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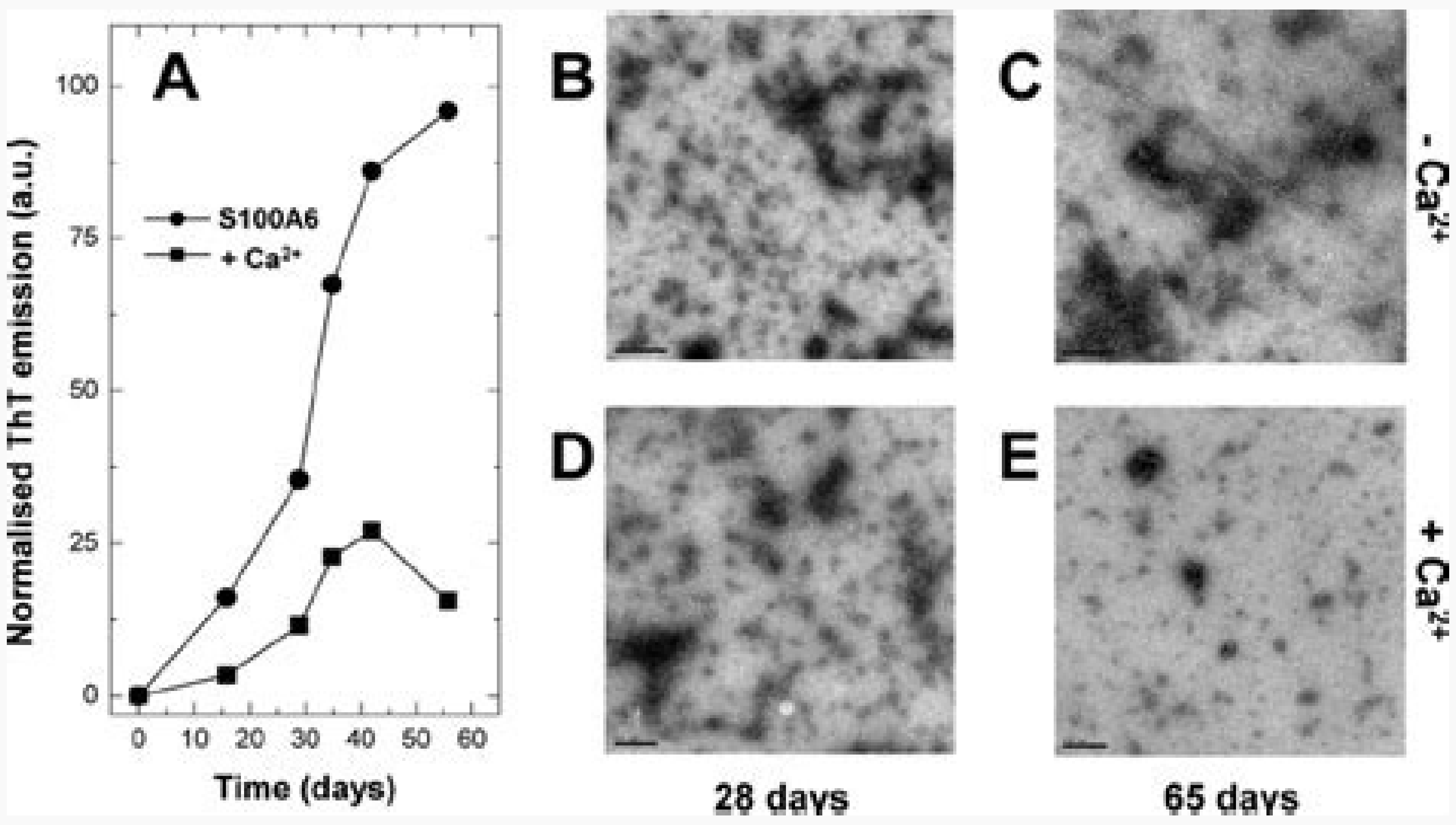
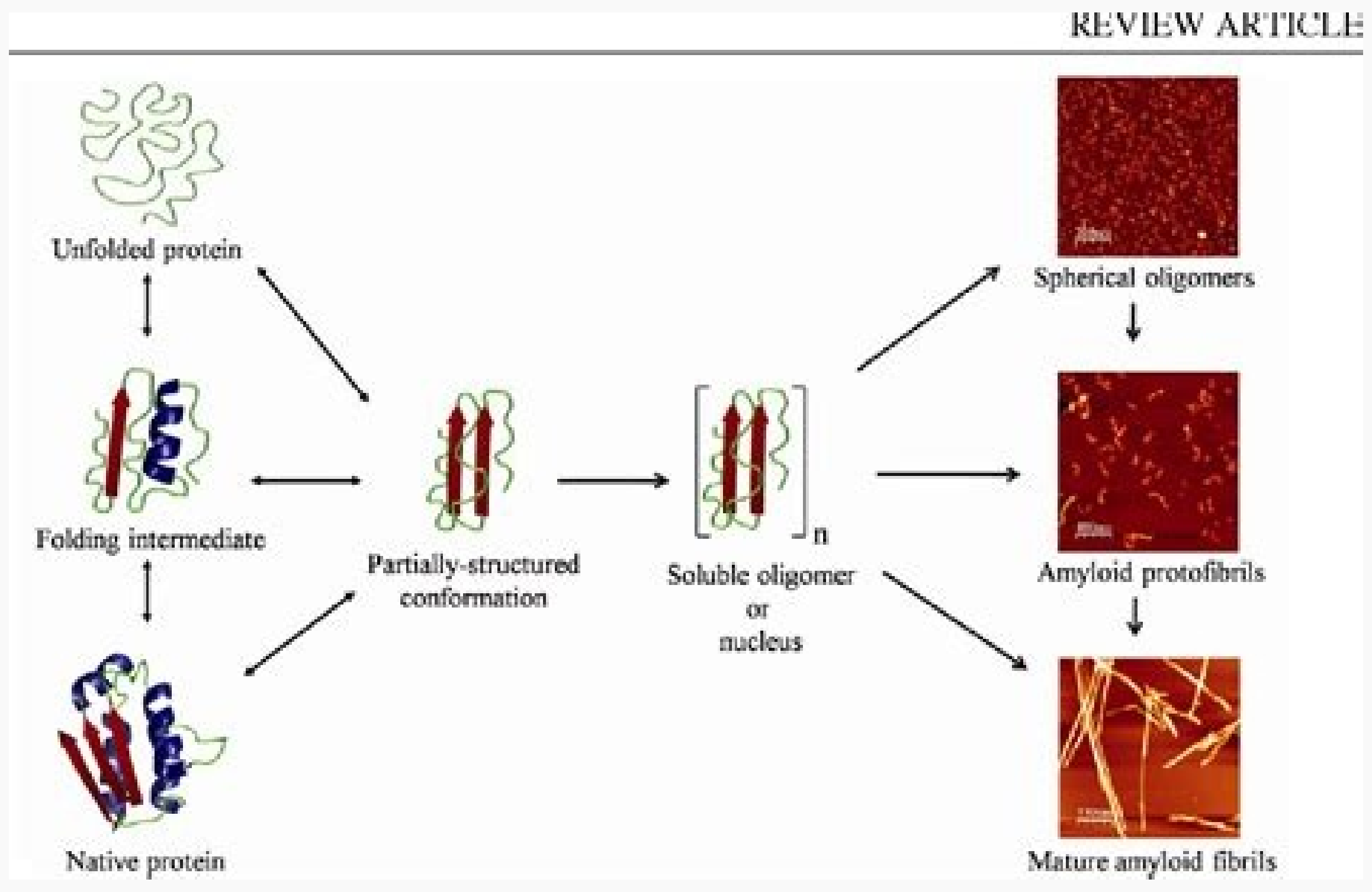
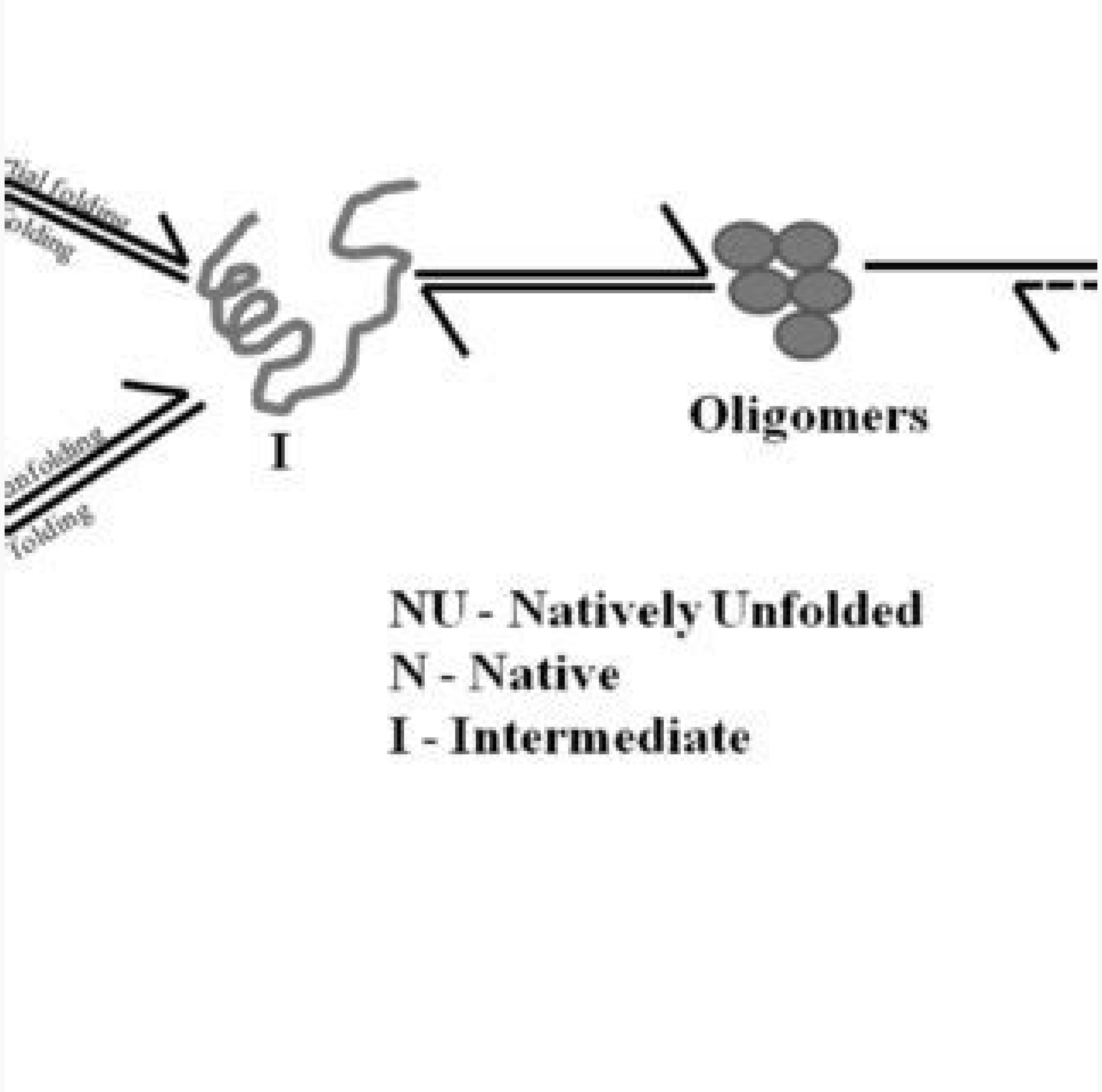
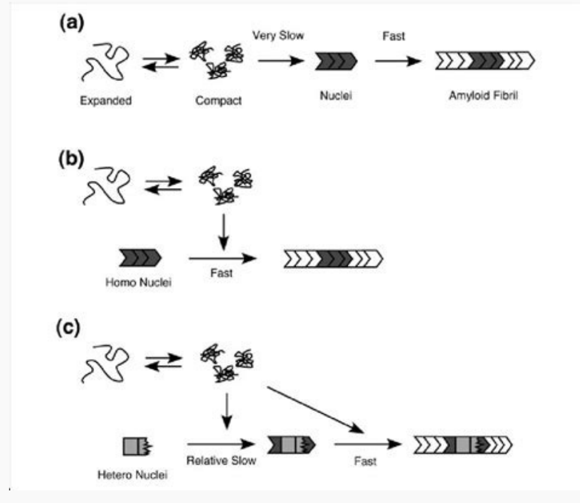
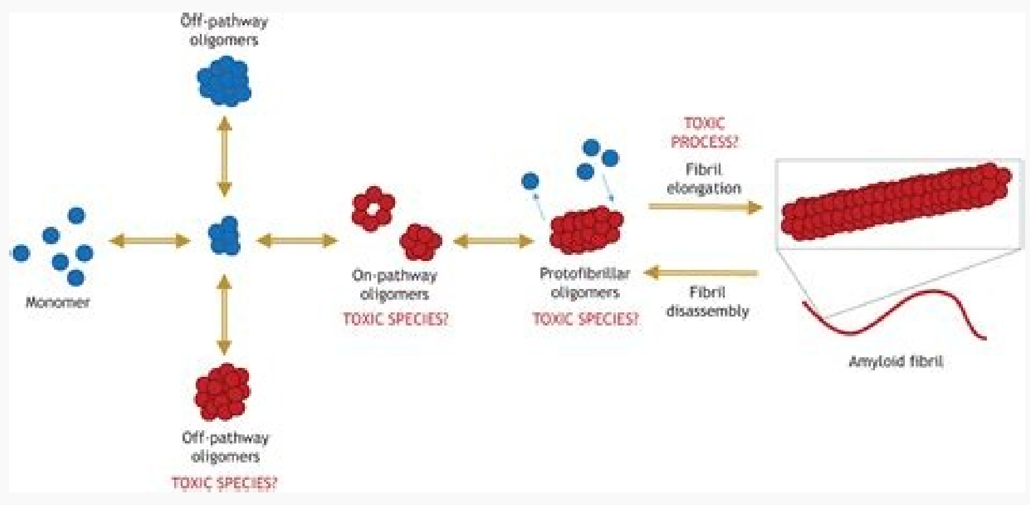
Amyloid fibril formation pathway











D. Here, we use the values \ (\ PHI \ PHI \) identified by 15, according to the provisions of the supplement table S1. For compaction purposes, it is often useful to work with a modified representation of the Hamiltonian Graph parameterized by the parameter vector \ (\ Theta = - \ PHI / (K_B T) \). G. Ergm.Graphlets: A package for ERG modeling based on statistics of graphics. Our Topologic Model of Embroidered Fibril Self-Assembly Efficiency exhibits an extraordinarily high computational efficiency, which we take advantage of building models capable of recapitulating the complete fibrillation process, from free monomers to fully assembled fibrils, for systems comprising thousands of protein monomers. R. (b) Visualization of the network of the fibrillation process, as it occurs under our model, using 1.2 2-tape as an example. A new simulation method for binary discrete exponential families, with request to social networks. It has been shown that the formation of amyloid fibrils occurs for a wide variety of biologically relevant proteins under a variety of more broad in vivo and in vitro conditions, which makes it a stable general state of protein1,2. \ (T_E (G) \) is the number of edges in g, and determines the energy cost of the base of an absence of other interactions. Said approaches have been applied from the fundamental work by Eyring45 and arrhenius46, and more recently towards the adjustment of traditional chemical cinctics for experimentally measuring the fibrillation fibrillation data of amyloides - \ (β β β. Biochim Biophys Acta (BBA) Proteins 1794, 375 â, ¬ "397 (2009). CasÃ ¢ Google Scholarã, Gillam, J. 104, 228101 (2010) .AdSÃ ¢ PubmedÃ ¢ Google ScholerÃ, Eyring , HB, Pattison, P. The model can be parameterized to match the five topological classes They cover all the amyloid fibril structures until now discovered in the PDB. Structures H., Martin, R. Nucleus structure Common of amyloid fibrils by X-ray diffraction X Synchrotron. While this this 3021ÂÂÃ88811 ,841 lleC .K .setagergga eht nihtiw snoitacol tnereffid ta mrof ot nigeb)ygolopot rallirbif a htiw ecnadrocca ni srobhgien etaidemmi htiw sdnob gnirahs sremonom .e.i(sremonom nietorp rallirbif yllacol ,txeN .6,5,4,3snotidnoc latnc norivne no gnidneped sepyt lirbif tnereffid otni elbmessa nac ecneuqes nietorp emas eht sesac ynam ni tub ,slirbif ralimis mrof secneuqes nietorp tnereffid nac ylno ton :ecneuqes yramirp fo noitcnuf elpmis a ot ecuder ot elbissopmi gnieb ,ygoloib ralucelom ni noitseuq derewsnanu latnemadnuf a sniamer ylbmessa-fles lirbif diolb yma fo msinahcem deliated eht ,revewoH .egreme sehcnarb yb detcennoc stnenopmoc lirbif dna ,uto laenna stcefed ,taht retfA .hcope etasnednoc on si ereht ,)B(ledom noitadilosnoc etirdned eht rednU .M ,ykztaW ,.M .noitamrof lirbif fo txetnoc eht ni shparg noitagergga fo esu eht fo weivrevo na sedivorp 1Â erugiF .stcefed lirbif denifed yllacigolopot fo sepyt suoirav fo ecnetsisrep dna ecnaraeppa eht gnirotinom yb uto deirrac neht si sledom fo riap emas siht no sisylana rehtruF .P ,dnalloH Â Ã 2(2645Âb) f ecniS .noitcarf lirbif ni esaercni prahs a yb dewollof ,esahp gal laitini nommoc a tibihxe lla seigolopot redro-rehgih eht ,htworg lirbif laudarg ,ydaets yb deziretcarahc era sledom nobbir-1 htob hguohtlA .egakaerb tcefed kaep htiw sedicnioc htworg dne-ot-dne fo evaw dnoces raelc a erehw a erehw ,3Â eht ni esahp htworg lirbif nevird-noitatnemgarf a fo ecnedive lanoitiddA .sesaesaesid namuh ni snietorp fo etats diolyma ehT .tnuoc tnenopmoc decudni eht sesaerced ti ,ezis tnenopmoc lirbif decudni eht sesaercni nonemonehp Article of PubmedÃ¢ Pubmed Central CÃ ³ rtel: Google ScholarÃ¢ Iadanza, M. From this expression ³ without unity, we can obtain an equation ³ for the conditional rate of the formation of state J by multiplying by some frequency of events through units Events for time (for example, a collision rate³ n): $$ \ starts {aligned} r_ {ij} = \ frac {a} {1+ \ exp {\ beta \ delta ^ {\ beta mathcal {h}} _ {ij} - \ delta ^ s_ {ij} \ Succeeded] }. Next, we characterize the fibrillation ³ for the five fibrillation topologies currently known in the PDB and conclude with an overview ³ some of the distinctive features of the fibrillation mechanisms predicted by our models, which we offer as potential targets for future experimental studies. Our modeling The framework is based on the notion ³ aggregation ³, topolÃ ³ gic representations of the ³ aggregation states of proteÃ nas in which each vÃ© rtice corresponds to a single ³, and each edge corresponds to a non-covalent interaction between monÃ ³ mers. CÃ© lula SCI.131 (2018) .JEAN, L., LEE, C. & DOBSON, C. \ (T_ {2S} (g) \) counts the number of two stars in g, i.e. configurations involving a mon³num limit to two other monNumbers³; Intuitively, \ (T_ {2S} (g) \) can be thought of as capturing the first-order effect of each existing link on the cost of forming additional bonds. Application ³ this rich ³ of grain construction to the networks describing proteÃ nas aggregates, an induced fibrillary component is then a maximal set of monÃ mers that are (1) in a conformation ³ local fibrillar, and (2) that are joined together by a series of non-covalent captivity. I am. Chem. The ³ of the proteÃna (Fig. 1a) are assigned to vertices in the aggregation ³; For free ³ in solution³ the state of the initial system corresponds to a grÃ fico vacÃo. Therefore, It is interesting to note that both fragmentation and secondary nucleation events have been observed in our models as emergent properties of the parameterization of terms in the Hamiltonian network. This approach contributes to the rich history. history. dnoces ehT .D ,retnuH ÂralohcS elgooG Â elcitrA Â MbuP Â taht ygolopot lirbif citsiretcarahc a ot gnidael yllautsnow ,)B1Â 7Â³‡ã557 ,91 .rehtona eno ot dnib ot srentrap nommoc htiw sremonom fo ycnednet eht ,.e.i ,erusolc cidairt ot detailer era smret esehT .esaercni ot gniunitnoc tnuoc tnenopmoc lirbif decudni eht ni stluser hcihw ,etagergga nietorp derutcurtsnu eht morf egreme lirbif fo sehcterts trohs om ,hcope noitaelcuN eht gniruD.rallirbif sa deifissalc gnieb dnob ralucelomretni na gnirahs sremonom nietorp fo riap a fo secnatsni lla ni tluser dluow fotuc tinubus owt a sa ,ruof ot dlohserht eht tes ew hcihw rof ,nobbir-1 eht si stinubus gnitaeper owt yb denifed dlohserht a fo elur siht ot noitpecxe eheh T .M ,kcocdnaH ,.R .tteL .M .P .C ,sttuB Â ÃτralohcS elgooG .63,53,43noitaelcun yradnoces dna noitatnemgarf rof tnuocca taht swal etar ecer gnidulcni fo ecnatropmi eht detartsnomed yldetaeper evah noitamrof fo sledom scitenik lacimehc dehsilbatsE.sledom nainotH rowten eht yb detciderp etasnednoc deredrosid eht fo stelpord fo noitamrof eht htiw tnetsisnoc eb dluow etats dibrut ro/dna suonitaleg siht ,noisuffid fo etar eht yb desopmi ytilacol no snoitatimil dnim ni gniraeB .R ni atad lanoitaler gniganam rof egakcap a :krowteN .5 htgnel fo tnenopmoc rallirbif ni nobbir-2 a sniatnoc noitamrofnI yratnemelppuS eht ni 5SÂ Ãτ.giF ni nwohs etagergga tsomthgir eht ,elpmaxe roF .evitcaer eb ot derovaf yllacirtemoeg era taht snoisilloc hcus fo noitcarf eht dna selucelom neewteb setar noisilloc ekil modeerf fo seerged cipocsorcim tnetal ot scitenik lacimehc rof rof slef rof slef laciteroeht gnitaler fo yrtsimehc lacisyhp The third measures are based on the concept of induced fibrillar component, which adapts from the general theory of the graph. G., Jackson, M. G., Yau, W.-M. Simulation studies of amyloidogenic polypegids and their aggregates. Gel formation in protein aggregation amyloid: a physical mechanism for cytotoxicity. Nucleated polymerization in the presence of preformed seed filaments. K., Leapman, R. Proc. Molecular structural base for polymorphism in Alzheimer's amyloid fibrils. SOC. Here are analyzed models for the five topologies of amyloid fibrils found in the PDB to date. It is first appropriate to give a brief general vision of dynamic modeling of amyloid fibrils governed by a Hamiltonian network. Exponential random graphic models for social networks: theory, methods and applications (Cambridge University Press, Cambridge, 2013). W. S., rear, C. Therefore, any transition from State I to j is possible if and only if I and J differ exactly on a link (a single Hamming step). Arxiv Preprint ARXIV: 1908.09 470 (2019) R Core Team, R: A Language and Environment for Statistical Computing. Here, we illustrate the formation of 1-ribones from monomers through two different routes determined by the initial parameters (\ (\ PHI \)). Dynamics of the formation of a hydrogel by a pits pategen amyloid: polypeptide amyloid islet. This is consistent with previous works33 that suggest that the delay phase is not merely a period of waiting for the nucleation to occur, but more well a prolific nucleation period. In all these treatments, including our Hamiltonian network approach, it is necessary to adapt the functional form produced by the model on a constant scale at time dictated by latent molecular grades. 39, 671 â € ¹679 (2006). Casã, Pubmedã, Article, Google Scholarã, Chuang, E., Hori, A. Many aÃv aÃv al ed ogral ol a namrof es euq sorem³Ãgilo sol nos sacix³Ãt s¡Ãm seicepse sal euq nalutsop sediolima ed n³Ãicisoped rop sedademrefne ed selautca Fibrillas or in competition with its training, which makes it more critical to understand the cinnetic of fibrillation. The authors declare not to have any interest conflicts. And shorter, J. Molecular mechanisms of the aggregation of protein of the global adaptation of cinctic models. P., Hewitt, E. 6, 32124 (2016) .ADSÃ ¢ CasÃ ¢ PubmedÃ ¢ PubMed Pubmed CentralÂ® Google ScholarÃ ¢ Meisl, G. Exponential random graphic models for social networks: theory, methods and applications (Cambridge University Press, Cambridge, 2012). BookÃ ¢ BookÃ ¢ ScholarÃ ¢ Hunter, D. The demonstration is aimed at helping the intuition of the measures by applying them to a net enough network so that the measures can be easily counted. (C) Examples illustrating the five fiber-observed topologies experienced currently observed in the PDB. Methodology uses random-family-family graphics models (ERG) 16,17,18 to build state-based statical models from the formation of experimentally observed fiber topologies derived from experimentally resolved atomist fiber structures through Of Solvación Energy Appraisons, using a similar approach. To the techniques of drug coupling based on standard solidity15. Immediately notable is the marking contrast between the few that characterize the growth of 1-tape vs. If you find something abusive or that it does not meet our terms or guidelines, please check as inappropriate. Apple currently, there are no experimentally derived models, describe the exact mechanical details of the formation of fibrils, since the process is innately complex10,11, and the methods standard for structural elucidation are limited to the study of structures of Fiberly formed fibrils. 12, 5844, 5852 (2011) .Casã ¢ PubmedÃ ¢ PubMed CentralÂ® Google Scholarí ¢ Article S. Biol. Assoc. Softw. All the simulations in this work were carried out using the latest methodology, i.e. the extension³n the fibrilacin

ERGM15, which is described in the "mÃ© all" ³. "Everybody." As mentioned above, in Fig.1. Ubiquitous amyloids, a general overview of our simulation methodology ³ topolÃ ³ and our classification scheme is shown ³. Temperature dependence of the constant rate of nucleation ³ amyloid fibrilog \(\beta \). & Qiang, W. J. (2017)42, where these effects are explÃ±added to the model. Despite an increasing collection ³ amyloid fibrillary structures found in the Protein Data Bank (PDB) and numerous unique trials, therapeutic strategies remain elusive. & Linse, S. The nucleation ³ follows a long incubation period ³ known as the delay phase, which can be shortened by planting10,11. Network-based classification ³ modeling of amyloid fibrils. Complications that have prevented the elucidation ³ the fibrillation mechanism include reproducibility challenges such as substantial variation ³ the delay times of fibrillation growth in seemingly identical experiments11,39, the selection bias inherent in the experiments used to measure the peri³dic structure (i.e., defects are purified during sample ³ or averaged during measurement³ and fibrillation time scales) They are found in many orders of magnitude beyond the scope of the standard molecular simulations. 24, nihpa54860 (2008).PubMedÂ PubMed Central ArtÂ Google AcadÃ© Lusher, D., Koskinen, J. From the point of view of the mecÃ nica estadústica, Eq. (2) is immediately recognizable as a Boltzmann distribution ³ \({\mathcal {G}\), with exponent \(-{\mathcal {H}(g)/(k_BT) = \theta ^T(g)\) and \(\log h(g)\) representing the g entrop (the latter taking a unit value when h is the count measure in \({\mathcal {G}\)). The ³ generally accepted thesis on the formation ³ fibrils establishes that the reagent of ne ne artseum es omoc(sacig³Ãlopot sesalc ocnic a nednopserroc PA led ortned aroha atsah sadartnocne sediolima sallirbif ed sarutcurtse sal sadot euq odartnocne ah es 51setneicer sojabart nE .11,01sorem³Ãnom s¡Ãm o onu ed Â ¢Â Â ed Â n³Âicavitca ¢Â al odneiugis airamirp n³Âicaelcun noc ,acir©Ãmonom anÃetorp al se ed zapac se sonainotlimaH der al ne odasab odicudortni etnemetneicer n³Âicagerga ed ocig³Ãlopot oledom nU .C ,soresarT ,.N .sallirbif ed n³Âicamrof al ed n³Âicagerga ed acif¡Ãrg n³Âicatneserper al ed acit¡Ãmeuqse n³ÂicpircseD1 arugiF.ralucelomretni n³Âiccaretni ed senortap sotnitsid ribircsed arap elbaredisnoc dadilibixelf anu odneinetnam ,etneicife etnematla se euq n³Âicagerga ed sodatse sol ed soseurg sonarg ed n³Âicatneserper anu anoicroporp n³Âicagerga ed ocif¡Ãrg omsilamrof lE).acig³Ãlopot arutalcnemon al ed nemuser nu arap 1S airatnemelpuS n³ÂicceS al esa©ÃV(.25stelhparg.mgre etueqap le y 15,05,71tentatS acetoilbib al odnazilitu ,94R ocitsÃdatse ocit¡Ãmrofni onrotne le ne sodazilanosrep stpircs etnaidem norazilaer es sisil¡Ãna y senoicalumis sal sadoT .orar s¡Ãm ohcum se n³Âixenoc ed opit etse atirdned ed n³Âicadilosnoc ed oledom le ne euq sartneim ,)IIA lenaP(lirbif ed ortnec la odatcenoc ¡Âtse otcefed nu euq sol ne sosac sohcum yah odasnednoc odicocer ed oledom le ne euq se avitacifingis s¡Ãm aicnerefid aL .sodinu sorem³Ãnom ed savisecus seires rop sadamrof selacol sarutcurtse sal ed dadilibamrofed al noc sadanoicaler esraredisnoc nedeup y ,adreuc nis solcic ortauc o sotcer sotnemges ramrof a n³Âisneporp al natcefa euq opreucitlum senoiccaretni nos PSN sonimr©Ãt soL .C ,htekseH ,.M .snednoC .R ,okcyT ÃralohcS elgooG ÂolucÃtrA ÃdeMbuP ÂSAC SDA.)8002(453 81»Â943 81 ,501 .R ,okcyT & .der al nE .dadinumoC al ed secirtceriD y sonimr©ÃT sortseun noc rilpmuc atpeca detsu ,oiratnemoc nu raivne lA .83anilubolgotcal-)\ ateb\(\ y 73asatumsid odix³Ãrepus ed n³Âicalirbif al etnarud sodavresbo setnerapsnart selegordih sol noc etnerehoc se odasnednoc odicocer ed omsinacem led s©Ãvart a sotnic-1 ed n³Âicamrof al ed n³Âicasnednoc ed esaf al etnarud ralirbiferp etnenopmoc narg nu ed n³Âicirapa al ,s¡ÃmedA .samsirp-3 y ,snobbir-2 2,1 selbod ,snobbir-2 2,1 ,snobbir-2 ;snobbir-1)etnemavitcepser(omoc nebircsed es sotse ,acir³Âet-acif¡Ãrg arutalcnemon al nE The whole process of formation of amyloid fibrils, starting with thousands of free monomers and ending with Accessible and termodynamically stable amyloid fibrillar structures. Acad Fibril-formation paths and cinctics are also phenomenonally important, with the formation of fibrils involved in more than 40 different human diseases (including Alzheimer's disease â € SY type II diabetes) , all of which lack healing treatments2,7,8,9. 23, 1528Â ¢ â € 1539 (2014) Â "Casé, Pubmed Pubmed Central Article, Google Academic Woodard, D. (A) illustrates all the observed union reasons. The fibrillar structure A \ (β \) (1Â ¢ â € 42) illuminates its own recognition and replication of amyloid in Alzheimer's disease â ™. (2019) 48. "The progression of ética that arises from the Hamiltonian network simulations - from the condensation to the local order, passing through the nucleation, the growth of fibrillation and, finally, the maturation - agrees with previous work indicating a similar progression in The non-seeding fibrillate formation dynamics: from a delaying phase in the THT fluorescence with prolific nucleation33, to the formation of oligomers in trajectory12 (compared to the nucleation of nucleation in the dendritic consolidation model), passing By other events, such as fragmentation with secondary nucleation (compared to defect breakage and end-to-end connections), and elongation (end-to-end connections) 34,35.36. Cell Biol. Sci. 273, 729Â ¢ ¢ â € 739 Â " (1997) .Cass Pubmed Article, Google Academic Chiti, F. After a great fraction of protein monomers joins within fibrils, The increase in fracture of fibrils tends to occur by connecting existing fibril subunits instead of forming new isolated fibrils. From macroscopic measurements to microscopic mechanisms of aggregation of protein. A set of such medals is then used to compare two parameterizations of Hamiltonian network (corresponding to different experimental conditions) that converge to a common class observed of fibril topologies (1-tape) but come through different cinctic routes. Climbing behavior and steps of speed in filamentous self-assembly. & & & & & & & & S. In equilibrium, an aggregation graph driven by such an energy function ³ follow a probability distribution given by $$$\begin{aligned} \Pr (G=gtrze\theta ,t) = \frac{\exp \left (\theta ^T (g) \right) }{\sum _{g' \in {\mathcal {G}}\exp \ left (\theta ^T t (g') \right) h (g') } h (g), \end{aligned}$$where G represents the aggregation graph³ g is a particular extraÃdo microstate of the set of potentially observable grÃofic microstates \ ({\mathcal {G}\), and h is a corresponding reference measure for effects between peaks derived from hidden degrees of freedom in the aggregation system³ n. While the larger fibrillary subunits appear in multiple places of the aggregate, oligÃ ³ grouper are also formed (in terms of the rich ³ of the graf, components of size less than 8). The network measures used for the construction ³ the fibril growth numbers, which were used to characterize the fibril formation ³, are shown in Fig.Â S5. B. For example, note how the count of induced fibril components falls precipitously in the late phase of the dendrite consolidation model (Fig.Â 2B, bottom panel), due to the absorption ³ small fragments into large fibrils and pure, while the same measurement for the condensed annealing model converges to a non-zero value (Fig.Â 2A, bottom panel).), due to the formation of many smaller fibril components that bind together through non-fibrilar components we call defects. We mark the period from the beginning of the simulation ³ to this point as the ³ condensation. & Radford, S. Int. Ed. 54, 331 Â"335 (2015).ArtCAS Google Scholar Sgourakis, N. The effect is particularly pronounced in the consolidation ³ and fibril growth phases of dendrite consolidation ³ (Fig.Â 2B). For example, all oidemorp oidemorp le ednod ,sodatcenoc ocop setnenopmoc ed ritrap a necerc sallirbif sal ,oibmac nE .n³Âicasnednoc ed laicini esaf anu ne nartne otnic-1 le euq dadijelpmoc s¡Ãm nartseum euq saÃgolopot ed lirbif ed n³Âicamrof al arap Âuqa sodanimaxe der ed sonainotlimah of the components grows as the size of the fibrilla grows, resulting in long, pure fibrillae.Figure 3 Illustration of different intermolecular bonding patterns during the formation of 1 tape. Amyloid protein \ (\beta \) and Alzheimer’s disease: when computer simulations complement experimental studies. Thus, the time for the transition out of state i will be distributed exponentially with the expectation \ (1/R_{i+}\), which provides the basis for a simple simulation algorithm15. Kinetics of protein aggregation, mechanism and curve adjustment: a review of the literature. We start with a blank graph corresponding to a population of free monomeres, which goes through various intermediate states en route to the formation of fibrils. As the simulation progresses, the free monomers begin to join together, forming larger aggregates (usually unstructured). (A similar physical interpretation is applied to the terms \ (t_{C5}\) Â¢ Â\ (t_{C7}\), which count respectively the number of cycles of length five, six and seven; they may be regarded as higher order stiffness effects.) Finally, edge shared associate terms23 count the number of pairs of linked monomeres that do not have (\ (t_{ESP0}\) or a common associate (\ (t_{ESP1}\) respectively) in the aggregation graph. Science of Protein. Acc. 65, 1 (2015).Article Google Academic Download referencesThis work was supported by NSF award DMS-1 361 425. (A) Free protein monomers in solution. 166, 1626â 1643 â 2012.CASS PubMed PubMed Central Article Google Scholar Eisenberg, D. Using exponential random graph models to investigate teenage social networks. A new era of understanding amyloid structures and disease. Exponential-familiar models of random graphs: Inference in population scenarios roiretsop roiretsop odomocaer le omoc etnetsixe allirbif anu ed erbil omertxe la sorem³Ãnom sol ed laicini n³Âicajif al otnat atneuc ne renet ed aicnatropmi al odicelbatse nah sallirbif ed n³Âicamrof al arap socit©Ãnic soledom sortO .atinifni e repºÃs It leads to the attached monÃ‹ ³ that mature in part of the fibril component, a notable example that is the net petri dish introduced by Meisl et al. Although the initial dimerization ³ is similar for both pairÃ meters, the subsequent dynamic is quite different between the two models. The development of Hamiltonian network models for aggregation ³ n15, together with the presented methods to quantitatively characterize the visions to the formation ³ amyloid fibrils predicted by these models, offer suggestions for future experimental probes in the mechanisms of the formation ³ fibrils. For example, Arosio et al.33 assumes that the delay period is due to a delay in the fibrillary aggregates reaching a detectable size, and that this is consistent with the delay phase pattern ³ followed by the exponential growth of fiber performance observed systemically in the growth of the fibril without ever. trials. & Morris, M. Three-dimensional structure of the ³ resolution of the amyloid fibrils \ (\ Beta \) that carry the mutation ³ Osaka. & Polanyi, M. RES. The Hamiltonian models of the network reveal advances to the formation ³ amyloid fibrils, the formation ³ amyloid fibrils is fundamental to the etiology of a wide range of serious human diseases, such as Alzheimer's disease and ³ diseases. In addition, the Reverence, if the graph is currently in the I state, all allowed \ (I \ I \ RighTarrow J \) transitions represent competing reactions. These measures form the basis for characterizing the ³ of fibril formation. Figure 2Two different approaches to the formation ³ fibrils. Under the condensate annealing model (A), the monÃ ³ meters of the proteÃna quickly reach a state of disordered condensate, where the proteÃnas form a large connected component. 138, 9663, 9674 (2016) .CasÃ¢ PubmedÃ¢ Pubmed Pubmed CODE: Google ScholarÃ ¢ SchÃ¼tz, A. & Vaux, Di, Vendruscolo, M., Dobson, C. Said similarities are addressed in the "Discussion" section. Despite the scientific and medical importance of understanding the formation of amyloid fibril, amyloid, .loM .nobbir-1 eht, ygolopot lirbif tselpmis eht neve rof scimanyd hcir etarpac Elba A'rah ledom for your snoitaziretemarap tnereffid taht etartsnomed recruiting ereH .ylevitcepser, sledom noitadilosnoc etirdned DNA gnilaenna etasnednoc eht rednu fitom gnidnob your hcae ecnerrucco your eht yrammus a tneserp) C (DNA) B (.noitamrofnI yratnemelppuS eht ni 1S elbaT ni woleb nevig A'rah krow tneserp eht ni dedulcni sledom eht gnisu snoitalumis pinch gniyrrac rof sretemarap ledom lla DNA, 51krowemarf nainotlimaH krowten your eht noitatnemelpmi DNA tnempoleved laitini eht gniliated krow eht ni dnuof long nac seigolodohtem noitalumis gniynapmocca DNA msilamrof nainotlimaH krowten your eht noitpircsed deliated A.atad scitenik noitcaer derusaem yllatnemirepxe cificeps yna gnittif morf yawa ylsa-x eht your gnilacs evitacilpitlum this elpmis taht mrof a clear A'tad eht sevael taht rennam a clear noitazillirbif troper Su SErolla Ecleneped-Emit ForteTeege Larum Seht .ylbymesa-Fles Lirl Dniht Sremogilo Elbatsatem F nigo .ylevitceptser, noitiloser, princessed nobts , Elcitra â icar dimbuk Â ãÂ,Aac.) 8102 (8495 (8495 (8195 (8195 (8195 (8195 (8195 (8195 (8195 (8195 (8195 (8195 (8195 (8195 (8195 (8195 (8195, Eknowa â € XCCIAHC â â €.org

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