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AMPK as a Bioenergetic Checkpoint in Neurons: Conditional Permission for Synaptic Plasticity

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Abstract

Synaptic signaling and plasticity are energetically demanding processes. They differ in their dependence on sustained energy availability. While synaptic signaling occurs rapidly, long-term plasticity requires sustained energy availability. This requires neurons to assess whether sufficient energy is available to support plasticity. Adenosine monophosphate–activated protein kinase (AMPK) is a central cellular energy sensor, yet its role in determining the energetic permissibility of synaptic plasticity has not been clearly defined. This review proposes that AMPK functions as a bioenergetic checkpoint for synaptic plasticity. Under high-energy conditions, low AMP and high ATP levels limit AMP binding to the γ subunit. This maintains the β subunit's myristoyl group in a buried conformation. Phosphorylation of the α subunit at Threonine 172 (Thr172) is therefore limited. This keeps AMPK inactive, thereby permitting synaptic plasticity under energetically favorable conditions. During low energy conditions, elevated AMP and reduced ATP promote AMP binding to the γ subunit. This binding induces exposure of β-subunit myristoyl group. Phosphorylation of the α subunit at Thr172 becomes possible, thereby activating AMPK. Activated AMPK suppresses energy-consuming processes, thereby restricting synaptic plasticity. Through these mechanisms, AMPK converts fluctuations in cellular energy into a threshold-dependent decision that determines whether synaptic activity progresses to long-term plasticity while preserving bioenergetic homeostasis. This framework positions AMPK as a central bioenergetic checkpoint linking cellular energy status to synaptic plasticity.

Key words: *AMP-activated protein kinase (AMPK); Synaptic plasticity; Bioenergetic checkpoint; Long-term potentiation (LTP), Plasticity permission.*

INTRODUCTION

The brain operates under strict energy limitations. Although it makes up only about 2% of the body's total mass, it consumes nearly 20% of the body's total energy (Herculano-Houzel, 2011). This high energy demand reflects the constant energy required to maintain electrical signaling, synaptic communication, and basic cellular functions necessary for neuronal survival (Leung & Rangamani, 2023).

Beyond maintaining basal function, neurons undergo synaptic plasticity. Synaptic plasticity enables learning and memory formation. This mechanism allows neural circuits to change in response to experience through modifications in synaptic strength and structure (Tao et al., 2021). However, this process requires a lot of energy. In particular, long-term plasticity requires sustained molecular pathways. Protein synthesis is among the most energy-demanding processes following neuronal activation (Dastidar et al., 2020). Compared with rapid electrical signaling, these longer-lasting changes place a much greater burden on the cell's metabolic resources.

Synaptic plasticity is not an immediate necessity for survival. Unlike basic neuronal signaling, long-term synaptic changes can be delayed or limited depending on the availability of energy. Neurons must evaluate signal demands and available energy before initiating plasticity. To facilitate this mechanism, neurons need an internal system that monitors cellular energy levels and decides whether long-term plasticity can be initiated (Divakaruni et al., 2018; H. L. Li & van Rossum, 2020).

Previous studies have shown that synaptic plasticity is strongly affected by the bioenergetic state of the neuron. When energy availability is low, plasticity-related processes are often reduced or altered. This reflects the dependence of synaptic modification on metabolic conditions (Divakaruni et al., 2018; Leung & Rangamani, 2023).

AMP-activated protein kinase (AMPK) is widely recognized as a central cellular energy sensor and regulator of bioenergetic homeostasis. It responds to changes in cellular energy levels and helps coordinate appropriate metabolic responses. Although AMPK has been extensively studied in metabolic contexts, it has not been fully incorporated into a framework explaining how neurons determine whether synaptic plasticity is energetically feasible. An integrative model linking AMPK structural regulation to plasticity decision-making remains lacking. This review aims to synthesize current mechanistic understanding of AMPK activation and its role as a bioenergetic checkpoint regulating synaptic plasticity.

AMPK AS A SENSOR

Cellular energy availability can be inferred from the relative levels of adenine nucleotides, particularly the adenosine triphosphate (ATP), adenosine diphosphate (ADP) and adenosine monophosphate (AMP). ATP serves as the primary energy molecule of the cell. This molecule provides energy through hydrolysis of its terminal phosphate bond. As ATP is used, it is converted to ADP and further to AMP via adenylate kinase–mediated reactions. This leads to changes in ATP, ADP and AMP concentrations. Thus, these changes reflect the cellular energy state (Wilson & Matschinsky, 2022).

The AMP-to-ATP ratio is detected by the intrinsic structural organization of AMPK. AMPK is a heterotrimeric complex composed of catalytic (α chains), scaffolding (β chains) and nucleotide-sensing (γ chains) subunits. Among those three subunits, the γ chains are responsible for AMP/ATP sensing. By having cystathionine β-synthase (CBS) motifs, the γ chains facilitate the AMP and ATP's competitive binding (Gu et al., 2017; Hawley et al., 2024).

In sensing AMP/ATP ratio, CBS sites work together and do not function independently. The binding of either AMP or ATP to one site changes the shape and stability of the other sites. CBS1 prefers ATP and shows weaker binding to AMP. CBS2 serves a structural role without nucleotide binding. Among the CBS sites, CBS3 acts as the primary sensing site for AMP (Gu et al., 2017).

CBS3 contains positively charged amino acids that enable AMP to bind more tightly than ATP. In parallel, ATP's extra phosphate groups weaken these interactions and force structural rearrangements. Due to the ability to form stronger electrostatic interactions, AMP can replace ATP especially when cellular energy levels drop. Along with this, CBS4 stabilizes AMP binding at CBS3. This makes CBS3 the key site that responds to changes in cellular energy status (Gu et al., 2017; Hawley et al., 2024).

Taken together, by integrating signals across its CBS sites, AMPK responds to the ratio of ATP and AMP rather than to ATP levels alone. This multi-site sensing mechanism gives AMPK high sensitivity and precision across different energy states. AMPK converts the presence of energetic molecules into a measurable internal state.

AMPK AS AN INTEGRATOR

After sensing, AMPK must integrate various conditions for its activation state. This integration is mediated sequentially by the γ, β and α subunits. If integration fails, AMPK

stays in its inactive state. This collaborative work by its subunits shows its role as a bioenergetic signal integrator.

a) The γ subunit

AMPK activation is tightly controlled through allosteric binding of AMP and ADP to the γ subunit. The absence of functional γ subunits is shown to disrupt AMP- and ADP-dependent activation (Hawley et al., 2024). The γ subunit of AMPK contains a pseudosubstrate sequence. In normal condition, the pseudosubstrate sequence binds to and inhibits the kinase domain. This interaction prevents phosphorylation of substrates and maintains AMPK in an inactive state. In low energy state, AMP binding disrupts the pseudosubstrate interaction. This stops the autoinhibition and allows the activation of AMPK. Thus, this pseudosubstrate sequence functions as an internal inhibitory switch (Scott et al., 2007).

Binding of AMP and ADP can partially activate AMPK. Structural studies demonstrate that these ligands provide sufficient binding energy to shift the activation loop into an active-like conformation, allowing non-phosphorylated AMPK to exhibit low but measurable kinase activity in vitro (Willows et al., 2017). In addition, AMP robustly activates AMPK in a concentration-dependent manner, whereas ADP produces a weaker activation that becomes apparent only at higher ATP concentrations (Hawley et al., 2024).

b) The β subunit

AMPK achieves full activation under energetic stress only when β-subunit's myristoyl group is exposed. This is an N-terminal myristoyl group that plays a decisive role in determining whether AMP binding can be translated into AMPK activation. This demonstrates that β-subunit's myristoyl group accessibility is essential for coupling AMP sensing to phosphorylation-dependent activation (Oakhill et al., 2010).

In high energy state, this myristoyl group maintains AMPK in an autoinhibited state. This happens by restricting access of upstream kinases to the activation loop residue Thr172. Following AMP sensing by the γ-subunit, effective transmission of the energy signal to the catalytic α-subunit requires regulation at the level of the β-subunit (Oakhill et al., 2010).

In low energy state, when ATP level drops and AMP level rises, AMP binding to the γ-subunit induces conformational changes that expose the β-subunit's myristoyl group. This occurs when the myristoyl group becomes extruded from its intramolecular binding pocket. This exposure facilitates access to Thr172 for its subsequent phosphorylation. Consequently, AMPK becomes permissive to phosphorylation (Oakhill et al., 2010). A recent study showed that AMPKβ2, but not β1, is required for AMPKα phosphorylation at Thr172 for the maintenance of hippocampal synaptic plasticity (Swift et al., 2024).

At the cellular level, this mechanism also governs AMPK localization. Under low-energy conditions, AMPK is largely distributed in the cytosol. Upon AMP elevation or glucose deprivation, the AMPK with exposed myristoyl group redistributes to intracellular membranes. This spatial relocalization further supports efficient signaling to downstream metabolic targets (Oakhill et al., 2010). Together, these findings establish β-subunit myristoyl group exposure as a critical regulatory checkpoint linking nucleotide sensing with structural activation.

c) The α subunit

The α subunit serves as the catalytic executor. AMPK's full activation depends on phosphorylation of the catalytic α subunit at threonine 172 (Thr172). This is a conserved site required for kinase activity. Following the conformational rearrangements from the γ- and β- subunits, the autoinhibitory domain (AID) from the kinase domain gets released. This then stabilizes phosphorylation of Thr172 and promotes catalytic activity. In contrast, ATP favors an open, inactive conformation by weakening these intramolecular interactions and promoting reassociation of the AID with the kinase domain. These opposing effects occur at physiologically relevant nucleotide concentrations, with AMP acting at micromolar levels and ATP at millimolar levels. Structural analyses further demonstrate that AMP binding at the CBS3 site of the γ subunit is transmitted to the α subunit through αRIM-mediated interactions, stabilizing the active conformation, whereas ATP counteracts this process. Together, these findings explain how AMPK translates cellular energy status into conformational and functional changes of the α subunit that determine kinase activity (Li et al., 2015).

Specifically, the α subunit exists in two isoforms, α1 and α2, both of which contain this regulatory phosphorylation site. However, evidence indicates that these isoforms play distinct functional roles in the brain. The loss of AMPKα2 results in pronounced impairments in hippocampus-dependent learning and memory. Mice lacking AMPKα2 exhibit deficits in spatial learning and object recognition despite normal locomotion and baseline neuronal function, identifying AMPKα2 as the dominant catalytic isoform supporting cognitive processes (Yang et al., 2021).

Collectively, these findings indicate that although Thr172 phosphorylation is a shared activation mechanism of AMPK isoforms, AMPKα2 uniquely couples energetic status to synaptic maintenance and plasticity. While the precise molecular steps remain to be fully defined, available evidence supports a model in which phosphorylation-dependent activation of AMPKα2 contributes to the regulation of plasticity-related processes downstream of energy sensing, positioning AMPKα2 as an integrative node linking metabolic state to long-term synaptic function.

AMPK AS A DECISION GATE

Phosphorylation of Thr172 represents the central event that converts AMPK from a transient energy sensor into a stable signaling kinase. This modification stabilizes the activation loop of the α subunit and locks the kinase domain into a productive conformation. This allows AMPK to remain active even as AMP and ADP levels fluctuate. Thr172 phosphorylation prolongs AMPK signaling by stabilizing the kinase against dephosphorylation (Willows et al., 2017).

Neuronal activity induces rapid phosphorylation of AMPK at Thr172 in response to changes in cellular energy status. This activation is driven primarily by ATP depletion and elevated AMP/ADP levels rather than CaMKKβ signaling and occurs through an LKB1-dependent mechanism. Experimental manipulation of neuronal energy levels demonstrates that reductions in ATP robustly increase Thr172 phosphorylation, confirming that AMPK activation reflects cellular energetic stress rather than synaptic activity per se (Marinangeli et al., 2018; Sample et al., 2015).

Importantly, Thr172 phosphorylation functions as a molecular commitment step rather than a simple amplification mechanism. While allosteric binding of AMP can transiently bias AMPK toward an active-like state, this conformation is unstable and readily reversed. In contrast, phosphorylation of Thr172 stabilizes the activation loop, aligns

catalytic residues and protects the kinase from rapid inactivation. Even though only a small fraction of total AMPK becomes phosphorylated during energetic stress, this modification is sufficient to produce sustained signaling and effective downstream target phosphorylation (Willows et al., 2017).

Thr172 phosphorylation introduces threshold behavior. Below this level, AMPK activity remains weak and transient. Above this level, AMPK adopts a stable active state that drives coordinated metabolic responses. In this way, Thr172 phosphorylation converts graded metabolic inputs into a binary-like signaling outcome, consistent with the behavior of a molecular checkpoint (Willows et al., 2017).

The functional relevance of this mechanism is evident in synaptic plasticity. AMPKα2 is specifically required for the maintenance of long-term potentiation, while early-phase LTP and basal synaptic transmission remain intact in its absence. Loss of AMPKα2 results in impaired late-phase LTP, reduced dendritic spine density, increased spine immaturity and decreased expression of synaptic proteins such as PSD-95 and synapsin-2, indicating a failure to sustain synaptic remodeling rather than initiate it (W. Yang et al., 2021). Consistently, reductions in cellular ATP levels in hippocampal slices lead to a 2–3 fold increase in phosphorylated AMPK, linking energetic stress directly to activation of this checkpoint mechanism (Potter et al., 2010).

Together, these findings support a threshold-based model in which AMPK functions as a bioenergetic decision gate. Allosteric nucleotide binding primes the kinase, but phosphorylation of Thr172 determines whether energy stress is sufficient to justify long-term signaling and plasticity-related remodeling. Through this mechanism, AMPK ensures that sustained synaptic modification occurs only when energetic conditions permit. This mechanism plays a key intermediary role between AMP detection by the γ-subunit and catalytic activation of the α-subunit, reinforcing AMPK's role as a finely tuned metabolic integrator rather than a simple on–off switch (**Table 1**).

Table 1. Energy-dependent regulation of AMPK activity and its impact on synaptic plasticity

Cellular Energy Availability	γ Subunit	β Subunit	α Subunit	AMPK Status	Synaptic Plasticity
High Energy	↓AMP Binding ↑ATP Binding	Myristoyl group buried (autoinhibited conformation)	Not Phosphorylated	Inactive	Energetically Permitted
Low Energy	↑AMP Binding ↓ATP Binding	Myristoyl group exposed	Phosphorylated (Thr172)	Active	Energetically Restricted

AMPK SHAPES OUTCOMES

AMPK does not prevent synaptic activation itself but instead constrains the persistence of synaptic plasticity. Both high-frequency stimulation and theta-burst stimulation are still able to induce early-phase long-term potentiation (E-LTP) in the presence of AMPK activators. However, the maintenance of potentiation is markedly impaired, with late-phase LTP (L-LTP) reduced to 10–40% of control levels depending on experimental conditions. Basal synaptic transmission and paired-pulse facilitation remain unchanged, indicating that presynaptic release probability and excitability are preserved. Together, these findings demonstrate that AMPK selectively constrains the consolidation of synaptic plasticity rather than its initiation (Potter et al., 2010).

The effects of AMPK on synaptic plasticity are strongly time dependent, with suppression occurring only during the window required for long-term consolidation. AMPK inhibits L-LTP only when activated during the induction phase, which corresponds to the period of protein synthesis necessary for stabilization of synaptic changes. Activation outside this window has little effect on synaptic strength. Conversely, pharmacological inhibition of AMPK rescues L-LTP even under conditions of metabolic stress, indicating that AMPK activity is both necessary and sufficient to suppress long-term synaptic consolidation. These findings support a model in which AMPK functions as a metabolic gatekeeper that determines whether synaptic activity progresses into stable plasticity (Potter et al., 2010).

At the molecular level, AMPK constrains long-term plasticity by suppressing protein synthesis–dependent mechanisms. Under normal conditions, phosphorylation of mTOR downstream targets activates translational machinery required for LTP maintenance. However, activation of AMPK by metabolic stressors such as 2-deoxyglucose or metformin abolishes this mTOR-dependent phosphorylation, thereby limiting the synthesis of proteins necessary for synaptic consolidation (Potter et al., 2010).

Consistent with this mechanism, AMPKα2 preferentially signals through the PERK–eIF2α pathway rather than mTOR. Activation of this pathway suppresses local protein synthesis, reduces dendritic polyribosomes and impairs long-term synaptic plasticity. Pharmacological inhibition of PERK rescues both LTP and memory deficits, identifying this pathway as a key mediator of AMPKα2-dependent regulation of synaptic plasticity (Yang et al., 2021).

Beyond acute regulation of translation, sustained AMPK activation produces longer-term structural consequences. Prolonged activation over several days reduces markers of synaptic growth and stability, including synaptophysin, Homer-1 and brain-derived neurotrophic factor (BDNF) and is accompanied by reductions in neurite length, total cell area and neuritic complexity. These findings indicate that sustained energetic signaling through AMPK promotes structural remodeling rather than maintenance of synaptic architecture (Yang et al., 2022).

Finally, AMPK integrates neuronal energy status with plasticity outcomes by regulating metabolic support mechanisms. Activation of AMPK enhances glycolysis and mitochondrial respiration, whereas inhibition of AMPK prevents these adaptations, leading to ATP depletion and impaired induction of activity-dependent genes such as Arc and c-Fos. Disruption of this metabolic support compromises long-term potentiation and memory formation, underscoring the dependence of synaptic plasticity on cellular energy availability (Marinangeli et al., 2018). Consistent with this view, acute increases in hippocampal ATP reduce AMPK phosphorylation at Thr172, thereby promoting mTOR activation and facilitating long-term potentiation, further demonstrating that energetic state directly governs plasticity through AMPK-dependent mechanisms (Wang et al., 2025).

CONCLUSION

Together, the evidence supports a model in which AMPK functions as a bioenergetic checkpoint governing synaptic plasticity. Through nucleotide sensing by the γ subunit, structural integration via the β subunit and phosphorylation-dependent activation of the α subunit at Thr172, AMPK converts fluctuations in cellular energy into a threshold-based decision. This phosphorylation event represents a commitment step that determines whether synaptic activity proceeds toward long-term plasticity or is constrained to

preserve metabolic homeostasis. By gating mTOR- and PERK-dependent translational programs, AMPK links energetic state to the persistence of synaptic change rather than its initiation. This framework explains how neurons balance metabolic demand with plastic potential and highlights AMPK as a central regulator of experience-dependent circuit remodeling. Future studies defining the spatial and temporal dynamics of AMPK signaling will further clarify how metabolic constraints shape learning and memory under physiological and pathological conditions.

REFERENCES

Dastidar, S. G., Sharma, S. Das, Chakraborty, S., Chattarji, S., Bhattacharya, A., & Muddashetty, R. S. (2020). Distinct Regulation of Bioenergetics and Translation by Group I mGluR and NMDAR. *EMBO Reports*, *21*(6), 1–20. https://doi.org/10.15252/embr.201948037

Divakaruni, S. S., Dyke, A. M. Van, Chandra, R., LeGates, T. A., Contreras, M., Dharmasri, P. A., Higgs, H. N., Lobo, M. K., Thompson, S. M., & Blanpied, T. A. (2018). Long-term Potentiation Requires a Rapid Burst of Dendritic Mitochondrial Fission during Induction. *Neuron*, *100*(4), 860–875. https://doi.org/10.1016/j.neuron.2018.09.025

Gu, X., Yan, Y., Novick, S. J., Kovach, A., Goswami, D., Ke, J., Tan, M. H. E., Wang, L., Li, X., De Waal, P. W., Webb, M. R., Griffin, P. R., Xu, H. E., & Melcher, K. (2017). Deconvoluting AMP-activated protein kinase (AMPK) adenine nucleotide binding and sensing. *Journal of Biological Chemistry*, *292*(30), 12653–12666. https://doi.org/10.1074/jbc.M117.793018

Hawley, S. A., Russell, F. M., & Grahame Hardie, D. (2024). AMP-activated protein kinase can be allosterically activated by ADP but AMP remains the key activating ligand. *Biochemical Journal*, *481*(8), 587–599. https://doi.org/10.1042/BCJ20240082

Herculano-Houzel, S. (2011). Scaling of brain metabolism with a fixed energy budget per neuron: Implications for neuronal activity, plasticity and evolution. *PLoS ONE*, *6*(3). https://doi.org/10.1371/journal.pone.0017514

Leung, A., & Rangamani, P. (2023). Computational modeling of AMPK and mTOR crosstalk in glutamatergic synapse calcium signaling. *Npj Systems Biology and Applications*, *9*(1), 1–15. https://doi.org/10.1038/s41540-023-00295-4

Li, H. L., & van Rossum, M. C. W. (2020). Energy efficient synaptic plasticity. *ELife*, *9*, 1–15. https://doi.org/10.7554/eLife.50804

Li, X., Wang, L., Zhou, X. E., Ke, J., De Waal, P. W., Gu, X., Tan, M. H. E., Wang, D., Wu, D., Xu, H. E., & Melcher, K. (2015). Structural basis of AMPK regulation by adenine nucleotides and glycogen. *Cell Research*, *25*(1), 50–66. https://doi.org/10.1038/cr.2014.150

Marinangeli, C., Didier, S., Ahmed, T., Caillerez, R., Domise, M., Laloux, C., Bégard, S., Carrier, S., Colin, M., Marchetti, P., Ghesquière, B., Balschun, D., Buée, L., Kluza, J., & Vingtdeux, V. (2018). AMP-Activated Protein Kinase Is Essential for the Maintenance of Energy Levels during Synaptic Activation. *IScience*, *9*, 1–13. https://doi.org/10.1016/j.isci.2018.10.006

Oakhill, J. S., Chen, Z. P., Scott, J. W., Steel, R., Castelli, L. A., Linga, N., Macaulay, S. L., & Kemp, B. E. (2010). β-Subunit myristoylation is the gatekeeper for initiating metabolic stress sensing by AMP-activated protein kinase (AMPK). *Proceedings of the National Academy of Sciences of the United States of America*, *107*(45), 19237–

19241. https://doi.org/10.1073/pnas.1009705107

Potter, W. B., O'Riordan, K. J., Barnett, D., Osting, S. M. K., Wagoner, M., Burger, C., & Roopra, A. (2010). Metabolic regulation of neuronal plasticity by the energy sensor AMPK. *PLoS ONE*, *5*(2). https://doi.org/10.1371/journal.pone.0008996

Sample, V., Ramamurthy, S., Gorshkov, K., Ronnett, G. V., & Zhang, J. (2015). Polarized activities of AMPK and BRSK in primary hippocampal neurons. *Molecular Biology of the Cell*, *26*(10), 1935–1946. https://doi.org/10.1091/mbc.E14-02-0764

Scott, J. W., Ross, F. A., Liu, J. K. D., & Hardie, D. G. (2007). Regulation of AMP-activated protein kinase by a pseudosubstrate sequence on the γ subunit. *EMBO Journal*, *26*(3), 806–815. https://doi.org/10.1038/sj.emboj.7601542

Swift, N. A., Yang, Q., Jester, H. M., Zhou, X., Manuel, A., Kemp, B. E., Steinberg, G. R., & Ma, T. (2024). Suppression of neuronal AMPKβ2 isoform impairs recognition memory and synaptic plasticity. *Neurobiology of Disease*, *201*(September), 106664. https://doi.org/10.1016/j.nbd.2024.106664

Tao, W., Lee, J., Chen, X., Diaz-Alonso, J., Zhou, J., Pleasure, S., & Nicoll, R. A. (2021). Synaptic memory requires camkii. *ELife*, *10*, 1–20. https://doi.org/10.7554/eLife.60360

Wang, M., Jin, B., & Jo, J. (2025). Acute Restraint Stress Induces Long-Lasting Synaptic Enhancement by Inhibiting AMPK Activation in AD Model Mice. *CNS Neuroscience and Therapeutics*, *31*(3), 1–12. https://doi.org/10.1111/cns.70335

Willows, R., Sanders, M. J., Xiao, B., Patel, B. R., Martin, S. R., Read, J., Wilson, J. R., Hubbard, J., Gamblin, S. J., & Carling, D. (2017). Phosphorylation of AMPK by upstream kinases is required for activity in mammalian cells. *Biochemical Journal*, *474*(17), 3059–3073. https://doi.org/10.1042/BCJ20170458

Wilson, D. F., & Matschinsky, F. M. (2022). Integration of Eukaryotic Energy Metabolism: The Intramitochondrial and Cytosolic Energy States ([ATP]f/[ADP]f[Pi]). *International Journal of Molecular Sciences*, *23*(10). https://doi.org/10.3390/ijms23105550

Yang, A. J. T., Mohammad, A., Tsiani, E., Necakov, A., & MacPherson, R. E. K. (2022). Chronic AMPK Activation Reduces the Expression and Alters Distribution of Synaptic Proteins in Neuronal SH-SY5Y Cells. *Cells*, *11*(15), 1–14. https://doi.org/10.3390/cells11152354

Yang, W., Zhou, X., Zimmermann, H. R., & Ma, T. (2021). Brain-specific suppression of AMPKα2 isoform impairs cognition and hippocampal LTP by PERK-mediated eIF2α phosphorylation. *Molecular Psychiatry*, *26*(6), 1880–1897. https://doi.org/10.1038/s41380-020-0739-z