products with CTD function, which was previously implicated in such responses.

The RNA polymerase II CTD is found in an unphosphorylated form in transcription initiation complexes but is extensively phosphorylated during elongation[11,23]. Thus, CTD phosphorylation may regulate some event in transcription initiation. We tested whether SRB10/11 kinase has a role in CTD phosphorylation in the holoenzyme using purified RNA polymerase II holoenzyme from wild-type and *srb10* mutant cells. Figure 4 shows that CTD phosphorylation was reduced ~10-fold in the *srb10* mutant holoenzyme, indicating that the SRB10 kinase must be important for CTD phosphorylation; the reduced response of SRB10-deficient holoenzyme to galactose induction *in vivo* may therefore reflect its diminished ability to phosphorylate the CTD. The yeast general transcription factor TFIIH is present in the holoenzyme[1], and the TFIIH-associated kinase[24,25] may account for the residual CTD phosphorylation in the mutant holoenzyme.

The activities of wild-type and SRB10-mutant RNA polymerase II holoenzymes were compared in a reconstituted transcription assay (Fig. 4*c*); levels of basal and GAL4–VP16-activated transcription were similar for both holoenzymes. We could not find any defect in transcription *in vitro* comparable to the loss of CTD phosphorylation *in vitro* or of galactose induction *in vivo*. These results suggest that factors necessary to elicit the regulatory role of SRB10 are missing or not functional in our *in vitro* transcription systems. Alternatively, the holoenzymes may contain additional kinases that compensate for the loss of SRB10 function in these systems.

We have identified a kinase-cyclin pair in the RNA polymerase II holoenzyme, shown that it is involved in transcriptional regulation *in vivo* and in CTD phosphorylation *in vitro*. Although the exact role of CTD phosphorylation in transcriptional regulation is not known, our results demonstrate that the response of the transcription initiation apparatus to at least some regulatory signals *in vivo* involves the SRB kinase–cyclin pair. □

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ERRATA

Dimercaptan–polyaniline composite electrodes for lithium batteries with high energy density

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FIGURES 3 and 4 were accidentally transposed in this Letter. □

Incorporation of subgenomic amounts of DNA as compensation for mutational load in a gynogenetic fish

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IN Fig. 2*A* of this Letter, the designation of lanes above the gel was incomplete. The correct labelling is shown here. For example, lane 11 contains DNA from *Poecilia mexicana*, which was used as host species to produce the offspring, and not DNA from an offspring, as would have been inferred from the incomplete labelling. □