Staufen1 localizes to the mitotic spindle and controls the localization of RNA populations to the spindle

Sami Hassine, Florence Bonnet-Magnaval, Louis Philip Benoit Bouvrette, Bellastrid Doran, Mehdi Ghram, Mathieu Bouthillette, Eric Lecuyer and Luc DesGroseillers
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Original submission

First decision letter

MS ID#: JOCES/2020/247155

MS TITLE: Staufen1 localizes to the mitotic spindle and controls the transport of RNA populations to the spindle

AUTHORS: Sami Hassine, FLorence Bonnet-Magnaval, Louis-Philip Benoit-Bouvrette, Bellastrid Doran, Mehdi Ghram, Mathieu Bouthillette, Eric Lecuyer, and Luc DesGroseillers
ARTICLE TYPE: Research Article

We have now reached a decision on the above manuscript.

To see the reviewers' reports and a copy of this decision letter, please go to: https://submit-jcs.biologists.org and click on the 'Manuscripts with Decisions' queue in the Author Area. (Corresponding author only has access to reviews.)

I have now received comments on your manuscript from three experts. As you will see, all thought that the work was potentially quite interesting and significant but all also raised a number of concerns that must be dealt with before the manuscript can be reconsidered. Please address these issues as thoroughly as possible. I consider that no further experiments are required. Rather, you should tone down some of your conclusions and/or discuss the limitations of your results taking into account the reviewers' comments.

We are aware that you may currently be unable to access the lab to undertake experimental revisions. If it would be helpful, we encourage you to contact us to discuss your revision in greater detail. Please send us a point-by-point response indicating where you are able to address concerns raised (either experimentally or by changes to the text) and where you will not be able to do so within the normal timeframe of a revision. We will then provide further guidance. Please also note that we are happy to extend revision timeframes as necessary.

Please ensure that you clearly highlight all changes made in the revised manuscript. Please avoid using 'Tracked changes' in Word files as these are lost in PDF conversion.

I should be grateful if you would also provide a point-by-point response detailing how you have dealt with the points raised by the reviewers in the 'Response to Reviewers' box. Please attend to all of the reviewers' comments. If you do not agree with any of their criticisms or suggestions please explain clearly why this is so.

Reviewer 1

Advance summary and potential significance to field

In this paper, the authors present very convincing data supporting that STAU1 but not STAU2, is localized to mitotic spindle. In addition, the authors identified cellular transcripts enriched in mitotic spindle. They also observed that ribosome and OP-puromycin are enriched in mitotic spindle, suggesting that a subset of mRNAs are translated around mitotic spindle.

Comments for the author

Overall, the manuscript is well written and organized. The authors proposes a novel and intriguing notion for a translation associated with mitotic spindle.
Therefore, I have a few comments as listed below.

Specific comments:
1. Page 6, line 13: What does “OP-puromycin” stand for?
2. Supplementary Figure S1B: Authors should consider moving this figure to Supplementary Figure S3, because S1B supports proper purification of mitotic spindle.
3. Page 8, line 1: I agree that STAU2 is not enriched in mitotic spindle. But in case of STAU1-63, the input amount of STAU1-63 is relatively low, compared with STAU1-55.
4. Supplementary Figure S1B or S1D: I strongly recommend that authors present a whole gel image including a low molecular weight. In many cases for gene deletion using a CRISPR/Cas9 system, a truncated form is often detected.
5. Figure 4B: In the WT lane, many truncated form of STAU1 were detected. Are those simple degradation products during purification?
6. Figure 7C: OP-Pur corresponds to a truncated polypeptide and potentially misfolded polypeptide. These misfolded OP-Pur polypeptides are known to be targeted toward aggresome (Lelouard et al., 2004, J. Cell. Biol.; Park et al., 2017; Nat. Comm.), which overlaps with the microtubule organizing center.
Therefore, the authors’ assumption that the localization of OP-Pur corresponds to active translation site could be misleading. Instead, the OP-Pur synthesized in the cytosol may be transported to aggresome. Therefore, the authors’ conclusion in the Result section and Discussion section should be toned down.

Reviewer 2

Advance summary and potential significance to field

With their manuscript the authors dig deeper into the question of how and why RNAs are linked to the mitotic spindle; a phenomenon often described but poorly understood. They identify a specific isoform of the RNA binding protein Staufen1 to be important to enrich a set of RNAs, including mRNAs, at the spindle.
Interestingly, only the shorter isoform of Stau1 (Stau_55) is found on spindles; the longer Stau1_63 isoform or the paralog Staufen2 are missing. The authors further identify a short stretch of 11 amino acids, present in both Stau1 isoforms, that is crucial for the localisation of Stau1_55 but not Stau1_63 to mitotic spindles. Finally, data are presented to illustrate that spindle localised Stau1 overlaps with sites of active translation, indicating that fraction of Stau1_55 bound mRNAs could be translated on spindles.
The manuscript presents an important advance in understanding what recruits some RNAs to the mitotic spindle. Especially the validation of used antibodies is highly appreciated. Less clear is what is the importance of Stau1 during mitosis and the Stau1-mediated RNA recruitment to the spindle.

Overall, I do think the work is important and interesting, but some major points would need to be addressed.

Comments for the author

Major points

1) The title of the manuscript uses the word 'transport' which implicates active movements e.g. motor protein driven transport. After reading the manuscript, 'localisation' would certainly fit better as there is no data on active transport processes.
2) Given that all cell lines are at hand, the manuscript would greatly benefit from some characterisation of what Stau1 removal does to mitosis. Is it slowed down? Does spindle morphology change? Is there a higher fraction of defective spindles? This data should be straight forward to collect and would greatly support the relevance of the discovered Stau1_55 spindle-association.
3) P.6 line 1: A reference is given to unpublished work. As this work is not listed in the references, it's difficult to check whether it is on a preprint server. If this is not the case, the entire statement should be removed. Also, the statement based on this unpublished work is not clear; on one hand, there is a referenced statement in the introduction that Stau1 levels decrease during mitosis. But then the unpublished observation shows that Stau1 depletion impairs mitosis? How does this fit together?
4) What does the Stau1_55 N-terminus do? Can it directly bind microtubules?
Or does it bind to other microtubule-binding proteins which then cause the observed spindle binding? While the latter question might be too work intense to address within this work, it would be great to see, if the Stau1_55 N-terminus binds microtubules (and the Stau1_63 n-terminus not). Tubulin can be bought commercially, and fragments of bother termini should be straight forward to purify. A simple microtubule co-pelleting assay would then shed light on the nature of the interaction (indirect or direct?).
5) The author concludes that because the fluorescence of Stau1, tubulin and OP-Pur show some overlap, some Stau1-bound mRNAs on spindles might be locally translated. I think this is an extremely loose correlation. Unless higher resolution imaging data or data from translation sensors is presented this statement is superfluous and should be removed.
6) There is data on the Stau1_55 N-terminus discussed in the discussion that is found nowhere in the results part. (p.17, line 5). Either this reference to unpublished data should be removed or put into the results section together with the data.
7) There are more incidents of references to unpublished data in the discussion that should be removed unless the data is shown.

Reviewer 3

Advance summary and potential significance to field

In this manuscript Hassine and coworkers investigated the role of STAU1 in controlling localization of RNAs during mitosis of colorectal cancer cells. They report that a large fraction of STAU1 localizes in the spindles during mitosis which dependents on the first 88 N-terminal amino acids of the protein. They identified that a group of RNAs are enriched in spindle fractions and part of them are delocalized from the spindles when STAU1 is knockout. Finally, they showed that STOU1 could control the local translation of a subpopulation of its bound mRNAs in the spindles. Overall, this manuscript presents interesting data which should be of interest to the readers of the journal. The study is well designed and the data are appropriately interpreted. However, although the results include novel and interesting findings, several major concerns are remained.

Comments for the author

Main comments:
1. It is confusion for the function of STOU1 on cell proliferation. In the Instruction (line 4 of Page 5), the authors mentioned that "STAU1 is crucial for cell differentiation…and cell proliferation". While in the Result section, they generated STOU1-KO cell strain and concluded that "The growth rate of STAU1-KO

(clone CR1.3) HCT116 cells was similar to that of WT cells" (line 15 of Page 8).
2. It seems that STAU1 plays a role in spindle procession during mitosis through controlling localization and translation of different sub-types of pre-rRNAs and mRNAs. However, the physiological impact of this role on cell differentiation and growth has not addressed through their study. Without this, the significance of the study is weakened.
3. Line 7 of page 13, the authors mentioned that "we studied several RNAs whose amounts were decreased in spindle preparations of STAU1-KO cells compared to that of WT cells and are known targets of STAU1 binding". Does it mean that localization of the RNAs to the spindle is not solely dependent on STOU1? What is the cellular consequence of partly delocalizing these RNAs by STOU1 deletion or mutation?
4. In Fig 7;Using confocal microscopy, the authors analyzed the signals of O-propargyl-puromycin marker along with those generated by anti-tubulin and anti-STAU1 antibodies, they suggested that a subpopulation of STAU1-bound mRNAs is locally translated on the spindle. However, this conclusion may lack the support of biochemistry assays. Sucrose gradient assays of the cell spindle preparation should be performed to show whether STAU1 and its bound mRNAs are co-located in the polysomal fractions.

First revision

Author response to reviewers' comments

Reviewer 1 Advance Summary and Potential Significance to Field: In this paper, the authors present very convincing data supporting that STAU1, but not STAU2, is localized to mitotic spindle. In addition, the authors identified cellular transcripts enriched in mitotic spindle. They also observed that ribosome and OP- puromycin are enriched in mitotic spindle, suggesting that a subset of mRNAs are translated around mitotic spindle.

Reviewer 1 Comments for the Author:
Overall, the manuscript is well written and organized. The authors proposes a novel and intriguing notion for a translation associated with mitotic spindle. Therefore, I have a few comments as listed below.

Specific comments:
1. Page 6, line 13: What does "OP-puromycin" stand for?
We added this information in the text.

2. Supplementary Figure S1B: Authors should consider moving this figure to Supplementary Figure S3, because S1B supports proper purification of mitotic spindle.
We moved S1B to S3 as suggested.

3. Page 8, line 1: I agree that STAU2 is not enriched in mitotic spindle. But, in case of STAU1-63, the input amount of STAU1-63 is relatively low, compared with STAU1-55.
We agree that the amount of STAU-63 is always lower than that of STAU-55. However, we generated many gels where the amount of STAU in the spindle preparation is higher than that in inputs (for example, see old figure S1B – now S3C). Even then, STAU-63 was not present on spindle (while present in inputs). We are thus confident with the absence of STAU63 on spindles.

4. Supplementary Figure S1B or S1D: I strongly recommend that authors present a whole gel image including a low molecular weight. In many cases for gene deletion using a CRISPR/Cas9 system, a truncated form is often detected.
This referee probably means S4B,D. The guide RNA used to generate STAU1-CRISPR clones is located 34 amino acids downstream the first AUG codon. We never observed truncated fragments. We added a whole gel image of several clones to confirm (S4B).

5. Figure 4B: In the WT lane, many truncated form of STAU1 were detected. Are those simple degradation products during purification?

Yes. Purification is difficult and generates degradation products.

6. Figure 7C: OP-Pur corresponds to a truncated polypeptide and potentially misfolded polypeptide. These misfolded OP-Pur polypeptides are known to be targeted toward aggresome (Lelouard et al., 2004, J. Cell. Biol.; Park et al., 2017; Nat. Comm.), which overlaps with the microtubule organizing center. Therefore, the authors' assumption that the localization of OP-Pur corresponds to active translation site could be misleading. Instead, the OP-Pur synthesized in the cytosol may be transported to aggresome. Therefore, the authors' conclusion in the Result section and Discussion section should be toned down.
Colocalization was not only found at the centrosomes but also on mitotic fibers suggesting that aggresome cannot account for all staining. However, we did not further characterize these foci and therefore we toned down the sentences concerning local translation in Results and Discussion.

Reviewer 2 Advance Summary and Potential Significance to Field:
With their manuscript the authors dig deeper into the question of how and why RNAs are linked to the mitotic spindle; a phenomenon often described but poorly understood. They identify a specific isoform of the RNA binding protein Staufen1 to be important to enrich a set of RNAs, including mRNAs, at the spindle. Interestingly, only the shorter isoform of Stau1 (Stau_55) is found on spindles; the longer Stau1_63 isoform or the paralog Staufen2 are missing. The authors further identify a short stretch of 11 amino acids, present in both Stau1 isoforms, that is crucial for the localisation of Stau1_55 but not Stau1_63 to mitotic spindles. Finally, data are presented to illustrate that spindle localised Stau1 overlaps with sites of active translation, indicating that fraction of Stau1_55 bound mRNAs could be translated on spindles. The manuscript presents an important advance in understanding what recruits some RNAs to the mitotic spindle. Especially the validation of used antibodies is highly appreciated. Less clear is what is the importance of Stau1 during mitosis and the Stau1-mediated RNA recruitment to the spindle. Overall, I do think the work is important and interesting, but some major points would need to be addressed.

Reviewer 2 Comments for the Author:
Major points

1) The title of the manuscript uses the word 'transport' which implicates active movements e.g. motor protein driven transport. After reading the manuscript, 'localisation' would certainly fit better as there is no data on active transport processes.
We changed the title accordingly.

2) Given that all cell lines are at hand, the manuscript would greatly benefit from some characterisation of what Stau1 removal does to mitosis. Is it slowed down? Does spindle morphology change? Is there a higher fraction of defective spindles? This data should be straight forward to collect and would greatly support the relevance of the discovered Stau1_55 spindle-association.
STAU1 depletion has no effect in cancer cells. However, in non-transformed cells, STAU1 depletion impairs mitosis and cell proliferation. Time lapse videomicoscopy did not detect defective spindles or morphology changes. These results are described in Ghram et al that is now accepted for publication in JMB. We added this reference (previously cited as submitted).

3)P.6 line 1: A reference is given to unpublished work. As this work is not listed in the references, it's difficult to check whether it is on a preprint server. If this is not the case, the entire statement should be removed. Also, the statement based on this unpublished work is not clear; on one hand, there is a referenced statement in the introduction that Stau1 levels decrease during mitosis. But then the unpublished observation shows that Stau1 depletion impairs mitosis? How does this fit together?
This submitted reference is now published (JMB). We updated the citation accordingly. Also the discrepancy is due to STAUl complexity. Indeed, STAU1 expression decreases during mitosis as a physiological consequence of its degradation by APC/C. In non- transformed cells, non-physiologic STAU1 depletion (throughout the cell cycle) impairs mitosis and cell proliferation. This phenotype is not observed in cancer cells.

4) What does the Stau1_55 N-terminus do? Can it directly bind microtubules? Or does it bind to

other microtubule-binding proteins which then cause the observed spindle binding? While the latter question might be too work intense to address within this work, it would be great to see, if the Stau1_55 N-terminus binds microtubules (and the Stau1_63 n- terminus not). Tubulin can be bought commercially, and fragments of bother termini should be straight forward to purify. A simple microtubule co-pelleting assay would then shed light on the nature of the interaction (indirect or direct?).
This is an interesting project for future work (as mentioned by the referee). The role of STAU1 N-terminus is not clear. However, it is not clear that in vitro assay can answer this problematic as protein modification such as phosphorylation may be required in vivo to change the way STAU1 interacts with tubulin during mitosis. During interphase, STAU1 is indeed not localized to microtubules (Wickham 1999).

5) The author concludes that because the fluorescence of Stau1, tubulin, and OP-Pur show some overlap, some Stau1-bound mRNAs on spindles might be locally translated. I think this is an extremely loose correlation. Unless higher resolution imaging data or data from translation sensors is presented, this statement is superfluous and should be removed. We toned down the sentences concerning local translation.

6) There is data on the Stau1_55 N-terminus discussed in the discussion, that is found nowhere in the results part. (p.17, line 5). Either this reference to unpublished data should be removed or put into the results section together with the data.
This unpublished data was removed.

7) There are more incidents of references to unpublished data in the discussion that should be removed unless the data is shown.
Unpublished data were removed.

Reviewer 3 Advance Summary and Potential Significance to Field:
In this manuscript Hassine and coworkers investigated the role of STAU1 in controlling localization of RNAs during mitosis of colorectal cancer cells. They report that a large fraction of STAU1 localizes in the spindles during mitosis, which dependents on the first 88 N-terminal amino acids of the protein. They identified that a group of RNAs are enriched in spindle fractions and part of them are delocalized from the spindles when STAU1 is knockout. Finally, they showed that STOU1 could control the local translation of a subpopulation of its bound mRNAs in the spindles. Overall, this manuscript presents interesting data, which should be of interest to the readers of the journal. The study is well designed and the data are appropriately interpreted. However, although the results include novel and interesting findings, several major concerns are remained.

Reviewer 3 Comments for the Author: Main
comments:

1. It is confusion for the function of STOU1 on cell proliferation. In the Instruction (line 4 of Page 5), the authors mentioned that “STAU1 is crucial for cell differentiation…and cell proliferation”. While in the Result section, they generated STOU1-KO cell strain and concluded that “The growth rate of STAU1-KO (clone CR1.3) HCT116 cells was similar to that of WT cells” (line 15 of Page 8).
See comments to referee 1. The importance of STAU1 expression is different in cancer vs non-transformed cells. This is why we must indicate cell types when we refer to STAU1 functions.

2. It seems that STAU1 plays a role in spindle procession during mitosis through controlling localization and translation of different sub-types of pre-rRNAs and mRNAs. However, the physiological impact of this role on cell differentiation and growth has not addressed through their study. Without this, the significance of the study is weakened.
See comments to referee 2 (comment 2).

3. Line 7 of page 13, the authors mentioned that “we studied several RNAs whose amounts were decreased in spindle preparations of STAU1-KO cells compared to that of WT cells and are known targets of STAU1 binding”. Does it mean that localization of the RNAs to the spindle is not solely dependent on STOU1? What is the cellular consequence of partly delocalizing these RNAs by STOU1 deletion or mutation?

We assume that RNA localization to spindle is not solely dependent on STAU1. Indeed, several spindle-bound RNAs (identified by RNA-seq) are not delocalized in STAU1-KO cells, suggesting that their localization is not STAU1-dependent. The consequence of their delocalization is not clear. In cancer cells, there is no effect on cell proliferation. However, STAU1 overexpression, which also impacts the fate of RNAs on spindle, impairs cell proliferation and causes spindle defects. In non-transformed cells, STAU1 depletion causes mitotic defects and impairs cell proliferation.

4. In Fig 7；Using confocal microscopy，the authors analyzed the signals of O-propargyl- puromycin marker along with those generated by anti-tubulin and anti-STAU1 antibodies, they suggested that a subpopulation of STAU1-bound mRNAs is locally translated on the spindle. However, this conclusion may lack the support of biochemistry assays. Sucrose gradient assays of the cell spindle preparation should be performed to show whether STAU1 and its bound mRNAs are co-located in the polysomal fractions.
This is a good suggestion for future work. We toned down our conclusions.

Second decision letter

MS ID#: JOCES/2020/247155

MS TITLE: Staufen1 localizes to the mitotic spindle and controls the localization of RNA populations to the spindle

AUTHORS: Sami Hassine, Florence Bonnet-Magnaval, Louis Philip Benoit Bouvrette, Bellastrid Doran, Mehdi Ghram, Mathieu Bouthillette, Eric Lecuyer, and Luc DesGroseillers
ARTICLE TYPE: Research Article

We have now reached a decision on the above manuscript.

To see the reviewers' reports and a copy of this decision letter, please go to: https://submit-jcs.biologists.org and click on the 'Manuscripts with Decisions' queue in the Author Area. (Corresponding author only has access to reviews.)

As you will see, the reviewers recognize that most initial concerns have been addressed in your revised manuscript. However, reviewer #3 still raised a criticism that will require further amendments to your manuscript. I hope that you will be able to discuss in a revised version the issue raised by reviewer #3, because I would like to be able to accept your paper.

We are aware that you may currently be unable to access the lab to undertake experimental revisions. If it would be helpful, we encourage you to contact us to discuss your revision in greater detail. Please send us a point-by-point response indicating where you are able to address concerns raised (either experimentally or by changes to the text) and where you will not be able to do so within the normal timeframe of a revision. We will then provide further guidance. Please also note that we are happy to extend revision timeframes as necessary.

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Reviewer 1

Advance summary and potential significance to field

In this paper, the authors present evidence supporting STAU1-dependent localization of a subset of transcripts to spindle during mitosis.

Comments for the author

In the revised manuscript, the authors have satisfactorily addressed all comments of this reviewer.

Reviewer 3

Advance summary and potential significance to field

The authors well addressed my points, however, one major concern still remains.

Comments for the author

In the manuscript, the authors nicely showed the ability of STAU1 to regulate the transport/localization of different RNA biotypes into spindles, which may contribute to rRNA maintenance during mitosis. However, experiments were mostly performed in HCT116 colorectal cancer cells, in which STAU1 depletion does not impair mitosis and cell proliferation. Therefore, what is the relative physiological importance of STAU1 localizing RNA populations to the spindles of cancer cells? In other words, since STAU1 deletion only affects the cell mitosis and proliferation in non-transformed cells, using non-transformed cells to study the role of STAU1 on spindle morphology change or the biological consequence of STAU1-directed RNA localization into spindles would be a better choice.

Second revision

Author response to reviewers' comments

Reviewer 3 Comments for the Author:
In the manuscript, the authors nicely showed the ability of STAU1 to regulate the transport/localization of different RNA biotypes into spindles, which may contribute to rRNA maintenance during mitosis. However, experiments were mostly performed in HCT116 colorectal cancer cells, in which STAU1 depletion does not impair mitosis and cell proliferation. Therefore, what is the relative physiological importance of STAU1 localizing RNA populations to the spindles of cancer cells? In other words, since STAU1 deletion only affects the cell mitosis and proliferation in non-transformed cells, using non-transformed cells to study the role of STAU1 on spindle morphology change or the biological consequence of STAU1-directed RNA localization into spindles would be a better choice.
We only partly agree with this referee. We indeed showed that STAU1 knockout has no effect on cancer cell proliferation but that STAU1 depletion impairs mitosis in non-transformed cells. We agree that it is thus important to study the consequence of STAU1 depletion in non-transformed cells, what we did in Ghram et al 2020. In this paper, we showed that STAU1 depletion alters mitosis but not the structure of the spindle nor the capacity of cells to divide and survive. It is possible that STAU1 depletion in non-transformed cells interferes with some aspects of the mitotic checkpoint control, mechanisms that are usually lost in cancer cells. However, depletion/knockout of STAU1 is not the only way to modify the expression of STAU1-bound RNAs during mitosis. STAU1 overexpression also changes the fate of bound RNAs (localization, transport, decay, etc). Following overexpression, both mitosis and cell proliferation are impaired in cancer cells (but not in non-transformed cells) (Boulay et al 2014). We thus believe that studying STAU1 expression in the context of cancer cells is also relevant and important.

We modified the following paragraph in the manuscript (discussion p17) to discuss the consequence of manipulating STAU1 expression during mitosis.

"Thus, STAU1 controls, in different cellular compartments, differential sub-populations of pre-rRNAs and mRNAs that likely regulate cell decision during mitosis. The consequence of STAU1 depletion varies according to cellular context. While it has no observable effect in cancer cells, STAU1 depletion impairs mitosis progression and cell proliferation in non-transformed cells (Ghram, 2020). Nevertheless, spindle defects are not observed and both daughter cells survive. It is possible that STAU1 depletion interferes somehow with a mitotic checkpoint control, a mechanism that is often lost in cancer cells. Cancer cells are rather susceptible to STAU1 overexpression (Boulay et al., 2014). Overexpression, that also alters the fate of STAU1-bound mRNAs, hinders cell proliferation via a reduction in the number of cells that transit mitosis. Via the posttranscriptional regulation that it imposes to its bound RNAs, STAU1 regulates crucial functions and deregulation of this mechanism may explain the proliferation defects observed in non-transformed cells upon STAU1 depletion (Ghram, 2020) and in cancer cells upon STAU1 overexpression (Boulay et al., 2014)."

Third decision letter

MS ID#: JOCES/2020/247155

MS TITLE: Staufen1 localizes to the mitotic spindle and controls the localization of RNA populations to the spindle

AUTHORS: Sami Hassine, Florence Bonnet-Magnaval, Louis Philip Benoit Bouvrette, Bellastrid Doran, Mehdi Ghram, Mathieu Bouthillette, Eric Lecuyer, and Luc DesGroseillers
ARTICLE TYPE: Research Article

I am happy to tell you that your manuscript has been accepted for publication in Journal of Cell Science, pending standard ethics checks.