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CORRECTION

Distinct roles of Polycomb group gene products in transcriptionally repressed and active domains of *Hoxb8*

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There were errors published in *Development* **133**, 2371-2381.

The authors have become aware of errors with the display of data in Fig. 4E, Fig. 5A and Fig. 6. These errors are detailed below, and the authors have also provided the original data for these figures, which are included as part of the correction.

In Fig. 4E, the Rnf2 and trimH3-K27 lanes were spliced together without marking, removing intervening lanes from the original gel. In preparing the figure, the *Hoxb6* Rnf2 lanes were duplicated as *Hoxb8* Rnf2. In addition, contrast adjustments were applied to the original image that obscured weak bands in the *Rnf2*$^{-/-}$ lanes that we assume are the result of non-specific binding of chromatin to the beads during the immunoprecipitation procedure. Finally, the control lanes are wrongly labelled as *Hprt*; the negative control used for this experiment was in fact *Adam34*. These errors are corrected in the revised Fig. 4 and legend shown below, and the original data for this experiment are provided as 'Fig. 4E original data'.

Fig. 4 (revised)

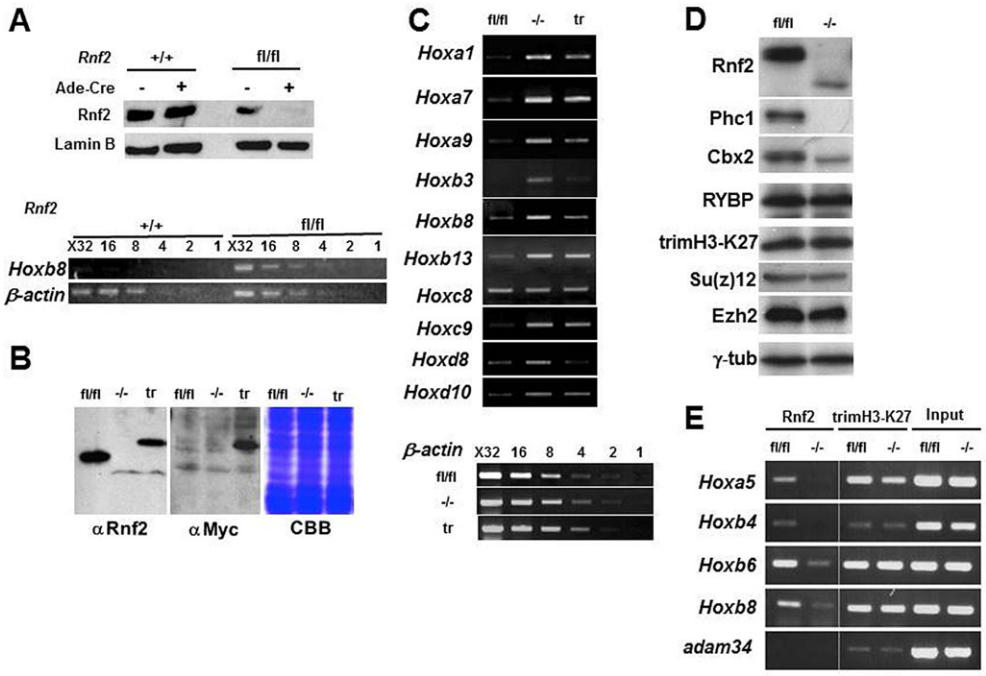

Fig. 4. De-repression of Hox genes in *Rnf2*$^{-/-}$ MEFs and ES cells. (**A**) Conditional depletion of Rnf2 leads to de-repression of *Hoxb8* in MEFs derived from the cranial part of *Rnf2*$^{fl/fl}$ 9.5 dpc embryos. (Top) Infection of Cre-expressing adenovirus vector to MEFs derived from *Rnf2*$^{fl/fl}$ embryos (fl/fl) depleted the Rnf2 gene products, whereas the wild-type (+/+) MEFs were unaffected. Lamin B was used as a control. (Bottom) The expression of Hoxb8 was induced by infection of Cre-expressing adenovirus vector in *Rnf2*$^{fl/fl}$ MEFs (fl/fl), but not in the wild type (+/+). (**B**) *Rnf2*$^{-/-}$ (–/–) ES cells were derived from *Rnf2*$^{fl/fl}$ (fl/fl) ES cells by transient overexpression of Cre-recombinase. Rnf2 was re-expressed by transfecting *Rnf2*$^{-/-}$ ES cells with a construct expressing Myc-tagged Rnf2 (tr). The expression of endogenous and transfected Rnf2 was examined by using anti-Rnf2 (left) and -Myc (middle) antibodies. CBB staining was used as a loading control (right). (**C**) The expression of Hox cluster genes in *Rnf2*$^{fl/fl}$ (fl/fl), *Rnf2*$^{-/-}$ (–/–) and Rnf2 transfected (tr) ES cells was compared by RT-PCR. The quantity of synthesized cDNA from respective cells was equalized by comparing the relative amounts of β-actin transcripts. (**D**) The expression of Phc1 and Cbx2 gene products was reduced in *Rnf2*$^{-/-}$ ES cells (–/–) in comparison with the wild type (fl/fl), whereas the expression of RYBP (another Rnf2-binding protein that is not found in hPRC-H complex or class 1 PcG proteins) was not altered. (**E**) Rnf2 association and H3-K27 trimethylation at Hox promoter regions were compared between *Rnf2*$^{fl/fl}$ and *Rnf2*$^{-/-}$ ES cells. For the 'Input', genomic DNA extracted from the original whole cell lysate equivalent to the 1/40 volume of that used for the ChIP analysis was subjected to the PCR. *Adam34* was used as a negative control. White lines indicate the removal of intervening gel lanes.

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In Fig. 5A, for all samples except *Hoxa4*, the order in which the lanes are shown in the figure does not represent the order in which they were run on the original gel, where samples were run in the order: Suz12, Eed3, Rnf2, trimH3-K27, Input. For *Hoxb1* and *Hoxb4*, the lanes were exchanged in the figure but this splicing was not appropriately marked. For *Hoxb6* and *Hoxb8*, we failed to exchange these lanes and therefore the labelling was incorrect in the published figure. In addition, the control lanes were wrongly labelled as *Hprt*; the negative control used was *Adam34*. These errors are corrected in the revised Fig. 5 and legend shown below, and the original data for this experiment are provided as 'Fig. 5A original data'.

Fig. 5 (revised)

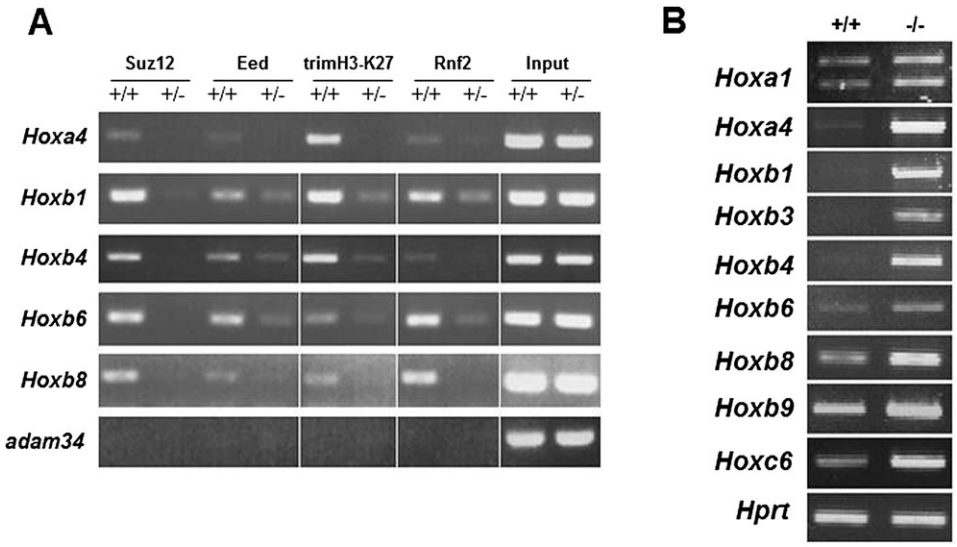

Fig. 5. De-repression of Hox genes in *Suz12*$^{-/-}$ ES cells correlates with reduction of Rnf2 association to Hox genomic regions. (**A**) The association of Suz12, Eed, Rnf2 and H3-K27 trimethylation at the Hox promoter regions in the wild-type and *Suz12*$^{-/-}$ ES cells. Whole-cell lysates prepared from approximately the same number of wild type (+/+) and *Suz12*$^{-/-}$ (–/–) ES cells were subjected to ChIP analyses using anti-Suz12, -Eed, -trimethylated H3-K27 and -Rnf2 antibodies. For the 'Input', genomic DNA extracted from the original whole cell lysate equivalent to the 1/40 volume of that used for the ChIP analysis was subjected to the PCR. *Adam34* was used as a control. (**B**) The expression of Hox cluster genes in the wild type (+/+) and *Suz12*$^{-/-}$ (–/–) ES cells was compared by RT-PCR. *Hprt* was used as a control. White lines indicate the removal of intervening gel lanes or their exchange.

In Fig. 6A, the original experiment included samples taken from *Rnf110*$^{+/-}$; *Bmi1*$^{-/-}$ animals. These were removed from the published version, but this splicing was not marked. In Fig. 6B, the original experiment included samples taken from *Phc1*$^{-/-}$; *Phc2*$^{+/-}$. We chose to show only data from the double heterozygote and double homozygote mutant, but in the case of *Hoxb8* accidentally removed the *Phc1*$^{+/-}$; *Phc2*$^{+/-}$ lanes instead of *Phc1*$^{-/-}$; *Phc2*$^{+/-}$. As can be seen in the original data, the results are highly similar for both genotypes. These errors are corrected in the revised Fig. 6 and legend shown below, and the original data for these experiments are provided as 'Fig. 6 original data'.

Fig. 6 (revised)

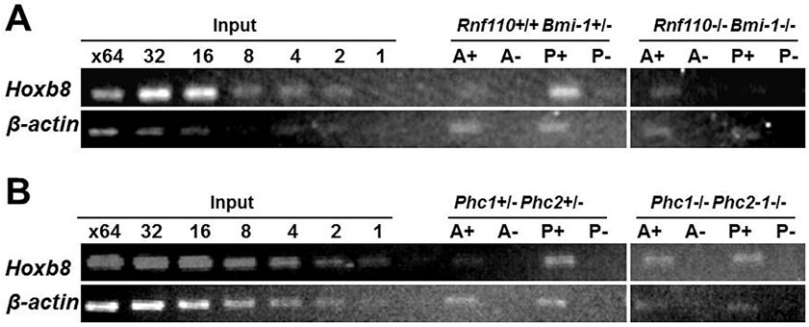

Fig. 6. Decreased H3-K9 acetylation at the first exonic region of *Hoxb8* in the posterior tissues of *Rnf110*$^{-/-}$*Bmi1*$^{-/-}$ and *Phc1*$^{-/-}$*Phc2*$^{-/-}$ embryos at 9.5 dpc. (**A**) Degree of H3-K9 acetylation in the anterior (A) and posterior (P) regions were compared in *Rnf110*$^{+/-}$*Bmi1*$^{+/-}$ and *Rnf110*$^{-/-}$*Bmi1*$^{-/-}$ embryos. The β-actin promoter was used as a positive control. (**B**) Degree of H3-K9 acetylation in the anterior (A) and posterior (P) regions were compared in *Phc1*$^{-/-}$*Phc2*$^{+/-}$, *Phc1*$^{+/-}$*Phc2*$^{-/-}$ and *Phc1*$^{-/-}$*Phc2*$^{-/-}$ embryos. The β-actin promoter was used as a positive control. In this study, the negative control ChIPs (A– and P–) were performed with rabbit IgG. White lines indicate the removal of intervening gel lanes.

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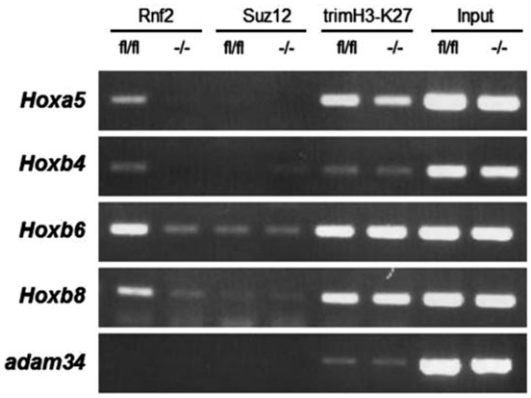
The authors apologise to readers for these errors. The editors of the journal and the authors' Research Integrity Office have examined this paper in detail and find that, although there were substantive errors in data presentation, these do not affect the conclusions of the paper.

Fig. 4E original data

Without lane splicing:



From laboratory notebook:

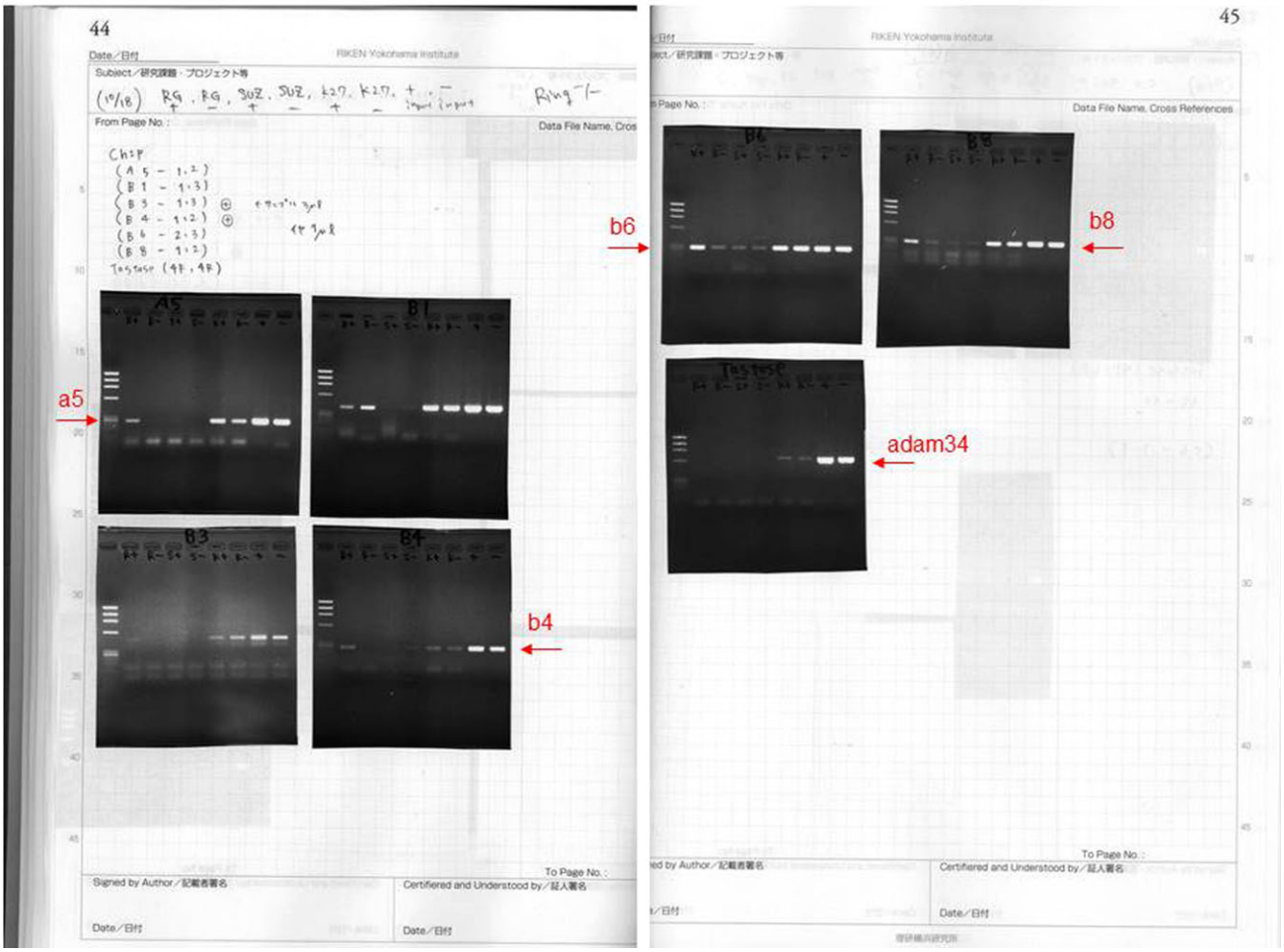


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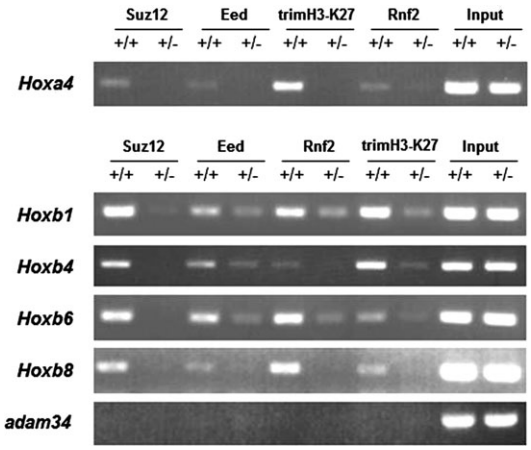
Fig. 5A original data

Without lane splicing:



From laboratory notebook:

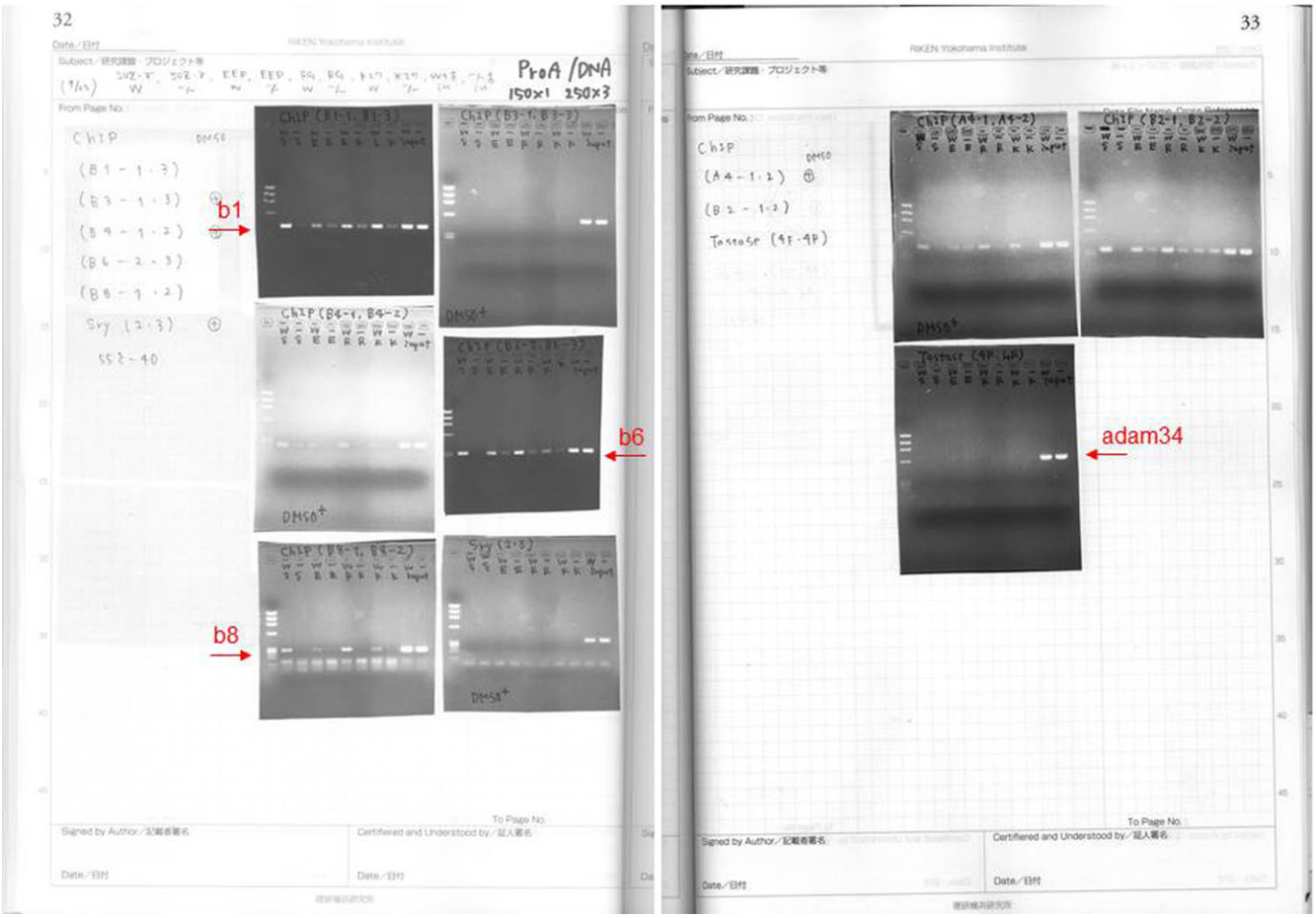


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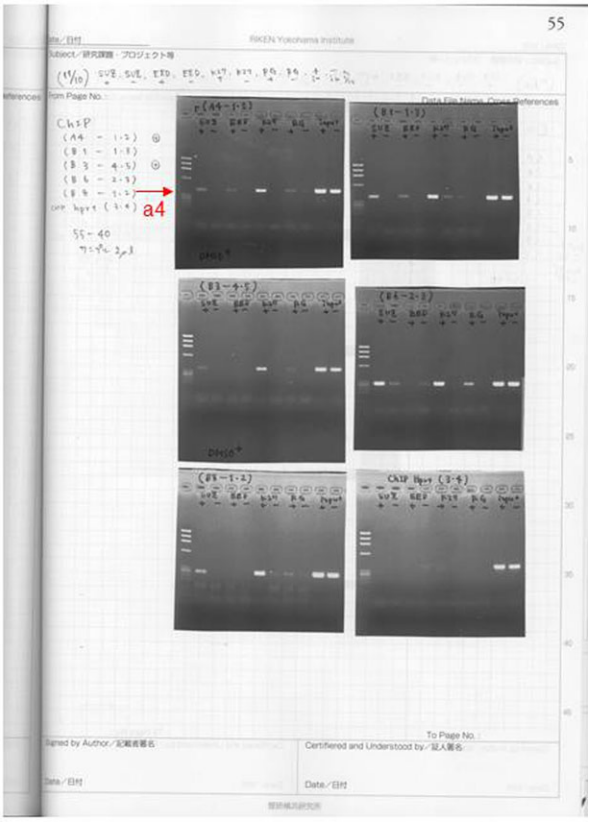
Fig. 5A original data (cont.)



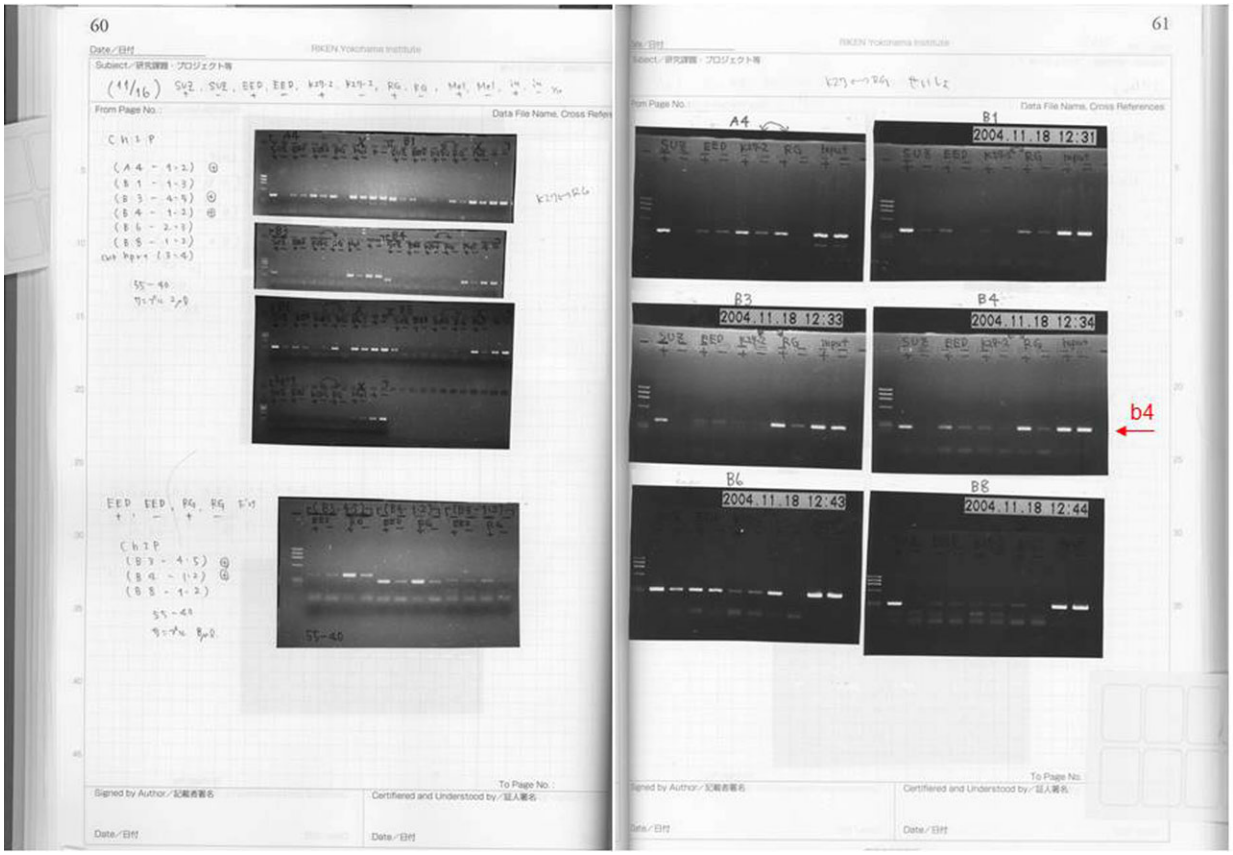


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Fig. 6 original data

Without lane splicing: