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CORRECTION

Correction: Phosphatase 2A and polo kinase, two antagonistic regulators of Cdc25 activation and MPF auto-amplification (*J. Cell Sci.* 112, 3747-3756)

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Journal of Cell Science was made aware by a reader of several issues concerning duplication of data in Fig. 3B,C and Fig. 5B,D in *J. Cell Sci.* (1999) **112**, 3747-3756.

The first author, Anthi Karaiskou, subsequently also contacted the journal to say that during figure compilation, some of the bands in western blots in the figures were duplicated during figure assembly. After further discussion, the journal referred this matter to Université Pierre et Marie Curie (UPMC, now Sorbonne Université), who investigated and cleared the authors of any wrongdoing. The UPMC committee decided that the conclusions of the paper were not affected by the errors and recommended correction of the paper (full reports available at: http://www2.cnrs.fr/sites/communique/fichier/rapport_conclusions.pdf and http://www2.cnrs.fr/sites/communique/fichier/rapport_analyse_detaillee.pdf).

The editorial policies of Journal of Cell Science state that: "Should an error appear in a published article that affects scientific meaning or author credibility but does not affect the overall results and conclusions of the paper, our policy is to publish a Correction…" and that a Retraction should be published when "…a published paper contain[s] one or more significant errors or inaccuracies that change the overall results and conclusions of the paper…". Journal of Cell Science follows the guidelines of the Committee on Publication Ethics (COPE), which state: "Retraction should usually be reserved for publications that are so seriously flawed (for whatever reason) that their findings or conclusions should not be relied upon". The standards of figure assembly and data presentation in this paper fall short of current good scientific practice. However, given that the investigating committee at UPMC declared that the conclusions of the paper were not affected by the errors, the appropriate course of action – according to COPE guidelines – is to publish a Correction, which the journal has made as detailed as possible.

Readers should note that the policy of the UPMC is that authors should retain original data for 10 years and that this paper falls outside this period.

Although the authors were unable to locate all the original data, they did produce replicates of experiments carried out at the same time showing similar results for most blots; the authors have therefore assembled new figure panels. However, they were unable to find the original data for the pTyr Cdc2 blot in Fig. 3B or Plkk1 blot in Fig. 3C and so have requested that these blots be disregarded. The authors state that as Cdc2 kinase activity is also shown in Fig. 3A, the conclusions are not affected by removal of the pTyr Cdc2 blot; the authors also state that the Plkk1 panel in Fig. 3C does not bring any input to the scientific conclusion.

The new panels for Figs 3 and 5 and the corrected legends are shown.

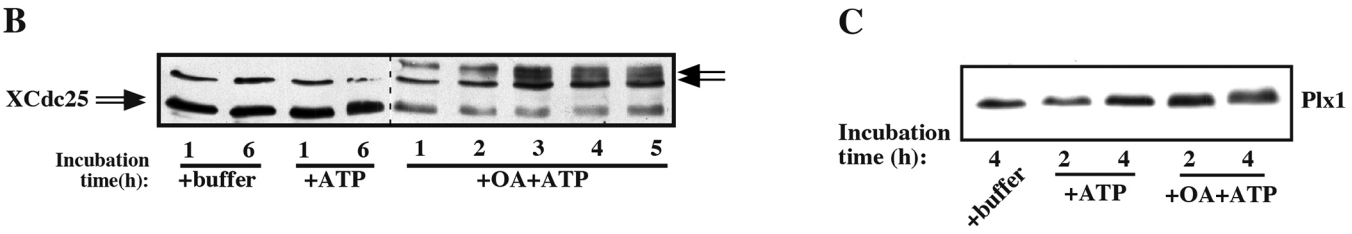

Fig. 3. Cdc2 kinase activation in the F40. (B) The F40 was incubated in the absence of effectors (+buffer), or in the presence of an ATP-regenerating system (+ATP), or 500 nM okadaic acid and an ATP-regenerating system (+OA+ATP). After incubation for the times indicated (hours), samples were analyzed by western blotting using anti-*Xenopus* Cdc25 antibody. (C) The F40 was incubated in the absence of effectors (+buffer), or in the presence of an ATP-regenerating system (+ATP), or 500 nM okadaic acid and an ATP-regenerating system (+OA+ATP). Samples were collected at various times, as indicated, and analyzed by western blotting using an anti-*Xenopus* Plx1 antibody.

As a result of corrections to these figures, readers of J. Cell Sci. **112**, 3747-3756 should ignore text related to the blot removed from Fig. 3B on p. 3750 (second column): 'This linear activation of Cdc2 kinase is correlated with a slight and progressive tyrosine dephosphorylation (Fig. 3B).' Also, p. 3750 (bottom of second column) should now read: 'After addition of ATP or ATP together with okadaic acid in the F40, Plx1 kinase is activated in parallel with Cdc2 kinase, as judged by its electrophoretic mobility and Plx1 kinase assay (Fig. 3C,D).'

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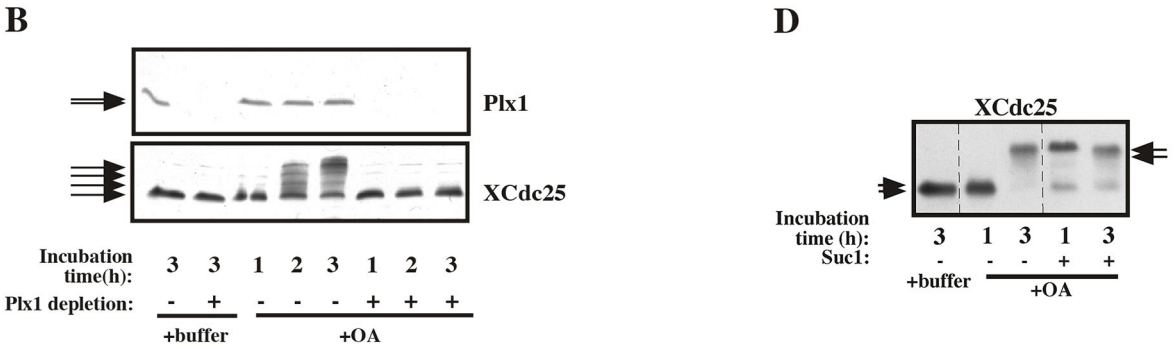

Fig. 5. Plx1 kinase and Suc1/Cks mediated Cdc2/Cdc25 association are necessary for the Cdc2/Cdc25 positive feed-back loop. (B) The mock-depleted or Plx1-depleted F40 was incubated in the absence of effectors (+buffer) or in the presence of 500 nM okadaic acid and an ATP-regenerating system (+OA). Samples were collected at various incubation times, as indicated, and immunoblotted with the anti-Plx1 antibody (upper panel) and the anti-Cdc25 antibody (lower panel). (D) The F40 was incubated in the presence of an ATP-regenerating system, supplemented with buffer, with 500nM okadaic acid (+OA) or with okadaic acid and 40 µg/ml p13Suc1 protein (Suc1). Samples were collected at various incubation times and submitted to western blotting with the anti-XCdc25 antibody. Arrows indicate the various migrating forms of XCdc25.

Readers should also note that there may be unmarked splicing of lanes from western blots in this paper, which although not acceptable by today's standards was prevalent in 1999 when the paper was published.

The authors apologise to the journal and readers for these errors.

Journal of Cell Science refers readers to other notices related to the UPMC investigation:

doi:10.1242/jcs.166553

doi:10.1242/jcs.222182

doi:10.1242/jcs.222240

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