In the United States, ovarian cancer ranks fifth as a cause of cancer-related deaths among females. In fact, it has the highest mortality rate among gynecologic tumors. Unfortunately, the majority of cases are diagnosed at an advanced stage. Despite optimal cytoreductive surgery and primary chemotherapy, 5-year survival rates of patients with advanced ovarian cancer remain <30% (1, 2). Therefore, novel approaches to treatment remain essential for effective, durable therapy.

Recent studies have focused on the role of adaptive immunity in epithelial ovarian cancer (3, 4). In fact, the presence or absence of specific T cell subsets has been correlated to prognosis in epithelial ovarian cancer (3, 4). In addition, to examine specificity, we analyzed whether ascites fluid could influence the activation of classic CD8+ T cells.

Experimental Design: To investigate the effects of ascites on NKT cell activation, we pretreated CD1d-expressing cells with the ascites and measured their ability to stimulate cytokine production in NKT cells.

Results: Pretreatment of CD1d-expressing cells with ascites from the majority of patients inhibited the ability of the cells to stimulate/activate NKT cells in a dose-dependent manner. Ascites treatment also partially blocked the ability of α-galactosylceramide–loaded CD1d-immunoglobulin–based artificial antigen-presenting cells (aAPC) to activate NKT cells. In addition, our data show that treatment with ascites does not inhibit HLA-A2–mediated activation of classic CD8+ T cells.

Conclusions: Together, these data suggest that ovarian and other cancers may have developed immune evasion mechanisms specifically targeting the CD1/NKT cell system.

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**Translational Relevance**

Natural killer T (NKT) cells are a unique subset of T cells that display markers characteristic of both natural killer (NK) and T cells. Circulating numbers of NKT cells are markedly decreased in patients with melanoma, prostate cancer, and numerous other cancers. In fact, activation of these cells through treatment with a synthetic glycolipid is currently being used in clinical trials. However, it is thought that the antitumor effects mediated by these cells are being hampered because the number and function of NKT cells are reduced in cancer patients. Tumor-associated glycolipids are frequently shed into the tumor microenvironment and can mediate immunosuppressive activity. These data suggest that one mechanism that tumors may have developed to evade immune detection is by shedding inhibitory lipids. In this study we found that ascites treatment specifically inhibits NKT cell activation. This study should provide a better understanding of NKT cell biology in terms of its role in anticancer immunity. This work has the potential to affect the development of new tests correlating NKT cell biology with patient outcomes, and may result in novel treatments that prevent disease progression.

(16). However, whereas numerous studies have examined the role of NKT cells in cancer in general, the specific role of these cells in the pathogenesis of ovarian cancer has yet to be elucidated.

It has been reported that ovarian malignancies may alter the local environment resulting in a suppression of the immune system (17). Many advanced ovarian cancer patients have a local accumulation of fluid called ascites that contains cellular components of the immune system such as lymphocytes and regulatory factors such as cytokines. Moreover, patients with advanced ovarian cancer have been reported to have higher levels of gangliosides in their plasma and ascites compared with the plasma samples of controls (18). Because NKT cells have been reported to recognize gangliosides in the context of CD1d (19, 20), we investigated whether ascites treatment of CD1d-expressing cells could alter their ability to activate NKT cells.

In addition, our group has developed noncellular, bead-based, artificial antigen presenting cells (aAPC), made by coupling HLA-immunoglobulin, along with other costimulatory molecules, onto magnetic beads. These aAPC have been shown to effectively expand cytomegalovirus (CMV)- and MART-1–specific CTL (21). In this study we developed CD1d-immunoglobulin–based aAPC. It has been reported that soluble forms of CD1d molecules loaded with lipid antigen are directly able to activate NKT cells (22–24). Because soluble forms of CD1d molecules loaded with lipid antigen can activate NKT cells (25), we investigated whether ascites treatment of CD1d-expressing cells could alter their ability to activate NKT cells.

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In this study, we sought to determine if treatment with ovarian cancer–associated ascites altered CD1d-mediated activation of NKT cells. Here, we report that treatment of CD1d-expressing cells with ascites from ovarian cancer patients and others abrogated their ability to activate both canonical and noncanonical NKT cells. This is the first report that ascites specifically inhibits the CD1d/NKT cell system.

**Materials and Methods**

**Patient samples.** Ovarian cancer–associated ascites and peripheral blood mononuclear cells were collected by the Kelly Gynecologic Oncology Service at Johns Hopkins Medical Institutions from patients undergoing primary cytoreductive surgery. Ascites was also collected from patients with hepatitis. All donors gave written informed consent before enrolling in the study. The Institutional Review Board of Johns Hopkins Medical Institutions approved this investigation.

**Cell lines.** Murine L cells transfected with vector alone or the wild-type cd1d1 cDNA in pcDNA3.1-neo (Invitrogen; CD1dwt) were kindly provided by R.R. Brutkiewicz (Indiana University School of Medicine, Indianapolis, Indiana; ref. 25), and were cultured in DMEM, supplemented with 2 mmol/L L-glutamine (BioWhittaker). 10% fetal bovine serum (FBS; HyClone), and ciprofloxacin (Serologicals Proteins). TAP (transporter associated with antigen processing)-deficient 17C4EM.T2 (T2) cells were maintained in Iscove’s modified Dulbecco’s medium, with the same supplements described above.

**NKT cells.** The Vo14+ NKT cell hybridoma cell lines DN32.D3 (26, 27), N38-2C12, N38-2H4, N38-3C3, and the CD1d1-specific NKT cell hybridoma N37-1A12 (Vo5+) have all been described (28) and were cultured in Iscove’s modified Dulbecco’s medium supplemented with 5% FBS and 2 mmol/L L-glutamine. For primary mouse NKT cells, liver mononuclear cells were isolated as described previously (29), then the cells were stained with antibodies against CD3 and NK1.1 (Pharmingen), and sorted using a FACSaria. Primary human NKT cells were isolated from peripheral blood mononuclear cells using the CD3+CD56+ isolation kit (Miltenyi).

**Generation of aAPC.** The preparation of CD1d-immunoglobulin–based aAPC was done according to the previously described method (21). Briefly, to conjugate hCD1d-immunoglobulin dimer molecules to beads, 50 μg of hCD1d-immunoglobulin (Pharmingen) were added to 0.5 mL of epoxy beads (Dynabeads, M-450, Exopyo, 4 × 108 beads/mL; Dynal) in sterile 0.1 mol/L borate buffer, pH 7.0 to 7.4. The bead protein mixture was mixed with rotation and incubated for 24 h at 4 °C. Then the beads were washed and the hCD1d molecules were loaded with lipid antigen-galactosylceramide (α-GalCer; 5 μg/mL in 1 mL PBS containing 5 × 108 beads; Axxora, LLC).

**Generation of CMV-specific CTL.** CMV-specific CTL were generated using peptide-pulsed HLA-A2-immunoglobulin–based aAPC, as previously described (21).

**Treatment of cells with tumor associated ascites.** The ascites were cleared of cellular debris by centrifugation at 250 × g for 10 min. The clarified supernatants were then stored at -20 °C. For experiments, the supernatants were thawed at 4 °C overnight, then L-vector, L-CD1d1wt, CMV-peptide pulsed T2 cells, or aAPC were incubated with ascites (2.5 × 105 cells per mL of ascites) for 4 h at 37 °C. The cells were subsequently washed thrice with 10 mL PBS, resuspended in Iscove’s modified Dulbecco’s medium, supplemented with 5% FBS and 2 mmol/L L-glutamine and cocultured with or without the indicated CD1d-immunoglobulin for 20 to 24 h at 37 °C. The cocultures with the primary T and NKT cells were incubated for 72 or 48 h, respectively. Cytokine release [interleukin-2 (IL-2), granulocyte macrophage colony-stimulating factor, or IFN-γ] was measured as an indication of NKT/T cell activation and was measured by standard sandwich ELISA.

**Results**

Pretreatment with tumor associated ascites inhibits CD1d-mediated activation of NKT cells. Ascites from a panel of ovarian cancer patients was used to treat CD1d expressing cells...
CD1d-mediated NKT cell activation.

clearly establish if nonmalignant ascites can regularly alter activation; therefore, it will require many more samples to ascertain if ascites can have an effect on CD1d-mediated NKT cell function. As shown in Table 1, we found that half of the ascites tested, as shown with samples OC-60 and OC-63, also contained immunosuppressive properties. Thus, we observed inhibitory effect is limited to the ascites or if it is a more global effect, we analyzed the inhibitory capacity of ascites obtained portal hypertension ascites from patients with hepatitis C. As shown in Fig. 2, pretreatment with serum does not inhibit CD1d-mediated antigen presentation to both canonical (Vα14+) DN32.D3, N38-3C3, N38-2C12, and N38-2H4, and noncanonical (Vα5+) N37-1A12 NKT cell hybridomas was inhibited by pretreatment with ascites (Fig. 1A).

Next, we sought to examine whether nonmalignant ascites also contained immunosuppressive properties. Thus, we obtained portal hypertension ascites from patients with hepatitis C. As shown in Table 1, we found that half of the ascites (3 of 6) did not inhibit the ability of the CD1d-expressing cells to stimulate the NKT cells. In fact, treatment with only one sample from the patients with hepatitis C resulted in >50% inhibition. Treatment with nonmalignant ascites can have an effect on CD1d-mediated NKT cell activation; therefore, it will require many more samples to clearly establish if nonmalignant ascites can regularly alter CD1d-mediated NKT cell activation.

To examine the specificity of inhibition, we analyzed the ability of NKT cells to produce other cytokines. NKT cell hybridomas have been reported to secrete granulocyte macrophage colony-stimulating factors (30), which is important for their maturation and effector functions (31, 32). We found that production of granulocyte macrophage colony-stimulating factor was also suppressed by the majority of the ascites samples tested, as shown with samples OC-60 and OC-63 (Fig. 1B). Ascites treatment of stimulatory cells not only inhibited NKT hybridoma cell lines, but even more importantly also inhibited the activation of primary mouse NKT cells (Fig. 1C) and primary human NKT cells (Fig. 1D). Taken together, these data show that ovarian cancer–associated ascites can profoundly affect the ability of APC to activate NKT cells.

Pretreatment with serum does not inhibit CD1d-mediated activation of NKT cells. Numerous reports have shown that cancer patients have a reduction in NKT cell number and function. As we have found that pretreatment with ovarian cancer–associated–ascites can reduce the stimulatory capacity of CD1d molecules, it is possible that this effect is not limited locally to the ascites. Therefore, to further analyze if the observed inhibitory effect is limited to the ascites or if it is a more global effect, we analyzed the inhibitory capacity of matched serum samples. As shown in Fig. 2, pretreatment with serum from patients or controls (human AB serum and FBS) (LCD1dwt), and the effect on CD1d-mediated antigen presentation was assessed (Table 1). Following treatment with ascites, LCD1dwt cells were washed extensively and cocultured with NKT cells. We evaluated the ability of the pretreated CD1d-expressing cells to stimulate the NKT cells by measuring the IL-2 released in the coculture supernatants by ELISA. We found that cytokine production was reduced by 10% to >95% following treatment with the majority of ascites samples examined, which is important for their maturation and effector functions (31, 32). We found that........................................................................................................................................................................

Table 1. Panel of ascites samples

<table>
<thead>
<tr>
<th>Code</th>
<th>Diagnosis</th>
<th>Histology</th>
<th>DN32.D3*</th>
<th>N37-1A12*</th>
<th>N38-2C12*</th>
</tr>
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<tr>
<td>OC-4</td>
<td>Ovarian cancer</td>
<td>High-grade serous carcinoma</td>
<td>-</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td>OC-6</td>
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<td>High-grade serous carcinoma</td>
<td>+</td>
<td>+</td>
<td>-</td>
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<td>High-grade serous carcinoma</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>OC-23</td>
<td>Ovarian cancer</td>
<td>High-grade serous carcinoma</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>OC-34</td>
<td>Ovarian cancer</td>
<td>High-grade clear cell carcinoma</td>
<td>+</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td>OC-36</td>
<td>Fallopian tube carcinoma</td>
<td>High-grade serous carcinoma</td>
<td>+</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td>OC-37</td>
<td>Ovarian cancer</td>
<td>Mixed clear cell and low grade serous carcinoma</td>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>OC-38</td>
<td>Fallopian tube carcinoma</td>
<td>Moderately differentiated serous carcinoma</td>
<td>+</td>
<td>+</td>
<td>++++</td>
</tr>
<tr>
<td>OC-40</td>
<td>Ovarian cancer</td>
<td>High-grade serous and clear cell carcinoma</td>
<td>+</td>
<td>+</td>
<td>++++</td>
</tr>
<tr>
<td>OC-46</td>
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<td>Atypical proliferative (borderline) serous tumor</td>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>OC-47</td>
<td>Ovarian cancer</td>
<td>MMMT, carcinosarcoma</td>
<td>+</td>
<td>+</td>
<td>++++</td>
</tr>
<tr>
<td>OC-48</td>
<td>Ovarian cancer</td>
<td>Well-differentiated endometrioid carcinoma</td>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>OC-55</td>
<td>Ovarian cancer</td>
<td>High-grade serous carcinoma</td>
<td>+</td>
<td>+</td>
<td>++++</td>
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<td>Primary peritoneal cancer</td>
<td>High-grade serous carcinoma</td>
<td>+</td>
<td>+</td>
<td>++++</td>
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<tr>
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<td>Ovarian cancer</td>
<td>High-grade serous carcinoma</td>
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<td>N.D.</td>
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<tr>
<td>OC-59</td>
<td>Ovarian cancer</td>
<td>High-grade serous carcinoma</td>
<td>+</td>
<td>+</td>
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<td>OC-60</td>
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<td>+</td>
<td>+</td>
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<tr>
<td>OC-62</td>
<td>Ovarian cancer</td>
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<td>-</td>
<td>+</td>
<td>N.D.</td>
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<tr>
<td>OC-63</td>
<td>Ovarian cancer</td>
<td>High-grade adenocarcinoma</td>
<td>+</td>
<td>+</td>
<td>N.D.</td>
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<tr>
<td>OC-64</td>
<td>Uterine cancer</td>
<td>High-grade serous carcinoma</td>
<td>+</td>
<td>+</td>
<td>N.D.</td>
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<tr>
<td>OC-65</td>
<td>Ovarian cancer</td>
<td>High-grade serous carcinoma</td>
<td>+</td>
<td>+</td>
<td>N.D.</td>
</tr>
<tr>
<td>OC-66</td>
<td>Ovarian cancer</td>
<td>High-grade serous carcinoma</td>
<td>+</td>
<td>+</td>
<td>N.D.</td>
</tr>
<tr>
<td>OC-67</td>
<td>Ovarian cancer</td>
<td>High-grade serous carcinoma</td>
<td>+</td>
<td>+</td>
<td>N.D.</td>
</tr>
<tr>
<td>OC-68</td>
<td>Ovarian cancer</td>
<td>High-grade endometrioid carcinoma</td>
<td>+</td>
<td>+</td>
<td>N.D.</td>
</tr>
<tr>
<td>OC-69</td>
<td>Ovarian cancer</td>
<td>High-grade serous carcinoma</td>
<td>+</td>
<td>+</td>
<td>N.D.</td>
</tr>
<tr>
<td>LP3</td>
<td>Hepatitis C</td>
<td>-</td>
<td>N.D.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LP4</td>
<td>Hepatitis C</td>
<td>+</td>
<td>N.D.</td>
<td></td>
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</tr>
<tr>
<td>LP5</td>
<td>Hepatitis C</td>
<td>-</td>
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<td>LP6</td>
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<td>N.D.</td>
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</tr>
<tr>
<td>LP7</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>LP8</td>
<td>Hepatitis C</td>
<td>+</td>
<td>N.D.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviation: N.D., not determined.

*Percent maximum inhibition of IL-2 production is shown as follows: -, no inhibition; +, <10%; ++, 10 to 30%; ++++, 31 to 50%; and +++++, >51%.
did not result in decreased stimulation/activation of NKT cells. Moreover, we found that serum pretreatment in some cases even augmented the ability of the APC to induce cytokine production from the NKT cells.

**Tumor-associated ascites inhibits presentation of α-GalCer by aAPC.** We developed bead-based CD1d-expressing aAPC (Fig. 3A). The system is based on directly coupling CD1d-immunoglobulin to magnetic beads and using them to stimulate the NKT cell hybridomas. Using this cell-free tool we can load CD1d-aAPC with α-GalCer, a potent stimulator of NKT cells, and determine if antigen processing or editing is necessary for the ascites-mediated inhibition. After establishing the system, the α-GalCer–loaded aAPC were treated with media or ascites for 4 hours, washed extensively, and cocultured with the NKT cell hybridomas, DN32.D3, N38-2H4; primary mouse NKT cells (C); or primary human NKT cells (D). Recognition of CD1d was assessed by measuring cytokine production in the supernatants by ELISA. NKT cells were cocultured in media alone or with L cells transfected with vector alone as controls. Spontaneous cytokine release and background levels of cytokine production (as measured by coculturing NKT cells with L cells containing vector alone) were subtracted before calculating percent inhibition.

---

**Fig. 1.** Treatment with ascites from ovarian cancer patients inhibits CD1d-mediated antigen presentation. A, LCD1dwt cells were treated with media or ascites from the indicated patients for 4 h, washed extensively, and then cocultured with a panel of NKT cell hybridomas, DN32.D3, N37-1A12, N38-3C3, N38-2C12, and N38-2H4. IL-2 was measured by standard cytokine ELISA, and data are shown as percent inhibition, normalized to cells treated with culture media as 100% or the maximum level of stimulation. The results are representative of more than 15 experiments, in which samples from 3 to 12 patients were tested in each experiment and each sample repeated at least twice. B, the ascites-treated LCD1dwt cells were cocultured with the Vs14+ NKT cell hybridomas, DN32.D3, N38-2H4; primary mouse NKT cells (C); or primary human NKT cells (D). Recognition of CD1d was assessed by measuring cytokine production in the supernatants by ELISA. NKT cells were cocultured in media alone or with L cells transfected with vector alone as controls. Spontaneous cytokine release and background levels of cytokine production (as measured by coculturing NKT cells with L cells containing vector alone) were subtracted before calculating percent inhibition.

---

**Fig. 2.** Treatment with serum from ovarian cancer patient does not inhibit CD1d-mediated antigen presentation. LCD1dwt cells were treated with 1 mL media containing 5% FBS (Media), FBS, human AB serum (ABS), or serum from the indicated patients for 3 to 4 h, washed extensively, and then cocultured with N37-1A12 and N38-3C3 NKT-cell hybridomas. IL-2 was measured by standard cytokine ELISA, and data are shown as percent control, normalized to cells treated with culture media as 100% or the maximum level of stimulation.

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**Ascites Blocks CD1d-Mediated NKT Cell Activation**
cells. Taken together, these data show that inhibition of antigen presentation by tumor associated–ascites is CD1d specific.

Pretreatment with ascites results in a dose-dependent inhibition of NKT cell activation. Further analysis of the ability of ascites to mediate inhibition of antigen presentation by CD1d was necessary to characterize the effect. We diluted the ascites with cell culture media and treated the LCD1dwt stimulator cells for 4 hours, washed them extensively, and cocultured them with NKT cells as described above. As shown in Fig. 5A to D, increasing concentrations of ascites resulted in a concomitant reduction in NKT cell activation, as measured by cytokine release, in all NKT cell hybridomas examined. Even when the APC were treated with 25% ascites fluid or less, the blocking effect on NKT cell activation was still observed. Although all of the NKT cell lines are CD1d-restricted, they vary in their antigen specificity. Namely, the noncanonical NKT cell hybridoma N37-1A12 does not recognize α-GalCer in the context of CD1d molecules; its activation, however, is similarly reduced in a dose-dependent manner. Interestingly, the dose curves are markedly different for each cell line. In addition, the ascites was passed through 40-μm filters to further characterize its inhibitory effects. There was no change its ability to block CD1d-mediated activation of NKT cells in vitro. This inhibition resulted in decreased production of multiple cytokines, IL-2, granulocyte macrophage colony-stimulating factor (in murine NKT cells; Fig. 1A-C) and IFN-γ (in primary human NKT cells; Fig. 1D). Interestingly, we found that this effect was limited to the ascites, as it was not found in the following treatment with serum-matched samples from the same patients (Fig. 2).

Ascites treatment of α-GalCer–loaded aAPC impaired their ability to activate NKT cells, which further suggests that the inhibition is independent of antigen processing (Fig. 3). Notably, in this study we used human CD1d-immunoglobulin dimers to generate aAPC. Thus, our study shows an inhibitory effect of ascites fluid on both mouse CD1d molecules (expressed on LCD1dwt cells) and human CD1d molecules (immobilized on aAPC). Moreover, because α-GalCer has been well-characterized as a high-affinity ligand and potent stimulator of NKT cells, the level of inhibition observed following treatment with ovarian cancer associated–ascites suggests that the putative inhibitory ligand has a very high affinity for CD1d.

Other groups have implicated a role for the shedding of glycolipids as a method of tumor cell evasion from immune detection (33). Whereas inhibition of CD1d-mediated antigen presentation by a glycosphingolipid (gangliotriaosylceramide) shedding from a murine lymphoma has been previously reported (25), the inhibition mediated by gangliotriosylceramide was specific to the invariant, canonical NKT cell

Discussion

In this study we have found that treatment of CD1d-expressing stimulator cells with ovarian cancer associated–ascites inhibited CD1d-mediated activation of NKT cells in vitro. This inhibition resulted in decreased production of multiple cytokines, IL-2, granulocyte macrophage colony-stimulating factor (in murine NKT cells; Fig. 1A-C) and IFN-γ (in primary human NKT cells; Fig. 1D). Interestingly, we found that this effect was limited to the ascites, as it was not found in the following treatment with serum-matched samples from the same patients (Fig. 2).

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population. In our study we have found dose-dependent inhibition of CD1d-mediated antigen presentation to both canonical and noncanonical NKT cells following pretreatment with ovarian tumor-associated ascites (Fig. 5A-D). Whereas CD1d molecules are known to traffic through endocytic compartments to present endogenous antigen to canonical NKT cells (34), presentation to noncanonical NKT cells (i.e., N37-1A12) is not dependent on antigen processing (35); this study suggests that treatment with ascites does not interfere with antigen processing, but rather may result in alterations in the expression of functional antigen-CD1d complexes on the cell surface. Our data using the α-GalCer–loaded aAPC (Fig. 3B) strongly support this hypothesis. Moreover, our findings in Fig. 5A to D are intriguing because the dose curves are markedly different for each NKT cell line examined. These data suggest that the discovery of natural ligands for NKT cells may reveal subtle differences in their antigen specificities that are not obvious with the potent agonist, α-GalCer.

Furthermore, the α-GalCer–loaded aAPC data (Fig. 3B), along with experiments showing that antigen presentation by HLA-A2 to classic T cells was not affected (Fig. 4), confirm that the reduction in antigen presentation is not due to the overall toxicity of the ascites or death of the treated stimulator cells. It is known that ovarian cancer patient ascites is rich in gangliosides (18), some of which have been previously shown to be presented by CD1d molecules (19, 20). We hypothesize that the mechanism of inhibition is the result of the binding of one or more gangliosides to the hydrophobic groove of CD1d, which blocks, covers, or competes with the binding of the endogenous ligand. This would explain the reduction in antigen presentation to all NKT cells examined (but not classic CTL) and account for the reversal of NKT cell activation using ascites-treated α-GalCer–loaded aAPC (Fig. 3B). It will be interesting to determine if ovarian cancer associated–ascites contains a specific family of gangliosides which mediate the inhibitory effects observed. Studies to identify the described inhibitory agent in the ascites are currently ongoing in the laboratory.

To our knowledge this is the first report showing that human tumor ascites specifically suppresses the CD1d/NKT system. Here, we present evidence which show that ascites, from cancer patients and possibly others, contain inhibitory substances (conceivably lipid antigens) that block CD1d-mediated activation of NKT cells. Thus, the presence of activated NKT cells may be a critical prognostic factor for ovarian cancer and more importantly, restoration of their function could be an effective therapeutic strategy.

Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

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References


Ascites Specific Inhibition of CD1d-Mediated Activation of Natural Killer T Cells

Tonya J. Webb, Robert L. Giuntoli II, Ophelia Rogers, et al.


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