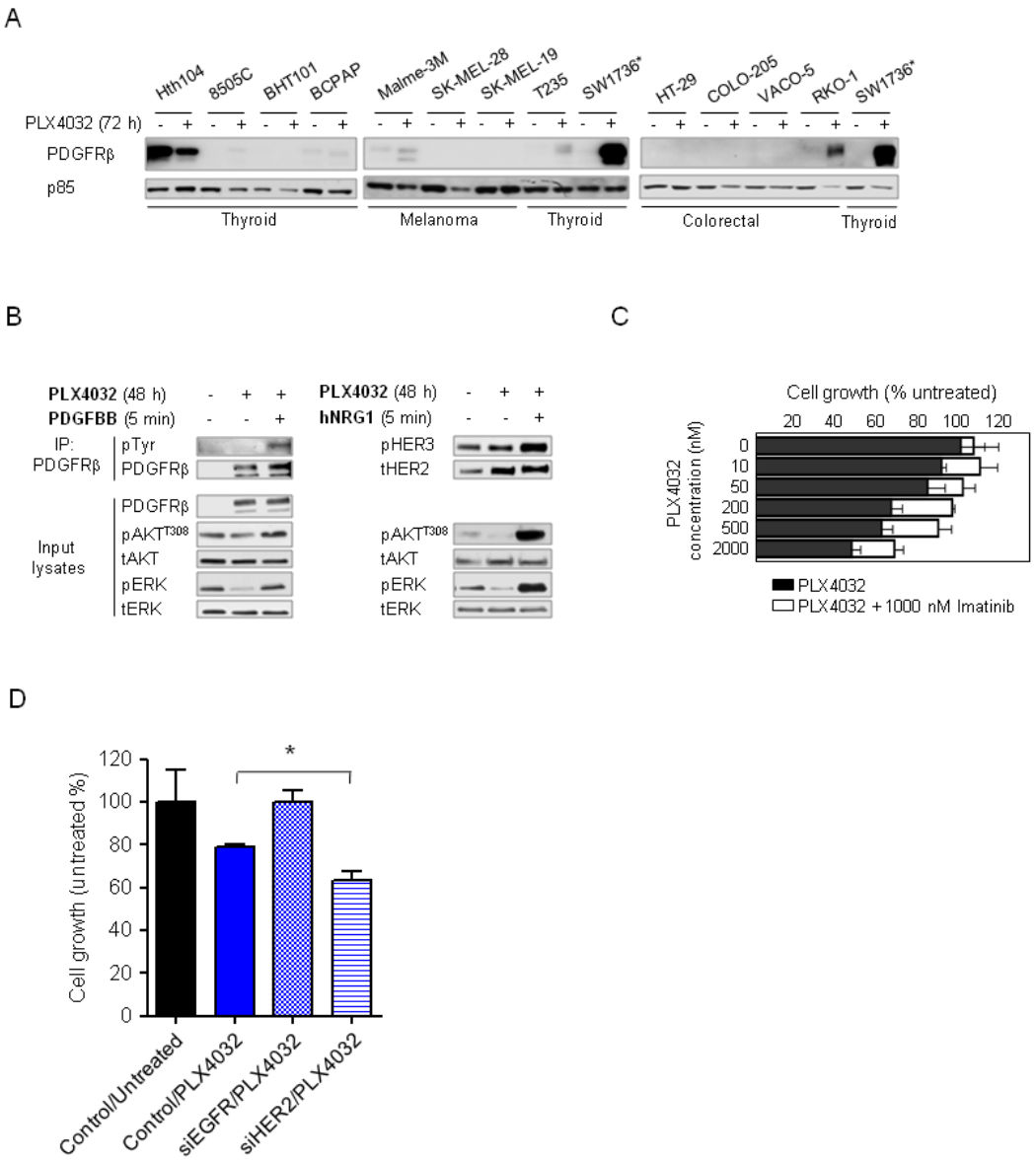


**Figure S7.** Role of PDGFRβ and EGFR in intrinsic resistance of thyroid cancer cells to RAF inhibitors. **A,** lysates of the indicated cell lines treated with or without 2 μM PLX4032 for 72 h were immunoblotted for PDGFRβ, and p85 (loading control). Lysates of SW1736 were used as an inter-blot control (*). PLX4032 induces expression of the receptor in SW1736 cells, whereas it is expressed constitutively in Hth104 cells. **B,** response of SW1736 cells to ligand induced activation of PDGFRβ or HER2/HER3 after exposure to PLX4032. Cells were treated with PLX4032 for 48 h, and then with or without 50 ng/ml

PDGFBB (left) or NRG1 (right) for 5 min. PDGFRβ phosphorylation was detected by IP followed by Western blotting with anti-pTyr. Lysates were immunoblotted with the indicated antibodies. Ligand induced HER3 activation induced more robust activation of signaling as compared to PDGFBB. **C,** SW1736 cells were treated with increasing concentrations of PLX4032 alone (grey) or in combination with 1 μM imatinib (open bars). Cells were collected and counted after 4 days of treatment. Bars represent percent change (mean +/- SD) in cell counts of triplicate wells compared to untreated cells. Imatinib did not sensitize cells to growth inhibition by vemurafenib. **D,** 8505C cells were transfected with control siRNA or siRNA to EGFR or HER2 (brown) for 16 h, and then treated with 2 μM PLX4032 for 3 days. Bars represent percent change (mean +/- SD) in cell counts of triplicate wells compared to untreated cells transfected with control siRNA. EGFR knockdown did not sensitize thyroid cells to growth inhibition by vemurafenib, whereas HER2 enhanced its effects. *$p<0.05$