Table 7
The results of a logistic regression analysis with backward selection.

	B	SE*	Wald (*df* = 1)	OR	95% CI
The age of initial HFASD diagnosis	0.18	0.05	13.34***	1.20*	(1.1–1.3)
Neglect	1.85	0.77	5.70**	6.34*	(1.4–28.8)
Physical abuse	1.32	0.57	5.30**	3.73*	(1.2–11.4)
Bullying	-0.84	0.49	2.96**	0.43	(0.2–1.1)
Parental divorce	1.01	0.59	2.96**	2.74	(0.9–8.6)
$\chi^2(5)$	49.5***				
AIC	138.4				
Correct classification (%)	85.7				

* Significant at the 0.05 level, two tailed.
** Significant at the 0.01 level, two tailed.
*** Significant at the 0.001 level, two tailed.

the age of initial HFASD diagnosis, neglect, physical abuse, bullying and parental divorce were significant. The model that included these five variables initially provided the best fit in this study ($\chi^2 = 49.5$, $df = 5$, $p < 0.001$, AIC = 138.4). Of these five variables, only the ORs for the age of initial HFASD diagnosis, neglect and physical abuse were significant. Individuals who experienced childhood neglect or physical abuse were most likely to have criminal behaviours later in life. Criminal behaviour was 6.3 times more likely to occur in those who experienced neglect and 3.1 more likely in those who experienced physical abuse compared with the control group. There was a 1.2-fold increase in criminal behaviours for each year that the psychiatric diagnosis was delayed.

4. Discussion

4.1. Characteristics of individuals with HFASD who exhibit criminal behaviours

In this study, the most common criminal behaviour was theft (55.6%), followed by sexual misconduct (25.0%), violence (25.0%), and running away (19.4%). Theft is also the most common criminal behaviour in the general Japanese population (Ministry of Justice, Japan, 2008) and in many European countries (e.g., Junger-Tas et al., 2010). The rates of sexual misconduct, violence, and running away in the general Japanese population are not high (Japanese Ministry of Justice, 2008); thus, the relatively high rate of sexual misconduct in this study may by a characteristic of individuals with HFASD. Hellemans, Colson, Verbraeken, Vermeiren, and Deboutte (2007) interviewed 24 adolescents and adults with HFASD regarding their sexuality and reported that approximately one-third required sexual development or behavioural interventions. Indeed, previous case studies have reported that individuals with HFASD and excessive sexual interest engaged in sexual misconduct (e.g., Kohn et al., 1998; Murrie et al., 2002). Accordingly, a feature of ASD was derived: Restricted and repetitive patterns of behaviour, interests, and activities may take on a sexual aspect, and their unique or intense sexual interests may lead to criminal behaviour (Murrie et al., 2002).

In the present study, 94.4% of participants in the criminal group exhibited "multiple recurrent incidents with a current episode of criminal behaviour" or "multiple recurrent incidents without a current episode;" 5.6% of participants reported only once and no recurrences illicit behaviours at the time of the assessment." This result corresponds with many previous case studies reporting that individuals with HFASD repeat criminal behaviours (e.g., Baron-Cohen, 1988; Chen et al., 2003; Mawson et al., 1985). As mentioned earlier, restricted and repetitive patterns of behaviour, interests, and activities might contribute to recurrent criminal behaviour. Moreover, a lack of empathy for others (Wing, 1981), which relates to severe and sustained impairments in social interactions, might also be related to recurrent criminal behaviour (Woodbury-Smith et al., 2005).

More importantly, these individuals often repeat criminal behaviours even though they were seeing child psychiatrists and receiving traditional interventions. These findings reflect the difficulty of intervening in cases of criminal behaviour. Preventive approaches should focus on ASD traits.

4.2. Criminal behaviour risk factors in individuals with HFASD

The results indicated that the age at which HFASD was first diagnosed, physical abuse and neglect significantly predicted criminal behaviour in individuals with HFASD. This finding corresponds with previous case reports suggesting that a delayed initial diagnosis and appropriate treatment lead to violent behaviours (Mukaddes & Topcu, 2006). Our findings demonstrating that neglect and physical abuse have significant effects are also in agreement with previous results from the general population. For example, childhood neglect and physical abuse significantly predict aggression that results in violent crime arrests in adulthood (Maxfield & Widom, 1996). Thus, neglect and physical abuse are significant risk factors of criminal behaviour in the HFASD population. Neglect and physical abuse exert a large influence on children's physical and psychological development. For instance, when children experience neglect or physical abuse, their physical growth is stunted, and their mental status is unstable; these children are more likely to have mental disorders such as depression or aggression towards others (Child Welfare Information Gateway, 2008). Such problems exert negative influences on their

emotional regulation, friendships and adjustment to school (Sroufe, Egeland, Carlson, & Collins, 2005), which may result in criminal behaviour.

This study is one of the few to assess the relationship between the age of initial HFASD diagnosis and the likelihood of criminal behaviour. The results show that a later diagnosis is correlated with an increased prevalence of criminal behaviour. In general, the presence of HFASD is easily overlooked in young children (De Giacomo & Fombonne, 1998) because speech delays, a common characteristic of ASD, are unlikely to be observed. Later diagnoses lead to a lack of early medical and educational interventions and perhaps the inability to acquire social skills and adapt to society (e.g., Lord, 1995). It is critical that children with ASD increase their repertoire of appropriate behaviours at an early age (Howlin, 1997; Richman, 2001); thus, late diagnoses might be a significant risk factor of social adaptation failures in individuals with ASD.

Parents of children with ASD typically report higher levels of parenting stress and affective symptoms compared with parents of normally developing children and those of children with other disabilities (e.g., Bristol & Schopler, 1984; Dumas, Wolf, Fisman, & Culligan, 1991). Moreover, Hastings and Johnson (2001) found that parental stress correlated with levels of autism symptoms. From these findings, one might associate a delayed ASD diagnosis with criminal behaviour. A delayed diagnosis leads to a poor prognosis (Lord, 1995) and elevates parental stress; the parents may also become depressed or apathetic, which leads to harsher disciplines that could develop into child abuse (Sullivan & Knutson (2000). Child abuse exacerbates the child's socio-emotional development, which might lead to criminal activity.

The additional CA categories, hyperactivity and being bullied, were not significantly correlated with criminal behaviour; however, researchers have observed that there are significant correlations among these variables in the general population. For example, children who lack control at age three exhibit aggression later in life (Caspi, Henry, McGee, Moffitt, & Silva, 1995). Moreover, alienation from friends was positively correlated with aggression (Schwartz, McFadyen-Ketchum, Dodge, Pettit, & Bates, 1998). One possible explanation for the present study's non-significant results is the high percentages of hyperactivity and bullying in both the criminal and control groups. The lack of a significant between-group difference with regard to hyperactivity and bullying may have masked the relationship between these factors and risk of criminal behaviours. Hyperactivity is often observed in children with ASD (e.g., Wing, 1996), and these children are often ridiculed and become targets of bullying because they fail to comprehend the intentions of others (Heinrichs, 2003; Yoshida & Uchiyama, 2004). Therefore, although hyperactivity and bullying were not significantly correlated with criminal behaviour in individuals with HFASD in the present study, practitioners must still consider these factors when working with this population.

4.3. Limitations

This study selected several CAs to predict criminal behaviour in individuals with HFPDD. However, many of these CAs are environmental factors; the only individual factors were physical illness and hyperactivity. As a result, other individual factors (e.g., hereditary) were not taken into account. In addition, the interaction between individual factors and environmental factors was not examined. Therefore, future studies are needed to examine the influence of biological and environmental factors on criminal behaviour in individuals with HFASD.

Except for the age of initial HFASD diagnoses, all the CAs were rated as either present or absent, even though the severity of these factors may differ among individuals. Sampson and Laub (1994) assessed quantitative variables similar to CAs and explained that family poverty interrupted informal social control processes in the family. The lack of informal social control in families increased the risk for delinquency. Thus, insufficient family functioning (e.g., economic adversity, parental criminality and family violence) might postpone the timing and age at which HFASD is first diagnosed. Therefore, studies that rate the presence and level of child abuse or neglect using multilevel rather than dichotomous scales may reveal additional details regarding the relationships among criminal behaviours and these variables. Moreover, prospective studies might provide additional information on this topic.

Acknowledgements

This study was supported by the Research Institute of Science and Technology for Society in the Japan Science and Technology Agency (JST)'s grant FY2007-2012, "Prevention of Victimisation and Criminal Offences" (principal investigator, Masatsugu Tsujii) from the Research and Development Program of the JST, "Protecting Children from Crime".

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ORIGINAL ARTICLE

Microglial Activation in Young Adults With Autism Spectrum Disorder

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Context: A growing body of evidence suggests that aberrant immunologic systems underlie the pathophysiologic characteristics of autism spectrum disorder (ASD). However, to our knowledge, no information is available on the patterns of distribution of microglial activation in the brain in ASD.

Objectives: To identify brain regions associated with excessively activated microglia in the whole brain, and to examine similarities in the pattern of distribution of activated microglia in subjects with ASD and control subjects.

Design: Case-control study using positron emission tomography and a radiotracer for microglia—[^{11}C](*R*)-(1-[2-chrorophynyl]-*N*-methyl-*N*-[1-methylpropyl]-3 isoquinoline carboxamide) ([^{11}C](*R*)-PK11195).

Setting: Subjects recruited from the community.

Participants: Twenty men with ASD (age range, 18-31 years; mean [SD] IQ, 95.9 [16.7]) and 20 age- and IQ-matched healthy men as controls. Diagnosis of ASD was made in accordance with the Autism Diagnostic Observation Schedule and the Autism Diagnostic Interview–Revised.

Main Outcome Measures: Regional brain [^{11}C](*R*)-PK11195 binding potential as a representative measure of microglial activation.

Results: The [^{11}C](*R*)-PK11195 binding potential values were significantly higher in multiple brain regions in young adults with ASD compared with those of controls ($P < .05$, corrected). Brain regions with increased binding potentials included the cerebellum, midbrain, pons, fusiform gyri, and the anterior cingulate and orbitofrontal cortices. The most prominent increase was observed in the cerebellum. The pattern of distribution of [^{11}C](*R*)-PK11195 binding potential values in these brain regions of ASD and control subjects was similar, whereas the magnitude of the [^{11}C](*R*)-PK11195 binding potential in the ASD group was greater than that of controls in all regions.

Conclusions: Our results indicate excessive microglial activation in multiple brain regions in young adult subjects with ASD. The similar distribution pattern of regional microglial activity in the ASD and control groups may indicate augmented but not altered microglial activation in the brain in the subjects with ASD.

JAMA Psychiatry. 2013;70(1):49-58.
Published online November 26, 2012.
doi:10.1001/jamapsychiatry.2013.272

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AUTISM SPECTRUM DISORDER (ASD) is a group of neurodevelopmental disorders characterized by pervasive abnormalities in social interaction and communication and by repetitive and restricted behavioral patterns and interests. Autism spectrum disorders include autistic disorder, Asperger disorder, and pervasive developmental disorder not otherwise specified.[1] Recent population-based surveys[2,3] showing that ASD is more common than previously believed have aroused serious public concern worldwide. Although the neurobiologic basis for ASD remains poorly understood, a growing body of research[4,5] suggests that immune abnormalities are a contributing factor to the development of ASD. Several genetic studies link ASD with genes that are associated with various immune functions,

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including the HLA antigen[6] and the major histocompatibility complex class III molecule, such as complement C4B.[7,8] Systemic abnormalities of the immune system have been one of the most common and long-standing reported findings in subjects with ASD.[9,10] Notably, increased production of cytokines (eg,

Table 1. Demographic Characteristics of the Subjects[a]

Variable	Mean (SD) [Range] Control (n = 20)	ASD (n = 20)
Age, y[b]	22.6 (5.3) [17.8-35.5]	23.3 (4.0) [18.6-31.9]
WAIS-III full IQ[c]	102.8 (12.5) [81.0-131.0]	95.9 (16.7) [81.0-140.0]
ADI-R		
Social	NA	20.6 (5.1) [10.0-29.0]
Communication	NA	15.2 (4.4) [8.0-24.0]
Stereotype	NA	4.3 (2.2) [3.0-10.0]
ADOS		
Social	NA	6.4 (3.0) [4.0-11.0]
Communication	NA	6.2 (2.7) [2.0-13.0]
Stereotype	NA	1.0 (0.9) [0-3.0]
Faux Pas Test	NA	21.2 (8.6) [3.0-34.0]
Y-BOCS	NA	11.0 (6.4) [0-28.0]
DCDQ-J total	NA	60.4 (12.0) [42.0-73.0]

Abbreviations: ADI-R, Autism Diagnostic Interview–Revised; ADOS, Autism Diagnostic Observation Schedule; ASD, autism spectrum disorder; DCDQ-J, Japanese version of the Developmental Coordination Disorder Questionnaire; NA, not applicable; WAIS-III, Wechsler Adult Intelligence Scale, third edition; Y-BOCS, Yale-Brown Obsessive Compulsive Scale.

[a]All subjects were men.

[b]$P = .63$.

[c]$P = .15$.

interleukin 6 [IL-6], tumor necrosis factor, and macrophage chemoattractant protein-1) has been observed in peripheral samples and the brains of ASD subjects.[11-16] In general, plasma cytokine levels in ASD subjects are widely distributed and show substantial overlap with control subjects, implying that there is a subset of ASD subjects with high levels of such cytokines. In addition, several studies[17-20] have identified specific antibodies against human brain epitopes in the serum of mothers of children with ASD, as well as in children with ASD, although autoantibodies are found in only 10% to 15% of the children with ASD. These findings argue in favor of the participation of the immune system in the pathogenesis of a subset of ASD subjects.

Microglia are resident brain cells that sense pathologic tissue alterations.[21,22] The first microglial precursors colonize the brain during the embryonic and fetal phases of development.[23,24] They develop into brain macrophages and perform immune functions. Upon exposure of the brain to any form of insult, such as infection, trauma, or ischemia, the microglia are rapidly activated. When activated, microglia produce neurotoxic substances, including proinflammatory cytokines (ie, tumor necrosis factor and IL-1β) and oxygen species (ie, hydrogen peroxide and superoxide). However, under certain conditions, activated microglia can produce anti-inflammatory cytokines such as IL-10 and transforming growth factor-β, which have neuroprotective effects in experimental animal models of traumatic injury and stroke.[25,26] Furthermore, experimental studies[27,28] have demonstrated that microglia play a role in the maintenance of synaptic integrity in the uninjured brain.

Recently, Vargas and colleagues[16] determined the magnitude of neuroglial and inflammatory reactions and their cytokine expression profiles in brain tissues from the cerebellum, midfrontal, and cingulate gyrus obtained at autopsy from children and adults with ASD. Immunocytochemical examination revealed marked activation of microglia and astroglia. Microglial responses were diffusely distributed in the cortex and subcortical areas, as well as the cerebellum, and were present as microglial nodules or as part of a prominent accumulation of perivascular macrophages. More recently, Morgan and colleagues[29] quantitatively assessed activated microglia in the dorsolateral prefrontal cortex of postmortem brains from children and adults with ASD. They found that the microglia were markedly or marginally activated in most cases examined. Transcriptomic analysis of the autistic brain by Voineagu and colleagues[30] has shown the presence of 2 modules in the ASD brain: a neuronal module enriched for known autism susceptibility genes, including neuronal-specific factors, such as ataxin 2-binding protein 1, and a module enriched for immune genes and glial markers. The latter immune-glial module has a less pronounced genetic component and thus is most likely either a secondary phenomenon or the result of environmental factors. Despite the striking features of microglial activation in the pathogenesis of ASD, to our knowledge, there is no information on the patterns and characteristics of the distribution of microglial activation in the whole brain in ASD subjects.

To address this issue, we conducted a positron emission tomography (PET) analysis using the radiocarbon ([^{11}C])-labeled (*R*)-(1-[2-chrorophynyl]-*N*-methyl-*N*-[1-methylpropyl]-3 isoquinoline carboxamide) ([^{11}C](*R*)-PK11195), a radiotracer that specifically binds to activated microglia.[31-33] This procedure permitted us to visualize the activated microglia in vivo in the whole brain. In this study, we initially determined the distribution of [^{11}C](*R*)-PK11195 binding potential (BP) in the whole brain of young adults with ASD and then identified several brain regions associated with the activation of microglia. Subsequently, we compared the levels of [^{11}C](*R*)-PK11195 BP in the identified brain regions. Because microglia may be prenatal in origin,[23,24] and because ASD is typically diagnosed by 3 years of age, we hypothesized that the regional variability of the [^{11}C](*R*)-PK11195 BP in the identified brain regions is similar between ASD and control subjects, whereas the magnitude of [^{11}C](*R*)-PK11195BP in ASD subjects is greater than that of controls in all the regions. To test this hypothesis, we carefully recruited subjects with no history of epilepsy or medication because epileptic seizures and psychotropic drugs can influence the condition of microglial activation.[34-37]

METHODS

SUBJECTS

The ethics committees of the Hamamatsu University School of Medicine approved this study. Written informed consent was obtained from all subjects and their guardians after they had been provided a detailed explanation of the study procedures. Twenty men with ASD and 20 age- and IQ-matched typically developing male subjects participated in this study (**Table 1**). All subjects were right-handed and had an IQ of greater than 80. None of the subjects were tobacco smokers, and none were

taking any medication, including psychotropic drugs. All of them were physically healthy. At the time of scanning, all the subjects had no symptoms of inflammation and were not under stressful conditions. All the subjects with ASD were diagnosed by 2 trained child psychiatrists (K.N. and T.S.) according to the *DSM-IV-TR*.[1] The ASD diagnosis was confirmed for all cases using the Autism Diagnostic Interview–Revised (ADI-R)[38] and the Autism Diagnostic Observation Schedule (ADOS)[39] module-4 by trained clinicians (K.J.T. and K.M., respectively). As a result, 15 of 20 ASD subjects were diagnosed as having autistic disorder and the remaining 5 were considered to have pervasive developmental disorder not otherwise specified on the basis of the ADOS scores, although all 20 subjects met the ADI-R criteria for autistic disorder. None of the ASD subjects was classified as having regressive autism, the classification of which was based on clinical characteristics using both parental reporting and answers to questions on the ADI-R regarding language loss (question 11) and social skills (question 25). The ASD subjects did not have any other psychiatric comorbidity disorders, as confirmed by the Structured Clinical Interview for *DSM-IV* Axis I disorders.[40] In addition, they had no notable dysmorphism, neurocutaneous abnormalities, significant neurologic deficits, history of epileptic seizures, or disorders known to be associated with autism, such as fragile X syndrome, neurofibromatosis, or tuberous sclerosis. Fragile X syndrome was excluded by determining the CGG repeat number in the *FMR1* gene. We measured the markers of inflammation in the blood in the ASD subjects, including the serum C-reactive protein and white blood cell count. Both levels in all the ASD subjects were within normal range. None of the ASD subjects had any history of inflammatory or allergic diseases, except 2 subjects who had had atopic dermatitis in their childhood. One of the ASD subjects had a family history of major depression (his mother). In the remaining 19 subjects, there was no family history of any chronic inflammatory diseases or neuropsychiatric conditions. In the ASD subjects, the social cognitive disability and the degree of repetitive and/or obsessive behavior and interests were evaluated by the Faux Pas Test[41] and Yale-Brown Obsessive Compulsive Scale,[42,43] respectively. Current motor coordination problems were assessed by the Japanese version of the Developmental Coordination Disorder Questionnaire.[44] All control subjects were found to be mentally and physically healthy on the basis of comprehensive assessments of their medical histories and neuropsychiatric examinations.

MAGNETIC RESONANCE IMAGING AND PET PROCEDURES

As described previously,[33,45] we obtained 3-dimensional magnetic resonance images (MRIs) just before PET measurements using a 0.3-T MRI unit (MRP7000AD; Hitachi Medical) and a high-resolution brain PET scanner having an intrinsic resolution of 2.9 × 2.9 × 3.4 mm at full-width at half maximum and a 163-mm axial field of view, and yielding 47 PET images simultaneously (SHR 12000; Hamamatsu Photonics), respectively. All MRI and PET scans were set parallel to the anterior-posterior intercommissural line.[45] Before dynamic PET scanning, a 20-minute transmission scan was performed for attenuation correction using a $^{68}Ge/^{68}Ga$ source. Then, after a bolus intravenous injection of a 350-MBq dose of [^{11}C](*R*)-PK11195, we performed 32 serial PET scans (time frames: 4 × 30 second, 20 × 60 second, and 8 × 300 second) for 62 minutes. In quantitative PET brain imaging, the motion artifact is the important degrading factor. Therefore, we fixed the head of each subject by using a thermoplastic face mask, observed subjects carefully during each scan, and confirmed that all the subjects had remained immobilized.

IMAGE ANALYSIS AND KINETIC MODELING

The brain, particularly in cortical subregions, is known to be sensitive to a partial volume effect that sometimes occurs during the measurement of small brain structures and that leads to an underestimation of tracer activity. In this study, we used the following previously described procedure to minimize the contribution of the partial volume effect.[33,45] First, we adjusted the MRI voxel size to the PET voxel size 3-dimensionally using image-processing software (DrView; Asahi Kasei) on a Sun workstation (HyperSPARC ss-20; Sun Microsystems). Then, these reformatted MRIs with 3-dimensional scales and coordinates identical to those of the PET images were used as anatomic landmarks for the regions of interest (ROIs) setting. Subsequently, by referring to areas on the MRIs as anatomical landmarks, the ROIs were carefully drawn to avoid the involvement of either the sulci or ventricles. An investigator masked to the subject's condition placed 3 ROIs over the bilateral cerebellar cortices, midbrain, and bilateral thalami on the MRIs. These ROIs were then transferred onto the corresponding dynamic [^{11}C](*R*)-PK11195 images.

To assess activated microglial density in the brain, we analyzed the [^{11}C](*R*)-PK11195 time-activity curves (TACs) on the basis of a simplified reference tissue model[46,47] because the regional brain [^{11}C](*R*)-PK11195 BP (a ratio of binding and dissociation rate constants, k_3/k_4) estimated by the simplified reference tissue model is reported to correlate with the magnitude of microglial activity.[33,48] Because the decrease of TACs was sharpest in the cerebellar ROI among the 3 ROIs examined in the control group, we assumed that the specific binding would be the least in this region. A normalized input curve was first created by averaging the TACs from the ROIs placed over the bilateral cerebellar cortices in the control group. Then, the normalized mean input curve was used as the reference input function of the simplified reference tissue model in the ASD and control subjects because a desirable reference region free from specific binding was not evident in the ASD subjects.

Using biomedical imaging software (PMOD, version 3.0; PMOD Technologies), we constructed whole-brain parametric maps of the [^{11}C](*R*)-PK11195 BP for the subsequent voxel-based analysis using Statistical Parametric Mapping software (SPM5; http://www.fil.ion.ucl.ac.uk/spm). The [^{11}C](*R*)-PK11195 BP maps were normalized to the Montreal Neurological Institute space, as defined by the MRI T1 template implemented in SPM5. The extracerebral structures were then masked by demarcating cerebral regions on spatially normalized MRIs. Finally, the normalized and masked BP maps were smoothed with an 8-mm full-width at half maximum gaussian filter.

In addition to the voxel-based analysis, which is suitable for an exploratory examination of altered tracer distribution in the brain, we performed a volume of interest (VOI)–based analysis because it enabled us to generate quantitative differences in [^{11}C](*R*)-PK11195 BP in specific regions. For this purpose, we placed additional spherical VOIs of 5-mm radius, which centered on the peak voxel derived from the results of the voxel-based analysis, on [^{11}C](*R*)-PK11195 BP maps for each of the subjects. The VOIs selected were the bilateral cerebellum, brainstem, splenium of the corpus callosum, bilateral fusiform gyri, bilateral superior temporal gyri, and the bilateral anterior cingulate, bilateral orbitofrontal, left midfrontal, and right parietal cortices. Averaged [^{11}C](*R*)-PK11195 BP values for each VOI were obtained in the ASD and control groups.

VOXEL-BASED MORPHOMETRY

To investigate possible differences in brain structure between the ASD and control groups, we conducted voxel-based morphometry. For this purpose, we used a 3-T MRI scanner (Signa Excite;

General Electric Medical Systems) to obtain T1-weighted volumetric images scanned by the inversion recovery-prepared fast spoiled gradient recalled acquisition protocol as follows: repetition time = 11.0 milliseconds, echo time = 5.0 milliseconds, preparation time = 450 milliseconds, flip angle 20°, number of excitations = 1, field of view = 24.0 cm, matrix = 256 × 256, auto-zero-fill interpolation = 512, location per slab = 160, slice thickness = 1.2 mm, and voxel size = 0.94 × 0.94 × 1.2 mm. The T1-weighted volumetric images were analyzed using the VBM5.1 toolbox (http://www.fil.ion.ucl.ac.uk/spm/ext/) implemented in SPM5 with the default parameters. Estimates of the absolute gray matter (GM), white matter (WM), and cerebrospinal fluid (CSF) volumes were obtained after the automatic brain segmentation procedure had been carried out by VBM5.1. The total intracranial volume was calculated as the sum of the volumes of the GM, WM, and CSF.

STATISTICAL ANALYSIS

The demographic and clinical variables of the ASD and control groups were compared by the unpaired *t* test using statistical software (PASW Statistics version 18; SPSS Japan Inc). The level of statistical significance was set at $P < .05$.

The voxel-based analyses of the [^{11}C](*R*)-PK11195 BP maps were conducted using SPM5. For the SPM5 analysis of the [^{11}C](*R*)-PK11195 BP maps, between-group comparisons were performed to explore regional differences in the [^{11}C](*R*)-PK11195 BP using the *t* test for each voxel without a proportional scaling of the [^{11}C](*R*)-PK11195 BP maps. We also performed exploratory correlation analyses between the regional changes in [^{11}C](*R*)-PK11195 BP values and the severity of clinical features in ASD subjects using SPM5. The scores on the ADOS, ADI-R, Faux Pas Test, Yale-Brown Obsessive Compulsive Scale, and the Japanese version of the Developmental Coordination Disorder Questionnaire were variables of interest. To test hypotheses about the region-specific effects of these variables, the estimates were compared using 2 linear contrasts (positive or negative correlation). In the SPM5 analyses, values of $P < .05$ were statistically significant after adjustment for the false discovery rate in the whole-brain multiple comparisons.

In the VOI-based analyses, we tested the main effect of the diagnosis of ASD on [^{11}C](*R*)-PK11195 BP values derived from 13 brain regions using 2-way analysis of variance, in which statistical significance was set at $P < .05$. For comparisons of clinical variables between subgroups of ASD subjects, a Mann-Whitney test was performed.

To assess the differences in segmented brain volumes between groups in the voxel-based morphometry analysis, we conducted a multivariate analysis of covariance using PASW software with group (ASD and control) as a between-subject factor, segmented brain regional absolute volume (GM, WM, and CSF) as a within-subject factor, and intracranial volume as a covariate. The statistical significance level was set at $P < .05$. Second, for the GM analysis, the normalized, modulated, and smoothed GM image segments in each group were entered into a voxel-wise 2-sample *t* test analysis in SPM5. An absolute threshold mask of 0.30 was used to avoid possible edge effects around the border between GM and WM. The statistical threshold was set at $P < .05$ after the false discovery rate correction. Data were presented as mean (SD).

RESULTS

Characteristics of all the subjects are summarized in Table 1. There was no significant difference in age or IQ between the 2 groups.

COMPARISON OF [^{11}C](R)-PK11195 BP BETWEEN ASD SUBJECTS AND CONTROLS

The tissue TACs of [^{11}C](*R*)-PK11195 are shown in **Figure 1**A. After the administration of [^{11}C](*R*)-PK11195, the radioactivity in 3 ROIs over the cerebellum, midbrain, and thalamus of a representative control subject decreased with time. The TACs in an ASD subject decreased less sharply than those in the control subject, indicating a time-course accumulation of [^{11}C](*R*)-PK11195 in the respective brain structures. Figure 1B shows MRI-PET fusion parametric images of [^{11}C](*R*)-PK11195 BP in the representative control and ASD subjects. A marked increase in [^{11}C](*R*)-PK11195 binding was observed across widespread areas of the brain of the representative ASD subject.

In the voxel-based analysis, we found greater [^{11}C](*R*)-PK11195 BP in multiple brain regions in the ASD group than in the control group; the brain regions with increased [^{11}C](*R*)-PK11195 BP included the cerebellum, brainstem (midbrain and pons), subcortical region (corpus callosum), limbic region (anterior cingulate cortex), and the frontal, temporal, and parietal regions (**Table 2** and **Figure 2**). Among the brain regions, the left cerebellum showed the most prominent *z* score. There were no voxels in which controls had a significantly higher [^{11}C](*R*)-PK11195 BP compared with that of the ASD group. In the ASD group, there was no significant difference in [^{11}C](*R*)-PK11195 BP between the 2 diagnoses—that is, autistic disorder (n = 15) or pervasive developmental disorder not otherwise specified (n = 5).

On the basis of the results of the voxel-based analysis, we then conducted VOI-based analysis. We placed 14 spherical VOIs of 5-mm radius, which centered on the peak voxels listed in Table 2. In accordance with the findings derived from the voxel-based analysis, the [^{11}C](*R*)-PK11195 BP was significantly higher in ASD subjects than in control subjects throughout all VOIs (**Figure 3**; $F_{13,532} = 17.62, P < .001$). As shown in Figure 3, the mean [^{11}C](*R*)-PK11195 BP was highest in the brainstem, followed by the left cerebellum, right orbitofrontal cortex, right anterior cingulate cortex, and other regions in the control group. The corresponding rank order was essentially the same in the ASD group. Thus, the pattern of distribution of [^{11}C](*R*)-PK11195 BP values throughout the VOI was quite similar between the 2 groups. **Figure 4** shows a scatterplot of [^{11}C](*R*)-PK11195 BP from the 4 VOIs (the left cerebellum, midbrain, right orbitofrontal cortex, and right anterior cingulate cortex) in the ASD and control groups. Although the overall average level of [^{11}C](*R*)-PK11195 BP was higher in the ASD group than in the control group, the BPs of some ASD subjects overlapped those of the controls in the 4 VOIs.

CORRELATION BETWEEN [^{11}C](R)-PK11195 BP AND SYMPTOMS IN ASD

Relationships between the regional changes in [^{11}C](*R*)-PK11195 BP values and the clinical features of ASD subjects were evaluated by voxel-based exploratory correlation analyses using SPM5. There was no voxel for which significant correlations were observed between [^{11}C](*R*)-

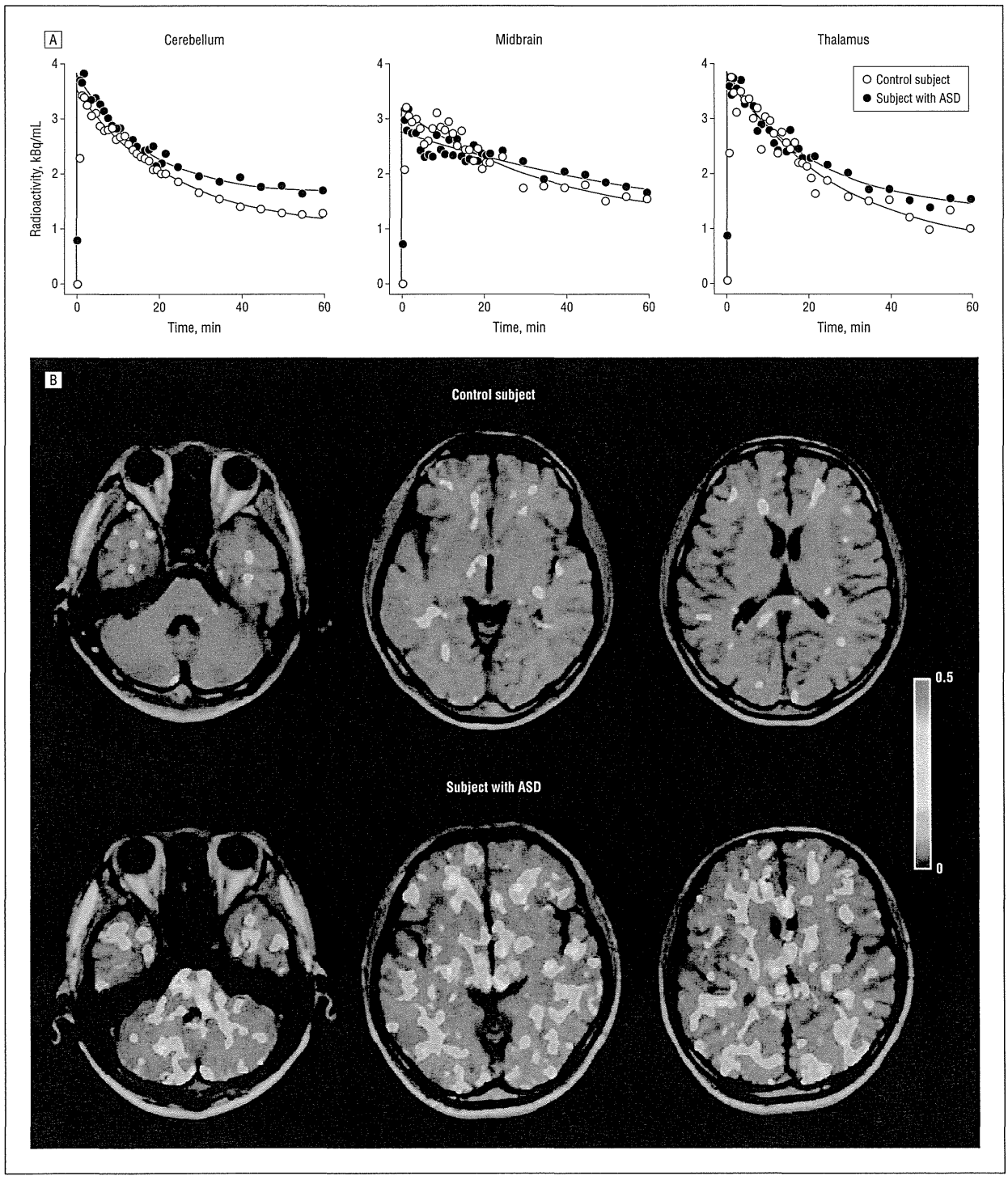

Figure 1. Results of positron emission tomography image analyses in a healthy control subject and a subject with autism. A, Scattergrams showing the time-activity curves of radiocarbon (^{11}C)-labeled (*R*)-(1-[2-chrorophynyl]-*N*-methyl-*N*-[1-methylpropyl]-3 isoquinoline carboxamide) ([^{11}C](*R*)-PK11195) for regions of interest in the cerebellum, midbrain, and thalamus in a subject with autism spectrum disorder (ASD) and a control subject. B, Magnetic resonance imaging–positron emission tomography fusion parametric images of [^{11}C](*R*)-PK11195 binding potential in a subject with ASD and a control subject. The left brain is shown on the right. The color bar indicates a level of binding potential.

PK11195 BP and the scores on the Faux Pas Test, Yale-Brown Obsessive Compulsive Scale, ADI-R, ADOS, or the Japanese version of the Developmental Coordination Disorder Questionnaire after the correction of whole-brain multiple comparisons (data not shown).

In the VOI-based analysis, we also conducted correlation analyses between [^{11}C](*R*)-PK11195 BP in each VOI and clinical valuables, and we found no significant correlations. We divided the ASD group into 2 subgroups, a High-BP and Not-High-BP group, on the basis of the

Table 2. Results of the Whole-Brain Voxel-Based Statistical Parametric Mapping Analyses of [^{11}C](*R*)-PK11195 Binding Potential: Increase in Binding in the Subjects With ASD[a]

Brain Regions	Coordinates			Voxel Level	
	x	y	z	Corrected *P* Value	*z* Score
Cerebellum					
Left lobuli 7, 8, and 9–	10–	58–	38	.03	4.82
Right lobuli 7 and 8	32–	76–	48	.04	3.77
Brainstem (midbrain and pons)	10–	38–	42	.03	4.56
Frontal region					
Left middle frontal gyrus, BA10, BA46–	44	50	12	.03	3.89
Left orbitofrontal cortex, BA11–	8	48–	4	.03	3.93
Right orbitofrontal cortex, BA47	14	30–	16	.03	4.32
Temporal region					
Left superior temporal gyrus, BA22–	52–	28	4	.03	3.67
Right superior temporal gyrus, BA22	50–	20–	6	.03	4.22
Left fusiform gyrus, BA37–	48–	60–	14	.03	4.16
Right fusiform gyrus, BA37	38–	58–	16	.03	4.30
Parietal region					
Right parietal cortex, BA40	28–	48	54	.03	3.70
Limbic region					
Left anterior cingulate cortex, BA32–	6	38	18	.03	4.12
Right anterior cingulate cortex, BA32	18	10	46	.04	3.47
Subcortical region					
Corpus callosum–	2–	26	16	.03	4.11

Abbreviations: ASD, autism spectrum disorder; BA, Brodmann area; [^{11}C](*R*)-PK11195, radioactive carbon-labeled (*R*)-(1-[2-chrorophynyl]-*N*-methyl-*N*-[1-methylpropyl]-3 isoquinoline carboxamide).

[a]The significance thresholds at the voxel cluster levels were $P < .05$ after false discovery rate correction for multiple comparisons across the whole brain. Coordinates are given in millimeters based on the Montreal Neurological Institute brain template. Each location is a peak within a cluster (defined as the voxel with highest *z* score).

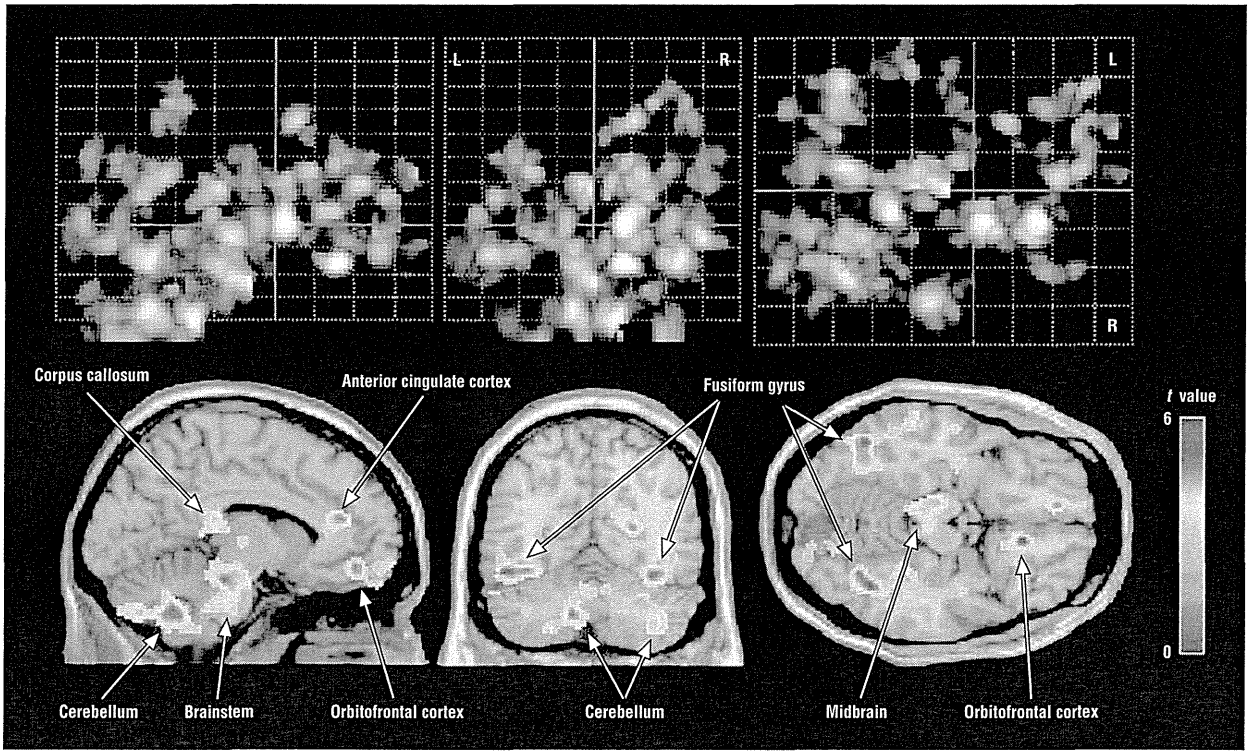

Figure 2. Results of the whole-brain voxel-based statistical parametric mapping analysis of the [^{11}C](*R*)-PK11195 binding potentials. Locations of clusters with significant increases in the group with autism spectrum disorder compared with the control group ($P < .05$, false discovery rate corrected) are shown on glass brain images and superimposed onto normal-template magnetic resonance images. L indicates left; and R, right.

[^{11}C](*R*)-PK11195 BPs in 4 VOIs respectively located in the left cerebellum, midbrain, right orbitofrontal cortex, and right anterior cingulate cortex. In the VOI at the left cerebellum, 12 ASD subjects had BPs that were more

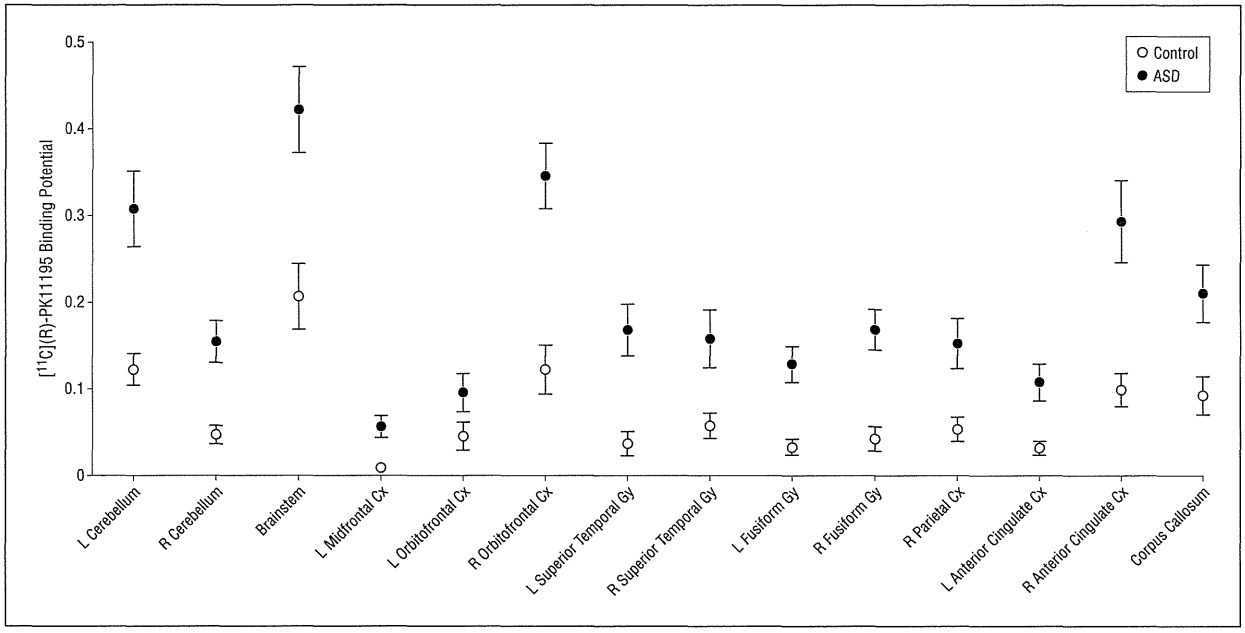

Figure 3. Regional brain [^{11}C](*R*)-PK11195 binding potential in the autism spectrum disorder (ASD) and control group. Subjects with ASD had significantly higher [^{11}C](*R*)-PK11195 binding potentials than those of controls ($F_{12,456}$ = 24.59, $P < .001$). Error bars represent the SEM. Cx indicates cortex; Gy, gyrus; L, left; and R, right.

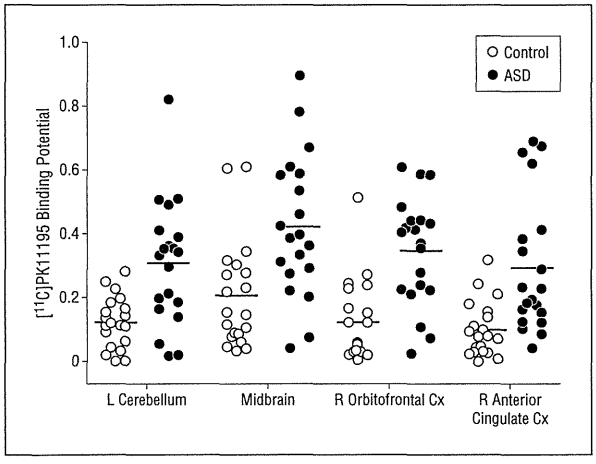

Figure 4. Scatterplot of regional [^{11}C](*R*)-PK11195 binding potential in the autism spectrum disorder (ASD) and control groups in 4 spherical volumes of interest placed over the left cerebellum, midbrain, right orbitofrontal cortex, and right anterior cingulate cortex.

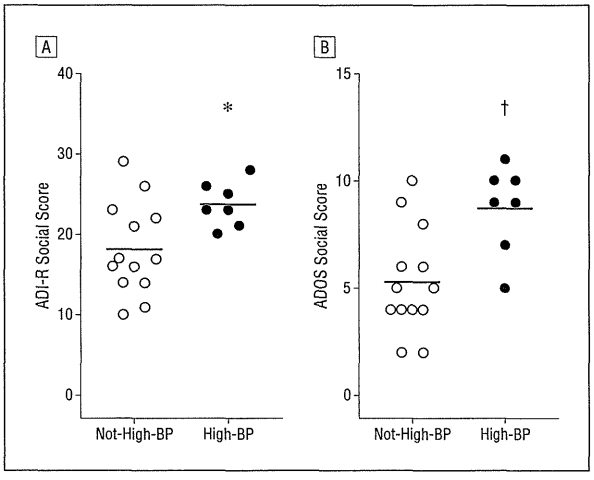

Figure 5. Comparison of social domain scores from Autism Diagnostic Interview–Revised (ADI-R) (A) and Autism Diagnostic Observation Schedule (ADOS) (B) between the High-Binding Potential (BP) and Not-High-BP subgroups in subjects with autism spectrum disorder. *P = .03 and †P = .006.

than 2 SDs higher than the mean BP of controls. The number of ASD subjects who had BPs that were more than 2 SDs higher than the mean value of the controls was 6 for the VOI in the midbrain, 10 for the VOI in the right orbitofrontal cortex, and 8 for the VOI in the right anterior cingulate cortex. Subjects with ASD who exhibited high BPs in at least 3 of the 4 VOIs were classified into a High-BP group (n = 7), and the remaining subjects were classified into a Not-High-BP group (n = 13). When clinical variables were compared between the High-BP and Not-High-BP groups, statistically significant differences were observed for the social scores of the ADI-R (U = 19.0, P = .04) and the ADOS (U = 13.0, P = .01) (**Figure 5**), suggesting that social disabilities might be more severe in the High-BP group.

COMPARISON OF REGIONAL VOLUME BETWEEN ASD SUBJECTS AND CONTROLS

The absolute volumes of the segmented brain regions were estimated in the control and ASD groups (GM: 676.3 [50.3] vs 705.8 [78.2] [control vs ASD]; WM: 421.7 [42.3] vs 439.7 [48.4]; CSF: 405.1 [47.1] vs 426.0 [50.2]; and intracranial volume: 1503.1 [123.7] vs 1571.5 [161.7]). The multivariate analysis of covariance revealed no significant differences in volume between the 2 groups (GM: $F_{1,37}$ = 0.006, P = .94; WM: $F_{1,37}$ = 0.209, P = .65; CSF: $F_{1,37}$ = 0.036, P = .85). A voxelwise 2-sample t test analysis of normalized and smoothed

GM images revealed no significant differences in GM volume between the 2 groups (data not shown).

COMMENT

Our PET measurements revealed that young adults with ASD had significantly increased [^{11}C](*R*)-PK11195 BP, a representative measure of the activation of microglia, in a wide range of brain areas, including the cerebellum, brainstem, anterior cingulate cortex, frontal cortex (orbitofrontal and midfrontal), temporal cortex (superior temporal and fusiform), parietal cortex, and corpus callosum. The microglial activation was greater in the ASD group than in the control group across all regions tested, although the most prominent increase was evident in the cerebellum. To our knowledge, this is the first in vivo evidence of the presence of excessive microglial activation in ASD subjects, and these findings support the contention that microglial activation may play a role in the pathogenesis of ASD.[16,29]

When we performed a VOI-based analysis on the [^{11}C](*R*)-PK11195 BPs for different brain regions associated with microglial activation, the pattern of distribution of [^{11}C](*R*)-PK11195 BP values throughout the VOIs was quite similar between the ASD and control subjects. The similar distribution of regionally activated microglia in the ASD and control groups may indicate the augmented but not altered microglial activation in the brain in the ASD subjects. Resident microglia, which are embryonic and fetal in origin, can be replenished intrinsically and do not require significant turnover from circulating blood progenitors (monocytes)[49] (see also the review by Chan et al[50]). Under pathologic conditions, however, microglia in neonates and adults are considered to derive from circulating blood monocytes originating primarily within the bone marrow.[50] In brain tissues from children and adults with ASD, macrophage chemoattractant protein-1, which can facilitate the infiltration and accumulation of blood monocytes in the brain,[51,52] is greatly increased.[16] It is also possible that microglia might respond to prolonged aberrant neuronal functioning in the ASD adults, providing trophic support to damaged cells or engaging in synaptic stripping to protect against excitotoxicity.[25-28] Taken together, the excessive activation of microglia in ASD subjects could begin in the prenatal period and last until adulthood. However, we propose that the critical period for the occurrence of excessive activation of microglia as a possible pathogenic factor for ASD may be during prenatal and early postnatal development of the brain because symptoms of ASD are manifested very early in life, typically by 3 years of age. To better understand the detailed mechanism underlying the long-running microglial activation, further studies, including experiments in animal models, may be helpful.

In the present PET assessment, young adults with ASD showed a prominent activation of microglia in the cerebellum. The cerebellum has been one of the foci of postmortem studies of autistic children and adults. Of the 30 postmortem cases of autism in which the cerebellum has been studied, 22 (73%) showed a reduced number of Purkinje cells, particularly in the hemispheres.[53-56] Pathologic abnormalities have been observed in both childhood and adult cases, with and without a history of seizures or medication usage. It is not known whether cerebellar lesions might have been present in the high-functioning young adults with ASD recruited for this study. Nonetheless, cerebellar activation of the microglia may reflect an association with cerebellar pathologic abnormalities, because when *N*-acetylaspartate, a putative marker of neuronal loss, was assessed by proton magnetic resonance spectroscopy, levels were significantly decreased in high-functioning adults with ASD.[57] An in vitro study has demonstrated that microglial activation can promote the death of developing Purkinje cells via reactive oxygen species[58]; however, it remains unclear whether this microglia-mediated mechanism would apply in cases of ASD.

The voxel-based correlation analysis failed to find a cluster in which [^{11}C](R)-PK11195 BP correlated significantly with any of the clinical features evaluated by the Faux Pas Test, Yale-Brown Obsessive Compulsive Scale, ADI-R, and ADOS. However, when ASD subjects were divided into High-BP and Not-High-BP subgroups before being entered into the VOI-based analysis, social disabilities as assessed by ADI-R and ADOS in the High-BP subgroup were significantly more severe than in the Not-High-BP subgroup. The results suggest that ASD subjects carrying more microglial activation may be more impaired in their cognitive skills. In a previous study, immune abnormalities in peripheral blood from severely affected children with ASD, especially the regressive type of autism, appeared to correlate with the disturbance of cognitive skills.[13,59] Considering the positive observation of the VOI-based analysis and the previous data in the ASD children with regression, the failure of the voxel-based correlation analysis was probably due to the selection of the ASD subjects, all of whom were high-functioning ASD subjects with no regression. Namely, the subject selection may have been inappropriate for comparison with studies of severely affected cases. The small subject population may be another reason for the lack of voxel-based correlation analysis. In this study, there was no correlation in the cerebellum between the [^{11}C](R)-PK11195 BP and motor coordination as assessed by the Developmental Coordination Disorder Questionnaire. Again, the selection of the high-functioning subjects and the small sample size may have contributed to the absence of correlation. Although there was no correlation of microglial activation with any of the clinical features, this could not exclude the recently emerging evidence that microglia play a crucial role in monitoring and maintaining synapses in the uninjured brain.[27,28] During development, microglia actively engulf synaptic material and play a major role in synaptic pruning.[60,61] Microglial activation might have led to impairment of synaptic function in the corresponding brain regions being associated with clinical features in ASD.[62-67]

Several limitations of our study bear mention. Our study was performed on a population basis and the subject group consisted entirely of high-functioning ASD subjects. That is, this study did not include ASD subtypes in which immunologic abnormality may be more prominent, although greater microglial activations are more

likely to occur in more severe subtypes. Another potential weakness was the nature of the tracer used in this study, which has a significant nonspecific binding. Future studies on a wider range of autistic phenotypes using a new ligand with more specificity would be warranted.

In conclusion, the present PET measurements revealed marked activation of microglia in multiple brain regions of young adults with ASD. The results strongly support the contention that immune abnormalities contribute to the etiology of ASD. The similar patterns of distribution of regionally activated microglia in these ASD and control groups may indicate the augmented but not altered microglial activation in the brain in the ASD subjects.

Submitted for Publication: November 22, 2011; final revision received March 27, 2012; accepted March 30, 2012.
Published Online: November 26, 2012. doi:10.1001/jamapsychiatry.2013.272
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Author Contributions: Drs Suzuki and Sugihara contributed equally to this work.
Conflict of Interest Disclosures: None reported.
Funding/Support: This work was supported by the Strategic Research Program for Brain Sciences ("Integrated research on neuropsychiatric disorders") and a Grant-in-Aid for Scientific Research from the Ministry of Education, Culture, Sports, Science, and Technology, Japan, and by the Research on Brain Science Funds from the Ministry of Health, Labour, and Welfare, Japan.
Role of the Sponsors: These funding sources played no role in the design or conduct of the study; collection, management, analysis, or interpretation of the data; or preparation, review, or approval of the manuscript.
Additional Contributions: We are grateful to Toshihiko Kanno, MS, Etsuji Yoshikawa, MS, Wataru Ishida, MA, Taro Takahashi, PhD, Utako Matsuo, MA, Kaori Inaho, MA, and Manami Ogawa, MA, for their excellent assistance.

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リレー寄稿
地域生活支援を考える

発達障害の人たちのひとり暮らしを地域で支援するために ～横浜市のサポートホーム事業からの一考察～

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1. はじめに

PDDサポートセンター　グリーンフォーレストは、平成17年に広汎性発達障害の方々を支援するために立ち上がった特定非営利活動法人です。平成24年6月現在、2つの地域活動支援センターを運営し、3つのグループホーム・ケアホームをバックアップしています。

今回は平成21年1月～平成23年3月に横浜市のモデル事業で行った、「生活アセスメント付き住居でのひとり暮らし支援事業（通称サポートホーム事業）」の取り組みをご紹介しながら、発達障害の方々の地域でのひとり暮らしの支援について考えさせていただきます。

2. ひとり暮らし支援の必要性の発見

当法人の地域活動支援センター　オフィス　ウイングは、平成18年より高機能広汎性発達障害の方々に、パソコンでのテープ起こし業務やデータ入力業務等の仕事を提供しています。「就職する前にもう少し経験を積みたい」という方々が利用しており、月曜から金曜まで、9時から17時のフルタイムで働いている方も少なくありません。

オフィス ウイングの利用者の中でも、決まった時間に出勤し、与えられた仕事ができる人たちは、学生のときは無遅刻無欠席だった方が多く、大変まじめな性格の人ばかりです。一方でアルバイト経験はほとんどなく、今の生活に困っていなければ、自分から経験を広げることはなかなかしないことが、特徴としてあげられます。

そんな人たちに、ある日、「一週間にいくらあったら生活できますか？」と尋ねてみました。すると、「使うお金は、ゲームに1日200円。1週間に1000円あれば生活できます」。ほかにも「仕事をしている人は、月に100万円くらい稼いでいるんでしょう」、「ひと月にかかる生活費は1億円くらいですか」など、あまりにも現実離れした答えが返ってきました。あわせて将来について、「5年後、どういう生活をしていると思いますか？」と尋ねると、「5年後になってみないとわかりません」。「将来、お母さんがいなくなったらどうするんですか？」と聞くと、「母は殺させません！」。

このような日々のやり取りの中から、発達障害の方々の中には、将来の生活がイメージしにくいため、家族がいなくなったら今の生活が変わらざるを得ないという事実に気づいていない方や、金銭的にも生活にも現状は困っていないため、将来に備えて準備をする必要性に気づいていない方々がいることがわかりました。また、将来に不安を抱えていたとしても、どこに、どのようにニーズを発したら解決につながるのかのイメージもつかず、そのままにしておきがちなこと、迷惑行為がなければ周囲からも放っておかれがちなことも明らかになりました。そこで、将来の生活イメージを経験で補完できるようなシステムが求められるという考えに至ったのでした。

3. サポートホーム事業について

A.事業概要

発達障害の方が将来の生活イメージを経験で補

完するシステムとして、以下のような事業を提案しました。

① ひとり暮らしができる1Kアパート（=サポートホーム）を提供する。

② サポートホームの支援者は、サポートホーム入居者の生活をアセスメントし、日常生活をサポートしながら、ひとり暮らしに必要な社会資源のサービスを入居者に提案していく。サポートホーム入居者は、地域の社会資源を使いながらサポートホームでひとり暮らしの経験を積む（図1）。

③ 約1年後、サポートホーム入居者は、ひとり暮らしの経験を携えて、好きな地域を選んでひとり暮らしをしていく。支援者はサポートホームでの経験を生かして生活ができるように社会資源をコーディネートし、ケース会議などで継続的に関わりながら、間接的にひとり暮らしをサポートし続ける（図2）。

なお、サポートホームはあくまでも「お試し」のひとり暮らしのため、敷居を低くしたいという思いから、家電を完備し、家賃は3万円に設定しました。水道光熱費や生活にかかる実費は自己負担としました。

B.サポートホームでの支援

◆アセスメントとアプローチ

サポートホームでひとり暮らしをサポートするのにあたり、「食事」「衛生管理」「健康管理」「金銭管理」「人との関わり」の5つのポイントから、支援者は入居者をアセスメントしていきました。アセスメントを通して見えてきた課題については、入居者の考えを取り入れた「折り合い点（目標）」を設定し、それに向けたアプローチを展開しました。その際、支援者の個人的な生活の価値観を入居者に押し付けることのないよう、以下の3点の視点を用いました。

① 生活のしやすさ…「家事のやり方がわからない」といったことに対し、マニュアルを作成するなど、構造化や視覚化を用いて具体的な解決策や方法を提示する。ひとり暮らしに必要な生活全般の基盤づくりに向けた視点。

② 生活の豊かさ…「買い物はこの店しかできない」というような、経験の狭さや偏りからくる言動

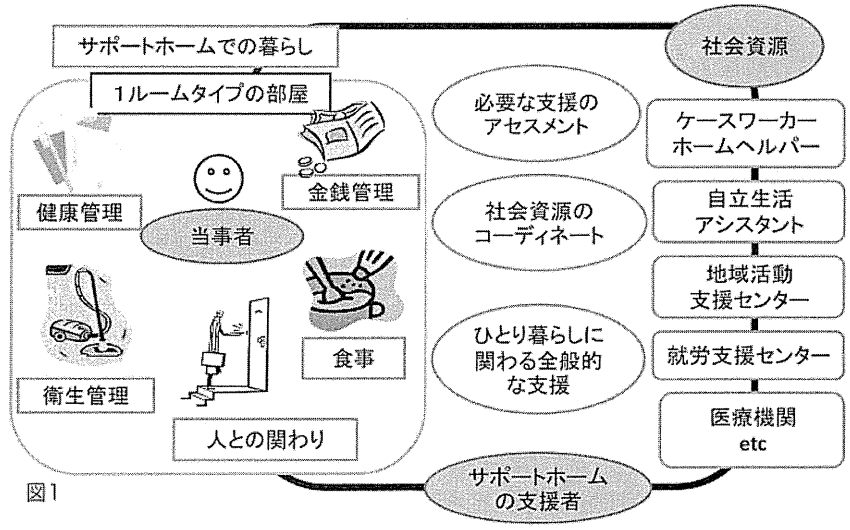


図1

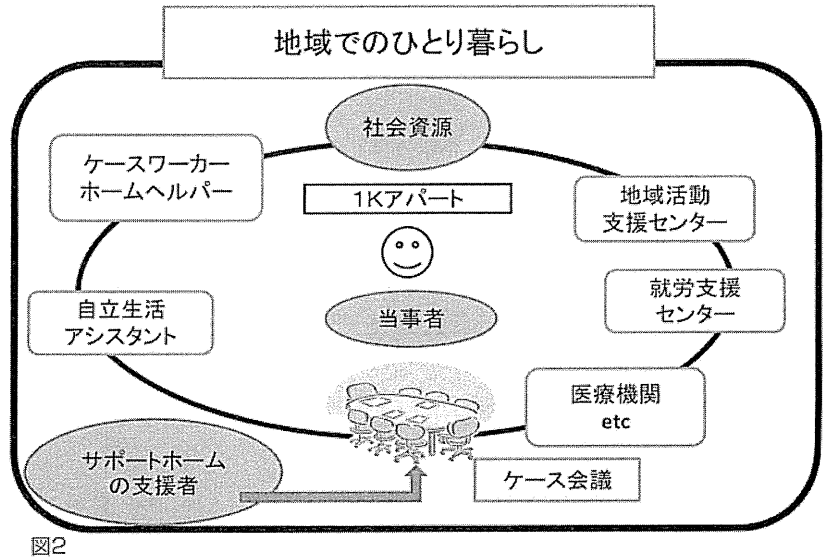


図2

に対し、入居者が広がりのある複数の選択肢の中から、自分の希望や意思に沿って物事を選べるように支援することで、生活を豊かにしようとする視点。

③ 社会との繋がり…サポートホームの支援者と家族以外の、第三者との関わりがスムーズにできるように支援することで、地域の中で孤立せずに生活できる環境をつくることを志向する視点。

これらのアセスメントは、アセスメントシート（図3）に記入していきました。時間を追って目に見える形で記録していくことで、入居者に必要な支援の中身や量を把握することができました。また、入居者の了承のもと、このアセスメントシートをほかの社会資源と共有することで、入居者に有効なアプローチを相互に確認する手段としても使うことができました（図4）。

◆記録とフィードバック

サポートホームの支援者は、訪問によって入居者のひとり暮らしのアセスメントをしました。訪問の頻度は入居者のニーズによって違いますが、基本的には最初は毎日数時間から始めました。そして最終的に地域でひとり暮らしを始めるまでには、月1度の訪問で生活が把握できるよう、計画的に訪問頻度を減らしていきました。

支援者が訪問をしなくても、入居者が自分の生活を振り返ったり、支援者が入居者の日々のニーズを把握できるように、基本的には3種類の記録をつけていただきました。

① 行動記録（図5）…睡眠リズムや食事の時間などの生活を記録するもの

② 家計簿…ひと月ごとの収入と支出、生活費が把握できるもの

③ 生活記録（図6）…食事や家事の内容と、気づいたことや新たな発見を書き留めておくもの

入居者にこれらの記録をつけてもらうことで、支援者は記載内容の意味や背景を理解しながら、ニーズの把握や解決策の提案をしていきました。支援者の訪問時に記録を使って話し合いをし、よくできていることや変化があったことを入居者にフィードバックすることで、入居者が自信を持って生活していけるように支

アセスメントシート

<　　編>

名前：○○さん	提案日：　　年　　月　　日（　）
テーマ：	
ねらい：	
現状：	
本人の考え（ニーズ・反応）	支援者の考え（提案）
折り合い点（目標）	
関わりのポイント	

折り合い（目標）にむけた課題・評価	実施期間

図3

アセスメントシート
<生活の豊かさ編>

名前：Aさん	提案日：2009年　7月30日（木）
テーマ：ホームヘルパーの導入	
ねらい：地域でのひとり暮らしを想定し、サポートホーム支援者以外の社会資源との関わりで、より幅広いメニューから食事を選べるようにする。	
現状：サポートホーム支援者の提案を受け入れレパートリーが広がってきたが、支援者の介入がなければ固定しがち。	
本人の考え・様子	支援者の提案
ヘルパーさんに細かいことを言われるのは嫌だけど、そういうことがなければ、まあ悪くはない。	自炊はよくやっていて素晴らしいけど、よりよい食生活をするために、ヘルパーさんと一緒に調理を教わるのはどう？
目標（折り合い点）	
利用してみて嫌であれば利用を中止することを前提として、一度導入する。	
関わりのポイント	
ヘルパー導入により、具体的にどういった変化があるのか等、具体的な見通しをもたせて伝える。	

目標（折り合い）にむけた課題・評価	実施期間
より栄養バランスのとれた食事や、手の込んだ料理を生活に取り入れられるという視点で、ホームヘルパー利用の提案をした。本人は最初、ヘルパーさんから何か注意されるのではないかという誤解（イメージ）があったが、丁寧に説明したところ、安心した表情を見せた。また「女性ヘルパーさん」という言葉に時折嬉しそうな表情もみせていた。 →本人も導入に関して「構わない」ということを言ってくれたので実際の導入の検討をしていく。	7月30日 （提案日）
ヘルパー導入のアンケートをお願いした。 「本当にヘルパーさんを使わなきゃいけないんですか…」とアンケート記入を渋る様子をみせる。調理というよりも、ヘルパーさんと自分の趣味の話をしたいという希望が大きい。ヘルパーさんに作ってもらう材料費が実費ということに、渋い表情をする。 「食材を使いまわしてもらえれば逆に食費が浮く」などヘルパーさんに来てもらうメリットを再度話し、「1度来てもらって嫌だったらまた考えよう」ということで本人も了承した。	9月3日
明日、ヘルパーの契約があることを伝えた。不安なのか拒否気味の様子だったので、まずは1度導入してみて、嫌だったらキャンセルしてもいいことを伝えた。	10月1日
契約日。 ヘルパーが入ることで、「メニューを決めるのがストレス」「何をどれくらい買えばいいのか」「いくらかかるのか」「食材が余ったら困る」「保存はどれくらいできるのか」などの不安を持っていた。契約時にヘルパーを交え、メニューの話をし、具体的な買い出しリストを渡すと「意外と買うものが多くない、こんなんでいいんですね」と、見通しがたって少し表情が和らいだ。 →嫌だから拒否というより、わからないから拒否、という部分が強かったと思われる。	10月2日
ヘルパー導入初日。（引き継ぎのためヘルパーさんと一緒に訪問） 本人は食材の賞味期限を気にし、「（実家へ帰宅する）週末までに食べきらなければ…」という意識が強かった。しかし「この食材は1週間もつ」、「この食材は冷蔵庫に入れておけば腐らない」など、1つずつ説明し、本人の「食べきらなければ」という意識をやわらげ、安心して食材を残せるように伝えていった。 ヘルパー導入までは不安な様子であったが、買うものの見通しも立ち、実際にヘルパーさんとの関わりを楽しめた様子。次回の訪問を楽しみにしていた。ヘルパー導入前はメニューを自分で決められなかったが、本人から「次週は鮭を焼いてみたい」との希望があった。	10月7日
ヘルパー2回目。（前回同様引き継ぎのためヘルパーさんと一緒に訪問） 訪問した際には、前回一緒に作ったポテトサラダをすでに自分で作り始めていた。予定通り「鮭」をヘルパーさんに教わりながら焼いた。 調理の教え方や接し方は特には問題ないが、次週のメニュー決めが課題になりそう。本人はすでに自分ひとりで作ったことのあるものや、ヘルパーさんと一緒に作って、ひとりでできるようになったものを選ぶことが考えられる。「ヘルパーさんと一緒に調理する時は、一品は自分ひとりで作れないものにする」ということを、本人・ヘルパーさんと確認した。	10月14日
ヘルパーさん単独で3度サービスに入ってもらった。その様子を聞き取る中で、「自分の趣味の話をする時間が多いかもしれない」「食事作り自体は楽しんでやれている。買い物も不安はない様子で問題なく行えている」ということがわかった。 慣れたメニューで早く終わらせて話の時間を多くもとうとすることが考えられ、エスカレートしていく可能性もあるため、サービスの時間や関わり方を再度検討していく。 （※導入時のサービスは週1回、2時間）	11月4日
ヘルパーと一緒に作った「ハンバーグ」を写真で撮ってメールしてくれた。 楽しく調理をしている様子。	11月11日
ヘルパー・本人と3者で決めたルール通り、基本的にヘルパーとは新しいメニューを作るということは守られている。本人も満足している様子。	12月3日
ヘルパーと一緒に作って覚えたものを、ひとりで作っていた。 また、ヘルパーと作ったものから、自分でアレンジして新しいメニューに挑戦していた。「料理に興味が出てきた」と言っている。	12月17日

図4

援していきました。

4. サポートホーム事業の成果と課題

サポートホーム事業は、20～30代で、福祉手帳を所持している4名の男性が利用しました。1人は高卒、3人は4年制大学卒の学歴を持っていました。入居条件の中には、日中の所属先・就労先があり、安定的に通えていることや、発達障害の診断があることを盛り込みました。

サポートホーム事業を実施した2年間で、4名のうち3名が実際に地域でのひとり暮らしへ移行しました。サポートホームでひとり暮らしを経験することで、ひとり暮らしに必要なスキルを獲得し、自信を持って地域に出ていくことができました。また、サポートホームで支援者と信頼関係を築き、必要なサポートがアセスメントされた状態での地域移行となったため、ケースワーカーや自立生活アシスタント※、ホームヘルパーや相談機関等、ひとり暮らしをサポートする地域の社会資源がネットワークを組んで、継続的にひとり暮らしを支える体制をつくることができました。

地域でのひとり暮らしに移行しなかった1名は、サポートホームでひとり暮らしを経験したことで、初めてひとり暮らしに必要な金額を把握

※単身等で生活する障害者が地域生活を継続するために、専門的知識と経験を有する「自立生活アシスタント」を派遣して、具体的な生活の場面での助言やコミュニケーション支援を行う横浜市の事業

年 月　日（月）	月　日（火）	月　日（水）	月　日（木）	月　日（金）	月　日（土）
0:00	0:00	0:00	0:00	0:00	0:00
1:00	1:00	1:00	1:00	1:00	1:00
2:00	2:00	2:00	2:00	2:00	2:00
3:00	3:00	3:00	3:00	3:00	3:00
4:00	4:00	4:00	4:00	4:00	4:00
5:00	5:00	5:00	5:00	5:00	5:00
6:00	6:00	6:00	6:00	6:00	6:00
7:00	7:00	7:00	7:00	7:00	7:00
8:00	8:00	8:00	8:00	8:00	8:00
9:00	9:00	9:00	9:00	9:00	9:00
10:00	10:00	10:00	10:00	10:00	10:00
11:00	11:00	11:00	11:00	11:00	11:00
12:00	12:00	12:00	12:00	12:00	12:00
13:00	13:00	13:00	13:00	13:00	13:00
14:00	14:00	14:00	14:00	14:00	14:00
15:00	15:00	15:00	15:00	15:00	15:00
16:00	16:00	16:00	16:00	16:00	16:00
17:00	17:00	17:00	17:00	17:00	17:00
18:00	18:00	18:00	18:00	18:00	18:00
19:00	19:00	19:00	19:00	19:00	19:00
20:00	20:00	20:00	20:00	20:00	20:00
21:00	21:00	21:00	21:00	21:00	21:00
22:00	22:00	22:00	22:00	22:00	22:00
23:00	23:00	23:00	23:00	23:00	23:00

図5　出典：「発達障害のためのライフハック」＜http://fagoa.blogspot.jp/

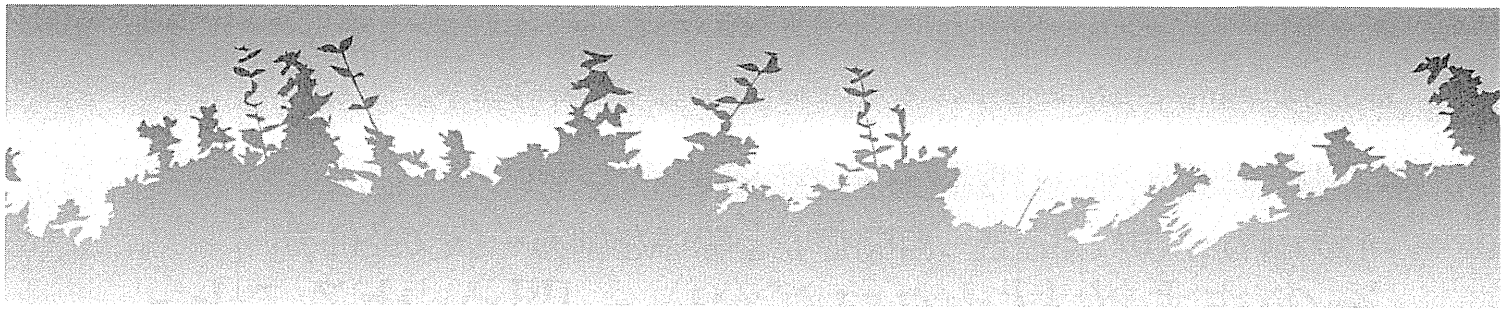
し、就労意欲も高くなりました。「お金を貯めてからひとり暮らしをしたい」という明確な希望があり、サポートホーム事業終了後は一度実家へ戻りました。支援者は、定期面談で継続的に就労できていることを確認しながら、ひとり暮らしへ向けて計画的に貯金をするサポートをし、結果、平成23年８月からひとり暮らしを開始しました。

発達障害の方のひとり暮らしの支援について、一見効果のありそうなサポートホーム事業ですが、今回の2年間のサポートホーム事業では、この事業が有効なタイプと、有効でないタイプも浮き彫りになりました。

まず、サポートホーム事業が有効なタイプは、①経験を積み上げることでイメージが補完できるタイプと、②支援者と振り返りができるタイプです。

①のタイプの方は、経験していないことに対してはイメージを持ちにくく、生活の選択肢が狭かったり偏りがある一方で、支援者が構造化や視覚化を用いてわかりやすく提案していけば、経験を積み上げて生活を豊かにしていくことができました。②のタイプの方は、知識として獲得する経験（間接経験）は豊富でも、直接経験は乏しく、誤った認識で行動することが多いため、問題や課題となる行動が多い方でした。それでも支援者と一つ一つの行動を振り返り、支援者の提案に折り合いをつけながら、サポートホームにおいて経験を積み上げていくことができました。

そしてサポートホーム事業が有効でなかったのは、支援者と振り返りができないタイプの方でした。サポートホームの支援者の介入を、本人は「抑制



される」ととらえてしまいがちで、前向きな話し合いがなかなかできませんでした。したがって経験を積んでひとり暮らしに生かしていくサポートホーム事業は、あまり有効ではありませんでした。このようなタイプの方の場合は、ひとり暮らしの経験を提供するよりも、困ったときに頼れる身近な相談者を置きながら実際にひとり暮らしをしていくのが、その方にとってはより有効なサポートになるのではないかと考えさせられました（サポートホーム事業によって、より有効な支援が浮き彫りにされたという意味では、一定のアセスメントの効果があったといえるかもしれません）。

5. 地域でのひとり暮らしを支援していくことについて

まず、今回のサポートホーム事業をやらせていただいて、発達障害の方々がひとり暮らしをする力を信じることの大切さを改めて実感しました。また、ひとり暮らしを経験できるサポートホームの存在が大きかったことはもちろんですが、「とりあえずやってみて、無理だったら家に戻れる」という状況で、家族の方々も一定の距離をとって見守ってくださっていたことが、発達障害の方が安心してサポートホーム事業を利用できる理由の一つとなっていたようでした。

地域でのひとり暮らしを志向するときは、地域の一員として、孤立することなく生活しないと意味がありません。そのことを念頭に置き、支援者は専門的な視点を持って、継続的に関われる支援体制を組み立てていく必要性があると思います。ひとり暮らしの中に入り込んでいく「おせっかい型」のサポートホーム事業だけでなく、多様な発達障害の方々に合った、地域でのひとり暮らしに向けた多様な支援が展開されていくことを望みます。

なお、サポートホーム事業は平成21年から平成23年の横浜市のモデル事業でしたが、今年度から新たに場所を設け、横浜市の新規事業として再スタートします。既存の制度を活用しながら、どこまで発達障害の方々のひとり暮らしを支援できるか、模索しながらの事業展開になりますが、また皆さんにご報告ができますよう、法人一同取り組んでまいりたいと思っています。

生活記録

自分の生活記録をつけることで、自己理解を深め、1日を振り返りましょう

記入日：　　年　　月　　日

	食べた物／やったこと	新しい発見（気づいたこと）や良かった点
＜食事＞		
＜家事＞		
＜その他＞		

記入日：　　年　　月　　日

	食べた物／やったこと	新しい発見（気づいたこと）や良かった点
＜食事＞		
＜家事＞		
＜その他＞		

図6

リレー寄稿
地域生活支援を考える

発達障害の人たちのひとり暮らしを地域で支援するために ～地域生活移行に向けた滋賀での取り組み～

松田裕次郎

社会福祉法人滋賀県社会福祉事業団
クリエートプラザ東近江「ジョブカレ」

はじめに

私が、福祉の世界に入ってそろそろ、20年が経過しようとしている。身体・知的・精神・発達障害の方々と10年近く関わる中で、私が感じる誰もが生きやすい社会とは、人が人を人として認める社会をどう実現するかということに収斂されるのではないかということである。

平成17年に高機能の発達障害のある人の支援を担当するようになってから、発達障害のある人のための支援の中には、障害の有無にかかわらず誰もが生きやすい社会を実現するためのエッセンスがあるのではないかと実感している。だからこそ発達障害のある人たちの地域生活を支える仕組みが大切だと思う。ここでは、本人たちが安心して地域で生活するためにどのような仕組みが必要なのかについて考察してみたいと思う。

滋賀県では、平成17年度より自閉症等発達障害支援体制整備事業の一環として、高機能自閉症地域生活支援モデル事業（～平成19年度）が実施された。後に、ひとり暮らしの支援を含めた、高機能自閉症地域生活ステップアップ事業（平成20年度～23年度）も実施された。これらの事業は滋賀県社会福祉事業団が委託を受け、平成17年度から23年度の7年間はグループホームのフレームを活用しながら、高機能の発達障害のある人たちの支援を行ってきた。そしてこれらの支援をより効果的に進めるために、今年度より発達障害者地域生活支援システム構築事業（通称「ジョブカレ」）に取り組んでいる。

前半では、モデル事業やステップアップ事業で行ってきた、発達障害のある人たちの地域生活を支える取り組みについて紹介しながら、本人たちが安心して地域で生活するためにどのような仕組みが必要なのかについて提案し、後半では現在取り組んでいるシステム構築事業について紹介してみたいと思う。

1「ホームかなざわ」について

平成17年に開始した高機能自閉症地域生活支援モデル事業は、滋賀県の甲賀福祉圏域において実施し、その拠点となるホームとして「ホームかなざわ」を用いた。ホームかなざわは、高機能に特化したグループホームであり本来的にはホーム利用後のひとり暮らしを目指し、およそ2年間を目途に生活スキルや対人関係スキル等の生活支援を行う通過型のホームという位置づけになる。

1）ホームかなざわ利用者について

ホームかなざわを利用された方は、次の①から④のケースに分けることができる。

① ひとり暮らしを目標とし、自立しようとするケース
② 自宅での生活がうまくいかず、自宅を出て生活しなければならないケース
③ ひとり暮らし等自立した生活を送っていたがうまくいかず、生活の立て直しを図るケース
④ 入所施設等で生活していたが、地域生活に移行を希望するケース

上記のように個々の事情は様々だが、皆地域生活を送りたいという希望を持ってこの事業を利用