Erratum

Focus on kidney cancer

In this Focus (Cancer Cell *6*, pp. 223–228), the legends for Figures 1 and 2 were reversed. They are reprinted below with the proper captions. Additionally, Figure 1 should be cited at the end of the second-to-last sentence of the section "Studies of dominantly inherited, epithelial forms of renal carcinoma," the citation at the end of the last paragraph of the section "VHL gene pathway: Opportunity for disease-specific approaches to therapy" should refer to Figure 2, not Figure 1, and the citation to Figure 2 at the end of the first paragraph of the section "Sporadic kidney cancer" should be disregarded.

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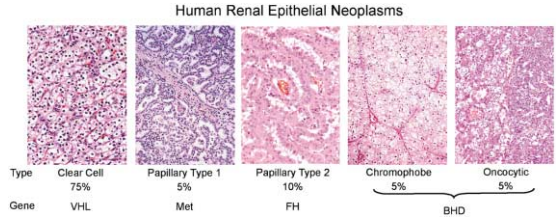

Figure 1. Kidney cancer is not a single disease, it is made up of a number of different types of cancer that occur in the kidney

These different types of kidney cancer are characterized by different histologies, have different clinical courses, respond differently to therapy, and are associated with alteration of different genes. Modified from Linehan et al. (2003).

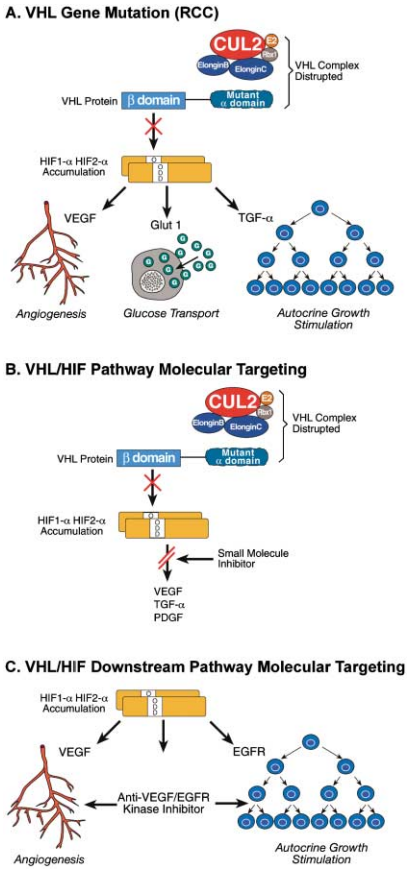

Figure 2. Molecular targeting of the VHL pathway in clear cell renal carcinoma

Mutation of the VHL gene in clear cell kidney cancer results in increased accumulation of HIF and the resulting increase in transcription of downstream targets such as VEGFR, EGFR, and TGFα (**A**). Mutation of the VHL gene in the α domain (shown here) inhibits binding to elongin C and formation of the VHL complex (Stebbins et al., 1999). Mutation in other parts of the gene, such as the β domain, prevents binding to and ubiquitin mediated degradation of HIF (Ohh et al., 2000). Potential disease-specific therapeutic approaches include agents which block the function of HIF (**B**), VEGFR, or EGFR (**C**). Modified from Linehan et al. (2002, 2003).