PublisherInfo		
PublisherName	:	BioMed Central
PublisherLocation	:	London
PublisherImprintName	:	BioMed Central

Protein folding

ArticleInfo		
ArticleID	:	4011
ArticleDOI	:	10.1186/gb-spotlight-20010313-01
ArticleCitationID	:	spotlight-20010313-01
ArticleSequenceNumber	:	82
ArticleCategory	:	Research news
ArticleFirstPage	:	1
ArticleLastPage	:	2
ArticleHistory	:	RegistrationDate : 2001–03–13 OnlineDate : 2001–03–13
ArticleCopyright	:	BioMed Central Ltd2001
ArticleGrants	:	
ArticleContext	:	130592211

Kenneth Lee
Email: kenlee_fr@yahoo.fr

The characteristic protein aggregates seen in the brains of patients with Alzheimer's or Creutzfeldt-Jakob diseases are caused by the proteins adopting abnormal shapes called amyloid fibrils. In the 8 March Nature, Christopher Dobson and colleagues at the Oxford Centre for Molecular Sciences, UK, report that proteins outside the brain are also capable of assuming abnormal 'amyloid' structures.

In a physiological environment the muscle protein myoglobin is globular and its structure does not suggest a tendency to form amyloid fibrils. But in a screening process in which temperature, pH and buffers were varied, Fändrich *et al* found a chemical environment - 50 mM sodium borate, pH 9.0 at 65°C - that favoured conversion of myoglobin from its native structure into amyloid fibrils (*Nature* 2001, **410:**165-166). They believe that organisms have evolved safeguards against this protein transition, but ageing or mutational changes could sometimes cause the protective mechanisms to break down.

References

1. Fändrich M, Fletcher MA, Dobson CM: Amyloid fibrils from muscle myoglobin. *Nature* 2001, 410:165-166., [http://www.nature.com/nature/]

2. Oxford Centre for Molecular Sciences, [http://www.ocms.ox.ac.uk/]