CYP1A2 Suppression in FQT/FQAD: Potential Mechanisms to Consider

- Person takes an FQ
  - FQ inhibits CYP1A2
    - Less CYP1A2, so cell sends out signal more is needed fast
      - More TOPO needed for the gene to make more CYP1A2
        - FQ binds to where more TOPOs are more active, ie, CYP1A2 gene transcription
          - FQ inhibition of CYP1A2 directly + FQ-TOPO stops CYP1A2 gene transcription
            - Drastically depleted CYP1A2 while on the drug
              - Rapidly dividing cells: may die if insult is great enough
                - Possibility of de novo mutations in CYP1A2 due to high TOPO activity while on the drug → functionality CYP1A2 compromised once drug stopped
                - Demethylases also inhibited by FQs resulting in long term or permanent epigenetic modifications → muting or suppression of CYP1A2
                - Autoimmunity to CYP1A2? → decrease/eliminate CYP1A2 function
                - Possible similar effects on TOPOs as well (similar flowchart could be written for TOPOs, as it’s required for its own transcription)
              - Possible upregulation or CYP1A2 induction as “rebound” effect when drug is stopped
                - Long term or permanent suppression of CYP1A2 → decreased ability to metabolize endogenous and exogenous substances (food metabolites, steroids, hormones, supplements, fatty acids, products of exercise) → build up of substrates → FQT/FQAD symptoms.
                - Methylxanthine sensitivities, both endogenous and exogenous
                - Build up of above substrates → “Invisible Line” or threshold I can’t cross for food, steroids, hormones, supplements, fatty acids, or exercise.

Flow Chart: FQ-Induced CYP1A2 Suppression

See website: search “CYP1A2 Flowchart” for more detailed description