Precursors of mast cells were defined as cells that formed mast-cell colonies in methylcellulose culture (CFU-mast). Mononuclear cells (MNC) were obtained from the bone marrow, peripheral blood, and small intestine of Ws/Ws rats with a small deletion at the tyrosine kinase domain of c-kit and of control normal (+/+). In the culture containing concanavalin A-stimulated spleen cell conditioned medium (ConA-SCM) alone, the numbers of mast-cell colonies produced by Ws/Ws MNC were comparable with those of +/+ MNC. In the culture containing both ConA-SCM and stem cell factor (a ligand of c-kit), however, the numbers of mast-cell colonies produced by +/+ blood MNC were 107 times as great as that of Ws/Ws blood MNC. Using this culture condition, we investigated changes in concentration of CFU-

mast in the marrow, blood, and intestine of +/+ rats after infection with *Nippostrongylus brasiliensis* (NB), which induced marked mast-cell accumulation in the small intestine. The concentration of CFU-mast in blood dropped to 21% of preinfection levels 1 week after the NB infection. In contrast, a sevenfold increase of CFU-mast occurred in the small intestine. The proportion of CFU-mast in S phase of the cell cycle remained at low levels in the marrow and blood after NB infection, but it increased significantly in the small intestine. The present result suggests that NB infection induces the invasion of CFU-mast into the intestine from blood and their subsequent proliferation in the tissue site.

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washed extensively in PBS containing 30 mM EDTA-Na₂ and 30 pg/mL gentamicin (three times, 15 min-
utes each). This treatment removed the villous epithelium but pre-
served the crypt epithelium, as seen in histologic sections. The frag-
ments were then transferred to flasks containing 25 mL α-MEM with collagenase (Sigma, Type I; 1 mg/mL), hyaluronidase (Sigma, Type I; 1 mg/mL), and 20% fetal calf serum (PCS; Hyclone, Logan, UT) at 37°C and incubated with magnetic stirring for 60 minutes. After the incubation, cell clusters were broken up by repeated aspiration through needles of decreasing size. The suspensions were passed through a glass wool column (0.5 g packed in a 10-mL syringe), and the cells were washed three times with α-MEM. The MNC fraction was obtained by centrifugation over a discontinuous density gradient of Percoll (Pharmacia, Piscataway, NJ). A stock solution of Percoll was prepared by mixing 90 mL Percoll with 8.96 mL calcium-free, magnesium-free Hanks’ balanced salt solution (CMF-HBSS; 10×), 1 mL HEPES buffer, and 455 μL HCl (1 N). To obtain the desired density, this stock solution of Percoll was diluted with CMF-HBSS (1×) supplemented with 1% HEPES buffer. The final density of Percoll was measured by PAAR Digital Density Meters (Anton Paar, Graz, Austria). Two milliliters of 1.085 g/mL Percoll was overlaid with 4 mL of 1.055 g/mL Percoll containing cell sus-
pension. After centrifugation at 600g for 20 minutes at 25°C, highly viable MNC could be recovered from the 1.055/1.085 g/mL inter-
face. Dead cells, debris, and epithelial cells were found at the upper surface, and red blood cells were found in the pelle. MNC at the interface were collected and washed twice with α-MEM. The procedure yielded 10⁶ to 3 × 10⁸ cells per rat. The purity of MNC was determined by examining cytocentrifuge specimens stained with May-Grünwald-Giemsa solution. More than 90% cells were MNC, and principal contaminants were crypt cells and eosinophils.

Spleen cell conditioned medium. Concanavalin A-stimulated spleen cell conditioned medium (ConA-SCM), in which T-cell–
derived cytokines such as IL-3 were reported to be present, was prepared as described by Broide et al with minor modifications. MNC (4 × 10⁶/mL) were isolated with LSM from spleen cells of +/+ rats and cultured for 3 days in 75-cm² tissue culture flasks (Corning, New York, NY) containing 3 μg/mL ConA (Sigma), 20% heat-inactivated horse serum (Irvine Scientific, Santa Ana, CA), and 10⁻² mol/L 2-mercaptoethanol (Sigma). The conditioned medium was centrifuged, filtered through a 0.22-μm filter (Millipore Corp, Bedford, MA), and stored at −80°C until use.

Clonal cell culture. Methylcellulose culture was performed ac-
cording to the method described by Nakahata and Ogawa with minor modification. MNC obtained from the bone marrow (10⁶), peripheral blood (5 × 10⁶), and lamina propria of small intestine (10⁷) were plated in 35-mm Falcon plastic dishes (Becton Dickinson Labware, Lincoln Park, NJ) in 1.0 mL of a mixture containing 0.9% methylcellulose (Shin-etsu Chemicals, Osaka, Japan), 30% FCS, 1% bovine serum albumin (Sigma), 10⁻¹ mol/L mercaptoethanol, 10% (vol/vol) ConA-SCM, and/or various concentrations of recombinant mouse SCF (rmSCF; a gift from Kirin Brewery Co Ltd, Tokyo, Japan). Dishes were incubated at 37°C in a humidified atmosphere with 5% CO₂. On day 10, colonies containing ≥50 cells were counted under the inverted microscope.

Staining of cultured cells. Individual colonies were lifted from the methylcellulose medium by using a 3-μL Eppendorf pipette under direct microscopic visualization and were collected in Eppendorf microcentrifuge tubes containing 0.3 mL of α-MEM. After washing two times with the medium, the same samples were immediately spun in a cytocentrifuge (Shandon Southern, Elliott, IL) at 600 rpm for 5 minutes. The slides were stained with May-Grünwald-Giemsa solution. The types of colonies were classified according to the presence or absence of mast cells. Colonies containing mast cells alone were counted as mast-cell (mast) colonies. Colonies consisting of granulocytes and/or macrophages were counted as granulocyte-
Table 1. Numbers of Mast-Cell and GM Colonies in the Bone Marrow, Peripheral Blood, and Small Intestine of Intact +/+ and Ws/Ws Rats in Various Culture Conditions

<table>
<thead>
<tr>
<th>Origin of Cells</th>
<th>Growth Factors†</th>
<th>Mast</th>
<th>Mast + GM</th>
<th>GM</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>+/+ marrow</td>
<td>ConA-SCM</td>
<td>5 ± 3</td>
<td>5 ± 3</td>
<td>730 ± 32</td>
<td>740 ± 32</td>
</tr>
<tr>
<td></td>
<td>rmSCF</td>
<td>228 ± 244</td>
<td>32 ± 9</td>
<td>620 ± 9</td>
<td>880 ± 23</td>
</tr>
<tr>
<td></td>
<td>Both</td>
<td>650 ± 224</td>
<td>85 ± 31</td>
<td>765 ± 31</td>
<td>1,500 ± 187</td>
</tr>
<tr>
<td>Ws/Ws marrow</td>
<td>ConA-SCM</td>
<td>5 ± 3</td>
<td>5 ± 3</td>
<td>790 ± 20</td>
<td>800 ± 20</td>
</tr>
<tr>
<td></td>
<td>rmSCF</td>
<td>0 ± 0</td>
<td>0 ± 0</td>
<td>198 ± 264</td>
<td>198 ± 264</td>
</tr>
<tr>
<td></td>
<td>Both</td>
<td>8 ± 25</td>
<td>5 ± 36</td>
<td>805 ± 32</td>
<td>818 ± 295</td>
</tr>
<tr>
<td>+/+ blood</td>
<td>ConA-SCM</td>
<td>0.3 ± 0.1</td>
<td>0 ± 0.2</td>
<td>0.3 ± 0.1</td>
<td>0.6 ± 0.2</td>
</tr>
<tr>
<td></td>
<td>rmSCF</td>
<td>11.9 ± 0.5</td>
<td>0.3 ± 0.1</td>
<td>1.3 ± 0.6</td>
<td>23.4 ± 2.3</td>
</tr>
<tr>
<td></td>
<td>Both</td>
<td>21.3 ± 2.7</td>
<td>0.9 ± 0.1</td>
<td>3.0 ± 0.1</td>
<td>23.4 ± 2.3</td>
</tr>
<tr>
<td>Ws/Ws blood</td>
<td>ConA-SCM</td>
<td>0.3 ± 0.2</td>
<td>0 ± 0.1</td>
<td>1.0 ± 0.1</td>
<td>1.3 ± 0.2</td>
</tr>
<tr>
<td></td>
<td>rmSCF</td>
<td>0 ± 0</td>
<td>0 ± 0.2</td>
<td>0.4 ± 0.2</td>
<td>0.4 ± 0.2</td>
</tr>
<tr>
<td></td>
<td>Both</td>
<td>0.2 ± 0.1</td>
<td>0 ± 0.1</td>
<td>0.6 ± 0.1</td>
<td>0.7 ± 0.2</td>
</tr>
<tr>
<td>+/+ intestine</td>
<td>ConA-SCM</td>
<td>0 ± 0</td>
<td>0 ± 0</td>
<td>0 ± 0</td>
<td>0 ± 0</td>
</tr>
<tr>
<td></td>
<td>rmSCF</td>
<td>10.8 ± 1.2</td>
<td>0 ± 0</td>
<td>10.8 ± 1.2</td>
<td>10.8 ± 1.2</td>
</tr>
<tr>
<td></td>
<td>Both</td>
<td>13.1 ± 0.7</td>
<td>0 ± 0</td>
<td>13.1 ± 0.7</td>
<td>13.1 ± 0.7</td>
</tr>
<tr>
<td>Ws/Ws intestine</td>
<td>ConA-SCM</td>
<td>0 ± 0</td>
<td>0 ± 0</td>
<td>0 ± 0</td>
<td>0 ± 0</td>
</tr>
<tr>
<td></td>
<td>rmSCF</td>
<td>0 ± 0</td>
<td>0 ± 0</td>
<td>0 ± 0</td>
<td>0 ± 0</td>
</tr>
<tr>
<td></td>
<td>Both</td>
<td>0 ± 0</td>
<td>0 ± 0</td>
<td>0 ± 0</td>
<td>0 ± 0</td>
</tr>
</tbody>
</table>

* Mean ± SE of four dishes. The result of a representative experiment is shown, but two other experiments gave similar results.
† Culture medium contained 10% ConA-SCM or 100 ng/mL rmSCF, or both.
‡ P < .01 by t-test when compared with the numbers of colonies produced by the same aliquot of cells in the culture containing 10% ConA-SCM alone.
§ P < .01 by t-test when compared with the numbers of colonies produced by +/+ cells in the same culture condition.

RESULTS

Effect of rmSCF on numbers of mast-cell colonies. MNC obtained from the bone marrow, peripheral blood, and small intestine were washed twice in serum-free α-MEM and then incubated for 60 minutes at 37°C in pre-warmed serum-free α-MEM with or without 200 μg/mL hydroxyurea (HU; Sigma) at a concentration of 104 cells per milliliter. After incubation, cells were washed three times in α-MEM supplemented with 2% FCS and plated in the methylcellulose medium containing 10% ConA-SCM and 100 ng/mL rmSCF. This concentration of HU was suitable for the suicide of hematopoietic cells that were in S phase of the cell cycle as reported by Kanakura et al.15

The numbers of mast-cell colonies were counted on day 10 after initiation of the culture. When marrow MNC or blood MNC were cultured with various concentrations of rmSCF, the numbers of mast-cell colonies increased with increasing concentrations of rmSCF up to 100 ng/mL (Fig 1). Few mast-cell colonies developed when marrow MNC and blood MNC were cultured with 10% ConA-SCM alone, but the addition of rmSCF markedly increased the numbers of mast-cell colonies. As the maximum number of mast-cell colonies were obtained when both 10% ConA-SCM and 100 ng/mL rmSCF were present, we thereafter used this culture condition to evaluate the numbers of CFU-mast in various organs and under various experimental conditions. In one experiment, we cultured marrow MNC of Ws/Ws rats instead of +/+ marrow MNC. Even 400 ng/mL rmSCF did not induce development of mast-cell colonies from Ws/Ws marrow MNC, and the addition of 400 ng/mL rmSCF to 10% ConA-SCM did not increase the numbers of mast-cell colonies produced by Ws/Ws marrow MNC (data not shown).

In addition to mast-cell colonies, colonies consisting of granulocytes and/or macrophages also developed. In the next experiment, we picked up all colonies and classified them by examining cytocentrifuge preparations. MNC derived from the bone marrow, peripheral blood, and small intestine of +/+ and Ws/Ws rats were cultured in three different conditions: (1) culture containing 10% ConA-SCM alone, (2) culture containing 100 ng/mL rmSCF alone; and (3) culture containing both 10% ConA-SCM and 100 ng/mL rmSCF. In the cultures containing 10% ConA-SCM alone, the numbers of mast-cell and GM colonies produced by MNC from Ws/Ws marrow and Ws/Ws blood were comparable with the numbers of colonies produced by +/+ marrow and +/+ blood MNC (Table 1). The numbers of GM colonies were greater than that of mast-cell colonies in both Ws/Ws and +/+ rats (Table 1). No colonies developed when MNC obtained from the small intestine of either +/+ or Ws/Ws rats were cultured with 10% ConA-SCM alone.

When MNC of +/+ and Ws/Ws rats were cultured with 100 ng/mL rmSCF alone, significantly greater numbers of mast-cell colonies were obtained when both 10% ConA-SCM and 100 ng/mL rmSCF were present, we thereafter used this culture condition to evaluate the numbers of CFU-mast in various organs and under various experimental conditions. In one experiment, we cultured marrow MNC of Ws/Ws rats instead of +/+ marrow MNC. Even 400 ng/mL rmSCF did not induce development of mast-cell colonies from Ws/Ws marrow MNC, and the addition of 400 ng/mL rmSCF to 10% ConA-SCM did not increase the numbers of mast-cell colonies produced by Ws/Ws marrow MNC (data not shown).

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When MNC of +/+ and Ws/Ws rats were cultured with 100 ng/mL rmSCF alone, significantly greater numbers of mast-cell colonies were obtained when both 10% ConA-SCM and 100 ng/mL rmSCF were present, we thereafter used this culture condition to evaluate the numbers of CFU-mast in various organs and under various experimental conditions. In one experiment, we cultured marrow MNC of Ws/Ws rats instead of +/+ marrow MNC. Even 400 ng/mL rmSCF did not induce development of mast-cell colonies from Ws/Ws marrow MNC, and the addition of 400 ng/mL rmSCF to 10% ConA-SCM did not increase the numbers of mast-cell colonies produced by Ws/Ws marrow MNC (data not shown).
MAST CELL PRECURSOR colonies developed from +/+ MNC than from Ws/Ws MNC (Table 1). Specifically, a large difference was observed in the numbers of mast-cell colonies, because no mast-cell colonies at all developed from Ws/Ws MNC in this culture condition (Table 1). When the numbers of mast-cell colonies produced by +/+ MNC were compared in different culture conditions, the numbers of colonies produced by marrow MNC and blood MNC were approximately 40-fold greater in the culture containing 100 ng/mL rmSCF than in the culture containing 10% ConA-SCM (Table 1). Addition of rmSCF to 10% ConA-SCM significantly increased the numbers of mast-cell colonies produced by +/+ MNC derived from the bone marrow, peripheral blood, and small intestine but not the numbers of mast-cell colonies produced by Ws/Ws MNC (Table 1).

Effect of NB infection. Rats of +/+ and Ws/Ws genotypes were infected with NB, and MNC were obtained from the bone marrow, peripheral blood, and small intestine at various times after the NB infection. MNC were plated in methylcellulose medium containing both 10% ConA-SCM and 100 ng/mL rmSCF, and the numbers of mast-cell colonies were counted under the inverted microscope. The numbers of mast-cell colonies produced by MNC of Ws/Ws rats remained at low levels even after the NB infection, and no significant effect of the NB infection was detectable (data not shown). Therefore, we will describe the NB infection-induced changes only in tissues of +/+ rats. The concentration of CFU-mast did not change significantly in the bone marrow, but a marked decrease was observed in the peripheral blood (Fig 2). The concentration dropped to 21% of preinfection levels on day 7 after the infection. This concentration of CFU-mast recovered gradually thereafter and reached preinfection levels 6 weeks after the infection. In contrast with the decrease of CFU-mast in the peripheral blood, the concentration of CFU-mast in the small intestine increased significantly; a sevenfold increase was observed on day 7 after the NB infection (Fig 2).

In the next experiment, the types of colonies were determined 1 week after the NB infection by picking up all colonies. In the bone marrow, the proportion of colonies of each type did not change significantly after the NB infection (Table 2). In the peripheral blood, the concentration of CFU-mast decreased, but that of CFU-GM did not change significantly. In the small intestine, CFU-GM were not detectable even after the NB infection (Table 2).

To examine whether CFU-mast proliferated after invading the small intestine, we examined the proportion of CFU-mast in S phase of the cell cycle before and 1 week after the NB infection. MNC from various tissues were incubated with or without HU. As a control, we used CFU-GM in the bone marrow of intact +/+ rats. The numbers of CFU-GM were significantly decreased by the HU treatment, and the proportion of CFU-GM in S phase was 24.3%. This is consistent with the values reported by Kanakura et al15 in the mouse bone marrow. In contrast to the HU-induced decrease of CFU-GM, the numbers of CFU-mast in the bone marrow, peripheral blood, and small intestine of intact +/+ rats were not decreased by the HU treatment (Table 3). One week after the NB infection, the proportion of CFU-mast in S

![Fig 2. Numbers of mast-cell colonies in the bone marrow, peripheral blood, and small intestine of +/+ rats at various times after the NB infection. MNC from bone marrow (10⁵), peripheral blood (5 × 10⁵), and small intestine (10⁵) of intact +/+ rats were cultured in methylcellulose medium containing both 10% ConA-SCM and 100 ng/mL rmSCF. Numbers of mast-cell colonies per 10⁵ MNC were calculated. Each point represents the mean of four dishes. The result of a representative experiment is shown, but two other experiments gave similar results. Bars show SE. At most points, SE was smaller than the size of each symbol (ie, ⊗).](image)
alone. In fact, mast cells are markedly deficient in tissues of SCM and SCF than in the culture containing ConA-SCM. The numbers of mast-cell colonies produced by rats. The tyrosine kinase activity of the c-kit receptor is impaired in both SCF appear to parallel the actual development of mast cells in tissues.

**DISCUSSION**

When bone marrow and peripheral blood MNC from Ws/ Ws and control +/+ rats were cultured in the methylcellulose medium containing ConA-SCM alone, the numbers of mast-cell colonies produced by Ws/Ws MNC were comparable with the numbers of mast-cell colonies produced by +/+ MNCs. This is consistent with the results of Suda et al. and Kanakura et al., who used W/W mice instead of Ws/Ws rats. The tyrosine kinase activity of the c-kit receptor is severely impaired in both W/W mice and Ws/Ws rats. Although the addition of SCF to ConA-SCM did not increase the numbers of mast-cell colonies produced by Ws/Ws MNC, the numbers of mast-cell colonies produced by +/+ MNC were much greater in the culture containing both ConA-SCM and SCF than in the culture containing ConA-SCM alone. In fact, mast cells are markedly deficient in tissues of W/W mice and Ws/Ws rats. Therefore, the numbers of mast-cell colonies in the culture containing ConA-SCM and SCF appear to parallel the actual development of mast cells in tissues.

Recently, Tei et al. examined the effect of ConA-SCM and SCF on suspension-cultured mast cells (CMC) of Ws/ Ws and +/+ rats. Their finding of a synergistic effect of ConA-SCM and SCF on the proliferation of +/+ CMC is consistent with our results. On the other hand, Ws/Ws CMC may survive at least 8 days in the suspension culture containing SCF alone, whereas no mast-cell colonies developed from Ws/Ws MNC in the methylcellulose culture containing SCF alone. Moreover, a weak synergistic effect of ConA-SCM and SCF was detectable in the suspension culture of Ws/Ws CMC. The clonal culture appears to be a more sensitive system than the suspension culture in demonstrating deficient development of mast cells in Ws/Ws rats.

In this study, we used ConA-SCM instead of IL-3 because (1) IL-3 of rat origin is rather difficult to obtain, and (2) in the case of mice, SCM is much better than recombinant IL-3 for development of CMC from bone marrow cells. In fact, Haig et al. examined the effect of recombinant rat IL-3 and lymph node-conditioned medium (LNCM) on development and phenotype of rat CMC. They obtained mesenteric lymph node cells from NB-infected rats and stimulated the lymph node cells by NB-antigen or ConA. The effect of rat recombinant IL-3 was comparable with LNCM. The use of ConA-SCM instead of IL-3 raises another problem: there is a possibility that ConA-SCM may contain GM colony-stimulating factor (CSF) that could inhibit mast cell outgrowth.

SCF and ConA-SCM showed an apparent synergism for development of mast-cell colonies. This is consistent with the results of Haig et al. and Tei et al. Both of them showed the synergistic effect using rat CMC. SCF shows synergism between various cytokines. For example, addition of SCF to GM-CSF, G-CSF, or IL-3 increased the numbers of GM or G colonies. Addition of SCF to erythropoietin increased the numbers of erythroid bursts, and addition of SCF to IL-7 enhanced proliferation of pre-B cells. Both IL-3 and SCF activate a common set of proteins. Both induce tyrosine phosphorylation of a common set of proteins, such as phosphatidylinositol-3'-kinase and mitogen-activated protein kinase. The synergistic effect between SCF and ConA-SCM may be explained at least partially by this mechanism.

NB infection induced a significant decrease of CFU-mast in the peripheral blood and a reciprocal increase in the intes-
tinal mucosa. The increase in the intestinal mucosa is consistent with the results of Guy-Grand et al., who evaluated the concentration of mast-cell precursors by limiting dilution analysis. The decrease of CFU-mast in the peripheral blood is unexpected and has never been reported. The numbers of mast-cell colonies produced by blood MNC are relatively small in the culture containing T-cell–derived cytokines alone, and we speculate it would be difficult to demonstrate a decrease from such a low starting value. The numbers of mast-cell colonies produced by blood MNC of +/- rats were rather large in the present culture containing both ConA-SCM and SCF. This may explain the successful demonstration of the decrease in blood CFU-mast after NB infection. The concentration of CFU-mast did not change in the marrow, and the turnover of CFU-mast in the marrow was not accelerated. Therefore, the decrease of CFU-mast in the peripheral blood appears to be attributable to the augmented recruitment of CFU-mast from the peripheral blood to the intestinal mucosa.

Both IL-34 and SCF5 have been reported to induce the migration of murine CMC. As the augmented production of IL-3 has been reported 1 week after NB infection, we suggest that IL-3 may induce the migration of CFU-mast from the blood to the intestinal mucosa. SCF may also play a role for the migration of CFU-mast. Mesenchymal cells are known to produce SCF. Longley et al. demonstrated the association of SCF with keratinocytes of the human skin. However, the association of SCF with the intestinal epithelium has not been reported. The cellular origin of SCF that may induce the migration of CFU-mast into the NB-infected intestine remains to be investigated. T-cell–derived cytokines such as IL-4, IL-9, and IL-10 enhance mast cell outgrowth in mice, but the effect of these cytokines on rat mast cells has not been reported. As NB infection appears to increase the production of these cytokines, there is a possibility that they may play significant roles for development of mast cells in the intestinal mucosa of NB-infected rats.

In addition to the increase of CFU-mast in the intestinal mucosa, NB infection augmented the proportion of CFU-mast in S phase in the intestinal mucosa. In contrast, the proportion of CFU-mast in S phase did not increase in the bone marrow and peripheral blood after NB infection. This suggests the presence of a local control mechanism in mast-cell differentiation. In fact, the numbers of mast cells increased in the intestinal mucosa, liver, and lung, but not in the skin, after NB infection. There is a possibility that the concentration of IL-3 and other T-cell–derived cytokines may be high enough only at the limited sites even after NB infection.

CFU-mast were not detectable in the small intestine of Ws/Ws rats even after NB infection. This does not appear to be consistent with our previous result that mucosal-type mast cells developed in the small intestine of the NB-infected Ws/Ws rats. Some CFU-mast of Ws/Ws rats may differentiate into mucosal-type mast cells after the invasion into the small intestine, but they appear to lose the colony-forming potential soon after the invasion. The discrepancy between differentiation and proliferation potentials remains to be investigated.

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ACKNOWLEDGMENT

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Infection with Nippostrongylus brasiliensis induces invasion of mast cell precursors from peripheral blood to small intestine

T Kasugai, H Tei, M Okada, S Hirota, M Morimoto, M Yamada, A Nakama, N Arizono and Y Kitamura