SILVIA TORNALETTI - FINE STRUCTURE OF DAMAGE/REPAIR

- → p53 mutation hot spots
  - many positions can be mutated in cancers (unlike RAS & others which only a few spots can be mutated)
  - skin cancer
    - many mutations are CC→TT (signature)

LIGATION MEDIATED PCR

- need break w/ 5'-P

Can you use this to detect uracil sites?? or AP sites??

mismatches?

CPD
↓ T T
↓ photolyase
↓ primer extension
↓ slow light linker
↓ PCR

6-4 -- background is too high w/ no damage (probably due to piperidine)

- good correlation w/ mutat. on hotspots & damage hotspots

DIFF DOSES?

- repair
  - nts << ts
  - slower repair at mutation hotspots

COULDN'T THIS BE A CORRELATION? MAYBE THE MUTATION HOTSPOTS HAVE TO DO W/ ABILITY TO REPLICATE PAST DAMAGE

- methylation
  - cut C's (only at unmethylated) and then do LMPCR
- use UV light as FOOTPRINTING agent

WHAT IS MUTATION PATTERN IN REPAIR DEFECTIVE CELLS?