

# 文献阅读教程

对大多数科研人来说，读英文文献都是科研的第一道坎儿，读文献时会遇到各种难题：阅读效率低，读不懂，读完没收获、没思路，读完记不住。为了帮助大家彻底解决这些读文献的烦恼，小绿鲸英文文献阅读器诞生了，下面为你介绍如何用小绿鲸高效读懂文献。

想必大家电脑都有个上百篇文献，不可能逐一仔细阅读，所以需要先阅读一下文章题目、摘要、前言以及文章中的图表，了解大致主要内容后进行文献分类，然后再按需进行不同形式的阅读。



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Heart and Lung  
transplantation  
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## Incidence and long-term outcome of heart transplantation patients who develop postoperative renal failure requiring dialysis

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**KEYWORDS:**  
heart transplantation;  
renal replacement  
therapy;  
rejection, dialysis;  
immunosuppression

**BACKGROUND:** Acute renal failure requiring dialysis after heart transplantation remains a significant clinical issue because of its increasing incidence. We aimed to investigate its time trends, clinical predictors, and long-term outcomes.  
**METHODS:** Adult heart transplantation recipients registered in the United Network for Organ Sharing registry between 2009 and 2020 were identified. The patients were grouped according to the requirement for dialysis in the postoperative heart transplantation period. The independent risk predictors were identified, and the association between post-heart transplantation renal failure requiring dialysis and long-term mortality accounting for re-transplantation was investigated.  
**RESULTS:** A total of 28,170 patients were included in the study, of which 3,371 (12%) required dialysis immediately post-heart transplantation. The incidence increased from 7.9% to 13.9% during the study period. Longer ischemic time, serum creatinine at transplantation >1.2 mg/dL, prior cardiac surgery, higher recipient body mass index, serum pre-operative arterial ventilation, history of dialysis, membranous glomerulopathy, and history of congenital heart disease or restrictive/hypertrophic cardiomyopathy were its predictors (all  $p < 0.05$ ). Patients on posttransplant dialysis had a higher risk of all-cause mortality (adjusted hazard ratio [aHR]: 5.2, 95% CI: 4.7-5.7,  $p < 0.001$ ), 30 day mortality (aHR: 7.7, 95% CI: 6.3-9.6,  $p < 0.001$ ) and 1 year mortality (aHR: 7.5, 95% CI: 6.6-8.6,  $p < 0.001$ ). Post-transplant dialysis was associated with a risk of treated rejection at 1 year.

**Data statement:** The data and materials used to conduct this research are available to researchers for purposes of reproducing the results or replicating the procedure on request.

**Reprint requests:** Alexandros Brasoulis, MD, Section of Heart Failure and Transplant, Division of Cardiovascular Diseases, University of Iowa

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**CONCLUSION:** Acute renal failure requiring dialysis after heart transplantation is associated with significantly worse 30 day and long-term mortalities, and thus, early identification of high-risk patients is crucial to prevent severe renal complications.

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Heart transplantation (HT) is a standard option for advanced heart failure.<sup>1</sup> Advances in immunosuppressive therapy such as calcineurin inhibitors (CNIs) continue to improve the long-term outcomes of HT recipients<sup>2</sup>, with the current median, 1 year, and 5 year survival rates being 10.7 years, 82%, and 69%, respectively.<sup>2–4</sup> However, these improved outcomes and prolonged survival have also increased the risk of comorbidities. Particularly, acute kidney injury (AKI) after HT leads to subsequent progressive chronic kidney disease (CKD) and end-stage kidney disease requiring dialysis has become an important complication owing to its increasing incidence.<sup>5</sup> The International Society of Heart and Lung Transplantation raised concerns for the increasing rate of renal-related complications as they are strongly associated with an increase in resource use, cost, and mortality.<sup>6</sup>

AKI occurs in 40% to 70% of HT patients, and it is associated with pre-existing comorbidities, right heart failure, major bleeding, or the use of CNIs.<sup>5,7,8</sup> However, little is known regarding its risk factors and its association with the long-term outcomes of HT patients, particularly in those requiring dialysis. In addition, there is also limited evidence on the clinical implications of more severe kidney injury because of its low incidence (about 10%). Although there have been observational studies that evaluated the risk factors for acute dialysis,<sup>9–11</sup> these studies were performed in a small population and were statistically underpowered to identify significant risk factors, suggesting the need for large-scale research. Furthermore, the long-term outcomes of HT patients who required dialysis have not been elucidated. Thus, this study aimed to assess the incidence, trends, and clinical predictors of acute renal failure requiring dialysis after HT and their association with the long-term outcomes among HT patients, using a large-scale cardiac HT registry.

### Methods

#### Study design and patients

The United Network of Organ Sharing (UNOS) registry is a large, ongoing, prospective registry that collects patient-level data in the entire United States transplant population. Data include information on donors, candidates, and recipients of every organ transplant that occurred since 1987.<sup>12,13</sup> Baseline characteristics at the time of transplantation, including demographic, clinical, laboratory parameters, and post-transplant follow-up data are collected.

This study evaluated adult patients aged ≥ 18 years who underwent isolated HT in the United States between January 1, 2009 and March 31, 2020. Patients who underwent multi-organ transplantation and those with missing data on the relevant donor and/or recipient demographics, including the information of acute

renal failure requiring dialysis after HT ( $n = 107$ , 0.4%), were excluded. The patients were grouped according to the requirement for acute dialysis in the postoperative HT period.

The study was approved by the institutional ethics review board of the University of Iowa and adhered to the Strengthening the Reporting of Observational Studies in Epidemiology reporting guidelines. The need for informed consent was waived because this was a secondary analysis of a de-identified dataset.

#### Variable definitions and outcome measures

Acute renal failure requiring dialysis after HT was defined as acute or worsening renal failure necessitating new renal dialysis during the admission of HT operation.<sup>14</sup> The indication for dialysis was determined according to the criteria of each participating institution.

The main outcome measures were 30 day, 1 year, all-cause mortality and treated acute allograft rejection episodes within the first year of heart transplantation. Deaths recorded in the UNOS dataset were ascertained from Organ Procurement and Transplant Network or verified from the Patient Status Confirmation List in the Secure Enterprise (current as of June 10, 2019). Treated rejection was defined as rejection that necessitated drug treatment within the first year after HT.<sup>15</sup>

#### Statistical analyses

The demographics and outcomes were compared between patients who did and did not develop acute renal failure requiring dialysis in the postoperative HT period. Normally distributed continuous variables were expressed as the mean with standard deviation and categorical variables as frequencies with percentage. Non-normally distributed continuous variables were expressed as median and interquartile range. Between-group comparisons of continuous variables were performed using Student's *t*-tests, while categorical variables were compared using Pearson's chi-squared test or Fisher's exact test, as appropriate. Multivariable logistic regression analysis was performed to identify the independent risk factors of acute renal failure requiring dialysis after HT. The variables in the model were determined according to the clinical relevance in association with the incidence of acute dialysis and the following recipient variables were included: age, sex, race, body mass index (BMI), diabetes mellitus (DM), prior history of CKD, surgery, primary cardiac diagnosis (ischemic cardiomyopathy, congenital heart disease, valvular cardiomyopathy, restrictive cardiomyopathy, hypertrophic cardiomyopathy, and other etiology), creatinine, calculated panel reactive antibody (PRA), extracorporeal membrane oxygenation (ECMO) at HT, inotrope use at HT, intra-aortic balloon pump (IABP) at HT, ventilator use at HT, and the use of induction therapy (antithymocyte globulin, interleukin 2 receptor antagonists, alemtuzumab, OKT3 or no induction therapy) after HT. For the donors, age, sex, and allograft ischemic time were included.

Thirty day, 1 year, and all-cause mortality were estimated with 95% confidence intervals (CIs) stratified by the presence of acute

看到这一大串英文，很多人都会感到犯难，但有了小绿鲸就不必头疼，强大的【翻译】功能帮你快速读懂英文文献。

## 1. 翻译

### 1.1 划词翻译

光标选中文献中需翻译的文字，右边即出现译文，可以边读边翻译。在译文上方可点击选择【鲸译AI通用版】或【鲸译AI学科版】，切换多个翻译引擎，提高翻译精准度。点击译文处【复制】按钮可直接复制粘贴；如原文格式或内容错误，可点击【修改】按钮进行修改后重新翻译。

**RESEARCH**

**PREGNANCY**

## ELABELA deficiency promotes preeclampsia and cardiovascular malformations in mice

Lena Ho,<sup>1</sup> 笔记 语料 引文 术语 单词 搜索 Messerschmidt,<sup>3</sup> Serene C. Chang,<sup>1</sup> Grace Hui-Yi Goh,<sup>1</sup> Gjjs B. Afink,<sup>2</sup> Chin Yan Lim,<sup>1</sup> N. Ray Dunn,<sup>1</sup> Davor Solter,<sup>1</sup> Barbara B. Knowles,<sup>1</sup> Bruno Reversade,<sup>1,2,3,4,5\*</sup>

Preeclampsia (PE) is a gestational hypertensive syndrome affecting between 5 and 8% of all

the presence of a CD31/Pecam<sup>+</sup> endothelial plexus, which subsequently fails to undergo remodeling and angiogenic sprouting to form organized vitelline vessels, dorsal aorta, outflow tract, and interisomitic vessels (Fig. 1, K to S, and fig. S1, E to J). The heart tube is poorly looped, with reduced smooth actin muscle (SMA) staining (Fig. 1, Q to S), and the most severely affected embryos (class 3) have pericardial edema (fig. S1, K and L). These cardiac defects are consistent with the first postgastrulation expression of *Ela* in the primitive foregut overlying the developing heart tube (Fig. 1, T and U) (6). Surprisingly, *Ela* is not detected in endothelial precursors of the yolk sac (Fig. 1W), whereas *Apnl* expression is ubiquitous in embryonic, allantoic, and yolk sac mesoderm,

还可以点击“跟随”，翻译界面就可以悬浮在文献上面随意移动，这样翻译固定的界面可以收缩起来使界面更加整洁

为了描述ELA对mam-malian发育的贡献，我们使用同源重组产生了Ela敲除(ElaD/D)小鼠，以删除编码成熟ELA肽的外显子3(图1, A和B, 以及图S1, B和C)。这一策略没有

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To delineate the contribution of ELA to mam-malian development, we generated Ela knockout (ElaD/D) mice using homologous recombination to delete exon 3 encoding the mature ELA peptide (Fig. 1, A and B, and fig. S1, B and C). This strategy did not

用下面这段摘要试一下吧！

## Abstract

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**CONCLUSION:** Acute renal failure requiring dialysis after heart transplantation is associated with significantly worse 30 day and long-term mortalities, and thus, early identification of high-risk patients is crucial to prevent severe renal complications.

## 1.2 学科翻译

在划词翻译时，如遇到一些专业词或长难词无法翻译或翻译不准确的情况，可选中对应内容点击下方工具栏中的【添加术语】，将其重新定义。然后点击右侧翻译引擎切换到【鲸译 AI 学科版】，勾选【术语库】，再次划词翻译就可以校正翻译结果。



### PREGNANCY

## ELABELA deficiency promotes preeclampsia and cardiovascular malformations in mice

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Preeclampsia (PE) is a gestational hypertensive syndrome affecting between 5 and 8% of all pregnancies. Although PE is the leading cause of fetal and maternal morbidity and mortality, its molecular etiology is still unclear. Here, we show that ELABELA (ELA), an endogenous ligand of the apelin receptor (APLNR or APRI), is a circulating hormone secreted by the placenta. Elabela but not Apelin knockout pregnant mice exhibit PE-like symptoms, including proteinuria and elevated blood pressure due to defective placental angiogenesis. In mice, infusion of exogenous ELA normalizes hypertension, proteinuria, and birth weight. ELA, which is abundant in human placentas, increases the invasiveness of trophoblast-like cells, suggesting that it enhances placental development to prevent PE. The ELA-APLNR signaling axis may offer a new paradigm for the treatment of common pregnancy-related complications.

the presence of a CD31/Pecam<sup>+</sup> endothelial plexus, which subsequently fails to undergo remodeling and angiogenic sprouting to form organized vitelline vessels, dorsal aorta, outflow tract, and inter-somitic vessels (Fig. 1, K to S, and fig. S1, E to J). The heart tube is poorly looped, with reduced smooth actin muscle (SMA) staining (Fig. 1, Q to S), and the most severely affected embryos (class 3) have pericardial edema (fig. S1, K and L). These cardiac defects are consistent with the first postgastrulation expression of *Ela* in the primitive foregut overlying the developing heart tube (Fig. 1, T and U) (6). Surprisingly, *Ela* is not detected in endothelial precursors of the yolk sac (Fig. 1W), whereas *Apj* expression is ubiquitous in embryonic, allantoic, and yolk sac mesoderm, which gives rise to endothelial cells (Fig. 1, V and X). The expression patterns of *Ela* and *Apj* suggest that the observed cardiac defects are partly due to insufficient blood flow to stimulate angiogenesis. Outside of the developing heart tube, *Ela* is first detected in the chorionic trophoblast of the developing placenta (Fig. 1U and fig. S1, M and N) and is robustly up-regulated after allantoic fusion (Fig. 2A), becoming restricted to syncytiotrophoblasts (STs) at E10.5

**syndrome**

英[syndrome] 美[syndrome]

n.综合征：综合症状；典型表现；

● 例句

- 1 Objective : To observe and analyze the effects of three operative methods on Carpal Tunnel Syndrome (CTS). 目的: 采用三种手术方法治疗腕管综合征(carpatunnel syndrome,CTS) 观察其疗效。
- 2 Objective To study the relationship between intervention treatment of Budd - Chiari syndrome (BCS) and pregnancy. 目的研究布-加氏综合征(Budd-Chiari Syndrome,BCS)介入治疗后与妊娠的关系。
- 3 Human immunodeficiency Virus ( HIV ) was the pathogen of Acquired Immunodeficiency Syndrome ( AIDS ). 艾滋病病毒 ( Humanimmunodeficiency Virus,HIV ) 是导致人获得性免疫缺陷综合症(AcquiredImmunodeficiency Syndrome,AIDS ) 的病原体。
- 4 The main problems included neurasthenia, depression,



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**译文**  术语库  锁定  逆翻  复制 14 \*

子痫前期(PE)是一种妊娠期高血压综合征，影响5 - 8%的妊娠。虽然PE是胎儿和产妇发病率和死亡率的主要原因，但其分子病因仍不清楚。

**原文** 选中下方词组添加为术语和单词  复制  修改

Preeclampsia (PE) is a gestational hypertensive syndrome affecting between 5 and 8% of all pregnancies. Although PE is the leading cause of fetal and maternal morbidity and mortality, its molecular etiology is still unclear.

## 1.3 单词翻译

鼠标左键双击单词，右侧展示单词翻译和例句，可以朗读发音，还能够添加单词本。

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**ions in mice**

**k,<sup>2,†</sup> Sam Tan Jian Chye,<sup>1,†</sup> Daniel M. Messerschmidt,<sup>3</sup> Ong,<sup>1</sup> Ling Ka Yi,<sup>3</sup> Souad Boussata,<sup>2</sup> Grace Hui-Yi Goh,<sup>1</sup> Lim,<sup>1</sup> N. Ray Dunn,<sup>1</sup> Davor Solter,<sup>1</sup> and Reversade<sup>1,2,3,4,\*</sup>**

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alian-specific organ from impaired migration of cardiac progenitors

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n.症状(symptom)的名词复数):征兆;心绞痛的症状可能和心脏病发作比较类似。

2 Experience flu symptoms, especially the fast attack of influenza symptoms must seek medical treatment immediately.

3 Most of swine lung plague is acute disease and pneumonic symptoms and septicemic symptoms. 其流行特征是以急性型最多,以肺炎症状和败血症症状为主,部分病畜康复后可再次感染。

## 1.4 跨段连续翻译

有些段落不在同一页面上,如果跨页选择会将页眉页脚上的不必要信息也复制过来,此时可以先选中前一页上需要翻译的文字,点击译文右上角【连翻】按钮,再选中后一页上需要翻译的文字,两段即可叠加翻译,再次点击按钮取消连续翻译。

ELABELA deficiency promotes hypoxia-induced angiogenesis in the mouse placenta

(hg) endoderm. Arrowheads indicate the start of *Ela* expression in the chorionic trophoblast. Scale bars, 100 μm. (W and X) RNAScope of *Ela* and *Apj* in E8 yolk sac layers adhering to underlying decidua. en, endoderm; me, mesoderm. Scale bars, 40 μm.

confounding transcriptional changes brought about by major cardiovascular anomalies seen at E10.5. *Ela*<sup>Δ/Δ</sup> placentas were categorized into class 1 or class 3 based on the gross morphology of the corresponding embryos (fig. S3A). RNA sequencing (RNA-seq) and principal component analysis revealed that both class 1 and 3 *Ela*<sup>Δ/Δ</sup> placentas clustered closer to each other than away from WT placentas (fig. S3B). Because class 1 placentas are grossly indistinguishable from WT counterparts, these results indicate that the observed transcriptional changes are due to ELA deficiency rather morphological defects already present at the time of specimen collection. Gene set enrichment analysis (GSEA) (7)

revealed that class 1 and 3 *Ela*<sup>Δ/Δ</sup> placentas have a gene signature indicative of an elevated hypoxic response (Fig. 3B; fig. S3, C and D; and table S1). Consistent with this observation, *Ela*<sup>Δ/Δ</sup> placentas have high levels of stabilized Hif1α (fig. S3, E and F) and decreased levels of pro-angiogenic genes (fig. S3, G and H). Concurrently, and possibly as part of the elevated hypoxic response, *Ela* deficiency results in an up-regulation of pro-angiogenic genes, even in class 1 placentas that are bereft of discernible vascular defects (fig. 3C).

A close examination of differentially regulated genes revealed a dramatic enrichment in genes and pathways defining endothelial tip cells (Fig. 3, D and E) (9). Tip cells form the leading edge of sprouting endothelial cells and migrate in response to pro-angiogenic signals (10). Functionally, in the same way as axonal growth cones, tip cells extend filopodia to determine the direction of the angiogenic sprout, whereas trailing stalk cells proliferate to enable lumogenensis and extension of the vascular sprout (11, 12). Gene ontology analysis confirmed functional hallmarks of tip cell identity such as vascular endothelial growth factor (VEGF) and semaphorin signaling, hormone secretion, axogenesis, and filopodia extension (Fig. 3D). Quantitative polymerase chain reaction (qPCR) analysis validated the

基因集富集分析(GSEA) (7)显示,第1类和第3类Ela/D/D胎盘具有指示低氧反应升高的基因特征(图3B;图S3, C和D)揭示了1类和3类Ela/D/D胎盘具有指示低氧反应升高的基因特征(图3B;图S3, C和D)揭示了1类和3类Ela/D/D胎盘具有指示低氧反应升高的基因特征(图3B;图S3, C和D)以及表S1)。与该观察结果一致,Ela/D/D胎盘具有高度稳定的Hif1α。

Gene set enrichment analysis (GSEA) (7) revealed that class 1 and 3 Ela/D/D placentas have a gene signature indicative of an elevated hypoxic response (Fig. 3B; fig. S3, C and D) revealed that class 1 and 3 Ela/D/D placentas have a gene signature indicative of an elevated hypoxic response (Fig. 3B; fig. S3, C and D) revealed that class 1 and 3 Ela/D/D placentas have a gene signature indicative of an elevated hypoxic response (Fig. 3B; fig. S3, C and D) and table S1). Consistent with this observation, Ela/D/D placentas have high levels of stabilized Hif1α.

用下面这段前言试一下吧!

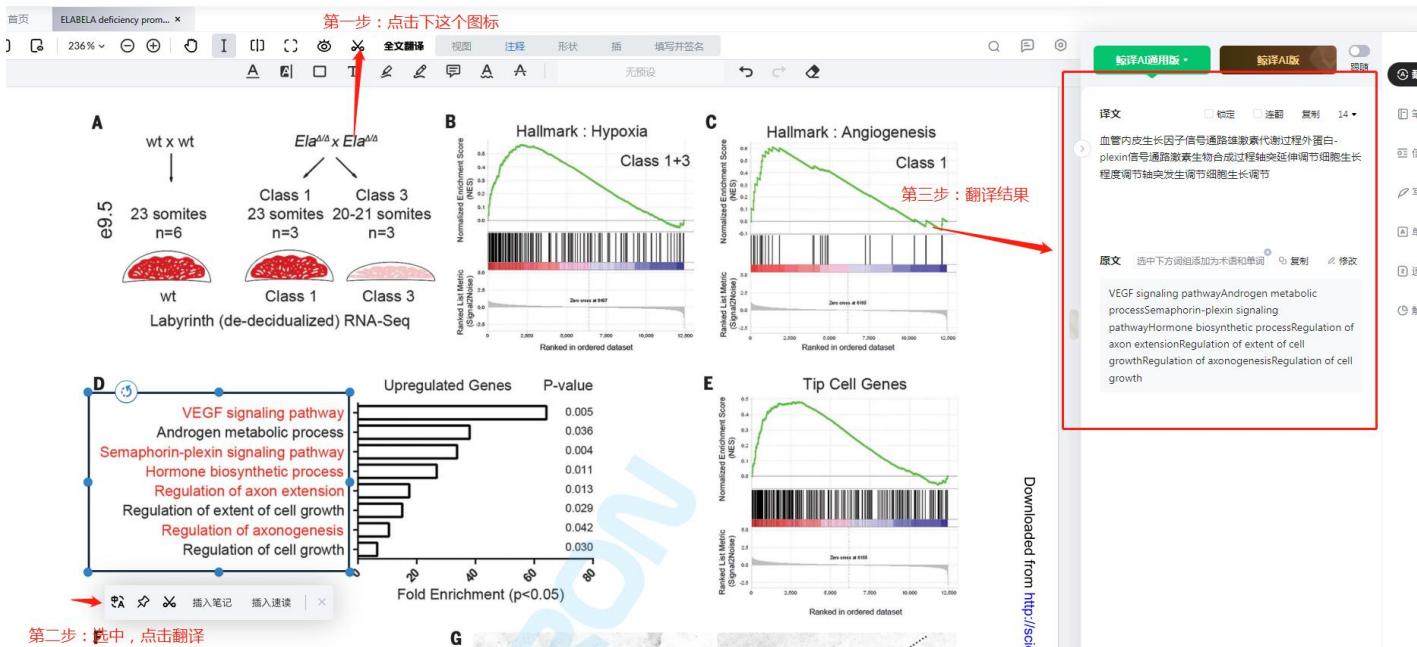
## Introduction

AKI occurs in 40% to 70% of HT patients, and it is associated with pre-existing comorbidities, right heart failure, major bleeding, or the use of CNIs. However, little is known regarding its risk factors and its association with the long-term outcomes of HT patients, particularly in those requiring dialysis. In addition, there is also limited evidence on the clinical implications of more severe kidney injury because of its low incidence (about 10%). Although there have been observational studies that evaluated the risk factors for acute dialysis, these studies were performed in a small population and were statistically underpowered to

identify significant risk factors, suggesting the need for large-scale research. Furthermore, the long-term outcomes of HT patients who required dialysis have not been elucidated. Thus, this study aimed to assess the incidence, trends, and clinical predictors of acute renal failure requiring dialysis after HT and their association with the longterm outcomes among HT patients, using a large-scale cardiac HT registry.

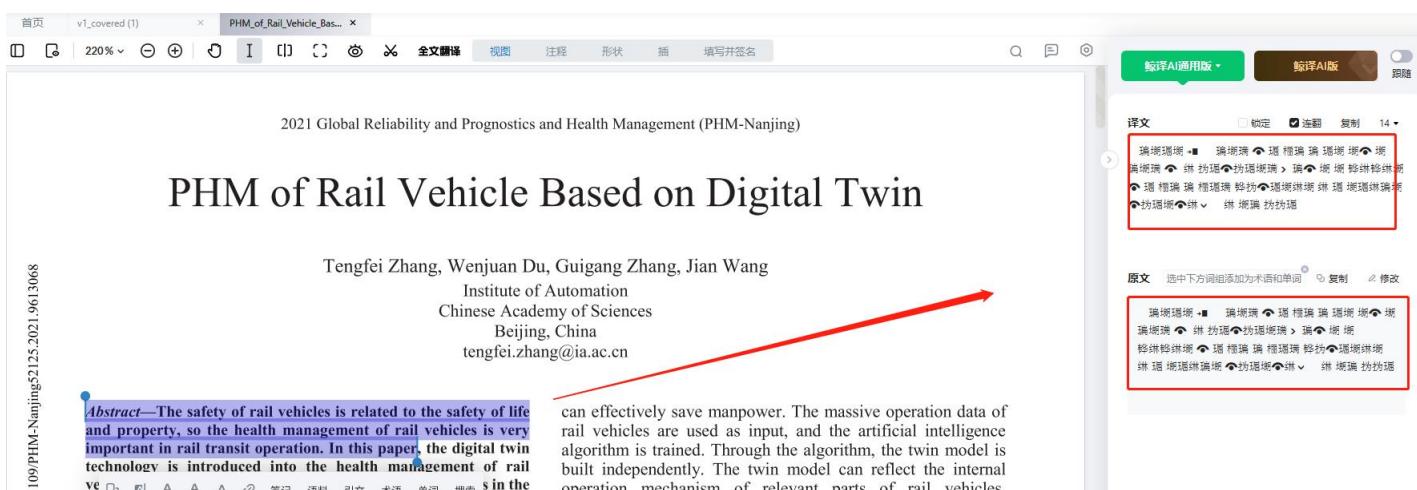
## 1.5 OCR 识别翻译

当遇到文献无法选中文本以及包含文字内容无法复制的图像区域，可以点击顶部菜单栏的【剪刀】图标，选中想要识别文字内容的区域，即可在右侧翻译栏得到可复制的原文与译文。



## 1.6 文献加密翻译

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2021 Global Reliability and Prognostics and Health Management (PHM-Nanjing)

## PHM of Rail Vehicle Based on Digital Twin

Tengfei Zhang, Wenjuan Du, Guigang Zhang, Jian Wang  
Institute of Automation  
Chinese Academy of Sciences  
Beijing, China  
tengfei.zhang@ia.ac.cn

**Abstract**—The safety of rail vehicles is related to the safety of life and property, so the health management of rail vehicles is very important in rail transit operation. In this paper, the digital twin technology is introduced into the health management of rail vehicles. Firstly, the related artificial intelligence algorithms in the field of digital Twin are combed. Then, a complex equipment health management architecture based on digital twin technology is proposed. The massive operation data of rail vehicles are used as input, and the artificial intelligence algorithm is trained. Through the algorithm, the twin model is built independently. The twin model can reflect the internal operation mechanism of relevant parts of rail vehicles, Demonstrate the status of rail vehicles and predict the future health of key components. Therefore, artificial intelligence algorithm plays an important role in digital twin technology.

can effectively save manpower. The massive operation data of rail vehicles are used as input, and the artificial intelligence algorithm is trained. Through the algorithm, the twin model is built independently. The twin model can reflect the internal operation mechanism of relevant parts of rail vehicles, Demonstrate the status of rail vehicles and predict the future health of key components. Therefore, artificial intelligence algorithm plays an important role in digital twin technology.

### 1.7 全文翻译

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The left screenshot shows the original English version of the article "An attachment perspective on loss and grief" from Current Opinion in Psychology. It includes the journal logo, author names (Mario Mikulincer and Phillip R. Shaver), abstract, and some text from the article. The right screenshot shows the same article in Chinese translation, with the title "从依恋角度看失去和悲伤" and the journal logo.

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## 2. 速读

### 2.1 图片速读

点击文献右侧【速读】，可以将文献中的图表和图注提取出来，让你快速预览所有图表中重要信息。点击【翻译图注】可以一键获取中文注释，读懂图表和文章主要内容，也可以全选下载下来进行保存。

The screenshot shows the 'Speed Reading' interface for a 'Public Health' article. On the left is the main document view, and on the right is a sidebar with a list of figures and their corresponding captions. Red arrows point to the '速读' (Speed Reading) button at the top of the sidebar and the '翻译' (Translate) button next to each figure caption. The sidebar also includes buttons for '收藏' (Bookmark), '下载' (Download), and '删除' (Delete).

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elicited by TMS delivered to the AH hotspot at 120% RMT with an interval of 3.3 s were averaged for the amplitude and latency measurements. MEPs were measured before the PAS stimulation (pre-PAS MEP), immediately (0 min), 30 min and 60 min after the PAS. The percentage ratio was calculated by dividing the post-simulation MEP (in  $\mu$ V) with the pre-stimulation MEP. The baseline MEP value was 100%. Values above 100% indicated MEP facilitation, and values below 100 % indicated MEP suppression. The stimulation parameters used for the subjects are given in Table 1.

**Table 1. Stimulation parameters used for individual subjects.**

Subjects	RMT (% SO)	PNS Intensity (mA)	F Latency (ms)	MEP Latency (ms)	ISI (ms)
S1	60	7	48.3	44.8	4
S2	55	9	54.5	42.7	12
S3	77	16	55.0	48.0	7
S4	64	13	54.0	43.6	10
S5	75	7	58.0	42.3	16

2.5. Long Intracortical Inhibition Protocol

The LICI protocol was applied with two TMS pulses delivered with a 100-ms ISI once every 3.3 s. The LICI was estimated immediately after the MEP measurements that were used to evaluate the PAS effects. The US and CS intensities were both 120% of the RMT. To evaluate the LICI effect, the MEP amplitude induced by CS was divided by the amplitude of US and multiplied by 100. MEPs recorded with the LICI protocol were analyzed with a custom-made script in Matlab 2021a (MathWorks, Inc., Natick, MA, USA).

## 2.2 参考文献速读

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Effects of repetitive transcranial magnetic stimulation on recovery in lower limb muscle strength and gait function following spinal cord injury: a randomized controlled trial

Journal: 2021 Spinal Cord

Author: Søren Krogh, P. Aagaard, A. Jønsson, K. Figlewski, H. Kasch

Abstract

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## 2.3 引证文献速读

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和摘要，快速了解参考文献主要内容。如果想要了解更多内容，可以点击文献标题，跳转文献官网去下载/查看更多。

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### 3. 笔记

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**ELABELA deficiency promotes preeclampsia and cardiovascular malformations in mice**

- 背景: ELABELA缺乏症可促进小鼠先兆子痫和心血管畸形
- 方法: 研究对象: 2000年3月25日—2022年3月25日所

B I S U A T 放大

工具栏

## 3.2 笔记模板

小绿鲸总结了一些常用的笔记模板，供大家选择，方便给大家梳理笔记思路，同时给不知道如何做文献笔记的同学一些参考，大家也可以将自己认为比较好的笔记保存为模板，方便下次直接使用哦~

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- 2 具体信息
- 3 文献基本信息
- 4 文献解构
- 5 本研究病例的亮点
- 6 写作方向
- 7 选题依据
- 8 发展脉络
- 9 本研究的实验方法
- 10 本研究的实验条件
- 11 基本信息
- 12 具体信息
- 13 基础研究

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## 3.3 思维导图

大纲式笔记可以转换为思维导图形式，更方便研究思路的梳理。

**ites**  
**scular**

**Messerschmidt,<sup>3</sup>**  
**race Hui-Yi Goh,<sup>1</sup>**

ing between 5 and 8% of all  
al morbidity and mortality,  
A (ELA), an endogenous  
mone secreted by the  
z-like symptoms, including  
al angiogenesis. In mice.

the presence of a CD31/Pecam<sup>+</sup> endothelial plexus, which subsequently fails to undergo remodeling and angiogenic sprouting to form organized vitelline vessels, dorsal aorta, outflow tract, and intersomitic vessels (Fig. 1, K to S, and fig. S1, E to J). The heart tube is poorly looped, with reduced smooth actin muscle (SMA) staining (Fig. 1, Q to S), and the most severely affected embryos (class 3) have pericardial edema (fig. S1, K and L). These cardiac defects are consistent with the first postgastrulation expression of *Ela* in the primitive foregut overlying the developing heart tube (Fig. 1, T and U) (6). Surprisingly, *Ela* is not detected in endothelial precursors of the yolk sac (Fig. 1W), whereas *Apj* expression is ubiquitous in embryonic, allantoic, and yolk sac mesoderm, which gives rise to endothelial cells (Fig. 1, V and X). The expression patterns of *Ela* and *Apj* suggest that the observed cardiac defects are partly due to insufficient blood flow to stimulate angiogenesis. Outside of the developing heart

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- 方法:  
研究对象: 2000年3月25日——2022年3月25日所  
有心血管畸形病人 分类

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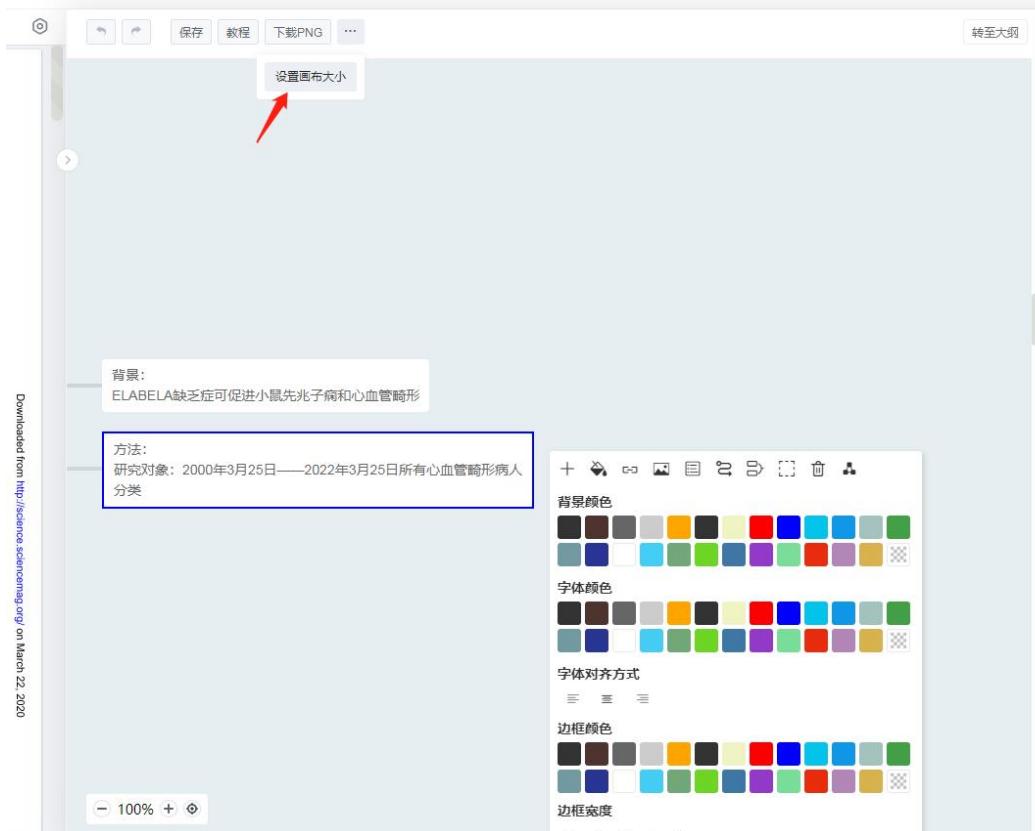
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- 背景:** ELABELA缺乏症可促进小鼠先兆子痫和心血管畸形
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• To delineate the contribution of ELA to mam- malian development, we generated Ela knockout (ElaD/D) mice using homologous recombination to delete exon 3 encoding the mature ELA peptide (Fig. 1, A and B, and fig. S1, B and C). This strategy did not

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方法：  
研究对象：2000年3月25日——2022年3月25日所有心血管畸形病人

分类：

- To delineate the contribution of ELA to mammalian development, we generated Ela knockout (Ela<sup>-/-</sup>) mice using homologous recombination to delete exon 3 encoding the mature ELA pep-tide (Fig. 1, A and B, and fig. S1, B and C). This strategy did not
- ● sia (PE) is a gestational hypertensive syndrome affecting between 5 and 8% of all pregnancies. Although PE is the leading cause of fetal and maternal morbidity and mortality, its molecular etiology is still unclear. Here, we show that ELABELA (ELA), an endogenous ligand of the apelin receptor (APLNR or APJ), is a circulating hormone secreted by the placenta. ELA is a pregnancy-associated hormone secreted by the placenta that exhibits peptide-like properties, including proteinuria and elevated blood pressure due to defective placental angiogenesis. In mice, infusion of exogenous ELA normalizes hypertension, proteinuria, and birth weight. ELA, which is expressed in the placenta and trophoblast stem cells, increases the invasiveness of trophoblast stem cells, suggesting that it enhances the APLNR signaling axis, may offer a new paradigm for the prevention of PE-related complications.

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**ELABELA deficiency promotes preeclampsia and cardiovascular malformations in mice**

背景：  
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step1

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K, Rakshit K, metabolic and Am J Physiol shed April 21, associated with diabetes mellitus influenced by regulate energy and pancreatic sed intake of ed role in the tion. Thus, in sis that dietary adoptions to nice to 12 wk with varying nt. Our results characteristic of

sity-mediated metabolic dysfunction may provide insights into prevention and treatment of T2DM.

The primary drivers of the obesity epidemic remain elusive. It is clear that genetic factors play an important role in the pathogenesis of obesity (13). However, genetic factors alone cannot account for a dramatic rise in obesity prevalence, suggesting that environmental and dietary influences likely play a contributory role (36). Alterations in diet macronutrient composition such as increased saturated fat content have been shown to promote adiposity and development of T2DM (36). In support of this premise, chronic exposure to saturated free fatty acids (i.e., lipotoxicity) recapitulates many pathophysiological aspects of obesity and T2DM, such as hepatic/skeletal muscle insulin resistance (31), adipose tissue dysfunction (36), and  $\beta$ -cell failure (30).

Conversely, evidence also suggests that increased intake of dietary carbohydrates and triglycerides play a

induced obesity

● is increased saturated fat content have been shown to promote adiposity and development of T2DM (36). In support of this premise, chronic exposure to saturated free fatty acids (i.e., lipotoxicity) recapitulates many pathophysiological aspects of obesity and T2DM, such as hepatic/skeletal muscle insulin resistance (31), adipose tissue dysfunction (36), and  $\beta$ -cell failure (30).

● eased intake of refined sugars has additional deleterious effects on promotion of adipogenesis, insulin resistance, and  $\beta$ -cell dysfunction (35).

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incidental development to prevent PE. The ELA-APLNR signaling axis  
the treatment of common pregnancy-related complications.

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from h  
red migration of cardiac progenitors  
(2, 3). Zebrafish lacking both Ela and Apelin  
(Apnl), the alternate ligand for Apnl, have defects  
in vasculogenesis owing to impaired migration  
of angioblasts to the midline (4). At present, the  
molecular effects of ELA signaling downstream  
of APLNR are unknown, and its involvement in  
mammalian development and physiology has  
not been addressed.

**to determine the role of Ela in mammalian development was generated Ela knockout**

Strategy did not result in nonsense-mediated decay of the *Ela<sup>+/+</sup>* mRNA (Fig. 1C) and presumably preserves the potential noncoding functions of the *Ela* transcript (5). Only half of the expected *Ela<sup>Δ/Δ</sup>* mice from heterozygous intercrosses were obtained at weaning (Fig. 1D) ( $P < 0.001$ , chi-square test with  $df = 1$ ). Notably, this reduced recessive Mendelian inheritance was even more pronounced for *Ela<sup>Δ/Δ</sup>* embryos carried by *Ela<sup>Δ/Δ</sup>* mothers (67%) than by *Ela<sup>Δ/+</sup>* mothers (31%) (Fig. 1D). This apparent maternal contribution is not due to *Ela* mRNA being deposited in the oocyte, because the onset of *Ela* transcription  
after allantoic fusion (Fig. 2A), becoming restricted to syncytiotrophoblasts (STs) at E10.5 (Fig. 2, C and C'). Accordingly, ELA protein is detected by immunohistochemistry in wild-type (WT) STs but not in *Ela<sup>Δ/Δ</sup>* placentas (Fig. 2, E and F). ELA-positive STs are juxtaposed to *Apnl*-expressing fetal endothelial cells (Fig. 2, B, D, and D'). Hence, ELA may signal to *Apnl*-expressing cells in a paracrine manner but may also be circulating systemically because the choriovillous placenta is perfused by maternal and fetal blood. Indeed, endogenous ELA is detected by enzyme-linked immunosorbent assay (ELISA) in the serum of pregnant females, peaking at midgestation, but not in nonpregnant mice (Fig. 3). Systemic ELA in a pregnant mother is contributed both maternally and embryonically (Fig. 3), the former reflecting secretion from the maternal endometrial stroma and kidneys (Fig. S2, A to C) and the latter from embryonically derived STs (Fig. 2C). We therefore conclude that ELA is a pregnancy-associated hormone secreted by the developing conceptus and placenta.

*Ela<sup>Δ/Δ</sup>* placentas from affected embryos have thin labyrinth (Fig. 2, I and J, and fig. S2, D and E) with poor vascularization (Fig. 2, K and L), increased apoptosis (Fig. S2, F and G), and reduced proliferation (Fig. S2, H and I). *Ela<sup>Δ/Δ</sup>* placentas from unaffected (class 1) or mildly affected (class 2) embryos, which are intermediate vascularized, nonetheless exhibit delayed

from h  
red migration of cardiac progenitors  
(2, 3). Zebrafish lacking both Ela and Apelin  
(Apnl), the alternate ligand for Apnl, have defects  
in vasculogenesis owing to impaired migration  
of angioblasts to the midline (4). At present, the  
molecular effects of ELA signaling downstream  
of APLNR are unknown, and its involvement in  
mammalian development and physiology has  
not been addressed.

**ELA normalizes hyp**

• **sia (PE) is a gestational hypertensive syndrome affecting between 5 and 8% of a**  
and maternal morbidity and mortality, its molecular etiology is still unclear. Here, we  
apelin receptor (APLNR, or APJ), is a circulating hormone secreted by the placenta.  
PE-like symptoms, including proteinuria and elevated blood pressure due to defectiv

ELA normalizes hy

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pathogenesis of obesity (13). However, genetic factors alone cannot account for a dramatic rise in obesity prevalence, suggesting that environmental and dietary influences likely play a contributory role (36). Alterations in diet macronutrient composition such as increased saturated fat content have been shown to promote adiposity and development of T2DM (36). In support of this premise, chronic exposure to saturated free fatty acids (i.e., lipotoxicity) recapitulates many pathophysiological aspects of obesity and T2DM, such as hepatic/skeletal muscle insulin resistance (31), adipose tissue dysfunction (36), and  $\beta$ -cell failure (30).

Conversely, evidence also suggests that increased intake of dietary carbohydrate (笔记 **添加标注至笔记**) erinsulinemia play a previously underappreciated role in the promotion of obesity and consequent metabolic dysfunction (18, 26). This view is supported by evidence that increased carbohydrate intake

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eased intake of refined motion of adipogenesis Furthermore, evidence  
hydrate diets, even in the ric intake (e.g., ketogen energy expenditure as ose homeostasis (11, 2

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后论文【写作】使用。

## 4. 写作

### 4.1 引文收藏、语料收藏

选取想要收藏的句子，点击下方工具栏中的【添加引文】和【添加语料】即可添加到引文库和语料库。  
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Dietary carbohydrates modulate metabolic and  $\beta$ -cell adaptation to high-fat diet-induced obesity

Tracy K. Her,<sup>1\*</sup> William S. Lagakos,<sup>1\*</sup> Matthew R. Brown,<sup>1</sup> Nathan K. LeBrasseur,<sup>2</sup> Kuntol Rakshit,<sup>1</sup> and Aleksey V. Matveyenko<sup>1,3</sup>

<sup>1</sup>Department of Physiology and Biomedical Engineering, Mayo Clinic School of Medicine, Rochester, Minnesota; <sup>2</sup>Department of Physical Medicine and Rehabilitation, Mayo Clinic, Rochester, Minnesota; and <sup>3</sup>Department of Medicine, Division of Endocrinology, Metabolism, Diabetes, and Nutrition, Mayo Clinic School of Medicine, Rochester, Minnesota

Submitted 10 December 2019; accepted in final form 15 April 2020

Her TK, Lagakos WS, Brown MR, LeBrasseur NK, Rakshit K, Matveyenko AV. Dietary carbohydrates modulate metabolic and  $\beta$ -cell adaptation to high-fat diet-induced obesity. *Am J Physiol Endocrinol Metab* 318: E856–E865, 2020. First published April 21, 2020; doi:10.1152/ajpendo.00539.2019.—Obesity is associated with several chronic comorbidities, one of which is type 2 diabetes mellitus (T2DM). The pathogenesis of obesity and T2DM is influenced by alterations in diet macronutrient composition, which regulate energy expenditure, metabolic function, glucose homeostasis, and pancreatic islet cell biology. Recent studies suggest that increased intake of dietary carbohydrates plays a previously underappreciated role in the

sity-mediated metabolic dysfunction may provide insights into prevention and treatment of T2DM.

The primary drivers of the obesity epidemic remain elusive. It is clear that genetic factors play an important role in the pathogenesis of obesity (13). However, genetic factors alone cannot account for the dramatic increase in obesity rates, suggesting that environmental and dietary influences likely play a contributory role (36). Alterations in diet macronutrient composition such as increased saturated fat content have been shown to promote adiposity and development of T2DM (36).

viewed, imaged, and analyzed using a Zeiss Axio Observer Z1 microscope (Carl Zeiss Microscopy, LLC) and ZenPro software (Carl Zeiss Microscopy).

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According to the former study, it showed/demonstrated/found that tal body lean and fat mass was assessed at study end point by quantitative magnetic resonance (EchoMRI,100). Assessment of oral glucose tolerance, in vivo insulin secretion, and insulin tolerance. To assess oral glucose

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The screenshot shows the Xilajing English Literature Reader interface. On the left, there's a sidebar with options like 'Recent阅读', 'My Documents', 'Notes', 'Search', 'Tags', 'Recycling Bin', and 'Journal Scan'. The main area displays a list of documents. One document is selected, showing its details: 'American Journal of Physiology-Endocrinology and Metabolism', volume 318, pages E856–E865, 2020. The right side has a 'Citation' panel with fields for page numbers, journal, impact factor, etc., and an 'Export' section with buttons for BibTeX, MLA, APA, and Chicago.

除了以上这些，小绿鲸还有【其他】一些实用的小功能。

## 5. 其他

### 5.1 对比阅读

点击上方菜单栏【对比阅读】可选择打开一篇文献，与当前文献进行内容对比阅读，两篇文献在同一界面打开，可以分别左右滑动查看。

This screenshot shows the toolbar of the Xilajing English Literature Reader. It includes icons for zoom (224%), orientation, text size, font style, and various reading and annotation features. A red arrow points to the 'Compare' icon, which allows users to open and compare multiple documents side-by-side.

*Am J Physiol Endocrinol Metab* 318: E856–E865, 2020.  
First published April 21, 2020; doi:10.1152/ajpendo.00539.2019.

**文献一**

**RESEARCH ARTICLE**

Dietary carbohydrates modulate metabolic and  $\beta$ -cell adaptation to high-fat diet-induced obesity

Tracy K. Her,<sup>1\*</sup> William S. Lagakos,<sup>1\*</sup> Matthew R. Brown,<sup>1</sup> Nathan K. LeBrasseur,<sup>2</sup> Kuntol Rakshit,<sup>1</sup> and Aleksey V. Matveyenko,<sup>1,3</sup>

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Conversely, evidence also suggests that increased intake of dietary carbohydrates plays a previously underappreciated role in the promotion of obesity (14,15). In addition to dietary factors, this study, we utilized mouse models to test the hypothesis that dietary carbohydrates modulate energetic, metabolic, and  $\beta$ -cell adaptations to high-fat diets. To address this, we exposed C57BL/6 mice to 12 wk of 3 enteral, high-fat diets (C-60% fat) ranging from 10 to 40% total carbohydrates (1–20%) to increase dietary complexity. Our results show that severe restriction of dietary carbohydrates characteristic of ketogenic diets reduces body fat accumulation, enhances energy expenditure, and reduces prevailing glycemia and insulin resistance compared with carbohydrate-rich diets. Moreover, severe restriction of dietary carbohydrates also results in functional, morphological, and molecular changes in pancreatic islets highlighted by restricted capacity for  $\beta$ -cell mass expansion and alterations in insulin secretory response. These studies support the hypothesis that low-carbohydrate/high-fat diets provide antidiabetogenic benefits and suggest further evaluation of the effects of these diets on  $\beta$ -cell biology in humans.

$\beta$ -cell; high-fat diet; insulin; ketogenic diet; obesity

**INTRODUCTION**

Obesity is a major health problem currently facing society, attributed largely to its causative association with metabolic

ity-mediated metabolic dysfunction may provide insights into prevention and treatment of T2DM. The primary drivers of the obesity epidemic remain elusive. It is clear that genetic factors play an important role in the pathogenesis of obesity (13). However, genetic factors alone cannot account for a dramatic rise in obesity prevalence, suggesting that environmental and dietary influences likely play a contributory role (36). Alterations in diet macronutrient composition such as increased saturated fat content have been shown to contribute to the development of T2DM (36). In support of this premise, direct exposure to free fatty acids (i.e., lipotoxicity) recapitulates many pathophysiological aspects of obesity and T2DM, such as pancreatic/skeletal muscle insulin resistance (31), adipose tissue dysfunction (36), and  $\beta$ -cell failure (30).

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**文献二**

Preprints are preliminary reports that have not undergone peer review. They should not be considered conclusive, used to inform clinical practice, or referenced by the media as validated information.

Does A One-Session Sexual Health Education Program Improve Sexual Confidence in Patients with Cervical Cancer? A Transtheoretical Model-based Clinical Study

Tao-Hsin Tung  
Hsiao-Wei Chen  
Hung-Huei Chou  
Jia-Ling Tsai  
Ya-Chieh Yang  
Jian Tao Lee (✉ jtlee@gap.cgu.edu.tw)  
Chang Gung University https://orcid.org/0000-0002-0144-9681

**Research Article**

**Keywords:** sexual self-efficacy, sexuality, health education, Transtheoretical Model, cervical cancer, gynecological cancer

**Posted Date:** January 11th, 2023

**DOI:** <https://doi.org/10.21203/rs.3.rs-2441010/v1>

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## 5.2 丰富的文献批注

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首页 Dietary carbohydrates m... ×

224% 148% 全文翻译 视图 **注释** 形状 插 填写并签名

Am J Physiol Endocrinol Metab 318: E856–E865, 2020.  
First published April 21, 2020; doi:10.1152/ajpendo.00539.2019.

**RESEARCH ARTICLE**

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## 5.3 文献共读

如果想要分享文献给其他小伙伴，只需点击右上角【设置】按钮，再点击【分享】即可。

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文献的数量越来越多，这时通过小绿鲸的【文献管理】功能，就可以从上百篇文献中快速找到目标文献，并回忆起文献重点信息。

## 5.4 图片钉住

有的时候想要图片悬浮在文献之上，那我们就可以点击顶部菜单栏的【剪刀】图标，选中自己所需要悬浮的图片，点击钉住的图标，就可以获取到悬浮的图片了。

**Fig. 1. Zygotic deletion of *Ela* causes midgestation lethality due to cardiovascular defects and phenocopies loss of *Apj*.**

(A) Exon 3 of murine *Ela* was flanked with loxp sites and excised with cre recombinase to generate the *Ela*<sup>Δ</sup> allele lacking the ELA mature peptide (MP) coding region.

(B) Schematic of cDNA from WT and *Ela*<sup>Δ</sup> alleles. SP, signal peptide.

(C) Semi-qPCR of *Ela* locus from genomic DNA (gDNA) and cDNA. Primer locations are indicated in (B).

(D) Distribution of genotypes at E10.5 and at weaning from intercrosses and *Ela*<sup>Δ/Δ</sup> (mother) x *Ela*<sup>+/Δ</sup> (father) crosses. %P, penetrance; L, number of litters. Data were tested using a chi-square test with 1 degree of freedom for significant deviation from the expected distribution.

(E to G) At E10.5, *Ela*<sup>+/Δ</sup> embryos are indistinguishable from WT, whereas 43% ( $n = 22$ ) of *Ela*<sup>Δ/Δ</sup> embryos and 14% ( $n = 3$  of 22) of *Apj*<sup>Δ/Δ</sup> embryos display cardiovascular defects along with IUGR. Scale bars, 1 mm.

(H to J) At E10.5, *Ela*<sup>+/Δ</sup> yolk sacs have normal vitelline vessels, whereas affected *Ela*<sup>Δ/Δ</sup> and *Apj*<sup>Δ/Δ</sup> embryos have avascular yolk sacs with a ruffled appearance. Scale bars, 1 mm.

(K to M) CD31 staining of *Ela*<sup>+/Δ</sup>, *Ela*<sup>Δ/Δ</sup>, and *Apj*<sup>Δ/Δ</sup> yolk sacs reveals poorly matured vasculature in mutant embryos. Scale bars, 50  $\mu$ m.

(N to P) CD31 staining of *Ela*<sup>+/Δ</sup>, *Ela*<sup>Δ/Δ</sup>, and *Apj*<sup>Δ/Δ</sup> head vasculature at E10.5.

	+/+	Δ/+	Δ/Δ	L	p-value	%P
e10.5	7	22	12	4	0.23	0
Wean	45	102	22	29	0.0002	51

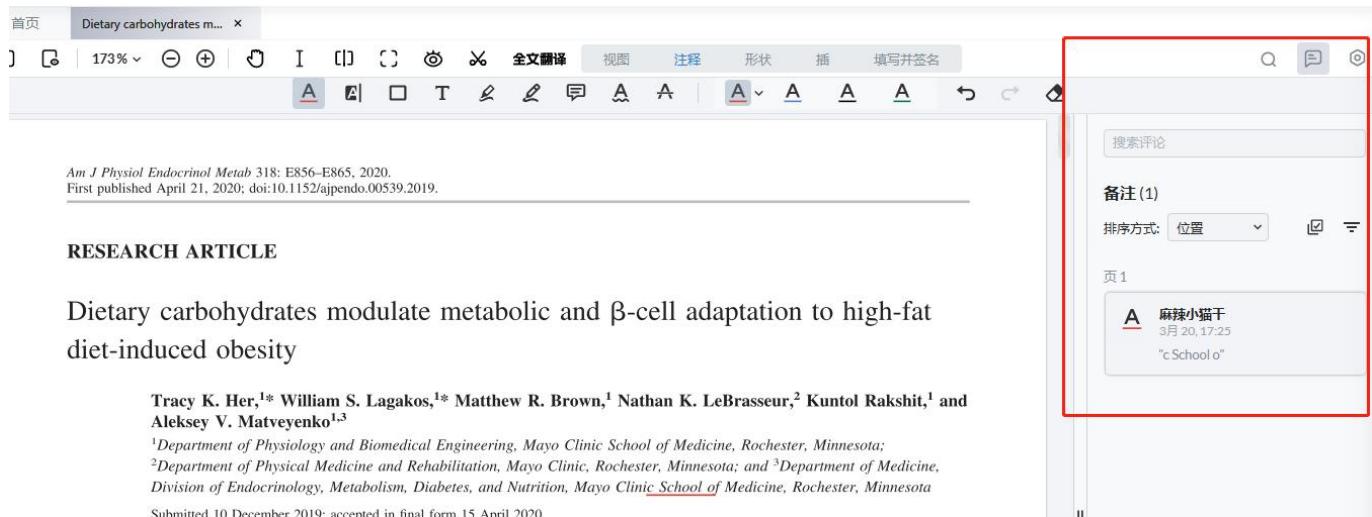
  

	Δ/+	Δ/Δ	L	p-value	%P
e10.5	45	35	9	0.26	22
Wean	73	24	17	1E-05	67

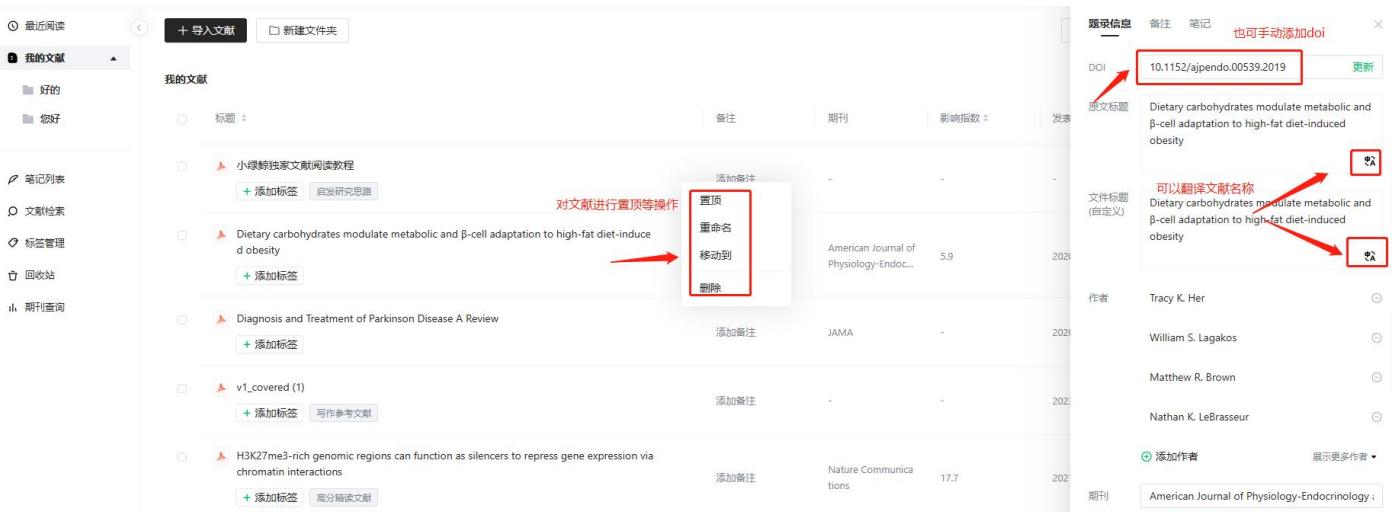
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## 6.1 标签、备注、标记重要度、信息预览

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v1_covered (1)	添加备注			2023	2023-03-20 17:02:53
H3K27me3-rich genomic regions can function as silencers to repress gene expression via chromatin interactions	添加备注	Nature Communications	17.7	2021	2023-03-20 17:02:58
Distributionally Consistent Simulation of Naturalistic Driving Environment for Autonomous Vehicle Testing	添加备注				2023-03-20 17:02:41

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## 7. 文献检索

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Nat Cell Biol. 2019 Jun;21(6):792. doi: 10.1038/s41556-019-0316-3.  
PMID: 30914825

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Regulatory T cell-derived Interleukin-10 Limits Inflammation at Environmental Interfaces

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and it therefore represents the best marker to date for this cell subset (Fontenot et al., 2003). Furthermore, genetic deficiency in Foxp3 leads to a highly aggressive fatal lymphoproliferative disease in mice, which is characterized by a massive lymphoid ablation of Treg cells in neonatal or adult mice resulting in a similar disease (Fontenot and Rudensky, 2006; Kim et al., 2007). In humans, Foxp3 deficiency in development of an autoimmune disease called IPEX syndrome (IPEX, Ichthyosis, Polyuria, Endocrinopathy, Enteropathy, X-linked): Autoimmune lesions associated with the Foxp3 deficiency are characterized by extensive infiltration of CD4+ T cells in the skin, gut mucosal layer of the skin and gastrointestinal tract, as well as humoral immune-mediated inflammation of multiple organs, including the brain, heart, lungs, kidneys, liver, and lungs (Birukow et al., 2007; Wilson and Fritsch, 2009). Interestingly, the absence of Foxp3 expression is necessary for suppressor function. Genetically marked cells expressing the Foxp3 gene lacking expression of Foxp3 protein fail to suppress T cell responses both *in vivo* and *in vitro* (Gavin et al., 2007; Lin et al., 2007). Furthermore, ablation of Foxp3 gene expression in mature peripheral T cells results in loss of their regulatory function (Wilson and

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