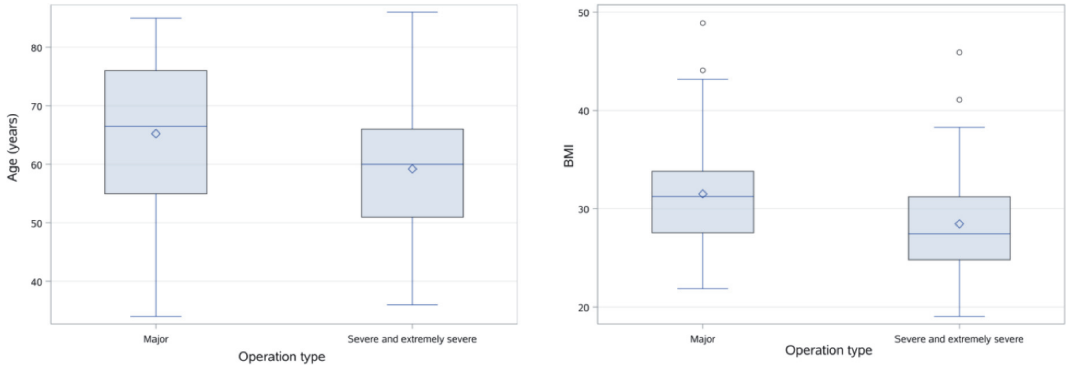
Abstract 245 Table 1 Characteristics of the study parameters, percentages are for the study population (N=97)

Characteristic (Median [range] or %)		Cancer sites		FIGO stage		Surgery type	
Age (years)	62 [34-86]	Endometrium	37%	I	55%	Major (2-3 hours)	52%
BMI (Kgr/m^2)	30 [19-49]	Ovarian	31%	II	11%		
Heart disease issues	51%	Cervical	14%	III	26%	Severe (3-5 hours)	38%
Smoking	31%	Sarcomas	7%	IV	8%		
Alcohol	5%	Vulvar	7%			Extremely severe (5+ hours)	10%
Central venous catheter	31%						



Abstract 245 Figure 1 Box and whisker plots for operation type vs. BMI and women age. Top and bottom of boxes indicate 25th and 75th percentiles, line within boxes indicate median value, diamond symbol indicates mean value and ends of whiskers are for minimum and maximum value (after exclusion of outliers, circles). For both Kruskal-Wallis comparisons: p<0.05.

figure 1). Extended duration surgeries required in FIGO III-IV stages compared to I-II (70%, 53% respectively, OR:2.1, p=0.1366), in ovarian and endometrial cases (73%, 47% respectively, OR:3.1, p=0.0317).

Median thromboprophylaxis duration was 31 days (27–35) and was not related to surgery duration (p>0.05). Three PE events occurred (3%), two in women >70 years and BMI >30 and one in a severe surgery. No bleeding events related to thromboprophylaxis were recorded.

Conclusions Higher BMI limited the possibilities to achieve extended surgical goals while younger age favored it. Intense postoperative thromboprophylaxis with tinzaparin (8,000IU) for 1 month, for women with active gynecological cancer and HTB (high BMI & age, >2 hours surgery, comorbidities, advanced FIGO stage) was effective and safe. Further research is needed.

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246 RESTRAINT STRESS PROMOTES TUMOR GROWTH IN A SYNGENEIC MOUSE MODEL OF OVARIAN CANCER

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There is growing evidence that links chronic stress to ovarian cancer (OC) progression. Cancer diagnosis, chemotherapy, and other traumatic life events can lead to altered psychological states, such as chronic stress. Chronic stress induces sustained activation of the sympathetic nervous system (SNS), releasing stress hormones that modulate physiological responses across different systems, including the immune system. Specifically, chronic SNS activation alters the distribution of T-cells, thus influencing clinical outcomes. The aim of this work was to determine the effects of restraint stress on a syngeneic mouse model of OC and characterize its effects on T-cell responses. We hypothesized that restraint stress would lead to accelerated growth of existing tumors and deregulation T-cell subpopulations, promoting an immunosuppressed tumor microenvironment. To determine the effect of daily restraint stress on tumor progression, we used 8 to 12-week-old female C57/BL6 mice. After three days of daily restraint stress, mice were injected with IG10 or ID8 (1 × 10^6 cells/ 100µL) murine ovarian cancer cells. Mice were sacrificed 8–12 weeks after inoculation. Once sacrificed, tumors were removed and weighted. Our results indicate that daily restraint stress increased OC growth in both IG10/ID8 groups. Moreover, IG10 tumor-bearing mice showed increased ascites production. These results suggest an association between psychological factors and disease progression. Future experiments will assess the effects of chronic stress on T-cell maturation, effector functions, exhaustion, and activation in these samples. Results obtained from this project will support future in vivo experiments regarding the effect of restraint stress and immunotherapy efficacy.