

# Regulation of Reversible Dissociation of LHCII from PSII by Phosphorylation in Plants

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## ABSTRACT

LHCII is a crucial light-harvesting pigment/protein complex in photosystem II (PSII) supercomplex. It also participates in the light energy redistribution between photosystems and in the photoprotection via its reversible dissociation with PSII and PSI (photosystem I). This reversible detachment of LHCII is regulated by phosphorylation of its own and PSII core protein. Under low light conditions, LHCII is phosphorylated and dissociated with PSII core protein complex and combined with PSI, which balances the excitation energy between PSII and PSI; Under high light environment, the phosphorylation of PSII core proteins makes LHCII detach from PSII. The dissociated LHCII presents in a free state, which involves in the thermal dissipation of excess excitation energy. During photodamage, dual phosphorylations of both PSII core proteins and LHCII complexes occur. The phosphorylation of D1 is conducive to the disintegration of photodamaged PSII and the cycle of repair. In this circumstance, the phosphorylation of LHCII is induced by reactive oxygen species (ROS) and then the phosphorylated LHCII migrates to PSI, into the repair cycle of damaged PSII. The ferredoxin (Fdr) and thioredoxin (Tdr) system may play a possible central role in the phosphorylation regulation on LHCII dissociation.

## KEYWORDS

Light-Harvesting Complex II (LHCII); Phosphorylation; Photosystems; Photoinhibition; Ferredoxin and Thioredoxin System

## 1. Introduction

Photosynthetic organisms utilize sunlight energy, CO<sub>2</sub> and H<sub>2</sub>O to synthesize carbohydrates. The photosynthetic process consists of light reaction and dark reaction. In plants, light reaction occurs inside chloroplasts, to generate NADPH and ATP by photosynthetic electron transfer chain and photophosphorylation. The resulting NADPH and ATP are used to converse carbon dioxide into carbohydrates in dark reaction. The electron transfer chain locates in the membrane of thylakoid, containing the two types of photosystems (PSII, PSI) and cytochrome b6-f (Cytb<sub>6</sub>-f) complexes. NADP production depends on the linear electron transfer involving PSII, Cytb<sub>6</sub>-f and PSI. Extra ATP is synthesized by the cyclic electron transfer

only involving PSI and Cytb<sub>6</sub>-f. There is a difference in light absorption wavelength between PSII and PSI. With these characteristics of photosynthesis, to acquire the high efficiency of light utilization, plants not only need to coordinate the action between light reaction and dark reaction, but also between PSII and PSI.

Light is the sole energy for plant photosynthesis, but it also damages the photosynthetic apparatus [1-4]. Under fluctuating light or strong light conditions, the imbalance allocation of excitation energy occurs between PSII and PSI, or the captured energy in light reaction beyond the need of dark reaction. The resulting excess energy in photosynthetic apparatus damages PSII complexes and reduces the photosynthetic efficiency. This phenomenon is so-called photoinhibition or photodamage. In order to avoid this injury, plants have developed a series of specific

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protection mechanism in chloroplasts during the long-term evolution [5-7]. This mechanism involves the architectural shifts in PSII complexes and thylakoid membrane [8]. One of them is the reversible detachment of light-harvesting pigment complex with PSII and PSI core complex [9-11]. Under the light condition with changing wavelength, the reversible detachment of LHCII complex makes the excitation energy redistributed between the two photosystems [10,12]. In continuous intensive light condition, a number of LHCII dissociate from PSII and present in a free state inside chloroplast stroma, preventing more excitation energy from destroying the reaction centers [13,14]. Several reports show that the detachment of LHCII also participates in PSII repair mechanism during photodamage [15,16]. In higher plants, the reversible dissociation of LHCII with the photosystems is regulated by phosphorylation [6,17], and the mechanism of phosphorylation according to the light intensity.

## 2. LHCII Phosphorylation in State Transition

The reaction centers of PSII and PSI have different light absorption characteristics, their absorption peak, respectively, at 680 nm and 700 nm. As green plants are subjected to light environment with fluctuating intensity, PSII and PSI are excited in out-balance and LHCII displaced from the over-excitation photosystems to the less-excitation photosystem, which is so-called state transition [12,18]. Under photoinhibition conditions, state transition is also considered as a very important photoprotection mechanism of PSII [19].

The reversible phosphorylation of LHCII complex regulates the reversible displacement of LHCII between two photosystems is regulated by the phosphorylation/dephosphorylation of LHCII complex [17], because the phosphorylation state of LHCII changes LHC II affinity with the two different photosystems [6]. As PSII complex is overly excited, LHCII is phosphorylated. And then the phosphorylated LHCII dissociates from PSII to move towards PSI, which is called state 2. When PSI is excessively excited, the phosphorylated LHCII is dephosphorylated and detached from PSI and then returns to PSII, which is called state 1 [11,20]. In unicellular organisms, the mobile fraction of LHCII during state transitions is approximately 80%, whereas in land plants the percent is only 15% - 20% [11].

The major function of photosynthetic electron transport chain is to synthesize NADPH and ATP. In some circumstances, plants need to strengthen cyclic photosynthetic electron transfer to generate more ATP. State transitions are found to participate in the regulation of ATP synthesis. Owens and Ohad (1982) [21] restricted ATP synthesis in *Chlamydomonas reinhardtii* by inhibiting chloroplast respiration, the phosphorylation level of LHCII significantly increased and accompanied by cytb<sub>6</sub>-f complex movement to PSI, which promoted the cyclic electron transfer and ATP generation. The similar phenomenon was observed in higher plants. The inhibition of ATP synthesis in tobacco plants induced the state transition and also accelerated the cyclic electron flow [22].

PSII is a dimeric supercomplex containing changeable number of outer LHCII complexes [23]. The LHC II consists of trimeric LHCII complexes, from 2 to 4 copies [24]. The polypeptides are encoded by the genes of lhcb1, lhcb2 and lhcb3 [25]. Lhcb1 and Lhcb2 have been well documented to reversibly phosphorylated/dephosphorylated, and Lhcb3 has not been found to have any phosphorylation site. After phosphorylated and dissociated from PSII, LHCII trimer is depolymerized and releases phosphorylated monomers that binds to PSI [10]. The connection between LHCII antenna and PSII core complex is through antenna proteins of CP29, CP26 and CP24, which are the products of genes of lhcb4, lhcb5 and lhcb6 [24,26,27]. In Arabidopsis, three isoforms of CP29 are found, named as Lhcb4.1, Lhcb4.2 and Lhcb4.3 [28]. Nevertheless, the difference in function of these protein isoforms has remained elusive.

The recent studies have revealed the protein kinases involved in PSII phosphorylation events [29]. In *Arabidopsis*, two orthologue thylakoid-associated serine/threonine kinases, called STN7 and STT7, were identified by mutant analysis approach and found to be the LHCII phosphokinase [6,9]. They are single-spanning membrane domain proteins. Their N-terminal regions locate in luminal side and C-terminal kinase domains in stromal side in chloroplasts. STN7 is essential for reversible LHCII polypeptide phosphorylation. This phosphorylation is required for state transitions in *Arabidopsis* [30-33]. The loss of STN7 in plants blocks the movement of LHCII complex from PSII core complex to PSI core complex [30,34]. Low light intensity activates STN7, but high light intensity deactivates it [31,35]. The redox state of mobile electron carriers between photosystems such as plastoquinone (PQH<sub>2</sub> and PQ) [35-39] and ferredoxin/thioredoxin system [12] regulate the activity of STN7. The lack of PQH<sub>2</sub> at the Q<sub>o</sub> site inside Cytb<sub>6</sub>-f complex results in STN7 deactivation in dark. Under short-wavelength light, PSII is over-excited, PQH<sub>2</sub> binding to the Q<sub>o</sub> site and causing a conformational change in Cytb<sub>6</sub>-f, which transfers the reduction signal from thylakoid lumen to the kinase domain in stromal side and activates LHCII kinase, resulting in LHCII phosphorylation [15, 40]. A new model is proposed by Puthiyaveetil (2011) [12]. In his model, when PSII is overly excited, PQH<sub>2</sub> released by Q<sub>o</sub> site reduces a disulfide bond between conserved luminal cysteine residues in STN7 and the

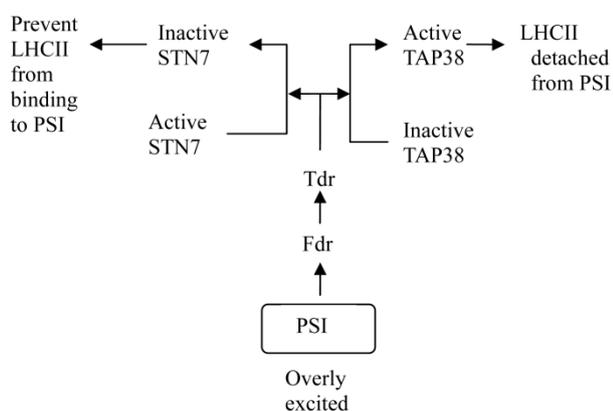
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kinase is activated. Under strong light, STN7 is inactivated via the ferredoxin/thioredoxin system by the reduction of stromal disulfide bond. This model better explained the inactivation of STN7. Moreover, we consider that the ferredoxin/thioredoxin system regulates both STN7 and phosphatase PPH1/TAP38 activity. STN7 can catalyze the phosphorylation of D1, D2 and CP43 of PSII core complex, but in a very low level.

CP29 and TSP9, the linker proteins [41,42] regulate state transitions by their differential phosphorylation [6, 43,44]. Under strong light condition, *stn8* and wild type *Arabidopsis thaliana* selectively increased the phosphorylation of CP29 isoforms Lhcb4.1 and Lhcb4.2 at threonine residues in five different positions, whereas in *stn7* and *stn7/stn8* mutant, the four phosphorylation positions were not found [45].

The dephosphorylations of Lhcb1 and Lhcb2 are majorly catalyzed by PPH1/TAP38 protein phosphatase. In mutants with loss of the activity of PPH1/TAP38, the size of PSI light harvesting complex grew and state transition1 is inhibited [46,47]. It is suggested that when PSI is over excited under long-wavelength light, PQH2 is oxidized and released from the Qo site, and the phosphatases in stromal side become active and catalyze the dephosphorylation of phosphorylated LHCII proteins [48, 49], LHCII dissociated from PSII.

When state 1 transition occurs, the phosphorylated LHCII is dephosphorylated and detached from PSI and then returns to PSII. For maintaining this process, it is essential for photosynthetic systems to enhance dephosphorylation reaction and inhibit phosphorylation reaction of LHCII. We propose that the ferredoxin/thioredoxin system plays a central role in this process (as shown in **Figure 1**). While overly excited, PSI generates more reduced ferredoxin (Fdr) and thioredoxin (Tdr), which activates the phosphatase PPH1/TAP38 and inactivates



**Figure 1.** A diagram of the ferredoxin and thioredoxin regulation on STN7 and TAP38 during state 1 transition. When PSI is overly excited and generates more reduced ferredoxin (Fdr) and thioredoxin (Tdr), which activate the phosphatase PPH1/TAP38 and inactivate STN7.

STN7 via the reduction of stromal disulfide linkage [12].

### 3. Phosphorylation of PSII Core Proteins in Photoprotection

The reversible dissociation of LHCII from PSII also occurs under high light intensity, but the mechanism differs from that in low light condition. The phosphorylation of core proteins in PSII complex occurs during photoprotection. The original and primary site of photodamage is locked in PSII [1]. In order to avoid this injury, plants need decreasing the excitation energy transferred to PSII reaction centers in high light condition. Therefore, LHCII detachment from PSII and PSI is conducive to this energy-decreasing process. But, under such high light condition, the phosphorylation of LHCII is restricted [20]. Several studies have shown that the ferredoxin and thioredoxin system at the side of PSI the down regulated the LHCII phosphorylation. The protein kinase STN7, which catalyzes LHCII phosphorylation, has disulfide bonds on the luminal side [17]. The disulfide bonds may be reduced to sulfhydryl group by ferredoxin-thioredoxin system, resulting in STN7 inactivation [12]. However, a fraction of LHCII are still detached from PSII under strong light condition, and the detached LHCII present in a free state, not binding to PSI [14]. The free LHCII dissipate the excitation energy via fluorescence release and thermal radiation. This process prevents the excitation energy which is not needed by reaction centers, from entering the reaction centers [14,50,51].

The protein phosphorylation of PSII core complex regulates the detachment of LHCII from PSII under strong light, when the phosphorylation of LHCII proteins is restricted. D1, D2, CP43 and PsbH of PSII core proteins were phosphorylated under high light intensity [14, 16,17]. PSII was overexcited in the mutants with loss of STN7 kinase under low intensity of light, but the overexcitation was not found under strong light. The mutant analyses on *Arabidopsis* indicate that the core proteins of PSII is phosphorylated by the protein kinase STN8, rather specific for these proteins [52,53]. This kinase is different from STN7 in structure. It has no disulfide bond in the luminal side so that the activity is not inhibited by ferredoxin-thioredoxin system. Consequently, core proteins of PSII complex are still phosphorylated by high irradiance condition. Zhang and Xu (2003) [14] found the core protein phosphorylation of PSII can induce the changes in the structure of PSII reaction center. These changes may loose the bounding of LHCII with PSII reaction center complex, in favor of LHCII dissociation. When green plants are moved to low intensity of light or dark condition, D1 protein is dephosphorylated by phosphatase [54], LHCII returning to PSII again. This phosphatase localizes in stacked thylakoid membrane and catalyzes the dephosphorylation of D1 in functional PSII

centers, independent of light, which is different from the phosphatase functioning in PSII repair [55]. In a reverse genetic screen, a PSII core protein phosphatase (PBPC) was identified, which counteracts the activity of STN8 kinases and is required for efficient dephosphorylation of PSII core proteins [56,57].

During high light, PSII and PSI need to prevent the accumulation of excess light energy. The detached LHCII must be prevented from phosphorylation again and blocked to bind PSI. The phosphorylated D1 proteins need to remain in phosphorylation state during high light. In this process, the ferredoxin/thioredoxin system may be a central coordinator (as shown in **Figure 2**). Under high light, PSII through PSI generates more reduced ferredoxin (Fdr) and thioredoxin (Tdr), which activate STN8 and inactivate PSII core protein phosphatase (PBPC) possibly by kinases. STN7 is also inhibited and prevented to phosphorylate LHCII, not associating with PSI. In spite of lacking of disulfide bond, STN8 activity is not inhibited by ferredoxin-thioredoxin system [52,53], but it may be regulated by phosphorylation [58].

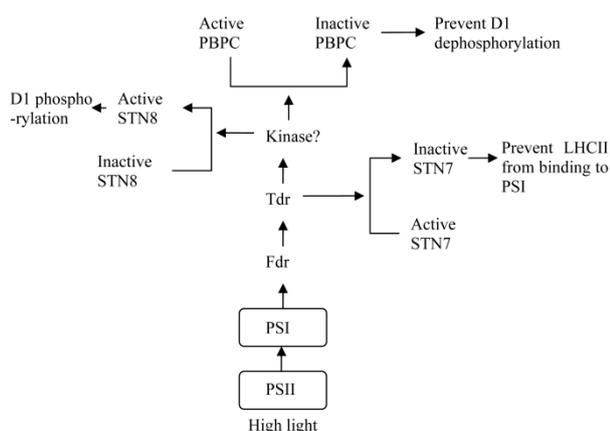
#### 4. LHCII Phosphorylation in Photodamage

In high light intensity, LHCII separates from PSII via the core protein phosphorylation of PSII in plants, and is avoided combining with PSI to prevent photoinhibition, even photodamage [3]. However, a number of studies have shown that, when plants are subjected to strong light condition, the phosphorylation of a fraction of LHCII complex remains to occur [16,39]. This phosphorylation may be due to the production of reactive oxygen species (ROS) in PSII reaction center. ROS serves as an oxidant that damages the PSII reaction centers and oxi-

dizes the sulfhydryl group to the disulfide bond of the inactivated LHCII kinase STN7 in thylakoids membrane, restoring the catalytic activity of the enzyme. Breitholtz *et al.* (2005) [39] found that, under high light intensity, in both Arabidopsis plants with higher excitation capacity of PSII and with defectiveness in non-photochemical reaction activity, the phosphorylation in LHCII occurred. Because the lower ability of non-photochemical quenching and higher excitation capacity of PSII result in more excess energy accumulation in PSII reaction centers, and the generating hydrogen peroxide and singlet oxygen restored the catalytic activity of the LHCII kinase. As a result, the phosphorylated LHCII migrates to the PSI enriched stromal region from the PSII enriched granal region of the thylakoid membrane and transfers the excitation energy to PSI [16]. Then, what function is the binding of LHCII to PSI and whether the binding leads to the damage to PSI?

The phosphorylation of LHCII induced by the high light intensity may involve in repair mechanism of PSII [15]. Of the core proteins of PSII complex, D1 is susceptible to be injured and the turnover rate is fast. Under prolonged high irradiance, ROS generated by PSII reaction center, can inevitably damage the D1 protein [59]. The rate of photodamage increases as irradiance intensity rises. On the other hand, the newly synthesized functional D1 displaced the injured one in PSII core complex and the repair kept pace with the damage. Otherwise, the structure and function of photosynthetic apparatus were severely disrupted. The repair process proposed includes: 1) disassembly of injured PSII-LHCII supercomplex into PSII and LHCII, and PSII core dimer into monomer; 2) movement of the PSII monomer from the stacked membrane region of thylakoid to the stroma region of non-stacked membrane; 3) D1, D2 and CP43 proteins were dephosphorylated; 4) degradation of injured D1 protein; 5) the newly biosynthesized D1 protein cotranslationally inserted PSII complex [60]. The repair cycle of inactivated PSII is associated with dephosphorylation of phosphorylated and photodamaged D1 and is a light-dependent reaction *in vivo* [55].

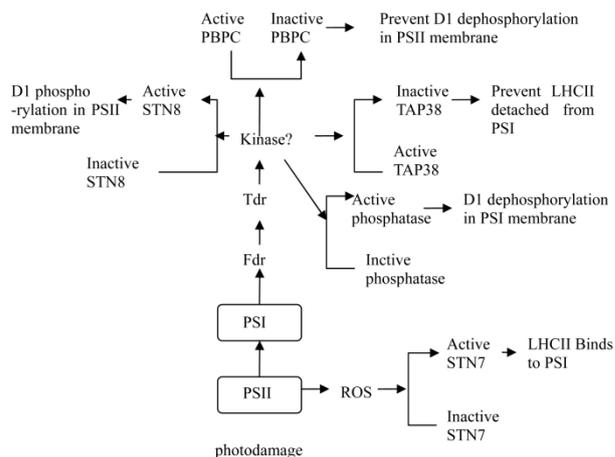
The PSII repair rate was decreased while the generation of ATP was blocked in *Spirodela oligorrhiza* [61,62] because the synthesis of ATP is indispensable for the D1 protein synthesis. The binding of phosphorylated LHCII to PSI accelerates the cyclic electron transfer and enhances the synthesis of ATP [21]. Consequently, the phosphorylation of LHCII is related to the transient repair of damaged PSII complex in photoinhibition. Murata *et al.* (2007) [62] proposed in his review that unfavorable environmental factors other than high light majorly inhibited the PSI repair cycle. For instance, high salt, low CO<sub>2</sub>, low temperature and mild heat stress all blocked *de novo* synthesis of proteins.



**Figure 2.** A diagram of the ferredoxin and thioredoxin regulation on STN8, PBPC and STN7 during high light. Under high light, PSII through PSI generates more reduced ferredoxin (Fdr) and thioredoxin (Tdr), which activate STN8 and inactivate PSII core protein phosphatase (PBPC) possibly by kinases. STN7 is also inhibited and prevented to phosphorylate LHCII.

During the photodamage, PSII core proteins are also phosphorylated like LHCII complex. These dual phosphorylations may more conducive to the repair of the injured PSII. Within chloroplasts, strong light causes the excitation imbalance between PSII and PSI leading to ROS generation and D1 photodamage. Plants prevent and recover from this damage by rearranging the structure of photosynthetic membrane [63]. The phosphorylation of PSII core polypeptides is of importance when the PSII core complex is rapidly photodamaged during high light stress [64]. The movement of impaired PSII complex from PSII-enriched regions to PSI-enriched regions for restore is facilitated by the core protein phosphorylation. A hypothesis supposed that the phosphorylation of injured D1 protein was a signal for the PSII complex movement to PSI-enriched regions from PSII-enriched regions [65,66]. In the PSI-enriched regions, proteases DegP and FtsH decomposed the damaged D1 protein [65,66]. In light condition, the decomposition of damaged D1 protein is indeed delayed in the mutants of *stm8* and *stm7/stm8*, other than in the plants of wild type and *stm7* [30]. The phosphorylation of PSII core protein participates in the change of thylakoid membrane contexture, which facilitated the transport of damaged PSII complex to the PSI-enriched regions for repair. Lack of STN8 impeded the shift of the stacked thylakoid membrane to non-stacked membrane [67]. The stack of photosynthetic membrane blocks the lateral displacement of thylakoid proteins and restrains the conversion of D1 proteins damaged by strong irradiance [67-69]. The stack of thylakoid membrane was not influenced by the lack of STN7 activity [30]. On the contrary, the translocation of movable LHCII fraction from PSII to PSI and state transition1 were impeded [42,34]. The release of PSII subunits was partially regulated by phosphorylation in PSII membranes [70,71]. Upon the phosphorylation of the PSII core proteins, the thylakoid membrane architecture was switched to a state with more fluid, as a result, promoting the cycle of PSII complex repair [72]. In summary, the dual phosphorylations of PSII core proteins and LHCII complexes enhance the dissociation of damaged PSII core protein with LHCII complexes and the migration of the PSII to stromal regions, via the conformational change of thylakoid membrane.

During photodamage occurs, chloroplasts not only need to prevent the accumulation of excess excitation energy in PSII, but also to repair the damaged PSII (as shown in Figure 3). In photodamage, PSII through PSI generates more reduced ferredoxin (Fdr) and thioredoxin (Tdr), which, possibly via kinase, activate STN8 and inactivate PSII core protein phosphatase (PBPC) in PSII membrane region and activates the inactive phosphatase in PSI membrane for D1 dephosphorylation and repair. STN8 catalysed D1 protein phosphorylation is neces-



**Figure 3.** A diagram of the ferredoxin and thioredoxin regulation on STN8, PBPC and STN7 under photodamage. When photodamage occurs, PSII produces reactive oxygen species (ROS) and through PSI generates more reduced ferredoxin (Fdr) and thioredoxin (Tdr). Fdr/Tdr activates STN8 and inactivates PSII core protein phosphatase (PBPC) in PSII membrane region possibly by kinases. The sulfhydryl group to the disulfide bond oxidized by ROS, the inactivated LHCII kinase STN7 become active. Active TAP38 may be inactivated via kinase (different from its activation). In addition, the inactive phosphatase in PSI membrane may be activated by Fdr/Tdr system via kinase for D1 dephosphorylation and repair.

sary for PSII repair, because damaged PSII repair depends on the dissociation of complexes. D1 phosphorylation is conducive to prevent excitation energy accumulation in PSII. In addition, under photodamage, PSII produces reactive oxygen species (ROS), oxidizing the sulfhydryl group to the disulfide bond and activates the inactivated LHCII kinase STN7. LHCII phosphorylation is not only beneficial to the disassembly of PSII complexes, but also enhances cyclic electron transport to synthesize ATP needed to repair damaged PSII complex. Active TAP38 may be inactivated via kinase, which is different from its activation.

## 5. Perspectives

Recent studies have shown that the regulation of reversible dissociation of LHCII is closely related to the site and amount of phosphorylation, but less is known about how plants differentially recognize the sites and control the amount. The regulation of light intensity to this process may be in a more complicated mechanism. In addition, during photodamage, the phosphorylated LHCII binds to PSI reaction center and transfers extra excitation energy to it, a potential damage. Therefore, we need more information about the mechanism of photoprotection of PSI reaction centers in such circumstance. And we know that the low light intensity condition is in favor of the repair of photodamaged PSII. This repair process, in-

volving the synthesis of D1 proteins *de novo*, needs extra ATP. But the low light intensity also activates phosphatases, resulting in the dephosphorylation of LHCII and the dissociation from PSII. As a result, the cyclic electron transfer and the synthesis of ATP will be decreased. Therefore, plants need a complicated system to ensure the process of D1 repair. Evidence shows that the phosphorylation regulation of reversible dissociation of LHCII with photosystems at least consists of ferredoxin/thioredoxin system and kinase system. Up to date, we have known little about kinases in this regulation network.

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