

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

## Circulating DNA fragments involved in vasculitis

ArticleInfo		
ArticleID	:	4145
ArticleDOI	:	10.1186/gb-spotlight-20010712-01
ArticleCitationID	:	spotlight-20010712-01
ArticleSequenceNumber	:	216
ArticleCategory	:	Research news
ArticleFirstPage	:	1
ArticleLastPage	:	2
ArticleHistory	:	RegistrationDate : 2001-07-12 OnlineDate : 2001-07-12
ArticleCopyright	:	BioMed Central Ltd2001
ArticleGrants	:	
ArticleContext	:	130592211

Tudor Toma

Email: ttoma@mail.dntis.ro

---

Vasculitis is a key pathogenic element in systemic lupus erythematosus (SLE) and results from activation of immune system triggered by unknown molecular mechanisms. In the July [Annals of Rheumatic Diseases](#), Miyata and colleagues at the Fukushima Medical University School of Medicine, Fukushima City, Japan, show that oligodeoxynucleotides (ODN) containing the CpG motif in the sera of patients with SLE may be implicated in vasculitis through activation of proinflammatory cytokines and expression of ICAM-1.

Miyata *et al.* transfected human umbilical vein endothelial cells (EC) with a CpG-ODN or a control analogue, GpC-ODN. They measured ICAM-1 expression by flow cytometry, and mRNA for interleukin 1 (IL1), IL6, IL8, tumour necrosis factor  $\alpha$  (TNF- $\alpha$ ), interferon  $\beta$  (IFN $\beta$ ) and ICAM-1 by semiquantitative reverse transcriptase polymerase chain reaction. The CpG-ODN transfection augmented the expression of ICAM-1 on EC and increased mRNA levels of IL6, IL8, TNF $\alpha$ , IFN $\beta$  and ICAM-1. GpC-ODN had no effect (*Ann Rheum Dis* 2001, **60**:685-689).

These results suggest the participation of circulating DNA fragments in the pathogenesis of vasculitis in SLE and the authors speculate that the pathogenic DNA fragments are derived either from CpG-rich islands or from microbial DNA.

## References

1. Miyata M, Ito O, Kobayashi H, Sasajima T, Ohira H, Suzuki S, Kasukawa R: CpG-DNA derived from sera in systemic lupus erythematosus enhances ICAM-1 expression on endothelial cells. *Ann Rheum Dis* 2001, 60:685-689., [<http://ard.bmjournals.com/cgi/content/abstract/60/7/685>]