Correction

MEDICAL SCIENCES. For the article “Cyclin-dependent kinase 6 associates with the androgen receptor and enhances its transcriptional activity in prostate cancer cells,” by Jin T. E. Lim, Mahesh Mansukhani, and I. Bernard Weinstein, which appeared in issue 14, April 5, 2005, of Proc. Natl. Acad. Sci. USA (102, 5156–5161; first published March 24, 2005; 10.1073/pnas.0501203102), the authors note that in Figs. 2C and 3A, the light and dark blue colors are reversed in the panels on “Relative Luciferase Activity,” due to a printer’s error. The related description of these results remains unchanged, as these errors do not affect the conclusions of the article. The corrected figures and their legends appear below.

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Fig. 2. Effects of mutations in CDK6 and the AR on the association between the two proteins and activation of the PSA promoter. (A) CDK6 associates with the AR. 293T cells were transfected with HA-tagged WT or point-mutated CDK6 plasmids plus or minus the AR plasmid. Lanes: 1, HA K6WT; 2, HA K6R31C; 3, HA K6D146N; 4, HA K6R60A,E61A; 5, AR; 6, AR and HA K6WT; 7, AR and HA K6R31C; 8, AR and HA K6D146N; and 9, AR and HA K6R60A,E61AA. Whole-cell extracts were immunoprecipitated with the HA (Upper) or the AR (Lower) antibody and immunoblotted with the HA or AR antibody. (B) The effect of CDK6 point mutations on activation of the PSA promoter. PC3 cells were transfected with the PSA–luciferase reporter and CDK6 WT or the CDK6 mutant constructs used in Fig. 2A. As indicated, the cells were also transfected with the AR expression plasmid. Relative luciferase reporter activity was measured after the cells were grown in the absence or presence of DHT for 20 h. (C) The effects of CDK6 truncation mutants on activation of the PSA promoter. PC3 cells were transfected with the PSA–luciferase reporter and full-length CDK6 or the indicated CDK6 truncation constructs. The AR expression plasmid was also cotransfected as indicated. Relative luciferase activity was measured after the cells were grown in the absence or presence of DHT for 20 h. (Lower) Extracts were also examined for CDK6 expression by using the HA-specific IgG antibody. Lanes: 1, HA K6Δ121-326; 2, HA K6Δ1161–326; 3, HA K6Δ221–326; 4, HA K6Δ261–326; 5, HA K6WT; 6, HA Lac-Z; 7, HA, and 8, no DNA. *, P < 0.05.

Fig. 3. Effects of CDK6 on various mutations in the AR and with respect to activation of the PSA promoter. (A) The effects of AR deletion mutants on CDK6 activation of the PSA promoter. PC3 cells were transfected with the PSA–luciferase and β-gal plasmids. As indicated, the cells were also cotransfected with one of the AR deletion constructs (AR Δ37–494, AR Δ653–910, AR Δ557–653, or the full-length AR) and the full-length CDK6 expression plasmid. Relative luciferase activity was then measured after the cells were grown in the absence or presence of 10 nM DHT for 20 h. (B) The effects of CAG polymorphism in the AR on CDK6 activation of the PSA promoter. PC3 cells were transfected with the PSA–luciferase reporter and β-gal plasmids. As indicated, the cells were also cotransfected with an AR plasmid encoding 48 (AR75), 20 (AR), or no (AR 70) CAG repeats and CDK6. (C) The effects of an AR mutant found in human prostate cancer on CDK6 activation of the PSA promoter in the presence of various steroids. PC3 cells were transfected with the PSA–luciferase reporter and β-gal plasmids. As indicated, the cells were also cotransfected with WT AR or the AR mutant AR T877A and CDK6 and grown in the absence or presence of 10 nM cortisol, DHT, β-estradiol, flutamide, or progesterone. *, P < 0.05.