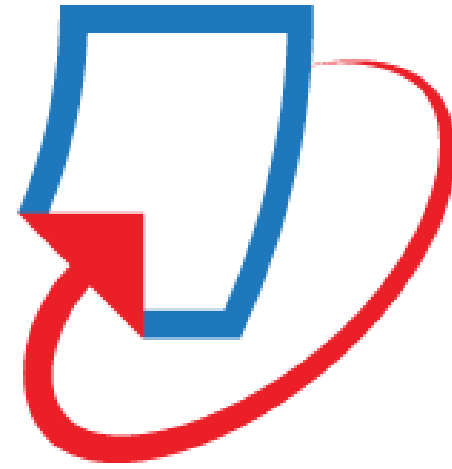




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# The Plagiarism Spectrum

The Plagiarism Spectrum identifies 10 types of plagiarism based on findings from a worldwide survey of nearly 900 secondary and higher education instructors. Each type has been given an easy-to-remember moniker to help students and instructors better identify and discuss the ramifications of plagiarism in student writing.

[www.turnitin.com/static/plagiarism-spectrum](http://www.turnitin.com/static/plagiarism-spectrum)

2021

# The Plagiarism Spectrum 2.0



The Plagiarism Spectrum 2.0 identifies twelve types of unoriginal work. Familiarity with traditional forms of plagiarism and emerging trends helps students develop original thinking skills and do their best original work.

<p><b>Original Thinking</b></p> <p>When someone submits assignments that are their own work, composed of original ideas built on attributed sources.</p>	<p><b>Student Collusion</b></p> <p>Working with other students on an assignment meant for individual assessment.</p>	<p><b>Word-for-Word Plagiarism</b></p> <p>Copying and pasting content without proper attribution.</p>	<p><b>Self Plagiarism</b></p> <p>Reusing one's previously published or submitted work without proper attribution.</p>	<p><b>Mosaic Plagiarism</b></p> <p>Weaving phrases and text from several sources into one's own work. Adjusting sentences without quotation marks or attribution.</p>	<p><b>Software-based Text Modification</b></p> <p>Taking content written by another and running it through a software tool (text spinner, translation engine) to evade plagiarism detection.</p>	<p><b>Contract Cheating</b></p> <p>Engaging a third party (for free, for pay, or in-kind) to complete an assignment and representing that as one's own work.</p>
<p><b>Inadvertent Plagiarism</b></p> <p>Forgetting to properly cite or quote a source or unintentional paraphrasing.</p>	<p><b>Paraphrase Plagiarism</b></p> <p>Rephrasing a source's ideas without proper attribution.</p>	<p><b>Computer Code Plagiarism</b></p> <p>Copying or adapting source code without permission from and attribution to the original creator.</p>	<p><b>Source-based Plagiarism</b></p> <p>Providing inaccurate or incomplete information about sources such that they cannot be found.</p>	<p><b>Manual Text Modification</b></p> <p>Manipulating text with the intention of misleading plagiarism detection software.</p>	<p><b>Data Plagiarism</b></p> <p>Falsifying or fabricating data or improperly appropriating someone else's work, putting a researcher, institution, or publisher's reputation in jeopardy.</p>	

## New Plagiarism Spectrum

[www.turnitin.com/resources/plagiarism-spectrum-2-0](http://www.turnitin.com/resources/plagiarism-spectrum-2-0)

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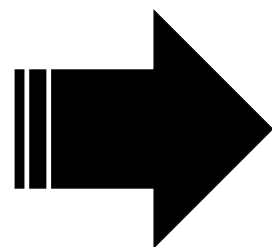
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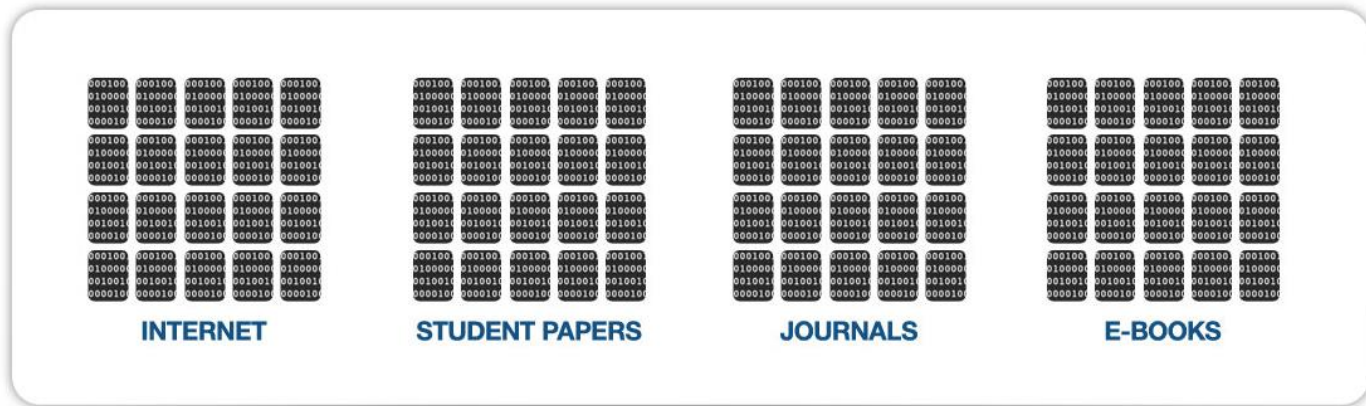


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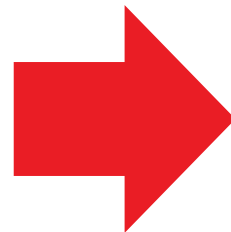
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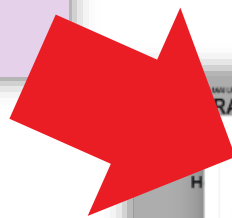
สวัสดีค่ะ คุณ [redacted] โปรดแจ้งวัตถุประสงค์การใช้งานในครั้งนี้อย่างละเอียดด้วยค่ะ  
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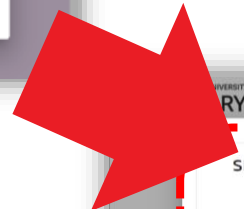


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01

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02

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บัญชีสถานะ  
Students

03

สำหรับผู้ที่มีบัญชี  
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แต่ต้องการใช้ใน  
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Please note that the key and pincode are case-sensitive. If you do not have this information, or the information you are entering appears to be incorrect, please contact your instructor.

Class ID

Class enrollment key



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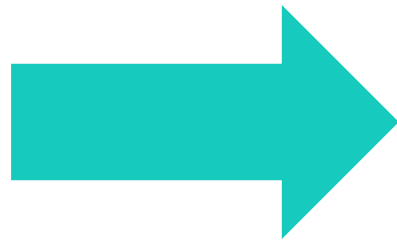
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Class ID	Class name	Instructor	Status	Start Date	End Date	Drop class
28693756	<b>(CMUL) Check Plagiarism : October 2022</b>	<b>CMU Library</b>	Active	15-Mar-2021	31-Jul-2021	
28630955	April 2021	Surintha Lasakun	Expired	09-Mar-2021	30-Apr-2021	

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28694031	<a href="#">(CMUL) Check Plagiarism : January 2022</a>	CMU Library	Expired	15-Mar-2021	01-Feb-2022	
28708220	<a href="#">(CMUL) Check Plagiarism : March 2022</a>	CMU Library	Expired	16-Mar-2021	01-Apr-2022	

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Enrollment key: \*

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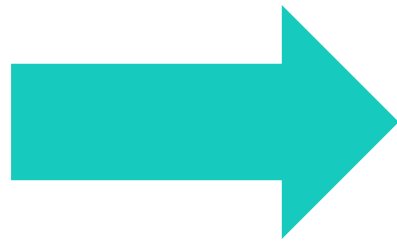
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32983930	(CMUL) Check Plagiarism : October 2022	CMU Library	Active	04-Jan-2022	02-Nov-2022	
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Student

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+ Add Class

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Class ID	Class name	Status	Start Date	End Date	Class Summary	Learning Analytics	Edit	Copy	Delete
28615472	Case study	Active	07-Mar-2021	31-Dec-2023					
32983930	(CMUL) Check Plagiarism : October 2022	Active	04-Jan-2022	02-Nov-2022					

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28693984	<a href="#">October 2021</a>	CMU Library	Expired	15-Mar-2021	31-Oct-2021	
28694031	<a href="#">(CMUL) Check Plagiarism : January 2022</a>	CMU Library	Expired	15-Mar-2021	01-Feb-2022	
28708220	<a href="#">(CMUL) Check Plagiarism : March 2022</a>	CMU Library	Expired	16-Mar-2021	01-Apr-2022	

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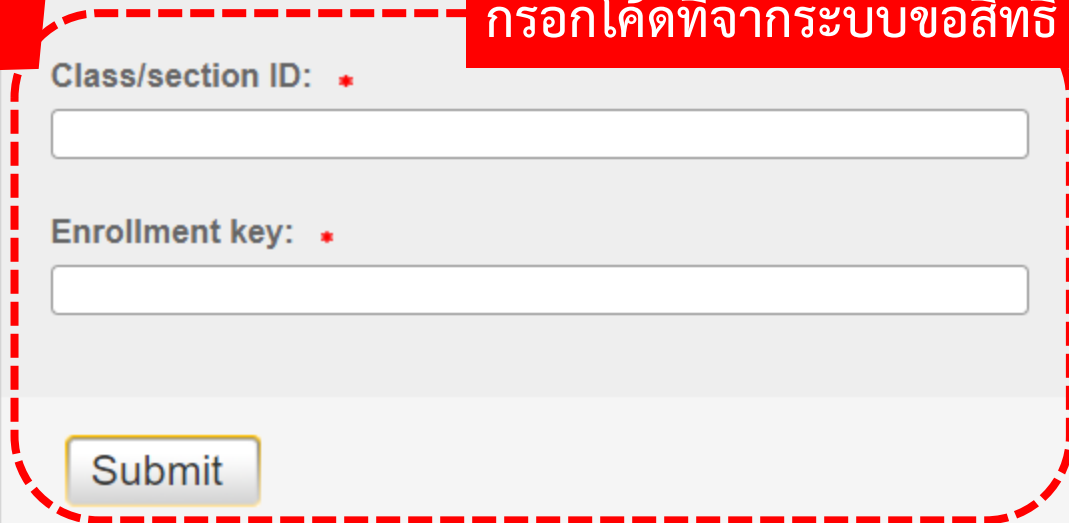
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Class/section ID: \*

Enrollment key: \*

Submit

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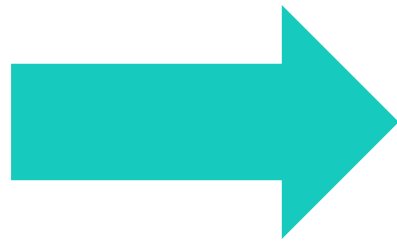
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28708220	(CMUL) Check Plagiarism : March 2022	CMU Library	Expired	16-Mar-2021	01-Apr-2022	

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






































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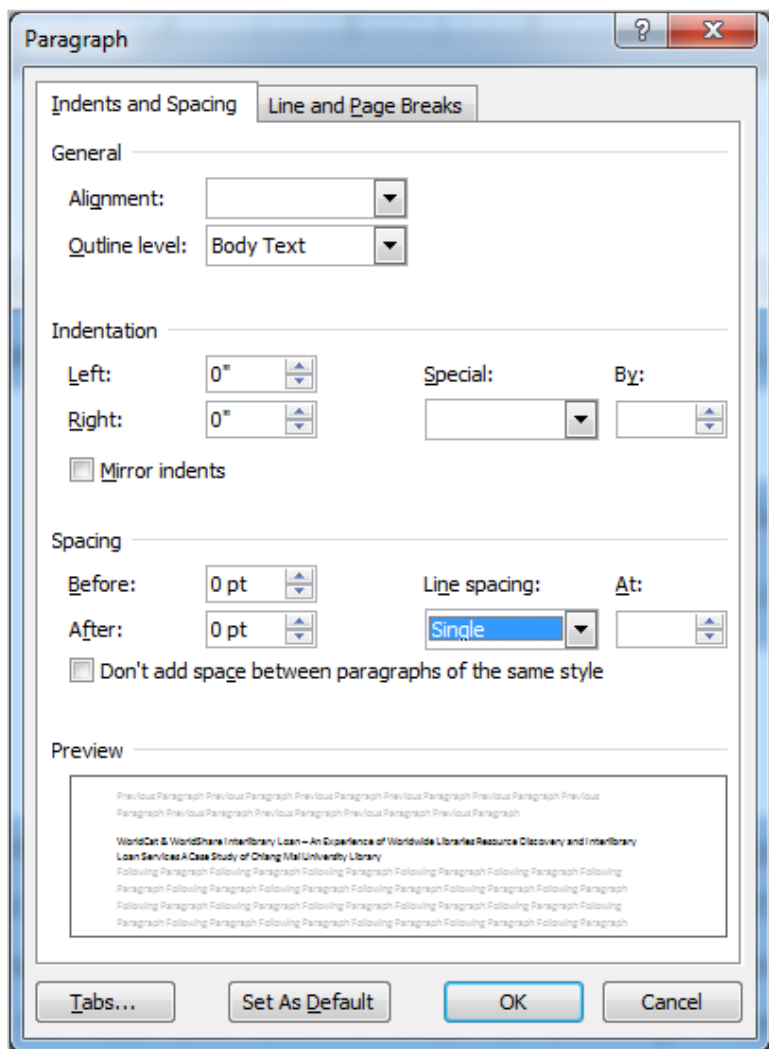
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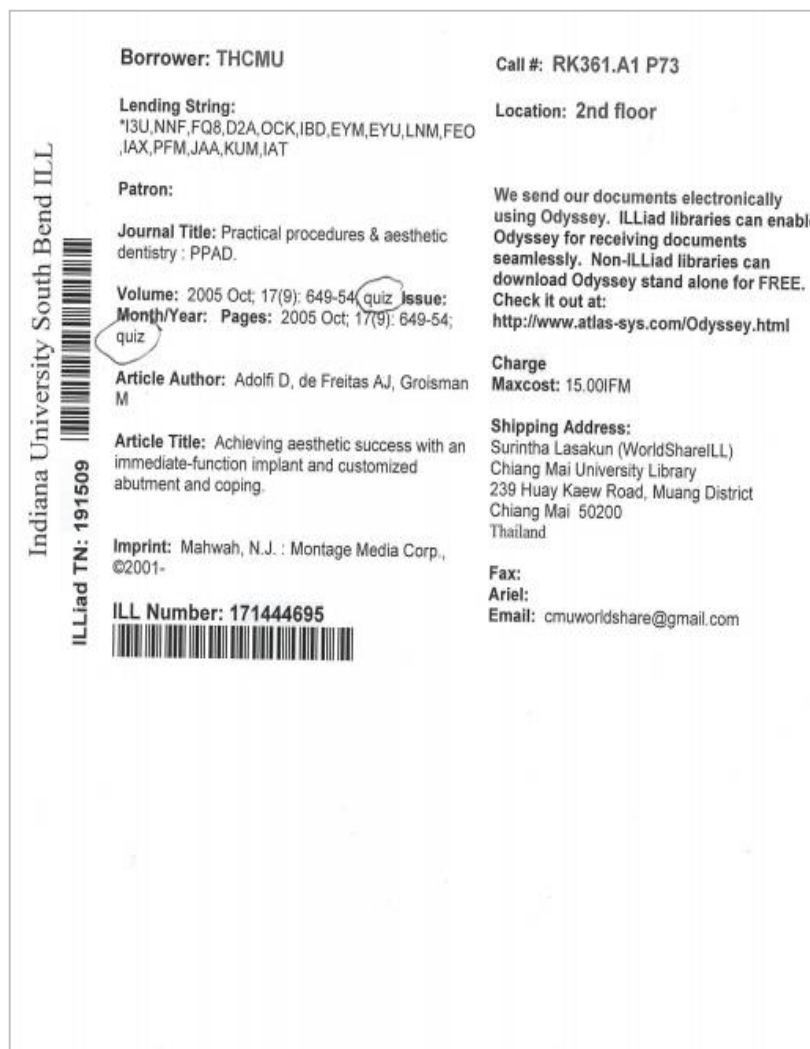
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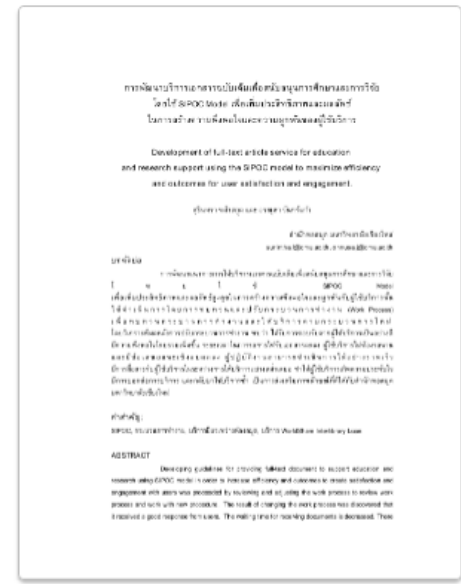
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รายละเอียดผลการตรวจ

68

ABSTRACT

The neurodegeneration, as indicated by brain dysfunction and cognitive complications following obesity and estrogen deprivation. Calorie restriction improved brain function in the neurodegenerative diseases. However, the comparative effects of a combined calorie restriction with exercise, calorie restriction, and exercise regime on brain/cognitive functions in the obesity with or without estrogen deprivation have not been investigated. We hypothesized that a combined therapy has greater benefit on rescuing brain/cognitive functions than monotherapy in both obesity with and without estrogen deprivation. Sixty female rats were nourished with a normal diet (ND) or a high-fat diet (HFD) for 27 weeks. At week 13, the ND-fed rats were assigned to a sham operation with sedentary lifestyle, and HFD-fed rats were assigned to be two groups: either a sham operation (HFS) or ovariectomy (HFO). At week 20, HFD-fed rats in each group were divided into four subgroups to receive either sedentary lifestyle, calorie restriction, exercise regime or a combination of calorie restriction and exercise for 7 weeks. Insulin resistance, cognitive decline and hippocampal pathologies were found in both HFS and HFO rats. HFO rats had higher level of insulin resistance and hippocampal ROS level than HFS rats. Calorie restriction decreased metabolic disturbance, decreased hippocampal oxidative stress, but failed to attenuate cognitive decline in HFS and HFO rats. Exercise attenuated metabolic/hippocampal dysfunctions, resulting in improved cognition in only HFS rats. Combined therapies restored brain function, and cognitive function in HFS and HFO rats. Therefore, a combined calorie restriction with exercise should be the greatest lifestyle modification to diminish brain pathologies and cognitive decline in obesity with or without estrogen deprivation.

Keywords: Obesity; Ovariectomy; Brain function; Caloric restriction; Exercise

1 Introduction

Several studies reported that excessive energy intake in conjunction with a sedentary lifestyle developed obesity, which can lead to develop pathophysiological conditions such as insulin resistance, type II diabetes, cardiovascular disease, and cognitive dysfunction [1-3]. Similarly, our previous studies established that long-term high-fat diet (HFD) consumption developed obesity, insulin resistance, brain oxidative stress, brain apoptosis, and synaptic dysfunction, leading to cognitive impairment [4-7]. Furthermore, an increase in the incidence of obesity, metabolic syndrome, and the neurodegenerative

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## ABSTRACT

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Lifestyle modification, including dietary intervention or exercise, has useful effects on the enhancement of metabolic and brain functions in cases of obesity, sex hormone deprivation, and aging [18-22]. A previous study found that calorie restriction had a beneficial impact on metabolic effects including reduced visceral adipose tissue deposition and hepatic glucose levels in obese rats [18]. In addition, calorie restriction inhibited the impaired insulin signaling in the skeletal muscle of ovariectomized model



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Duangkamol Mantor, Wasana Pratchayasakul, ...  
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ts under conditions of HFD-induced obese-insulin resistance. To study the combined effects of oophorectomy and obesity, many previous studies used the model of oophorectomy followed by obesity. Those studies and our previous study showed that obesity aggravated peripheral insulin resistance in the oophorectomy, as indicated by increased plasma insulin, increased hyperglycemia, increased HOMA index and impaired glucose tolerance, when

oxidative stress, brain apoptosis, and synaptic dysfunction, leading to cognitive impairment [4-7]. Furthermore, an increase in the incidence of obesity, metabolic syndrome, and the neurodegenerative disorders occurred during estrogen-deprived periods [8-10]. Previous studies and our study found that obesity aggravates the metabolic disturbance, cognitive decline, and brain pathology in estrogen-deprived rats [11-13]. Although estrogen supplement has been shown to attenuate the metabolic and brain dysfunction in estrogen-deprived condition [14-17], estrogen itself can lead to several adverse effects. Therefore, alternative therapies, which have less adverse effects and can reduce metabolic disturbance and brain dysfunction in estrogen-deprived condition, such as lifestyle modification, have become the fascinating topics for investigation.

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Hippocampal oxidative stress level subsequently decreased apoptosis, synaptic dysplasticity, and dendritic spine loss in hippocampus, which leads to improved cognition in HFD-fed group. Although we showed that exercise regime could enhance cognition, this enhancement did not recover the cognition to the basal level. A probable elucidation is that exercise regime did not completely recover brain function in HFD-fed rats, therefore exercise regime could not recover cognition in this condition. Although exercise regime also attenuated oxidative stress level in hippocampus of HFD-fed rats with estrogen deprivation, this oxidative stress level was still high. Therefore, we speculated that the high hippocampal oxidative stress level may decrease the beneficial effect of exercise on the attenuating of hippocampal apoptosis, synaptic dysplasticity and dendritic spine loss in HFD-fed rats with estrogen deprivation. Consequently, exercise failed to improve cognition in HFD-fed rats with estrogen deprivation.

Interestingly, we found that a combined calorie restriction with exercise regime over a short duration conferred the better profits on the decrement of hippocampal pathology and cognitive decline than monotherapy in both HFD-fed group with or without estrogen deprivation. It is possible that the additive effects of the combined therapies have the highest benefit on the reduction of ROS production in hippocampus, leading to the highest benefit on the decrement of apoptosis, synaptic dysplasticity and dendritic spine loss in both HFD-fed group with or without estrogen deprivation. All of these mechanisms are appropriate to restore cognition in sham-HFD-fed group and decrease cognitive decline in ovariectomized HFD-fed group following the combined therapy regime. Furthermore, a combined therapy exerted the greater attenuation in hippocampal pathologies than estrogen therapy, which lead to improve cognition better than estrogen therapy in HFD-fed rats with estrogen deprivation. Interestingly, estrogen therapy decreased only metabolic disturbance and hippocampal oxidative stress, but failed to improve the other hippocampal function and cognition in HFD-fed rats with estrogen deprivation. The possible explanations are that 1) combined therapy exerted the higher attenuation in hippocampal ROS level than estrogen therapy, resulting in decreased other hippocampal pathologies and improved cognition better than estrogen therapy; and 2) the dose of estrogen treatment which used in the present study might be enough only to decrease metabolic disturbance and hippocampal oxidative stress in HFD-fed rats with estrogen deprivation. Therefore, the high level of hippocampal apoptosis, synaptic dysplasticity, dendritic spine loss, and cognitive decline were still presented in estrogen-treated rats.

In conclusion, short-duration of calorie restriction alone attenuated metabolic dysfunction, and hippocampal oxidative stress, but failed to improve cognition in both HFD-fed group with or without estrogen deprivation. Exercise regime in the short duration decreased hippocampal pathology and cognitive decline in HFD-fed group but failed to decrease these brain pathologies in HFD-fed group with estrogen deprivation. Interestingly, the short-term combination of calorie restriction with exercise regime showed the higher improvement on all parameters than monotherapy and estrogen therapy in both HFD-fed group with or without estrogen deprivation. Therefore, this study supported the hypothesis that the use of these combined therapies over a short duration, rather than either of these regimes as a single therapy may be the best lifestyle modification for decreasing hippocampal pathology and cognitive decline from HFD consumption with or without estrogen deprivation.

## TEST

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## ABSTARCT

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## ก่อนสอบ



ตรวจเพื่อส่งผลการตรวจสอบ  
ให้อาจารย์ที่ปรึกษา

## สอบเสร็จ



มีการปรับรูปเล่ม  
ให้ตรวจและพิมพ์ผลสรุปแนบมา  
พร้อมเอกสารขอสำเร็จการศึกษา

## ส่งรูปเล่มฉบับสมบูรณ์

พร้อมไฟล์เพื่อขออนุมัติสำเร็จการศึกษา



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- หากส่งเอกสารเข้าไปตรวจกับ Turnitin ผ่าน Class ของห้องสมุดที่ชื่อ  
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# “การตรวจสอบด้วย Turnitin

เป็นการตรวจสอบ “ความเหมือน” ของข้อความเท่านั้น”



ไม่สามารถตรวจสอบการลอกเลียนผลงานได้ทั้งหมด  
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ดัชนีชี้วัดคุณภาพวารสาร

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เครื่องมือช่วยค้นคว้าวิจัย

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การเขียนรายงาน

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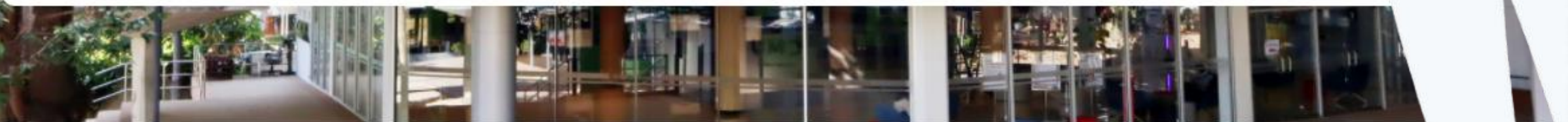
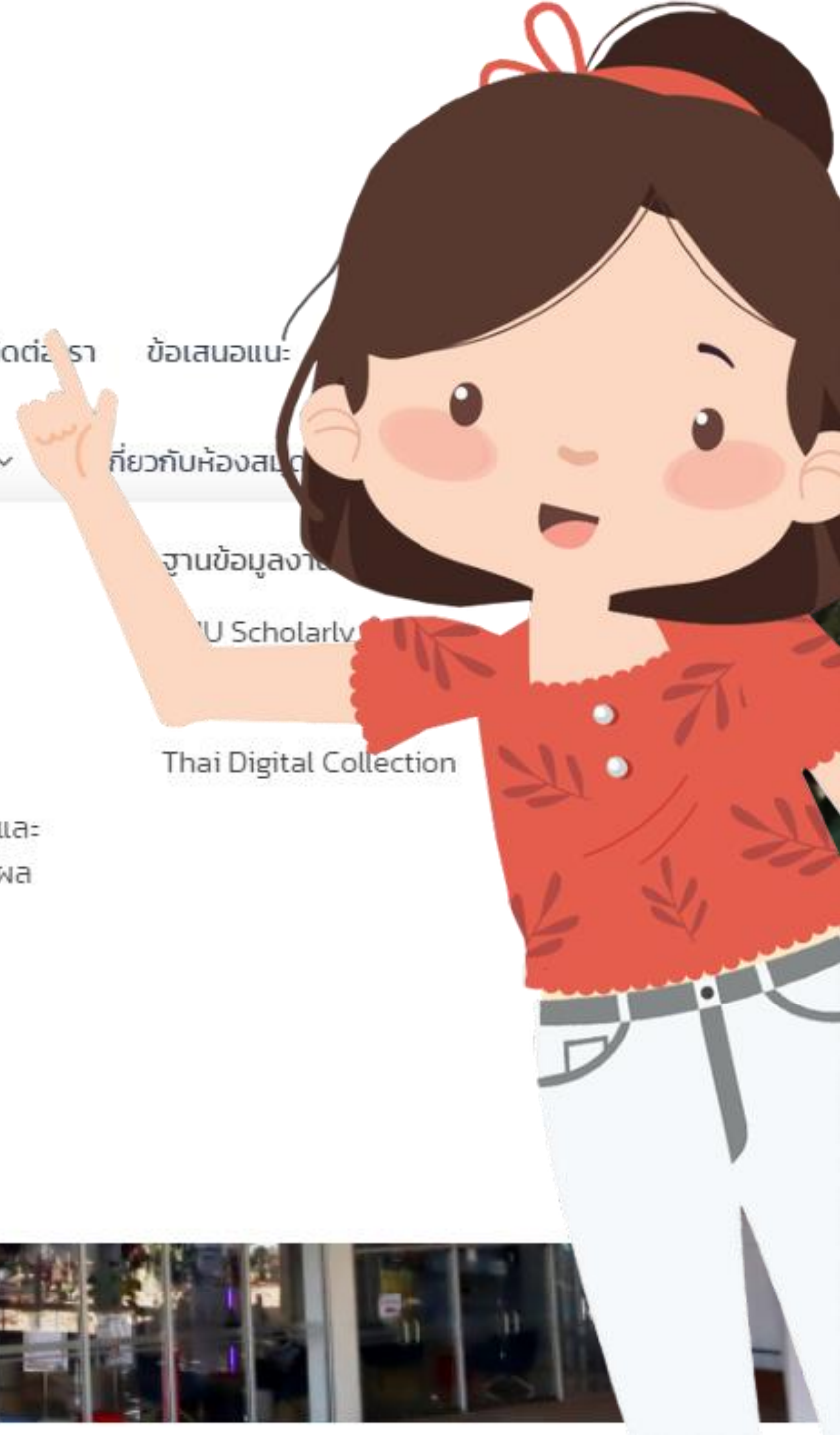
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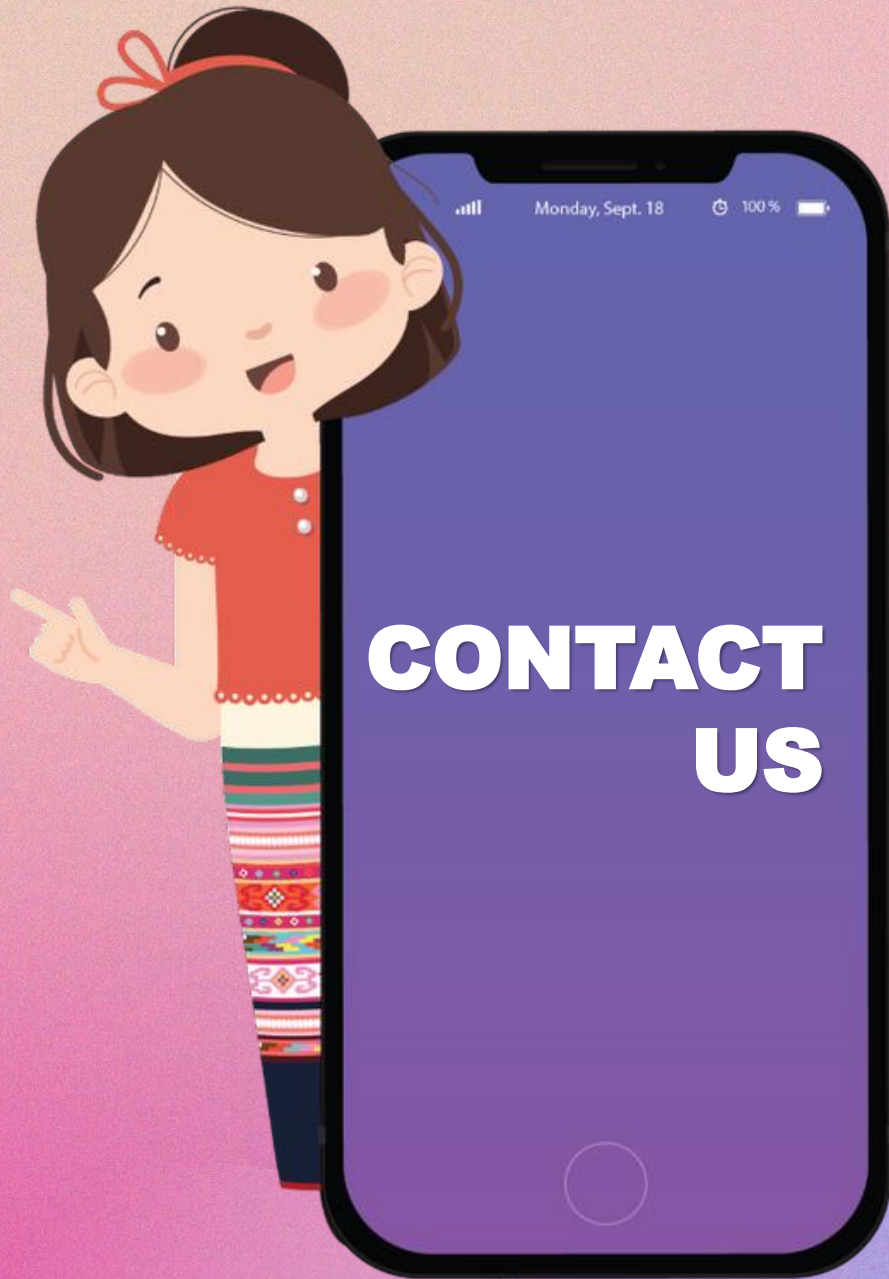




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