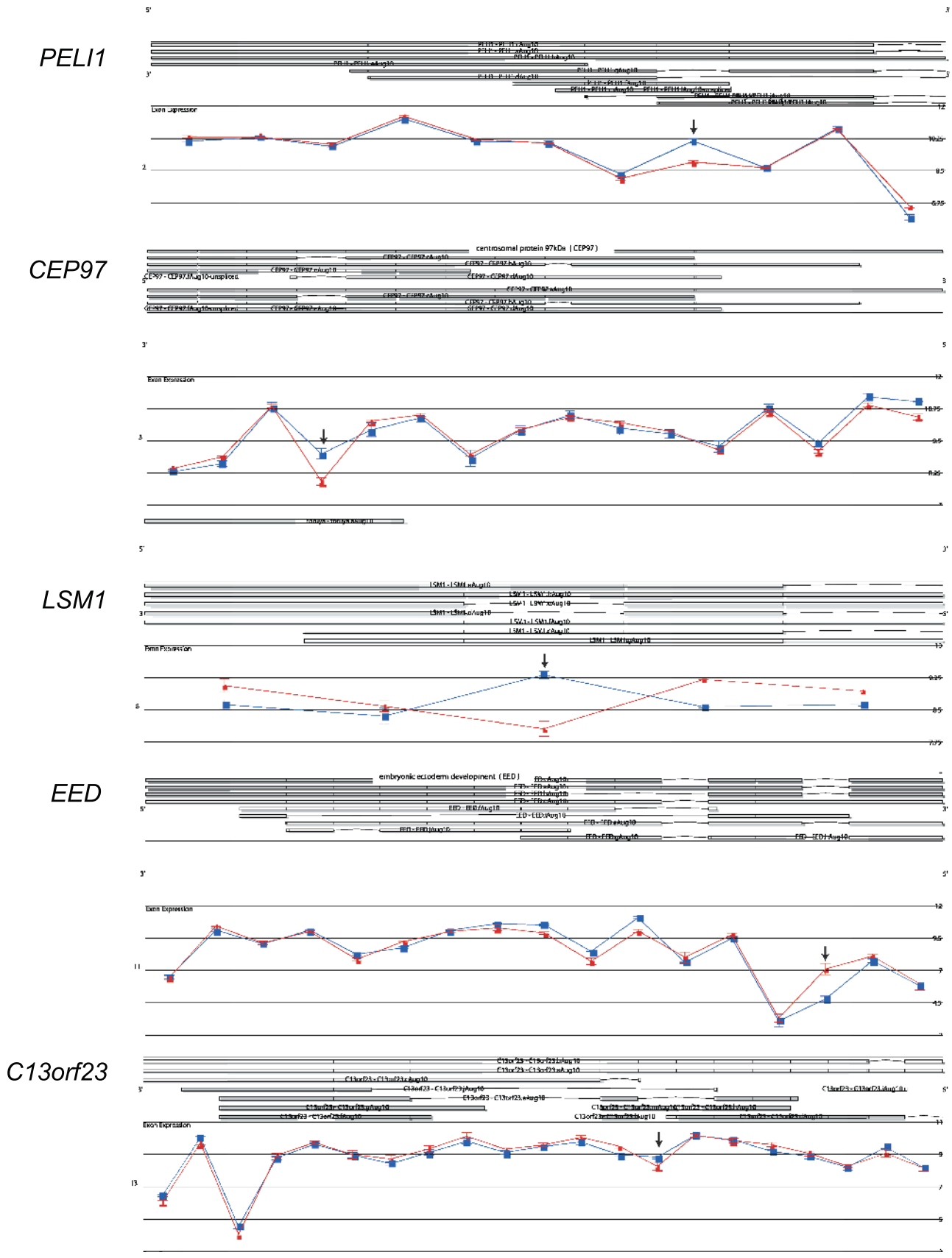

Figure W1. Partek representation of cisplatin-induced alternative splicing events. Splicing of the exons of selected genes (subsequently validated by PCR; Figure 6*C*) under normal and cisplatin-treated conditions is represented using plots generated by Partek genomics software on analysis of the exon microarray data. Each point represents the average expression level of the corresponding exon in each treatment group (three cisplatin *vs* three control samples). Representative transcripts for each gene are shown above the plots. Red represents cisplatin treatment, and blue represents normal conditions. The arrows indicate the alternatively spliced cassette exon.

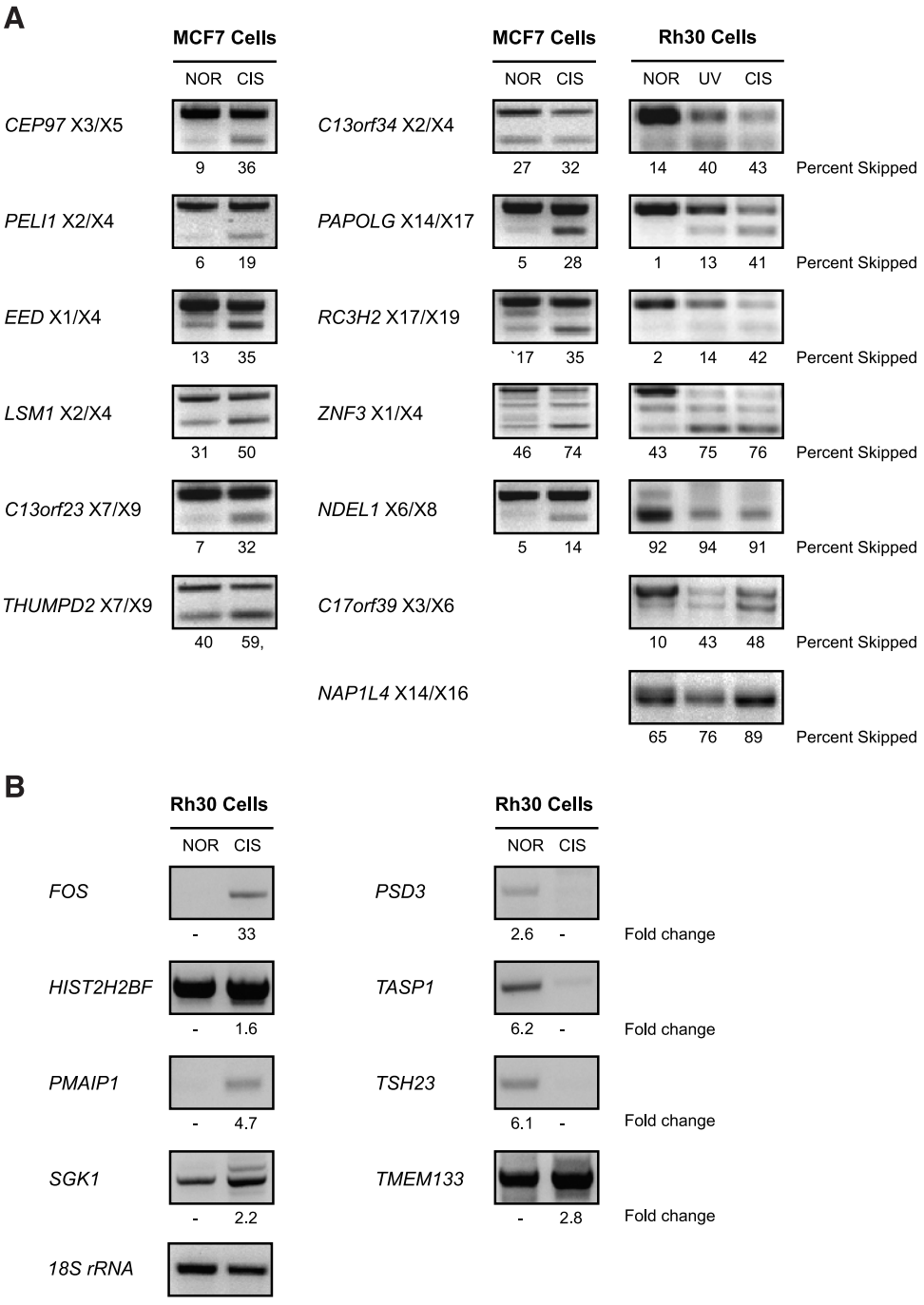

Figure W2. Validation of exon microarray targets in Rh30 and MCF7 cell lines. Rh30 and MCF7 cells were treated with 75 μM cisplatin or 50 J/m^2 UV as indicated for 24 hours, and total RNA was harvested. (A) Fifteen targets from the exon microarray that were predicted to show significantly altered splicing of cassette exons under stress were tested in these cell lines. Of the 15 targets, 12 were validated in Rh30 cells and 11 in MCF7 cells, and they recapitulated the splicing changes predicted by the exon array. The 11 targets validated in MCF7 cells and 7 of the 12 targets in Rh30 cells (the validation of the other 5 targets is shown in Figure 6) are shown here. (B) Eight targets that were predicted in the exon microarray to show significantly altered gene expression (increase or decrease) under cisplatin treatment were validated in Rh30 cells for differential expression between normal and cisplatin-treated conditions. Seven of the eight targets recapitulated the predicted expression changes (*TMEM133* being the exception).

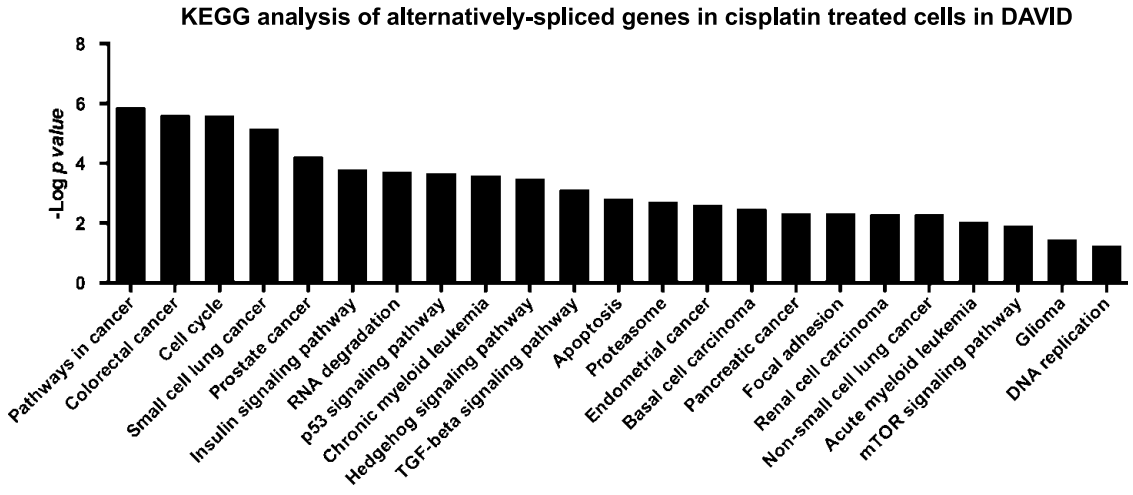

Figure W3. Pathway enrichment analysis of genotoxic stress–induced alternatively spliced genes reveals enrichment of genes involved cancer and related pathways. Alternatively spliced genes (alternative splicing $P < .05$ including FDR) were analyzed for ontology using DAVID bioinformatics tools. *Y* values indicate −log10 of the enrichment *P* values, denoting the magnitude of correlation between the alternatively spliced gene sets and established molecular pathways. While the genes showing alterations in splicing patterns under cisplatin treatment fell into a number of categories of biologic processes, we have represented here the most relevant and highly enriched pathways relating to cancer and associated cellular processes.

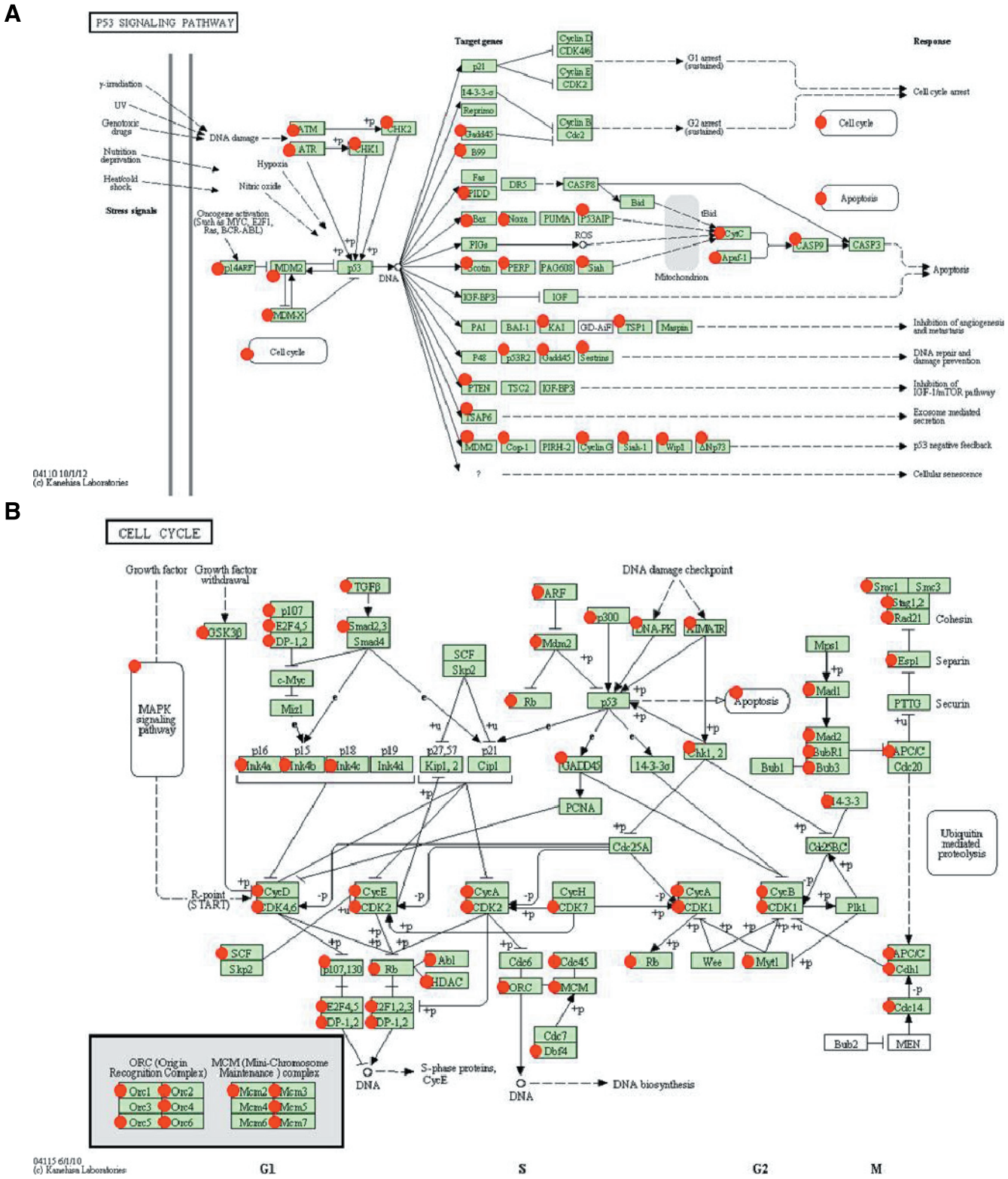

Figure W4. Enriched KEGG functional pathways of alternatively spliced genes under cisplatin treatment. KEGG functional pathway [31,32] analysis (part of the DAVID bioinformatics tools) was performed on the genes undergoing alternative splicing on cisplatin treatment (genes significantly differentially spliced are denoted by red circles): (A) p53 tumor suppressor pathway and (B) cell cycle control, two categories for which the stress-responsive alternative splicing events were highly enriched and which are relevant in the context of cancer.