Letter to the Editor

Dear *Cutis*®:

I enjoyed Elston's article, "What Is Your Diagnosis? Cowden Disease (Multiple Hamartoma Syndrome)" (*Cutis*. 2006;78:28, 51-52). The biological function of the phosphatase and tensin homolog PTEN was noted to be a protein tyrosinase phosphatase tumor suppressor gene. Early studies on PTEN suggested that this protein functioned as a protein tyrosinase phosphatase; however, those studies were incorrect.[1]

Later studies showed that PTEN functions as a phosphoinositide lipid phosphatase. This protein counteracts the phosphoinositide-3 kinase pathway by changing phosphatidylinositol 3,4,5 to phosphatidylinositol 4,5. Thereby, PTEN downregulates Akt signaling in cells and shifts signaling toward apoptosis.[2]

Sincerely,
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The author reports no conflict of interest.

REFERENCES

1. Li J, Yen C, Liaw D, et al. PTEN, a putative protein tyrosine phosphatase gene mutated in human brain, breast, and prostate cancer. *Science*. 1997;275:1943-1947.
2. Stambolic V, Suzuki A, de la Pompa JL, et al. Negative regulation of PKB/Akt-dependent cell survival by the tumor suppressor PTEN. *Cell*. 1998;95:29-39.

Author Response

I appreciate the comments from Dr. Seykora clarifying the role of PTEN.

Sincerely,
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The author reports no conflict of interest.