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ANTI-T CELL ANTIBODIES IN CHILDREN WITH JUVENILE RHEUMATOID ARTHRITIS (JRA). Karyl S. Barron, Earl J. Brewer, and William T. Shearer. Baylor College of Medicine, Houston, TX 77030, USA.

A few patients with JRA have been reported to have naturally occurring anti-T cell antibodies (Strelkauskas JI 120:1278, 1978). The present investigation was made to determine the prevalence of anti-T cell antibodies in 66 children with various connective tissue diseases. E-rosetted T cells were exposed to heat-inactivated serums from patients or controls at 4°C for 1 hour, washed, exposed to undiluted fresh rabbit complement at 37°C for 2 hours, and assessed for viability by Trypan blue dye exclusion. Anti-T cell antibodies were found in 43/44 JRA patients (mean cytotoxicity 15.2%±7.3) and in 10/10 children with SLE (mean cytotoxicity 20.0%±8.4), but in none of 15 normal controls or 12 children with other arthritides including: dermatomyositis, scleroderma, psoriatic arthritis, acute rheumatic fever, and episodic arthritis/arthralgia. There was no significant difference in mean % cytotoxicity among the JRA subclasses. In the JRA patients the % cytotoxicity was positively correlated with ESR ($p=0.01$), but not to the presence or absence of rheumatoid factor, antinuclear antibodies, or immune complexes (C1q-BA). The sera of 3 JRA patients (but not normal sera) repeatedly inhibited the stimulation of normal lymphocytes by mitogens and antigens by 47-99% (measured by the incorporation of ^{3}HdThd) when added to the culture system within the first 24 hours in a final dilution of 1:12 or 1:24 ($p<0.001$). Thus, anti-T cell antibodies were found in the majority of randomly selected JRA patients and exerted inhibitory effects upon selective aspects of normal lymphocyte function.

ALPHA INTERFERON IN JUVENILE ARTHRITIS. John J. Miller, III, and Ann M. Arvin. Children's Hospital at Stanford, Palo Alto, CA. 94304, USA.

Interferon has been reported to be present in sera and synovial fluids of about 50% of adults with systemic lupus erythematosus and other rheumatic diseases. We measured interferon in sera and synovial fluids of children with juvenile arthritis. Diluted samples were added to cultures of foreskin fibroblasts and protection against infection with vesicular stomatitis virus was measured. Interferon was found with a frequency similar to that in adult rheumatid disease and was not related to the type of juvenile arthritis:

Form of arthritis	Number elevated/number tested		
	Serum samples	Synovial fluid samples	Patients
Systemic	10/21	0/1	5/12
Systemic → poly	3/5	(none tested)	1/2
Polyarticular	6/17	3/6	6/14
Pauciarticular	9/22	4/7	8/16

Positive titers ranged from 8 to 46 units. The presence of interferon did not correlate with antinuclear antibodies, rheumatoid factor, C1q binding, the presence of C3d, "catabolin" (cartilage degrading activity), or with clinical disease activity.
The antiviral activity was destroyed by heat and acidity, which is characteristic of γ-interferon, but also by sheep antibody against human α-interferon. Thus, the interferon in these patients is similar to the unusual α-interferon found in adults with systemic lupus.

A COMPARISON OF ANTIBODIES TO NUCLEAR AND CYTOPLASMIC ANTIGENS AMONG CHILDREN AND ADULTS WITH SLE AND THEIR FIRST DEGREE RELATIVES. Thomas Lehman, Virgil Hanson, Nathan Zvaifler, Gordon Sharp, and Margaret Alspaugh, Bethesda, MD, Los Angeles and San Diego, CA, and Colombia, MO, USA

We compared 24 children with SLE and 94 of their asymptomatic first degree relatives, with 8 adults with SLE and 33 of their asymptomatic first degree relatives to determine whether the frequency of serologic abnormalities among children and adults with SLE or their assymptomatic first degree relatives differed. All were tested for anti-nuclear antibodies(ANA), anti-lymphocyte antibodies(ALA), antibodies directed against DNA(anti-DNA), RNP(anti-RNP), Sm(anti-Sm), SSA(anti-SSA), SSB(anti-SSB), PM-1, and SCl-70.

	Children with SLE	Adults with SLE	Children's relatives	Adults' relatives
ANA	24/24	8/8	29/94	7/33
ALA	7/24	4/8	15/94	1/33
Anti-DNA	6/24	7/8	26/94	13/33
Anti-RNP	2/24	4/8	3/94	0/33
Anti-Sm	2/24	4/8	1/94	0/33
Anti-SSA	5/24	3/8	4/94	1/33
Anti-SSB	2/24	0/8	0/94	0/33

Adults were more likely than children with SLE to have antibodies to DNA and RNP ($p<.01$ and $p<.005$ respectively). Among relatives there was a higher incidence of ALA among the relatives of children that among relatives of adults ($p<.05$). This may result from a higher intensity of exposure of relatives to child probands. No antibodies to PM-1 or Scl-70 were found.

SERIAL DETERMINATIONS OF CIRCULATING IMMUNE COMPLEXES (CIC) IN THE SERA OF CHILDREN WITH RHEUMATIC DISEASE. Donald A.Person, Gregory J. Buffone, Carolyn M. Leatherwood, Earl J. Brewer, and Edward H. Giannini, Baylor College of Medicine. Houston, TX 77030, USA.

Using the liquid phase C1q Binding Assay (C1qBA) we have serially studied CIC in the sera of 37 children with various systemic rheumatic disease. All patients included had at least three separate C1qBA tests done sometime during the course of their disease (3 months to more than 2 years). The patients included: 17 with systemic lupus erythematosus (SLE); 16 with juvenile rheumatoid arthritis (JRA: 9 polyarticular, 2 pauciarticular, and 5 systemic course); and two each with systemic vasculitis (SV) and scleroderma (SD). A total of 232 tests in these patients resulted in 75 positive tests (C1qBA > 8.8%). Thirteen of seventeen patients with SLE had CIC at one time or another during the course of their disease. Of the four without CIC three are now in remission and off all medications. In the patients with JRA 6/16 had CIC during the course of their disease (4 polyarticular girls and 1 girl and 1 boy with systemic course disease). Both girls with SV and both boys with SD had CIC at some point during the course of their disease. No single other laboratory parameter was predictive of CIC. Clinically CIC appeared to be related to increased disease activity. The two SLE patients with the highest C1qBA have died from their disease. The one patient with rheumatoid factor positive, progressive, erosive, polyarticular JRA had persistently positive CIC. Likewise, the child with SV who had persistent evidence of CIC had lost all of her fingers and toes to gangrene secondary to her disease. These results suggest that elevated CIC are related to disease activity in children with systemic rheumatic disease.