Life-Size

N.A.M.E.
1255 S. Wabash Ave. 4th Fl.
Chicago, IL 60605
Tel. 312.554.0671
Tuesday by appointment
Wed./Fri./Sat. 12-6pm
Thurs. 12-8pm

Uli Aigner
Jane Benson
Paul Kass
Dominic Kline
Audrius Plioplys
Life-Size

October, 18, 1996 - November, 16, 1996

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organized by Michael Hall

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Introduction:

What is Life-Size? For the purposes of this exhibition its the ability to reference the body without actually representing it. This enables the artists to place the work in relation to the viewer, forcing the scale and placement of the objects to become the key components to understanding the works. This allows the viewer to become the sole figurative aspect, allowing for a more participatory/experiential reading of the work.

Today representation is no longer a portrayal of life, but a subjectively experienced reality. Objects (Art) are blurring into our daily lives, simultaneously becoming functional and irrational. The true and the false are continually collaged into our daily lives by a variety of new and evolving media, our abilities to focus on the normal everyday and find the uncanny within, has sharpened our perceptual faculties. For both artists and viewers, procedures and practices have moved into real-time, real space.

The destruction of the apparatus* is a constant and consuming theme in a vast majority of contemporary works, and its apparent in varying degrees in this exhibition. Trashing the apparatus underscores the gap between form and content...and that gap must be the fundamental content and also the form. These days allegory is endlessly replayed where structural failure is a new kind of success in its own right. This structural failure is endemic to understanding our contemporary condition.

Michael Hall

Audrius Plioplys is both an artist and a Doctor of Neurology at Mercy Hospital in Chicago. Plioplys' thinking has always fed-off of both disciplines, making art that concentrates on thinking and the relationship between conceptual thought and neurology. His art is professional and academic but not overtly theoretical (like art theory), more phenomenological (like the physical sciences). Good art inherently has an abstract language and contains its own logic. Therefore, it would not be unreasonable to assume that other professions do as well. Of course I've only been discussing this work as text, but it's a little bit more complicated than that... I'm not even sure if I'm supposed to be reading all these papers. Because this text (academic papers) has been objectified by Plioplys' re-authoring of the text, of which he originally authored (and/or co-authored) are now from my perspective sculpture. Folded and stapled these papers hang on the wall and even though their depth is shallow, it has depth and weight and it's still about the space of the page. The narrative running from the front-side of the page to the backside of which you generally can't read, so in a literal sense the text is unreadable/ illegible, only the object nature of the work can be read. At this point Plioplys' project successfully blurs disciplines to an even greater length than the doctor/artist relationship one naturally infers. The line between theory and practice has dissolved into his trademark thinking about thinking.

Immunoglobin Reactivity in Autism and Rett's Syndrome

Audrius V. Plioplys, Adonna Greaves, Kamyar Kazemi, Earl Silverman

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Key Words. Autism · Immunoglobulins · Neuroimmunology · Rett's syndrome

Abstract. Blood samples were obtained from 17 patients with autism (8 – 23 years of age; 16 males and 1 female). B cell numbers as measured by anti-B1 antibodies were normal. B cell function (proliferation and in vitro IgG and IgM synthesis in response to pokeweed mitogen) was normal. Quantitative serum immunoglobulins (IgG, IgA and IgM) were normal. When tested against Western blots prepared from normal, human cerebellar tissue, there was an increased incidence of IgG anti-210K neurofilament subunit reactivity (41 vs. 7% in 348 controls; p < 0.001). IgM anti-210K reactivity occurred in 53% of the patients (22% in 111 controls; p < 0.05) with an overall incidence of anticerebellar Western blot banding of 88% (23% in controls; p < 0.001). IgG or IgM reactivity against front cortex Western blots was not observed. Similar investigations performed on 8 girls with Rett's syndrome failed to reveal any abnormalities.

Introduction

Autism is a syndrome characterized by social and communicative deficits of early onset accompanied by abnormal behaviors. There are many biomedical causes underlying autistic symptomatology [1], but in the majority of cases no clear etiology is ascertained. Immune system abnormalities have been associated with autism. Lymphocyte abnormalities have included inhibition of macrophage migration in response to human myelin basic protein [2], reduced mitogen-induced lymphocyte blastogenesis [3 – 5], decreased numbers of T lymphocytes with altered ratios of helper to suppressor T cells [5], and decreased natural killer cell activity [6].

Abnormalities in the circulating immune system have also been described in autism. There have been reports of defective antibody response to rubella vaccine [7] and the presence of circulating antibodies to serotonin receptors [8] and to neurofilament axonal proteins [4].
This study was undertaken in an attempt to better define possible circulating antibody abnormalities in autism. We specifically investigated B cell function and searched for the presence of circulating anticentral nervous system (CNS) antibody reactivity. For comparison, girls with Rett's syndrome were also investigated.

Materials and Methods

Blood samples were obtained from a total of 17 patients with autism. There were 16 males and 1 female. The age range was from 8 to 23 years with a mean age of 17. The diagnosis of autism conformed to the DSM-III-R criteria for autism. There were no identified bioculausal cancers of autism in any of the studied population. Parental consent was obtained prior to participation. This consent was approved by hospital ethics review committees. Simultaneously drawn blood samples from healthy young adults were used for the lymphocyte stimulation studies.

Blood samples were obtained from 8 Rett's syndrome girls with an age range of 2-15 years and a mean age of 8. The heparinized blood samples were obtained at different geographic areas in the United States and Canada and courier delivered to the laboratory for analysis. In all cases, a blood sample from a healthy young adult accompanied the Rett's syndrome sample, in the same package, to control for handling and shipping differences. In all cases, blood samples were received and processed less than 24 h from the time when they were drawn. Peripheral blood lymphocytes (PBL) were separated on a Ficoll-Hypaque density gradient. After washing, 2000 μl of cells were plated at a concentration of 5 x 10⁶ cells/ml in 96-well microplates in RPMI-1640 media with 10% fetal calf serum and L-glutamine. Triplicate wells were cultured in the presence or absence of pokeweed mitogen in concentrations of 1, 10, and 100 μg/ml at 37 °C in 5% CO₂. After 7 and 14 days, the supernatants were collected and quantitative IgG and IgM determinations performed using a Pandex nephelometer.

PBL cell surface phenotype was determined by indirect immunofluorescence. Briefly, 10⁶ isolated PBL were incubated with saturating amounts of murine monoclonal antibody. After washing, cells were then incubated with FITC-conjugated goat anti-mouse, isotype-specific immunoglobulin. The percentage of fluorescent positive cells was determined from a 2-parameter analysis of at least 10⁴ lymphocytes on a gated lymphocyte population. The green fluorescence intensity was detected as 488 nm with a laser power of 500 mW using a Coulter Epics V flow cytometer (Coulter Electronics, Hialeah, FL, USA). Murine monoclonal antibody B1 (pan B cell marker) was obtained from Coulter Immunochemistry (Hialeah).

Quantitative serum immunoglobulins were performed in the biochemistry laboratories of The Hospital for Sick Children using a Beckman array nephelometer. Immunoglobulin concentration with normative data from the biochemistry laboratories was used for comparison.

Brain samples were obtained at the time of autopsy from neurologically normal young adults who had died from non-neurologic causes. They were kindly provided by Dr. J. Deck of the neuropathology service at the Toronto General Hospital. The time of autopsy was not later than 12 h after the time of death. Routine neuropathologic examination was normal.

Results

In all cases of autism and Rett’s syndrome, B cell numbers as measured by anti-B1 antibodies were normal. B cell function (proliferation and in vitro IgG and IgM synthesis in response to two different concentrations of pokeweed mitogen) was also normal. Quantitative determinations of serum IgG, IgM, and IgA concentrations were normal.

When tested against Western blots prepared from normal, human cerebellum, there was an increased incidence of IgG anti-210K neurofilament subunit reactivity in the autistic (41 vs 7% in 348 controls; p < 0.001). Representative Western blots are illustrated elsewhere [13, 14]. Immunoreactive IgG bands against other cerebellar molecular weight epitopes was not observed. Western blots prepared from frontal cortex revealed no IgG-reactive banding. In a previously reported investigation of anti-CNS antibody reactivity in adults using identical techniques, the incidence of IgG anti-210K cerebellar reactivity in 18 patients with cerebellar ataxia was 17% [14].

IgM anti-210K cerebellar reactivity occurred in 53% of the autistics and in only 22% of 111 controls (p < 0.05). IgM binding at other molecular weights was also observed, with an overall incidence of 88% (23% in controls; p < 0.001). IgM reactivity against frontal cortex Western blots was not observed. No IgM immunoreactivity was detected in the Rett’s syndrome patients.

Of the autistic patients 6 (35%) had both IgG and IgM anti-210K cerebellar reactivity. These were all males with ages of 8, 12, 17, 18, 20, and 22 years (mean age of 19 years) and only 1 was taking a medication (methylphenidate). One (6%) had only IgG anti-210K cerebellar reactivity. This was a 14-year-old male who was taking pimozide. Three (18%) had only IgM anti-210K cerebellar reactivity. These were all males of ages 10, 16 and 18 years (mean age of 16 years) and none were taking medications. Seven (41%) had neither IgG or IgM anti-210K cerebellar reactivity. These included one female of 19 years of age and the remainder were males of 9, 14, 16, 17, 22 and 23 years of age (mean age of 17 years). Of these 4 were taking medications (1 was taking haloperidol, 1 carbamazepine and 2 thioridazine).

There was no correlation between any of the positive results and medication intake or age distribution. There was no correlation between the positive results and the presence of epilepsy since only 1 of the 17 autistics was being treated for seizures. In no case was there any clinical indication of cerebellar dysfunction.
No IgG or IgM immunoreactivity was detected in the Rett's syndrome patients when the serum samples were screened against blots prepared either from frontal cortex or cerebellum.

Discussion

Although antibodies recognizing serotonin receptors have been reported in 1 autistic child [8], a screening investigation of 20 patients with autism did not detect anti-CNS antibody reactivity [15]. However, the techniques that were used in this study were substantially different from those that we used. A membrane preparation made from human frontal cortex was used, not whole CNS tissue homogenates. Immunoreactivity against many CNS antigenic determinants, including cytoskeletal components such as neurofilaments, was not screened for.

Our results indicate that there is a significantly increased incidence of circulating anti-210K neurofilament immunoreactivity in autism. This finding is not specific for autism since it has been described in Creutzfeldt-Jakob disease [16, 17], Kuru [16], Parkinson's disease [18], the opsonophilic-myoclonus syndrome of childhood [19], and in normals [13, 14]. Furthermore, claims have been made that anti-210K immunoreactivity for both IgG and IgM classes of antibodies can be detected in virtually all normal individuals [20]. However, in our hands consistently the incidence of IgG anti-210K reactivity is low. In a study of 257 adults the incidence was 6% [14]. In a subsequent investigation of 358 children, the incidence was 3% when screened against Western blots prepared from frontal cortex, and 7% when screened against blots prepared from cerebellum [13]. For IgM, in 111 normal adults, the incidence of anti-210K reactivity was 22%. Thus our results of anti-210K reactivity in autism are statistically highly significant, but not specific for autism. It should be noted that none of our autistic patients had any clinical evidence of cerebellar dysfunction.

We did not detect immunoreactivity against frontal cortex blots which are also rich in neurofilaments. It is possible that there may be another substance in cerebellar tissue co-migrating at the same molecular weight as the 210K neurofilament subunit. Alternatively, antigenic epitopes on the 210K neurofilament subunit may be revealed in processing cerebellar tissue, an event which may not take place in preparing frontal cortex immunoblots. Nevertheless, a consistent finding was the fact that there was a high incidence of anti-cerebellar immunoreactivity.

The cerebellar specificity of our findings is particularly intriguing in light of a report suggesting cerebellar abnormalities in autism using brain-imaging techniques [21, 22]. These results are controversial and have not been confirmed by other investigators [23–25]. None of our studied autistic patients had brain MRI scans performed. It is possible that subsets of autistics may have differing neuropathologic findings on imaging studies. Ongoing anticerebellar immunoreactivity may play a role in the genesis and/or modification of cerebellar circuitry in autism.

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References


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Uli Aigner*/Jane Benson/Paul Kass/Dominic Kline/Audrius Plioplys

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L-Carnitine as a treatment of lethargy in children with chronic neurologic handi...