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instance, the knockdown of p21 in cancer cells can rescue them to proliferate under proteasome inhibition by bortezomib or MG132.^{^[@R2]^} Therefore, we further explored whether dephosphorylation of p21 played a role in the regulating P4H inhibition of cell cycle. The same Cdk inhibitors, including p21 and p27, were added to HCT116 cells in the presence of BCS or BCSN. The addition of Cdk inhibitors was able

to rescue the sub G0 or G0/G1 peaks or the cell cycle arrest induced by BCS or BCSN ([figure 5](#F5){ref-type="fig"}). These results indicate that dephosphorylation of p21 by PP2A or PP5 kinases is involved in P4H inhibition of cell cycle and cell death. ! [Dephosphorylation of p21 by PP2A or PP5 is involved in P4H inhibition of cell cycle and cell death. The inhibition of P4H by BCS or BCSN induced the

dephosphorylation of PP2A or PP5 substrates including p21, p27, and YAP1 in the absence of Cdk inhibitors. The phosphorylation of Cdk substrates was suppressed by the Cdk inhibitors, including p21 and p27. The addition of Cdk inhibitors could restore the normal cell cycle progression (A) and cell survival

(B).](rrtmj-4-0108-g005){#F5}

DISCUSSION {#S4} =====

The rapid proliferation of tumor

cells depends on the arrest of the cell cycle. Many antiproliferative pathways have been described in the context of cell cycle arrest.^{^[@R37],[@R38]^} Given the complex and tight regulation of cell cycle progression and apoptosis, the identification of novel regulators, and the confirmation of their critical role in the cell cycle are of great interest. Here, we demonstrated that P4H1 regulates the 3da54e8ca3

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