

Monday July 17

How to Write an Abstract



Abstract Structure

1. Background Info
2. Hypothesis, Objective, or Problem Identified
3. Some sort of methodology
4. Results
5. Conclusions and implications

Abstract Structure

1. Background Info

Context for the work

2. Hypothesis, Objective, or Problem Identified

What is your question and/or hypothesis?

3. Some sort of methodology

Key method, system, study design

4. Results

What are the findings?

5. Conclusions and implications

What do the findings mean, related to your purpose/hypothesis?

Things to keep in mind:

1. Consider your audience. Try to keep jargon to a minimum to engage a larger audience.
2. Make your hypothesis/purpose/objective very obvious.
3. Tie your results and conclusions back to your hypothesis/purpose/objective.
4. Make every word count.
5. Pay attention to instructions and length limitations!

Qiu X¹, Tan H¹, Fu D¹, Zhu Y¹, Zhang J¹.

⊕ **Author information**

Abstract

AIM OF STUDY: Breast cancer invasion and metastasis is the main reason for the failure, and laminin is involved in it. This study intends to explore the expression of laminin in breast cancer and normal breast tissue and its clinical significance.

MATERIALS AND METHODS: We use immunohistochemical assay for the detection of breast infiltrating ductal cancer tissues and normal breast tissues of laminin expression and discuss their role in breast cancer invasion and metastasis.

RESULTS: Our results showed that laminin was positive expressed in normal breast tissue, and strongly positive expressed but lost its' continuity in the breast cancer tissue.

CONCLUSION: This results revealed laminin is involved in breast cancer invasion and metastasis, and we can use this to determine whether the integrity of a basement membrane for differential diagnosis of benign and malignant breast tumors.

Prefrontal cortex (PFC) dysfunction is widely believed to underlie working memory (WM) deficits in people with schizophrenia (PSZ), but few studies have focused on measures of WM storage devoid of manipulation. Research in neurotypical individuals has shown that storage capacity is more closely related to posterior parietal cortex (PPC) than PFC, suggesting that reductions in WM storage capacity in schizophrenia that are associated with broad cognitive deficits may be related to neural activity in PPC. In the present human neuroimaging study, 37 PSZ and 37 matched healthy control subjects (HCS) of either sex completed a change detection task with varying set sizes while undergoing functional Magnetic Resonance Imaging. The task was designed to emphasize WM storage with minimal top-down control demands. Whole-brain analysis identified areas in which BOLD activity covaried with the number of items maintained in WM (K), as derived from task performance at a given set size. Across groups, K values independent of set size predicted BOLD activity in PPC, including superior and inferior parietal lobules and intraparietal sulcus, and middle occipital gyrus. Whole-brain interaction analysis found significantly less K -dependent signal modulation in PSZ than HCS in left PPC, a phenomenon that could not be explained by a narrower K -value range. The slope between K and PPC activation statistically accounted for 43.4% of the between-group differences in broad cognitive function. These results indicate that PPC dysfunction is central to WM storage deficits in PSZ and may play a key role in the broad cognitive deficits associated with schizophrenia.

Prefrontal cortex (PFC) dysfunction is widely believed to underlie working memory (WM) deficits in people with schizophrenia (PSZ), but few studies have focused on measures of WM storage devoid of manipulation. Research in neurotypical individuals has shown that storage capacity is more closely related to posterior parietal cortex (PPC) than PFC, suggesting that reductions in WM storage capacity in schizophrenia that are associated with broad cognitive deficits may be related to neural activity in PPC. In the present human neuroimaging study, 37 PSZ and 37 matched healthy control subjects (HCS) of either sex completed a change detection task with varying set sizes while undergoing functional Magnetic Resonance Imaging. The task was designed to emphasize WM storage with minimal top-down control demands. Whole-brain analysis identified areas in which BOLD activity covaried with the number of items maintained in WM (K), as derived from task performance at a given set size. Across groups, K values independent of set size predicted BOLD activity in PPC, including superior and inferior parietal lobules and intraparietal sulcus, and middle occipital gyrus. Whole-brain interaction analysis found significantly less K-dependent signal modulation in PSZ than HCS in left PPC, a phenomenon that could not be explained by a narrower K-value range. The slope between K and PPC activation statistically accounted for 43.4% of the between-group differences in broad cognitive function. These results indicate that PPC dysfunction is central to WM storage deficits in PSZ and may play a key role in the broad cognitive deficits associated with schizophrenia.

AURKA is an established target for cancer therapy; however, the efficacy of its inhibitors in clinical trials is hindered by differential response rates across different tumor subtypes. In this study, we demonstrate AURKA regulates amino acid synthesis, rendering it a vulnerable target in KEAP1-deficient non-small cell lung cancer (NSCLC). Through CRISPR metabolic screens, we identified that KEAP1-knockdown cells showed the highest sensitivity to the AURKA inhibitor MLN8237. Subsequent investigations confirmed that KEAP1 deficiency heightens the susceptibility of NSCLC cells to AURKA inhibition both in vitro and in vivo, with the response depending on NRF2 activation. Mechanistically, AURKA interacts with the eIF2 α kinase GCN2 and maintains its phosphorylation to regulate eIF2 α -ATF4-mediated amino acid biosynthesis. AURKA inhibition restrains the expression of asparagine synthetase (ASNS), making KEAP1-deficient NSCLC cells vulnerable to AURKA inhibitors, in which ASNS is highly expressed. Our study unveils the pivotal role of AURKA in amino acid metabolism and identifies a specific metabolic indication for AURKA inhibitors. These findings also provide a novel clinical therapeutic target for KEAP1-mutant/deficient NSCLC, which is characterized by resistance to radiotherapy, chemotherapy, and targeted therapy.

AURKA is an established target for cancer therapy; however, the efficacy of its inhibitors in clinical trials is hindered by differential response rates across different tumor subtypes. In this study, we demonstrate AURKA regulates amino acid synthesis, rendering it a vulnerable target in KEAP1-deficient non-small cell lung cancer (NSCLC). Through CRISPR metabolic screens, we identified that KEAP1-knockdown cells showed the highest sensitivity to the AURKA inhibitor MLN8237. Subsequent investigations confirmed that KEAP1 deficiency heightens the susceptibility of NSCLC cells to AURKA inhibition both in vitro and in vivo, with the response depending on NRF2 activation. Mechanistically, AURKA interacts with the eIF2 α kinase GCN2 and maintains its phosphorylation to regulate eIF2 α -ATF4-mediated amino acid biosynthesis. AURKA inhibition restrains the expression of asparagine synthetase (ASNS), making KEAP1-deficient NSCLC cells vulnerable to AURKA inhibitors, in which ASNS is highly expressed. Our study unveils the pivotal role of AURKA in amino acid metabolism and identifies a specific metabolic indication for AURKA inhibitors. These findings also provide a novel clinical therapeutic target for KEAP1-mutant/deficient NSCLC, which is characterized by resistance to radiotherapy, chemotherapy, and targeted therapy.

How can I make this more reader-friendly for an abstract book, for a broader audience?

AURKA is an established target for cancer therapy; however, the efficacy of its inhibitors in clinical trials is hindered by differential response rates across different tumor subtypes. In this study, we demonstrate AURKA regulates amino acid synthesis, rendering it a vulnerable target in KEAP1-deficient non-small cell lung cancer (NSCLC). Through CRISPR metabolic screens, we identified that KEAP1-knockdown cells showed the highest sensitivity to the AURKA inhibitor MLN8237. Subsequent investigations confirmed that KEAP1 deficiency heightens the susceptibility of NSCLC cells to AURKA inhibition both in vitro and in vivo, with the response depending on NRF2 activation. Mechanistically, AURKA interacts with the eIF2 α kinase GCN2 and maintains its phosphorylation to regulate eIF2 α -ATF4-mediated amino acid biosynthesis. AURKA inhibition restrains the expression of asparagine synthetase (ASNS), making KEAP1-deficient NSCLC cells vulnerable to AURKA inhibitors, in which ASNS is highly expressed. Our study unveils the pivotal role of AURKA in amino acid metabolism and identifies a specific metabolic indication for AURKA inhibitors. These findings also provide a novel clinical therapeutic target for KEAP1-mutant/deficient NSCLC, which is characterized by resistance to radiotherapy, chemotherapy, and targeted therapy.

How can I make this more reader-friendly for an abstract book, for a broader audience?

AURKA is an established target for cancer therapy;

Aurora Kinase A (AURKA) is a mitotic kinase. Elevated AURKA levels have been correlated with cell proliferation and chemotherapy resistance.

however, the efficacy of its inhibitors in clinical trials is hindered by differential response rates across different tumor subtypes.

However, in clinical trials, AURKA inhibitors have differential response rates across different tumor subtypes, making those inhibitors less useful for treatment.

How can I make this more reader-friendly for an abstract book, for a broader audience?

In this study, we demonstrate AURKA regulates amino acid synthesis, rendering it a vulnerable target in KEAP1-deficient non-small cell lung cancer (NSCLC). Through CRISPR metabolic screens, we identified that KEAP1-knockdown cells showed the highest sensitivity to the AURKA inhibitor MLN8237.

In this study, we examine the role of AURKA in amino acid synthesis; if amino acid synthesis is compromised, cells will be less able to proliferate.

Using a non-small cell lung cancer (NSCLC) cell line deficient in the tumor suppressor KEAP1, we did CRISPR metabolic screens while treating the cells with various AURKA inhibitors.

Aurora Kinase A (AURKA) is a mitotic kinase. Elevated AURKA levels have been correlated with cell proliferation and chemotherapy resistance. However, in clinical trials, AURKA inhibitors have differential response rates across different tumor subtypes, making those inhibitors less useful for treatment. In this study, we examine the role of AURKA in amino acid synthesis; if amino acid synthesis is compromised, cells will be less able to proliferate. Using a non-small cell lung cancer (NSCLC) cell line deficient in the tumor suppressor KEAP1, we did CRISPR metabolic screens while treating the cells with various AURKA inhibitors. We found that KEAP1-knockdown cells showed the highest sensitivity to the AURKA inhibitor MLN8237. Subsequent investigations confirmed that KEAP1 deficiency heightens the susceptibility of NSCLC cells to AURKA inhibition both in vitro and in vivo, with the response depending on NRF2 activation. Mechanistically, AURKA interacts with the eIF2 α kinase GCN2 and maintains its phosphorylation to regulate eIF2 α -ATF4-mediated amino acid biosynthesis. AURKA inhibition restrains the expression of asparagine synthetase (ASNS), making KEAP1-deficient NSCLC cells vulnerable to AURKA inhibitors, in which ASNS is highly expressed. Our study unveils the pivotal role of AURKA in amino acid metabolism and identifies a specific metabolic indication for AURKA inhibitors. These findings also provide a novel clinical therapeutic target for KEAP1-mutant/deficient NSCLC, which is characterized by resistance to radiotherapy, chemotherapy, and targeted therapy.

What if I don't have results yet?

Aurora Kinase A (AURKA) is a mitotic kinase. Elevated AURKA levels have been correlated with cell proliferation and chemotherapy resistance. However, in clinical trials, AURKA inhibitors have differential response rates across different tumor subtypes, making those inhibitors less useful for treatment. **In this study, we examine how the inhibition of AURKA affects gene expression.** Using a non-small cell lung cancer (NSCLC) cell line deficient in the tumor suppressor KEAP1, we **are doing** CRISPR metabolic screens while treating the cells with various AURKA inhibitors. Preliminary data suggest that KEAP1-knockdown cells show the highest sensitivity to the AURKA inhibitor MLN8237. We are testing the susceptibility of NSCLC cells to AURKA inhibition both in vitro and in vivo. We will be examining genes whose expression is upregulated and/or downregulated when AURKA is inhibited, focusing on genes in essential metabolic pathways that affect cell proliferation. **Our study aims to unveil the pivotal role of AURKA in metabolic pathways essential for cancer cell survival.** These findings could also provide a novel clinical therapeutic target for KEAP1-mutant/deficient NSCLC, which is characterized by resistance to radiotherapy, chemotherapy, and targeted therapy.

Abstract Rubric

Abstract Title:

First Author:

Grammar/Spelling/English Usage		
	No Problems	+2
	Minor problems, but does not affect understanding (may indicate carelessness)	+1
	Problems – makes abstract difficult to understand	+0
Components		
	Introduction	+1
	Statement of hypothesis/question/purpose	+2
	General methods used	+1
	Primary results (not necessary)	+0
	Primary Conclusion(s)	+1
	General statement of significance of work	+1
Necessary Attributes		
	<u>Concise</u>	
	All essential information, within word limit	+2
	Missing something / slightly over word limit	+1
	Too long / too short, so difficult to follow	0
	<u>Clear</u>	
	Easy to understand what abstract is about, no jargon, all terms defined	+2
	Generally able to understand, but some unclear terms	+1
	I'm lost!!!	0
Overall Readability / Interest		
	Do you want to read the paper / see the poster?	+3
	I've got all the information I need and it's fine	+2
	I know what's going on, but it's boring	+1
	This was too tough to read!	0
	OVERALL TOTAL	/15

[Qiu X¹](#), [Tan H¹](#), [Fu D¹](#), [Zhu Y¹](#), [Zhang J¹](#).

⊕ **Author information**

Abstract

AIM OF STUDY: Breast cancer invasion and metastasis is the main reason for the failure, and laminin is involved in it. This study intends to explore the expression of laminin in breast cancer and normal breast tissue and its clinical significance.

MATERIALS AND METHODS: We use immunohistochemical assay for the detection of breast infiltrating ductal cancer tissues and normal breast tissues of laminin expression and discuss their role in breast cancer invasion and metastasis.

RESULTS: Our results showed that laminin was positive expressed in normal breast tissue, and strongly positive expressed but lost its' continuity in the breast cancer tissue.

CONCLUSION: This results revealed laminin is involved in breast cancer invasion and metastasis, and we can use this to determine whether the integrity of a basement membrane for differential diagnosis of benign and malignant breast tumors.

Key words/concepts?

Breast cancer

Metastasis

Laminin

Laminin expression high in breast cancer tissue

Laminin is over expressed in breast cancer and facilitate cancer cell metastasis.

[Qiu X¹](#), [Tan H¹](#), [Fu D¹](#), [Zhu Y¹](#), [Zhang J¹](#).

⊕ [Author information](#)

Abstract

AIM OF STUDY: Breast cancer invasion and metastasis is the main reason for the failure, and laminin is involved in it. This study intends to explore the expression of laminin in breast cancer and normal breast tissue and its clinical significance.

MATERIALS AND METHODS: We use immunohistochemical assay for the detection of breast infiltrating ductal cancer tissues and normal breast tissues of laminin expression and discuss their role in breast cancer invasion and metastasis.

RESULTS: Our results showed that laminin was positive expressed in normal breast tissue, and strongly positive expressed but lost its' continuity in the breast cancer tissue.

CONCLUSION: This results revealed laminin is involved in breast cancer invasion and metastasis, and we can use this to determine whether the integrity of a basement membrane for differential diagnosis of benign and malignant breast tumors.

Key words/concepts?

Breast cancer

Metastasis

Laminin

Laminin expression high in breast cancer tissue

Aurora Kinase A (AURKA) is a mitotic kinase. Elevated AURKA levels have been correlated with cell proliferation and chemotherapy resistance. However, in clinical trials, AURKA inhibitors have differential response rates across different tumor subtypes, making those inhibitors less useful for treatment. In this study, we examine the role of AURKA in amino acid synthesis; if amino acid synthesis is compromised, cells will be less able to proliferate. Using a non-small cell lung cancer (NSCLC) cell line deficient in the tumor suppressor KEAP1, we did CRISPR metabolic screens while treating the cells with various AURKA inhibitors. We found that KEAP1-knockdown cells showed the highest sensitivity to the AURKA inhibitor MLN8237. Subsequent investigations confirmed that KEAP1 deficiency heightens the susceptibility of NSCLC cells to AURKA inhibition both in vitro and in vivo, with the response depending on NRF2 activation. Mechanistically, AURKA interacts with the eIF2 α kinase GCN2 and maintains its phosphorylation to regulate eIF2 α -ATF4-mediated amino acid biosynthesis. AURKA inhibition restrains the expression of asparagine synthetase (ASNS), making KEAP1-deficient NSCLC cells vulnerable to AURKA inhibitors, in which ASNS is highly expressed. Our study unveils the pivotal role of AURKA in amino acid metabolism and identifies a specific metabolic indication for AURKA inhibitors. These findings also provide a novel clinical therapeutic target for KEAP1-mutant/deficient NSCLC, which is characterized by resistance to radiotherapy, chemotherapy, and targeted therapy.

Keywords:

Aurora Kinase A, Non-small cell lung cancer (NSCLC), amino acid synthesis, asparagine, chemotherapy target

AURKA emerges as a vulnerable target for KEAP1-deficient non-small cell lung cancer by activation of asparagine synthesis

AURKA is an established target for cancer therapy; however, the efficacy of its inhibitors in clinical trials is hindered by differential response rates across different tumor subtypes. In this study, we demonstrate AURKA regulates amino acid synthesis, rendering it a vulnerable target in KEAP1-deficient non-small cell lung cancer (NSCLC). Through CRISPR metabolic screens, we identified that KEAP1-knockdown cells showed the highest sensitivity to the AURKA inhibitor MLN8237. Subsequent investigations confirmed that KEAP1 deficiency heightens the susceptibility of NSCLC cells to AURKA inhibition both in vitro and in vivo, with the response depending on NRF2 activation. Mechanistically, AURKA interacts with the eIF2 α kinase GCN2 and maintains its phosphorylation to regulate eIF2 α -ATF4-mediated amino acid biosynthesis. AURKA inhibition restrains the expression of asparagine synthetase (ASNS), making KEAP1-deficient NSCLC cells vulnerable to AURKA inhibitors, in which ASNS is highly expressed. Our study unveils the pivotal role of AURKA in amino acid metabolism and identifies a specific metabolic indication for AURKA inhibitors. These findings also provide a novel clinical therapeutic target for KEAP1-mutant/deficient NSCLC, which is characterized by resistance to radiotherapy, chemotherapy, and targeted therapy.

If you are submitting to

- SACNAS, 250 words is their limit.
- ABRCMS, the limit is 2500 characters not including spaces.
- For the VSSA poster session, the limit is 300 words.

With this structure in mind, draft an abstract

1. Background Info: 1-2 sentences
2. Hypothesis, Objective, or Problem Identified: 1-2 sentences
3. Some sort of methodology: What system?
Key method used
4. Results: Short sentences
5. Conclusions and implications: 1-2 sentences

Swap with your neighbor, and provide feedback

1. Background Info
2. Hypothesis, Objective, or Problem Identified
3. Some sort of methodology
4. Results
5. Conclusions and implications