

INFLAMMATORY INFILTRATE IN THE FELINE MAMMARY GLAND: HEALTHY, NON-NEOPLASTIC, BENIGN AND MALIGNANT LESIONS

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Introduction

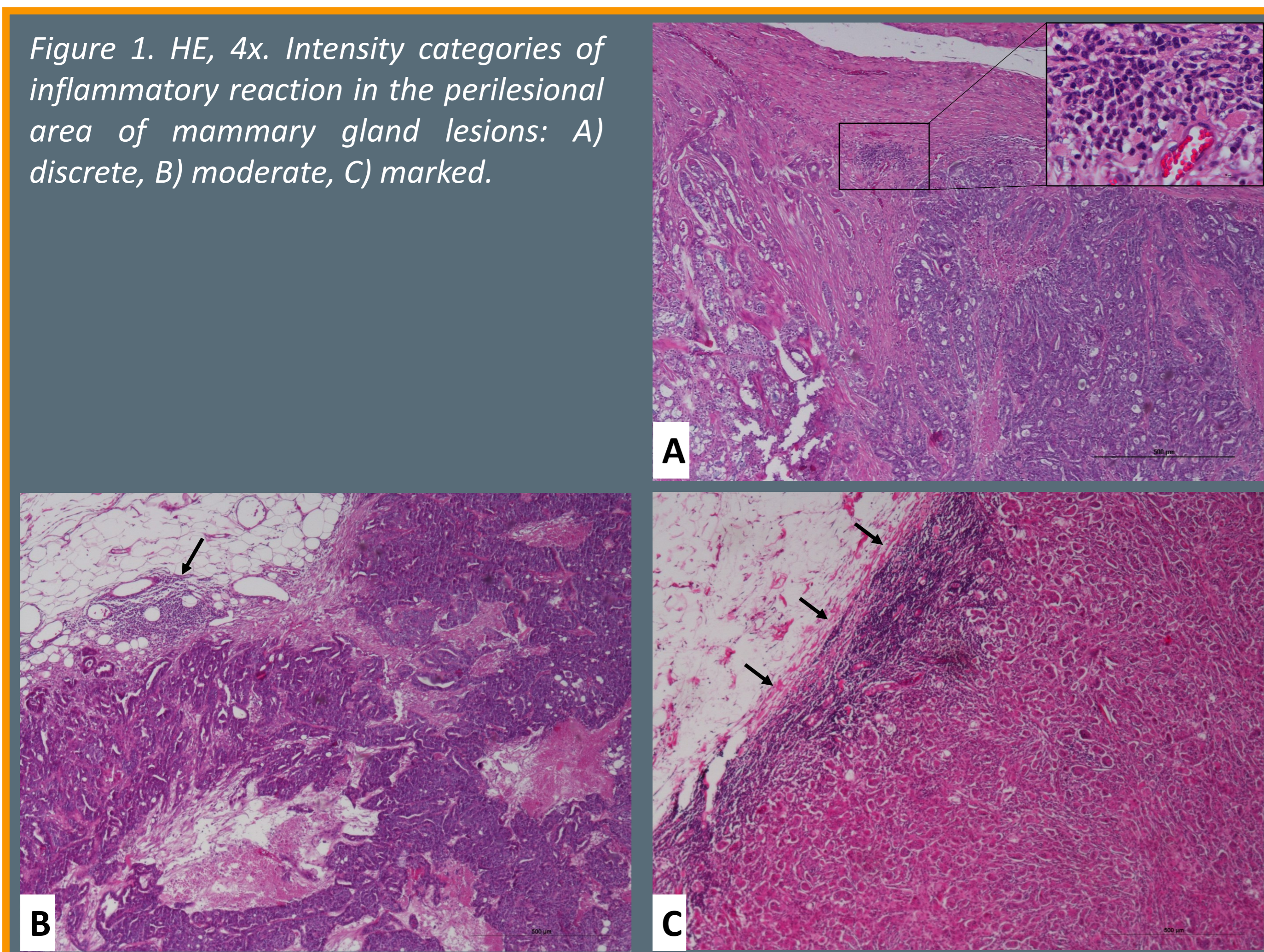
Inflammation is a frequent finding in feline mammary neoplasms. Recent research suggests that tumour-associated inflammation might play a significant part in the clinical outcome of feline mammary carcinomas, taking on a favourable or unfavourable role depending on the subset and location of the immune cells.

Objectives

The present study aimed at an overall assessment of inflammatory infiltrates in healthy, hyperplastic/dysplastic, benign and malignant lesions of the feline mammary gland.

Materials and Methods

Perilesional and intralesional inflammatory foci were evaluated in a whole section of each lesion and categorized according to distribution as: absent, focal (1-3 foci), multifocal (≥ 3 foci) or diffuse. Overall intensity was visually evaluated and recorded as discrete (Figure 1A), moderate (Figure 1B) or marked (Figure 1C). A control group included queens without mammary changes.



Results

The study enrolled 178 queens, encompassing 75 (25.2%) hyperplasias/dysplasias and 223 (74.8%) tumours (8.1% benign; 91.9% malignant). Perilesional inflammation was observed in 268 lesions (89.9%; 72% of non-neoplastic; 72.2% of benign; 98.1% of malignant). Hyperplasias/dysplasias and benign tumours displayed mostly focal to multifocal discrete perilesional inflammatory foci, whereas malignant tumours exhibited multifocal discrete to moderate foci. Most non-neoplastic and malignant lesions (74.6% and 87.8%, respectively) presented intralesional inflammatory infiltrates, while a considerably lower proportion of benign tumours (33.3%) did (Table 1). Inflammatory foci were predominantly focal in hyperplasias/dysplasias and benign tumours, and multifocal in malignant neoplasms. Intralesional immune cells were mostly discrete, irrespective of biological behaviour (Figure 2). The control group comprised 167 individual mammary glands, 28.1% of which presented scant individually disperse periductal/peritubular immune cells.

Figure 2. Intensity of perilesional and intralesional inflammatory infiltrates grouped as non-malignant lesions (hyperplasias/dysplasias and benign tumours) and malignant lesions.

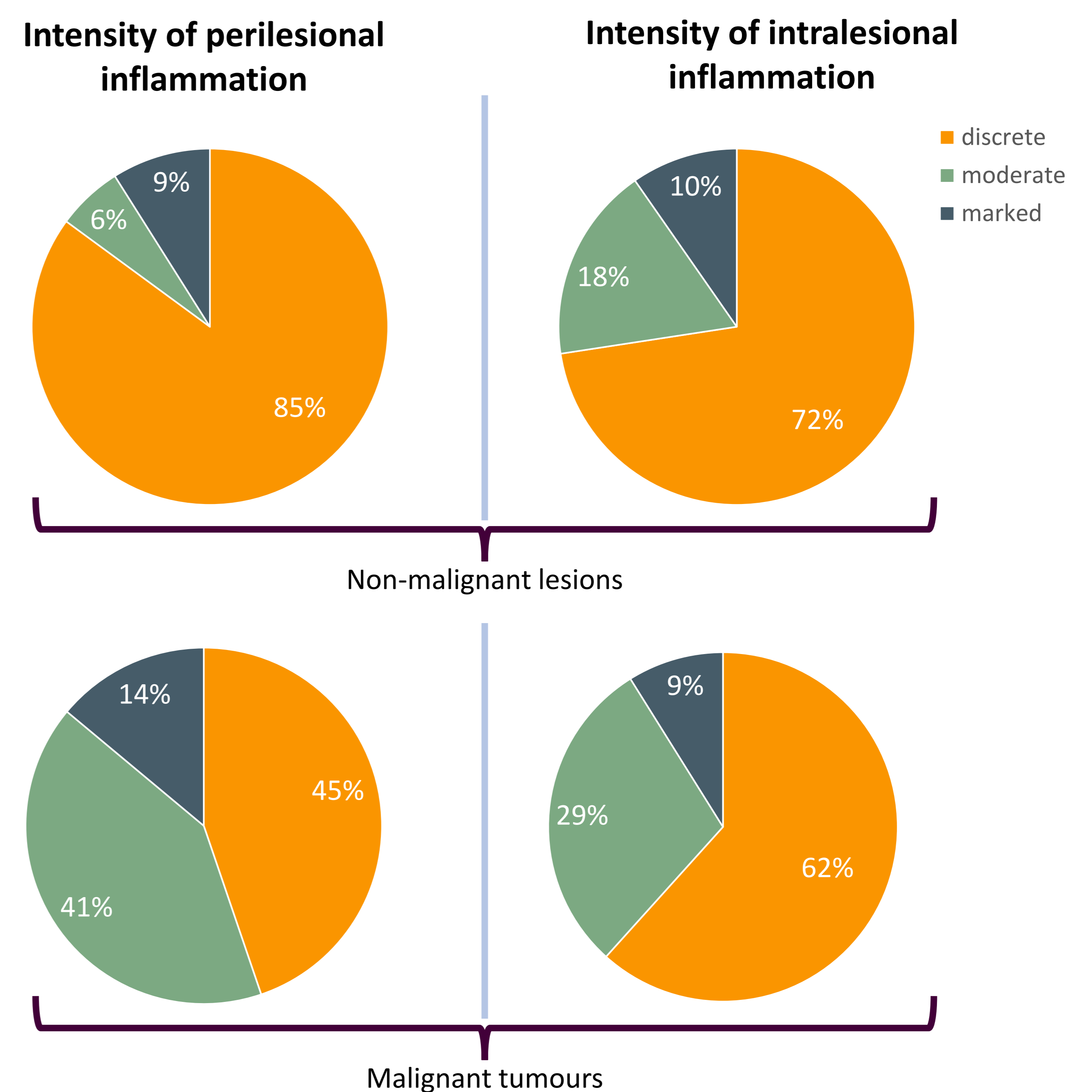


Table 1. Distribution of perilesional and intralesional inflammatory infiltrates grouped according to the biological behaviour of the lesion.

	Distribution			
	Absent	Focal	Multifocal	Diffuse
Perilesional inflammatory infiltrate				
Hyperplasias/dysplasias (n=75)	28%	32%	38,7%	1,3%
Benign tumours (n=18)	27,8%	38,9%	33,3%	-
Malignant tumours (n=205)	2%	9,3%	84,9%	3,9%
Intralesional inflammatory infiltrate				
Hyperplasias/dysplasias (n=75)	25,3%	41,3%	32%	1,3%
Benign tumours (n=18)	66,7%	27,8%	5,6%	-
Malignant tumours (n=205)	12,2%	25,4%	58%	4,4%

Conclusions

Differences observed in the abundance and distribution pattern of inflammatory infiltrate in different types of feline mammary lesions suggest that this component may be an active player in neoplastic transformation and progression in this species.

References

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