Normal salivary gland tissue has been recognized to positively stain for PSA, independently of gender, while immunostaining for AR has not been consistently found [6, 7]. Immunostaining for AR, and occasionally for prostatic acid phosphatase and PSA, has been reported in duct carcinoma and adenocarcinoma of salivary glands in both sexes [3–6, 8]. These findings suggest that, unlike prostate cancer, PSA expression could be independent of androgen stimulation in normal salivary gland tissue. However, the biological significance of PSA and AR expression in normal tissue and in SGCs, respectively, remains unknown.

In prostatic cancer, androgens play a role as both a survival and growth factor and androgen-deprivation therapy is successfully used.

Our experience suggests that a similar mechanism may be implicated in AR-positive salivary gland tumors. Studies looking at the underlying biological role of AR expression in SGCs are warranted.

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Successfully treated carcinoma erysipeloides from gastric cancer

In August 2000, a 59-year-old woman underwent a total gastrectomy for a poorly differentiated invasive adenocarcinoma (T2N2M0). After an uneventful postoperative recovery, she was discharged and followed up in the outpatient clinic. She presented with red-purple patches on the left supraclavicular region 2 years after surgery. Profuse granulation tissues extending around the lesions appeared. Skin biopsies showed massive and extensive infiltration of the dermis by clusters of poorly differentiated adenocarcinoma cells. Although the patient received palliative radiotherapy (46 Gy) and intravenous chemotherapy consisting of 5-fluorouracil (5-FU) and mitomycin, the skin lesions rapidly became exacerbated. The extensive lesions had irregular margins lacking clear borders with invasive erythematous plaques and profuse granulation tissues with bleeding, covering the entire left side of the neck extending to the infraclavicular area (Figure 1A).

The patient required hospitalization to change the dressings of her left chest tumors and morphine sulfate (50 mg/day) was required.

Figure 1. (A) Carcinoma erysipeloides, covering the entire left side of the neck extending to the infraclavicular area. (B) Six weeks after chemotherapy, the tumors dried and scab formation is seen.
to control the severe pain in the left chest wall. The patient received oral administration of 80 mg/day of 1 M tegafur–0.4M 5-chloro-2,4-dihydroxypyridine–1 M potassium oxonate (S-1) for 4 weeks as a final line chemotherapy because of hematological toxicity resulting from the previous chemotherapy. Six weeks later, the lesions dried, diminished remarkably in size and showed scab formation (Figure 1B). The left chest pain disappeared, and the administration of morphine sulfate became unnecessary. She was discharged, and is presently enjoying a normal life.

Metastasis to the skin is a comparatively rare complication of internal malignancies [1]. The clinical presentations appear as flesh-colored to red-purple or brownish solitary papules or nodules. Although carcinoma erysipeloides are frequently observed in patients affected by breast carcinoma, they are rarely observed during the course of other malignant tumors. These metastases are the clinical representation of the rapid spread of tumor cells along subepidermal lymphatic vessels. As a consequence of lymphatic blockage, edema, erythema and vesicles can appear [2]. Although both the appearance of tumors from gastric cancer and their successful treatment are very rare, S-1 was an effective anticancer drug in the present case.

S-1 is a newly developed oral anticancer drug, which consists of tegafur, gimeracil and oteracil potassium at a molecular ratio of 1:0.4:1, based on the biochemical modulation of 5-FU [3]. S-1 was more effective for lymph node metastasis than for the primary lesion, lung metastasis or liver metastasis in a phase II study [4]. Moreover, this oral anticancer drug has the advantage of not requiring hospitalization for patients with good performance status, because of its mild toxicity. The prognosis of gastric cancer with skin metastasis is generally poor, thus treatment preserving the quality of life of cancer patients should be considered positively.

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5. Dihydropyrimidine dehydrogenase activity before treatment ranged from 80 to 590 pmol/min/mg protein (mean 280, median 270). This range of DPD distribution, as well as the mean and median values, compare well to previous population studies of PBMC DPD activity in cancer patients [6]. PBMC DPD activity significantly decreased after platinum administration (Figure 1; paired-sample t-test, P = 0.010): the mean decrease in PBMC DPD activity was 73 pmol/min/mg protein (95% confidence interval 13.7–93.09). DPD decreased in 15/22 patients (68%; six out of nine patients with OXA and nine out of 13 patients with cisplatin). The DPD decrease was not related to the type of platinum complex administered (ANOVA, P = 0.60). In five patients, it was possible to perform a follow-up of DPD during the treatment course (four cycles of cisplatin–5-FU). All five had a DPD decrease that was maintained during this observation period.

These clinically-based observations concur with previous pharmacokinetic observations [4] and may have several pharmacological and clinical implications. First, they point to a

Impact of platinum complexes on dihydropyrimidine dehydrogenase activity in 5-fluorouracil-treated patients

The association cisplatin–5-fluorouracil (5-FU) in head and neck cancer [1], and the more recent combination between oxaliplatin (OXA) and 5-FU in advanced colorectal cancer [2], have largely proved their clinical efficacy. The origin of the pharmacological interaction between these associated drugs has not been fully elucidated. The enzyme dihydropyrimidine dehydrogenase (DPD) controls the catabolic route of 5-FU and thus influences 5-FU pharmacokinetics and pharmacodynamics [3]. Recent pharmacokinetic studies have suggested that OXA may inhibit 5-FU catabolism. This observation advocates a possible impact of OXA on DPD activity [4]. We recently conducted the following prospective clinical study to evaluate the impact of platinum complexes on DPD activity.

Twenty-two cancer patients who had not received prior chemotherapy treatment (13 head and neck cancer patients and nine advanced colorectal cancer patients) were included (19 men, three women). Mean age was 59 years (range 40–77). Most patients (73%) had a performance status <2. Cisplatin (80–100 mg/m²) in head and neck cancer patients and OXA (60–100 mg/m²) in colorectal cancer patients were given in association with 5-FU as previously described [1, 2]. Two blood samples were taken in the morning between 9.00 and 11.00 a.m., in order to limit the impact of the circadian influence reported for DPD activity; one just before starting platinum complex administration (day 1), and the second 24 h later (i.e. after platinum complexes administration and before starting administration of 5-FU). This was done during the first cycle of treatment. DPD activity was measured in peripheral blood mononuclear cells (PBMC) as previously described [5].