Beta-sheet protein fibrils, also known as amyloid or amyloid-like fibrils, have been associated with many important pathological conditions such as Alzheimer’s disease and type II diabetes. Fibril formation also poses an important challenge to the emerging field of peptide drug delivery as a major degradation pathway and those fibrils have gained bionanotechnological importance as bio-nanotubular scaffolds. Rational design of anti-fibrillation drugs, stable formulation of peptide drug molecules and development of bionanotechnological fibril devices all depend on understanding the structure of these fibrils. However, although the structures for some of those fibrils are experimentally determined, there are many others for which no structure is yet available. Therefore a computational method to model the structure of beta-sheet rich fibrils would be very valuable. In a few cases, research groups have successfully generated computational models for fibrils. However, their modelling approaches are typically specialized for a particular fibril system and are not easily generalizable to all beta-fibrils. In this seminar, I present a general computational procedure to model the structure of any beta-fibril starting from its sequence. The validity of this method is demonstrated by reproducing the experimentally determined structures from four different classes of fibrils.