SUPPLEMENTARY METHODS

Immunofluorescence

Cells were fixed, permeabilized and stained with the following primary antibodies: rabbit polyclonal anti-NR4A1/TR3 (Abnova, Taipei, Taiwan) (1:100 dilution), mouse monoclonal anti-Hsp60 (Santa Cruz Biotechnology, Santa Cruz, CA) (1:200 dilution), mouse monoclonal anti-cytochrome C (PharMingen, San Diego, CA) (1:100 dilution), rabbit polyclonal anti-Bcl-2 (Sigma Chemical Co.) (1:100 dilution), and mouse monoclonal anti-DDK (FLAG) (Origene, Rockville, MD). Binding of the TR3 primary antibody was detected with Alexa Fluor 488 anti-rabbit (Invitrogen Corp.) (1:200 dilution), while signal for Hsp60 and cytochrome C was detected with Alexa Fluor 594 anti-mouse (Invitrogen Corp.) (1:200 dilution). For assessment of TR3 subcellular localization, overlap of TR3 with mitochondrial Hsp60 and cytochrome C release, at least 100 cells were counted in 3 independent fields under high power (x40). GFP expression in clones transfected with ShRNA targeting TR3 or control, scrambled ShRNA was analyzed in the FITC channel.

Western Blotting

The following primary antibodies were used: rabbit monoclonal anti-TR3/nur77 (Epitomics, Inc., Burlingame, CA) (1:1000 dilution), rabbit polyclonal anti-Nurr1/NR4A2 (Epitomics, Inc.) (1:500 dilution), mouse monoclonal anti-NOR1/NR4A3 (Abnova) (1:100 dilution), rabbit polyclonal anti-PARP (Cell Signaling Technology, Beverly, MA) (1:1000 dilution), rabbit polyclonal anti-caspase-3 (Cell Signaling Technology) (1:1000 dilution), rabbit polyclonal anti-Bcl-2 (Sigma Chemical Co.) (1:500 dilution), rabbit polyclonal anti-phospho-JNK (Thr183/Tyr185) (Cell Signaling Technology) (1:5000 dilution), rabbit polyclonal anti-phospho-

Akt (Ser473) (Cell Signaling Technology) (1:5000 dilution), rabbit anti-phospho-serine (1:100 dilution) (Sigma Chemical Co.), and mouse monoclonal anti-β-Actin (Sigma Chemical Co.) (1:10000 dilution). We validated the efficiency of enrichment in subcellular fractionation experiments using mouse monoclonal anti-histone H3 (Cell Signaling Technology) (1:1000 dilution) and mouse monoclonal anti-α-tubulin (Sigma Chemical Co.) (1:1000 dilution) for the nuclear and cytoplasmic fractions, respectively.

Quantitative real time RT-PCR

Levels of mRNA expression for TR3 and NR4A2 were determined using Life Technologies (Grand Island, NY) TaqMan® gene expression assays Hs00374230_m1 and Hs00443062_g1, respectively, using the 7900HT real-time PCR machine (Applied Biosystems, Foster City, CA). Relative expression was measured compared to an internal GAPDH standard, TaqMan® gene expression assay Hs027589917_g1, and calculated using the $2^{-\Delta\Delta CT}$ method.

Case Selection and TMA generation

TMAs were generated in the Vanderbilt Translational Pathology Shared Resource. The REDCap® database available through the Vanderbilt Institute for Clinical and Translational Research was used to record patient demographic information, stage, grade, treatment history and outcomes. Progression-free survival was defined as the date of diagnosis to the first disease-free failure event (either recurrence as defined by RECIST criteria or date of death) or date of last contact. Overall survival was defined as date of diagnosis to either date of death or death of last contact. Platinum sensitivity was defined as a treatment-free interval of greater than 6

months after chemotherapy versus platinum resistance, as defined by a treatment-free interval of less than 6 months.

Immunohistochemistry

Immunostaining for the following primary antibodies was performed in the Vanderbilt Immunohistochemistry Core Facility: rabbit polyclonal anti-TR3/NR4A1 (Abnova, Taipei, Taiwan) (1:100 dilution), anti-pan-cytokeratin (Clone AE1/AE3, Dako, Carpinteria, CA) (1:500 dilution), anti-cleaved caspase-3 (Cell Signaling Technology) (1:300 dilution), or anti-mib-1/Ki67 (Vector Laboratories, Burlingame, CA) (1:1000 dilution). For assessment of cleaved caspase-3 and mib-1/Ki67 staining in ovarian xenografts, at least 100 cells were counted in 5 independent fields under high power (x40).