensional gel electrophoresis at low ethidium bromide concentration in the first dimension (top to bottom), and at increased EthBr concentration in the second dimension (left to right). Topoisomers on the right side of inflection point I were positively supercoiled under conditions of the first electrophoresis direction, topoisomers to the left were negatively supercoiled. Topoisomers above inflection point II were positively supercoiled under conditions of electrophoresis in the
second dimension, topoisomers below were negatively supercoiled. Hence, the linking number of topoisomers increase clockwise around the arc.

**Slide 18.** Gel purified nucleosome N1 (nucleosomes formed in four different positions on DNA element used) was digested for 1’ with exonuclease III after incubation with NURF and ATP for the indicated time. Note that after 5’ almost all nucleosomes have been repositioned into position N3. The multitude of bands in short intervals between bands labeled by red and blue dots suggests that the relocation of the nucleosome proceeds in small increments rather than in large jumps.

**Slide 20.** A biotinylated DNA molecule was bound streptavidin coated magnetic beads. The experiment on the left argues for removal of the nucleosome from the end by sliding toward the magnetic bead. If the nucleosome would have been removed by sliding off the end, the nucleosome would have been removed even after cutting with Nhe I first (lane 4), which was not the case. Only if the DNA is treated with SWI/SNF first and then cut with Nhe I is the nucleosome removed (lane 6). The inhibitory effect of the four-way junction in-cis argues for sliding as the mechanism of removal.

**Slide 21.** The hydroxyl radical abstracts hydrogen atoms from the ribose; the resulting chemical rearrangements lead to removal of the ribose. The reactive hydrogen atoms are those that are exposed to the solvent. On each strand, a strong cleavage site and two weak cleavages 7 and 8 bp 3’ of the strong cleavage site are observed. The two main cleavage sites on complementary strands are separated by three base pairs indicating that the dyad axis passes through the plane of a base pair rather than between two base pairs.

**Slide 23.** While ISWI slides nucleosome core particles toward the end of linear DNA molecules, the closely related remodelers SWI/SNF and RSC slide the histone octamer off the end by about 50 bp.

**Slide 24.** Digestion was in the presence of RSC and ATP for 60’ at 30 degrees C. These experiments demonstrate that DNA site are exposed due only to sliding of the core particle, but not on the surface of the core particle.