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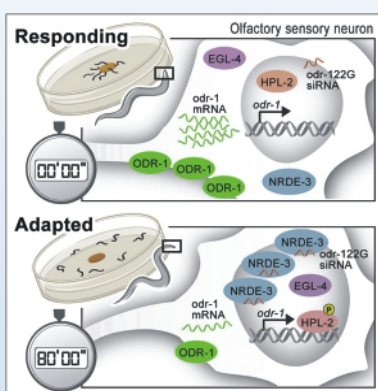


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研究興趣

• 利用嗅覺記憶可塑性來研究調控長期記憶的分子機制

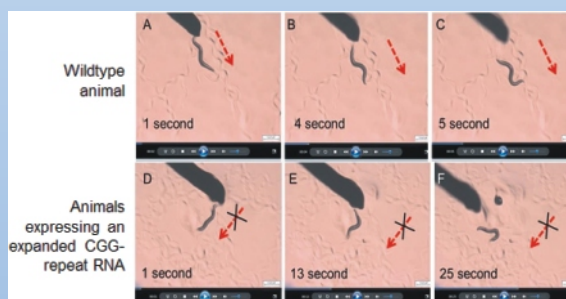
基因調控的表觀遺傳(epigenetic)控制不僅在細胞分化和生物體發育過程中扮演非常重要作用，近年來越來越多的證據顯示表觀遺傳調控在神經系統中如何因應環境變遷緊密相關。感覺神經元接受外來環境的刺激時，必須微調神經迴路(neuron circuit)的每一個神經細胞內的基因表現，做出適當的應對行為，這過程稱為神經和行為可塑性(neuronal and behavioral plasticity)。一旦喪失這可塑性，常常會造成神經和精神相關疾病。然而，表觀遺傳調控如何參與學習、記憶和神經病變的動態過程目前尚不清楚。我的實驗室利用線蟲(Caenorhabditis elegans)研究嗅覺神經對氣味有趨化性(chemotaxis)和長時間嗅覺刺激產生適應性(adaptation)的行為特徵，來探討不同的環境刺激因子和個別分子作用機制改變生物體的行為



做一系統性分析研究。這成果預期將可提供更多神經科學研究資訊，以期在成癮性和抑鬱症的治療上有新的對策。

• 建立人類疾病的模式生物來研究老年神經退化之分子機制

X染色體CGG序列誘發震顫/共濟失調綜合徵(Fragile X-associated tremor/ataxia syndrome)是一種老化神經變性疾病，是由FMR1基因的5'UTR不正常CGG序列重複導致，常發生在50歲以上的男性患者有平衡和認知失調的問題，然而台灣還沒有的基本病理機制研究團隊，因此許多患者常被誤診為帕金森氏症，延誤治療並造成醫藥浪費。針對這老化疾病的研究，我已經在線蟲的神經系統建立一個研究模式，預期利用遺傳篩選方式找到一個可能的治療標的物，對基因表達進行比對，快速找出可能的致病機制，並發展新的治療方法。





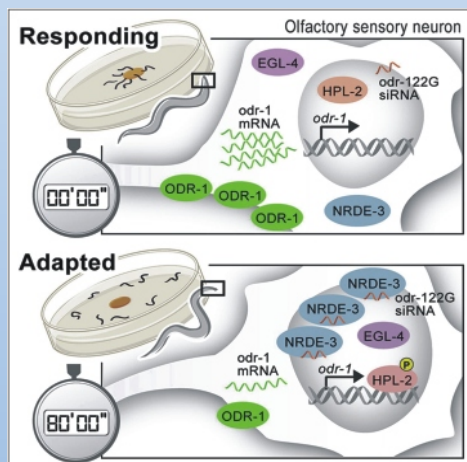
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Research Interests

- Investigation of epigenetic regulation in the olfactory plasticity in *Caenorhabditis elegans*

Epigenetic control of gene regulation is gaining importance in not only cell differentiation and organism development but also neuronal and behavioral plasticity. Loss of the plasticity has been tightly link to the pathogenic mechanisms of neurological and psychiatric diseases. However, it remains unknown how epigenetic regulation is involved in time-dependent and dynamic processes of learning, memory and even neurodegeneration. My lab has well-established olfactory behavior system in *Caenorhabditis elegans* which is inherently attracted to a variety of odors, but the animal ignores the odor after prolonged exposure to the stimuli. This decreased attraction is termed odor adaptation. This reliable system in neuronal plasticity allows us to understand the model of long-term memory in epigenetic dynamics.



- Epigenetic dynamics in a model of Fragile X-associated tremor/ataxia syndrome in *C. elegans*

Fragile X-associated tremor/ataxia syndrome (FXTAS) is a late onset neurodegenerative disorder and its clinical features including intention tremor, gait ataxia, and parkinsonism with associated cognitive impairment. Many patients have been reported to be misdiagnosed as Parkinson's disease. Thus, that is important to have model organisms to gain insight into its cellular and molecular mechanisms. Our research group has successfully established a human disease in *C. elegans'* neurons by expressing mRNA with the pathogenic expanded CGG repeats in the 5'UTR of the human fragile X mental retardation gene (*FMRI*). We found that the pathogenic gene caused movement defects which are observed in human. Thus, this nematode model is able to provide a reliable and fast platform for genetic, behavioral, and drug screenings.

