

## 2002 Spring Symposium Review

by Karen Steinmetz

GETA held its Spring Symposium on June 5, 2002, at ALZA Corporation in Mountain View, CA. The meeting was a huge success, combining excellent speakers with a wonderful lunch. The theme for the symposium was “Current Issues in DNA Repair: Models and Mechanisms.” The speakers that participated in the symposium were Drs Robert Tebbs, Lawrence Livermore National Laboratory, member of Larry Thompson’s Research Group; David Chen, Lawrence Berkeley National Laboratory; Wolf-Dietrich Heyer, UC Davis; Priscilla Cooper, Lawrence Berkeley National Laboratory, and Ms. Lisa DeFazio, Stanford University, Member of Gilbert Chu’s research group.

The symposium began with a brief introduction and overview by President-elect, Dr. Eva M. McGhee, from UCSF. She introduced information on how DNA repair plays an essential role in removing both endogenous and induced damage to the genome; models used to study DNA repair (yeast, mouse, and man); DNA repair mechanisms [direct reversal damage, Nucleotide Excision Repair (NER), Base Excision Repair (BER) Mismatch Repair, Recombination Repair (Homologous Recombination Repair and Nonhomologous Recombination Repair), Post-replicative repair and repair of damage to mitochondrial DNA].

The first speaker, Dr. Wolf Heyer (UC Davis) presented ongoing research in his laboratory investigating the Rad 54 protein involvement in the turnover of Rad 51-ds DNA filaments to provide an ordered transition to the resolution step, the final stage of homologous recombination.

Dr. Robert Tebbs (Lawrence Livermore National Laboratory) talked about some of his work on Xrcc1 mouse models with regard to base excision repair and DNA single-strand break emphasizing repair mechanisms that are essential for correcting endogenous DNA damage resulting from hydrolysis, oxidation, and simple alkylation.

Ms. Liza DeFazio (Stanford University) spoke about nonhomologous recombination repair, ongoing work in her lab, and how DNA-PKcs bring two DNA ends together and then undergo activation of its kinase presumably to regulate subsequent steps for processing and ligation of the ends.

Dr. Chen presented work from his research group on functions of DNA-dependent protein kinase complex in non homologous DNA end joining and telomere maintenance. He talked about how TRF1 and Ku interact to form a telomeric complex.

The last speaker was Dr. Cooper, who spoke on “Base excision repair.” Dr Cooper presented work from her laboratory on the identification of a novel transcription-coupled repair (TCR) pathway in human cells that preferentially removes oxidative base damage in active DNA through base excision repair.