# Algoritmi terapeutici 2026: terapia anticoagulante

## (nuovi) Anticoagulanti per la terapia del TromboEmbolismo (nelle neoplasie ematologiche)

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PROMISE
Università degli Studi di Palermo

Hematology, ASH Education Program ISSUES CME ABOUT

DECODING THE ENIGMA: CHALLENGES IN CLASSICAL HEMATOLOGY CONSULTATIONS | DECEMBER 5, 2025

# Anticoagulation in malignancy: the patient who bleeds and clots simultaneously

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*Hematology Am Soc Hematol Educ Program* (2025) 2025 (1): 176–182.

https://doi.org/10.1182/hematology.2025000703

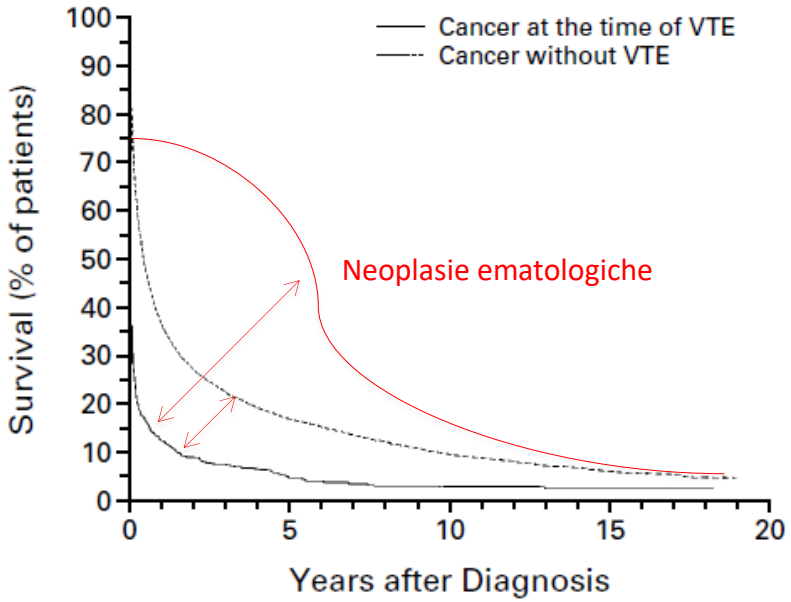
1. **Rilevanza del TEV in (onco)-ematologia**

2. **Attuali e nuove terapie**

# Tumore come (non unica) causa di morte nel paziente oncologico

Circa il 25% del TEV oggi diagnosticato in Italia è correlato a neoplasie

Esso incide sulla sopravvivenza del paziente oncologico




| No. at Risk | | | | |
|---|---|---|---|---|
| Cancer at the time of VTE | 668 | 23 | 10 | 3 |
| Cancer without VTE | 6668 | 913 | 338 | 87 |

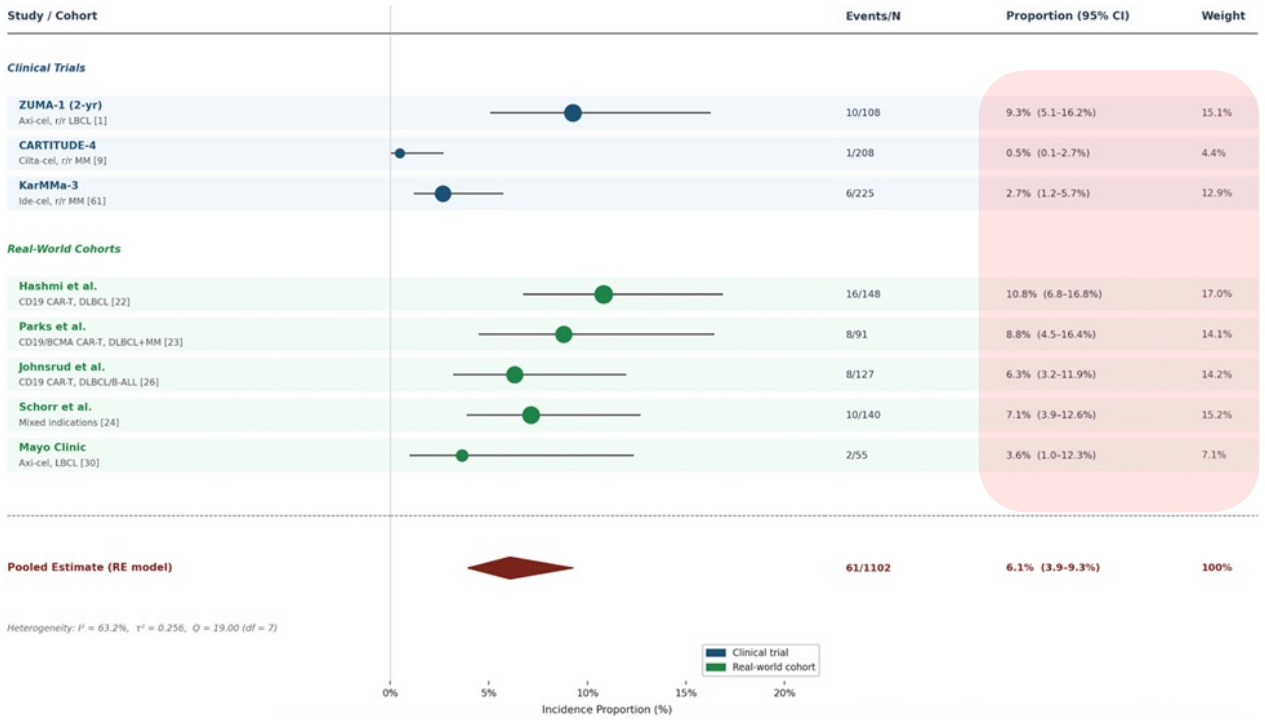


*Hematology Am Soc Hematol Educ Program* (2025) 2025 (1): 80–86.

# Q1. Secondo voi il rischio tromboembolico è sottostimato tra gli oncologi/ematologi?

L'incidenza del tromboembolismo venoso (TEV) nelle neoplasie ematologiche è significativamente più alta rispetto alla popolazione generale, con alcuni studi che riportano un rischio fino a 4 volte superiore o addirittura fino a 7 volte superiore nei pazienti sottoposti a chemioterapia. I linfomi (Hodgkin e non-Hodgkin) hanno un'incidenza del TEV di circa il **10%**. I pazienti con mieloma multiplo, specialmente se trattati con polichemioterapia che include talidomide e steroidi ad alto dosaggio, hanno un rischio elevato, stimato intorno al **28%.** Le LA incidenza pari al **4-11%**

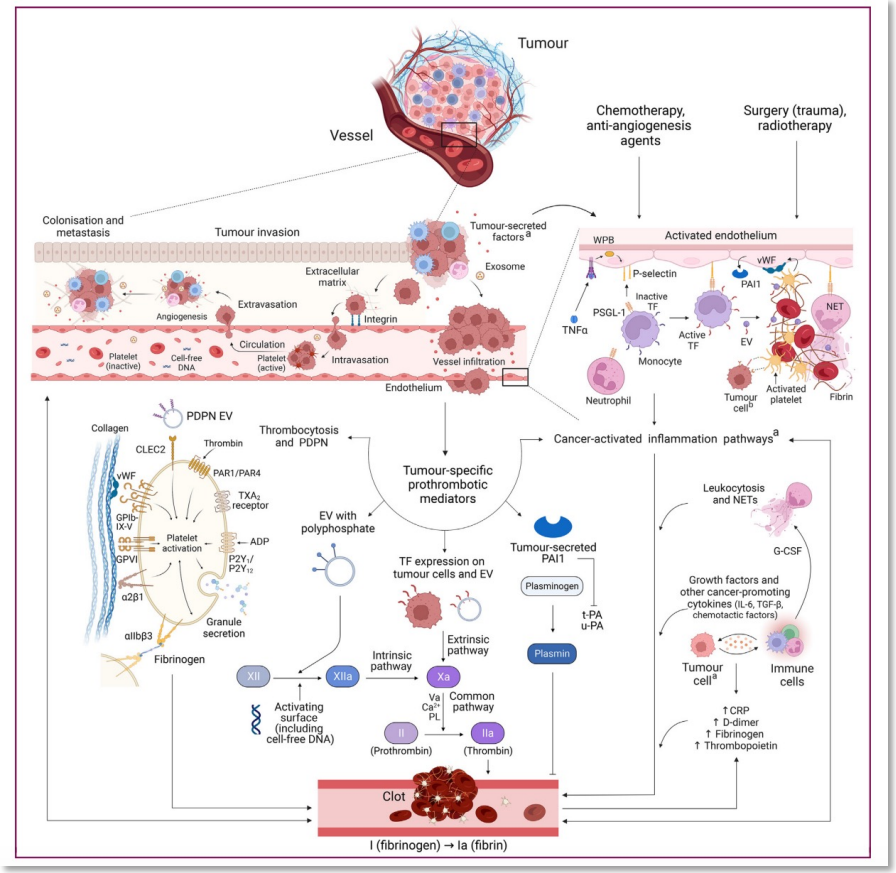
# Thrombotic events in pivotal/registrative CAR-T clinical trials and real life cohorts

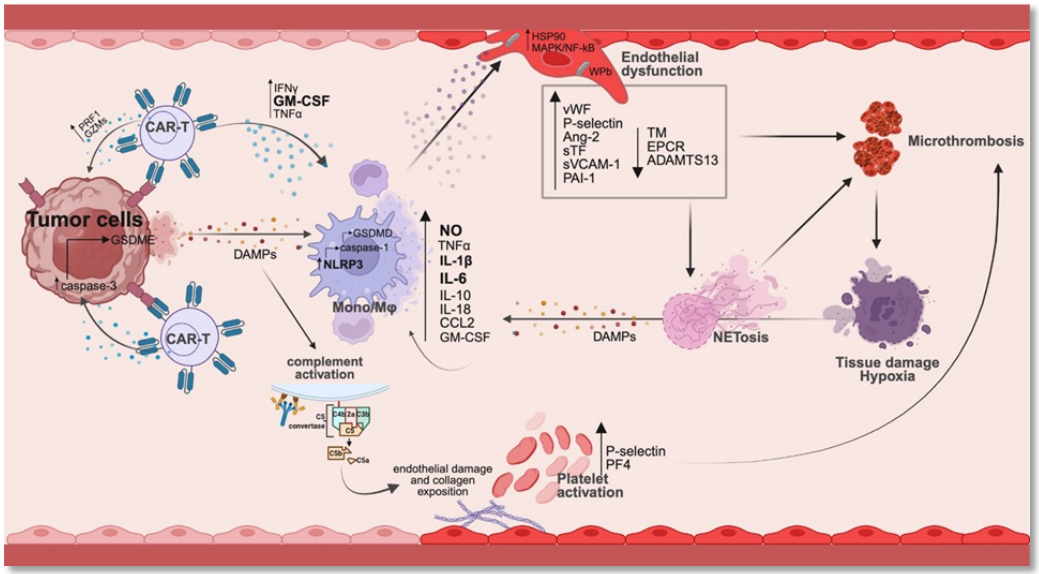



7%

*Maziarz RT, et al. J Clin Oncol 2026;44:86–91.*

# (nuova) Fisiologia della trombosi nel cancro




*Romano F et al. CAR T-cell–associated coagulopathy (CARAC): clinical spectrum, pooled incidence and management. BMT 2026 submitted*

1. Rilevanza del TEV in (onco)-ematologia

2. **Attuali e nuove terapie**

**THERAPEUTIC ANTICOAGULATION FOR VTE (CONTINUED)[§]**

| Agent(s) | Contraindications and Warnings | |
|---|---|---|
| LMWH: Dalteparin and enoxaparin | • Use with caution in patients with renal dysfunction. Consider dose adjustments or alternative therapy for patients with severe renal dysfunction (CrCl <30 mL/min).[j]<br>• Follow package insert for renal dysfunction dosing.<br>• Anti-Xa monitoring (peak and trough) of LMWH has been recommended for patients with severe renal dysfunction, although limited data are available to support the clinical relevance of anti-Xa levels.<br>• Absolute contraindication: recent/acute HIT<br>• Relative contraindication: past history of HIT<br>• Pork product allergy | |
| Fondaparinux | • Contraindicated in patients with CrCl <30 mL/min<br>• Use with caution in patients with moderate renal insufficiency (CrCl 30–50 mL/min), weight <50 kg, or age >75 y | |
| UFH | • Absolute contraindication: recent/acute HIT<br>• Relative contraindication: past history of HIT | |
| Warfarin | Relative contraindications:<br>• Concomitant inhibitors and inducers of CYP2C9, 1A2, or 3A4 | |
| DOACs: Apixaban, dabigatran, edoxaban, and rivaroxaban | Contraindications:<br>• Antiphospholipid syndrome (APS)[k]<br>• Pregnancy or breastfeeding<br>• Stage IV/V chronic kidney disease:<br>▸ Apixaban[l]: CrCl <30 mL/min[4]<br>▸ Dabigatran, edoxaban,[26] rivaroxaban[12]: CrCl <30 mL/min<br>• Active/clinically significant liver disease:<br>▸ Apixaban[6]: Child-Pugh Class B or C or alanine aminotransferase (ALT)/aspartate aminotransferase (AST) >3x upper limit of normal (ULN); total bilirubin >2x ULN<br>▸ Rivaroxaban[12]: Child-Pugh class B or C or ALT/AST >3x ULN<br>▸ Dabigatran[19,27-29]: Child-Pugh class C or ALT/AST >2x ULN or active/acute hepatitis or cirrhosis<br>▸ Edoxaban[8]: Child-Pugh class B or C or AST/ALT >3x ULN and bilirubin >2x ULN, cirrhosis, or active hepatitis<br>▸ Strong dual inhibitors/inducers of CYP3A4 and P-gp: see prescribing information for rivaroxaban and apixaban<br>• Inducers/inhibitors of P-gp: see prescribing information for dabigatran and edoxaban | Relative contraindications, use with caution:<br>• DOACs have been associated with an increased risk of GI and possibly genitourinary tract bleeding, and should be used with caution in patients with genitourinary or GI tract lesions, pathology, or instrumentation.<br>• Use with caution in patients with compromised renal or liver function.<br>• For patients receiving nephrotoxic or hepatotoxic chemotherapy, consider monitoring patients more closely with laboratory testing.<br>• Consider drug-drug interactions. |

Tinzaparina

# LMWH > VKA in the treatment of cancer-associated VTE

LMWH 100 UI/Kg/12h per il primo mese -> dose al 75%

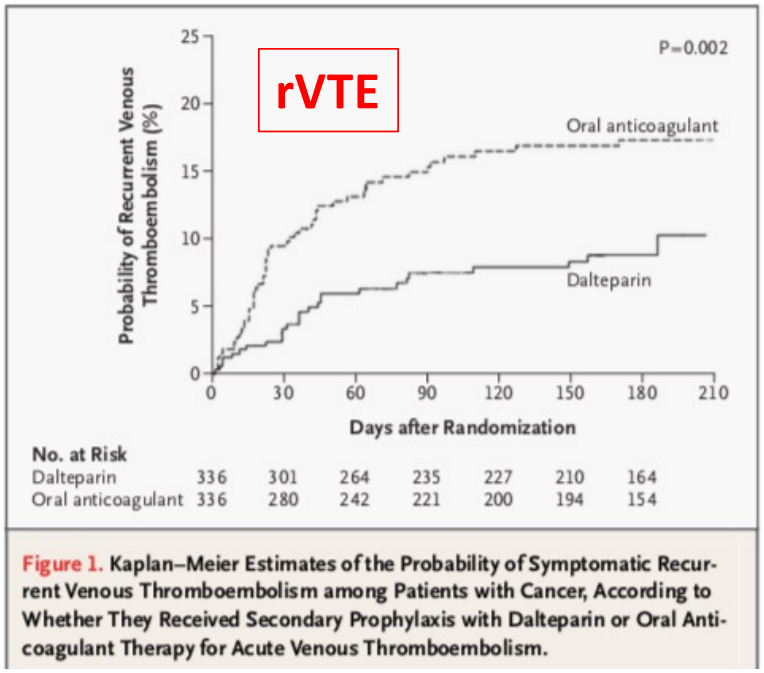



**Figure 1.** Kaplan–Meier Estimates of the Probability of Symptomatic Recurrent Venous Thromboembolism among Patients with Cancer, According to Whether They Received Secondary Prophylaxis with Dalteparin or Oral Anticoagulant Therapy for Acute Venous Thromboembolism.

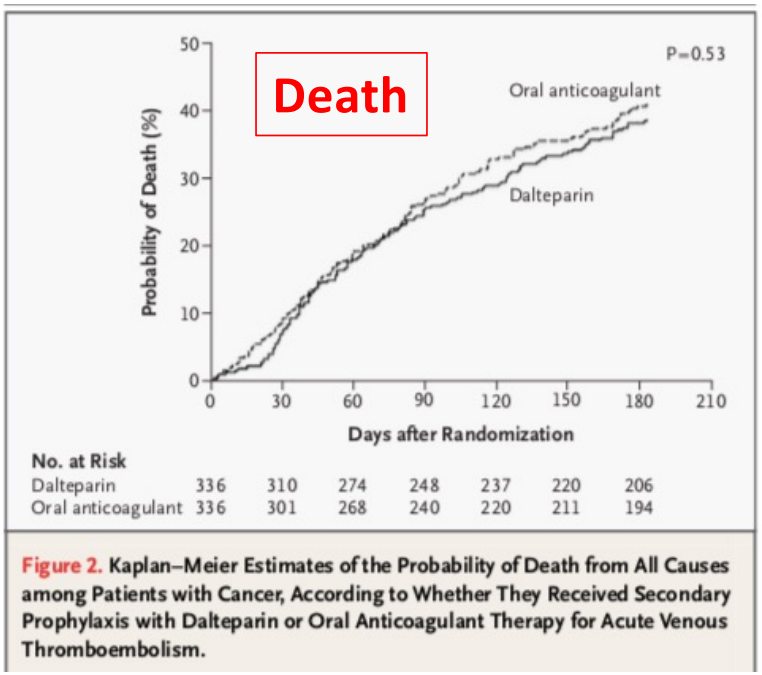



**Figure 2.** Kaplan–Meier Estimates of the Probability of Death from All Causes among Patients with Cancer, According to Whether They Received Secondary Prophylaxis with Dalteparin or Oral Anticoagulant Therapy for Acute Venous Thromboembolism.

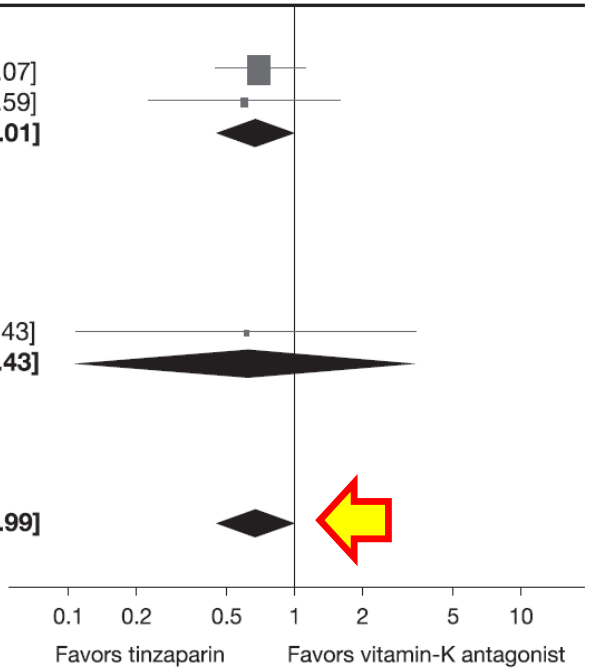
*Lee A et al. NEJM 2003; 349(2):146-53.*
*Napolitano M et al. J Clin Oncol 2014;32:3607-3612.*

# Q2. Le EBPM sono tutte uguali

## (soprattutto nel paziente oncologico)?

# Review Sistematica e meta-analisi: Tinzaparina nella CAT

| Study | Tinzaparin Events | Tinzaparin Total | Vitamin-K antagonist Events | Vitamin-K antagonist Total | Weight (%) | Risk ratio M-H, random [95% CI] |
|---|---|---|---|---|---|---|
| **Tinzaparin vs warfarin** | | | | | | |
| CATCH trial 2015 | 31 | 449 | 45 | 451 | 78.9 | 0.69 [0.45, 1.07] |
| LITE trial 2006 | 6 | 100 | 10 | 100 | 16.0 | 0.60 [0.23, 1.59] |
| **Subtotal [95% CI]** | | **549** | | **551** | **94.9** | **0.68 [0.45, 1.01]** |
| Total events | 37 | | 55 | | | |
| Heterogeneity: Tau²=0.00; Chi²=0.07, df=1 (*P*=0.79); I²=0% | | | | | | |
| Test for overall effect: Z=1.92 (*P*=0.05) | | | | | | |
| **Tinzaparin vs acenocoumarol** | | | | | | |
| Romera 2009 | 2 | 36 | 3 | 33 | 5.1 | 0.61 [0.11, 3.43] |
| **Subtotal [95% CI]** | | **36** | | **33** | **5.1** | **0.61 [0.11, 3.43]** |
| Total events | 2 | | 3 | | | |
| Heterogeneity: Not applicable | | | | | | |
| Test for overall effect: Z=0.56 (*P*=0.58) | | | | | | |
| **Total [95% CI]** | | **585** | | **584** | **100.0** | **0.67 [0.46, 0.99]** |
| Total events | 39 | | 58 | | | |
| Heterogeneity: Tau²=0.00; Chi²=0.08, df=2 (*P*=0.96); I²=0% | | | | | | |
| Test for overall effect: Z=2.00 (*P*=0.05) | | | | | | |
| Test for subgroup differences: Chi²=0.01, df=1 (*P*=0.91), I²=0% | | | | | | |




Martinez-Zapata MJ et al. Clin Appl Thromb Heamost 2018:24:226-234; Lee AY et al. JAMA2015;314:677–86; Hull RD et al. Am J Med 2006;119:1062–72; Romera A et al. Eur J Vasc Endovasc Surg 2009;37:349–56;

# Renal clearance among anticogaulants



**Table 1.** Comparison of the characteristics of the major anticoagulant classes.

| | Vitamin K Antagonists | Low Molecular Weight Heparins | Direct Oral Anticoagulants |
|---|---|---|---|
| Route of intake | Oral | Subcutaneous injection | Oral |
| Problems with oral intake in cancer | Yes | No | Yes |
| Problems with absorption in cancer | Yes | No | Yes |
| Renal clearance | No | Yes (except tinzaparin) | Yes |
| Food interactions | Yes | No | Yes |
| Influence of fasted/fed status | No | No | Yes * |
| Pharmacokinetic drug–drug interactions | Yes, with chemotherapeutics | No | Yes |
| Need to monitor | Yes | Not routine | No |

* Rivaroxaban should be taken with food. Prolonged fasting in patients on DOAC treatment may affect drug absorption leading to ineffective therapy [60].

# Q3. Secondo voi questi farmaci (VKA, LMWH & DOACs) sono interscambiabili nella pratica clinica? Abbiamo bisogno di «personalizzare» la terapia anticoagulante?

# Direct Oral Anticoagulants (DOACs) and Cancer

**Table 1. Recurrent VTE and major bleeding event rates in cancer patients enrolled in the RECOVER, EINSTEIN-DVT, EINSTEIN-PE, AMPLIFY, and Hokusai trials[33-36]**

| | VTE or VTE death, patients, n/N (%) | | Major bleed, patients, n/N (%) | |
|---|---|---|---|---|
| **RECOVER** | | | | |
| Cancer status* | Dabigatran | Control | Dabigatran | Control |
| No cancer | 58/2380 (2.4) | 50/2392 (2.1) | 18/2297 (0.8) | 33/2310 (1.4) |
| Active cancer | 10/173 (5.8) | 12/162 (7.4) | 6/159 (3.8) | 7/152 (4.6) |
| **EINSTEIN** | | | | |
| Cancer status† | Rivaroxaban | Control | Rivaroxaban | Control |
| No cancer | | | | 58/3832 (1.5) |
| Cancer at entry | | | | 8/196 (4.1) |
| Cancer diagnosis | | | | 6/82 (7.3) |
| **AMPLIFY** | | | | |
| Cancer status‡ | | | | Control |
| No cancer | | | | 45/2609 (1.7) |
| Active cancer | | | | 4/80 (5.0) |
| **Hokusai** | | | | |
| Cancer status§ | | | | Control |
| No cancer | 103/3658 (2.8) | 99/3629 (2.7) | 39/3658 (1.1) | 48/3629 (1.3) |
| History of cancer | 10/269 (3.7) | 21/294 (7.1) | 5/269 (1.9) | 10/294 (3.4) |
| Active cancer | 4/109 (3.7) | 7/99 (7.1) | 5/109 (4.6) | 3/99 (3.0) |

Studi con i DOACs: pochi pazienti oncologici, pochissimi oncoematologici

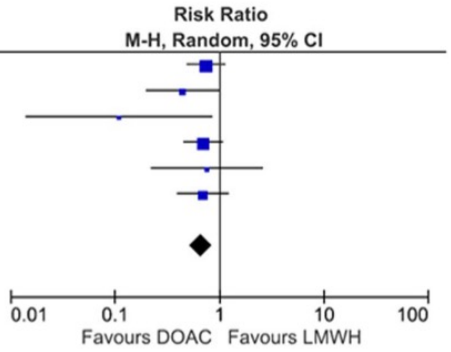
*Active cancer defined as having metastatic disease, recurrent cancer, or having been diagnosed or received treatment for cancer within 5 years prior to study enrollment.
†Active cancer was not defined.
‡Active cancer defined as having metastatic disease, recurrent cancer, or having been diagnosed or received treatment for cancer within 6 months prior to study enrollment.
§Active Cancer was defined according to the discretion of the local investigator.

# DOACs vs LMWH for the treatment of CAT
## (systematic review and meta-analysis of randomized controlled trials)

### A. Recurrent venous thromboembolism

| Study or Subgroup | DOAC Events | DOAC Total | LMWH Events | LMWH Total | Weight | Risk Ratio M-H, Random, 95% CI |
|---|---|---|---|---|---|---|
| HOKUSAI-VTE CANCER | 34 | 522 | 46 | 524 | 33.5% | 0.74 [0.48, 1.14] |
| SELECT-D | 8 | 203 | 18 | 203 | 9.3% | 0.44 [0.20, 1.00] |
| ADAM-VTE | 1 | 145 | 9 | 142 | 1.4% | 0.11 [0.01, 0.85] |
| CARAVAGGIO | 32 | 576 | 46 | 579 | 32.0% | 0.70 [0.45, 1.08] |
| CASTA-DIVA | 4 | 74 | 6 | 84 | 4.1% | 0.76 [0.22, 2.58] |
| CANVAS | 20 | 330 | 27 | 308 | 19.6% | 0.69 [0.40, 1.21] |
| **Total (95% CI)** | | **1850** | | **1840** | **100.0%** | **0.67 [0.52, 0.85]** |
| Total events | 99 | | 152 | | | |

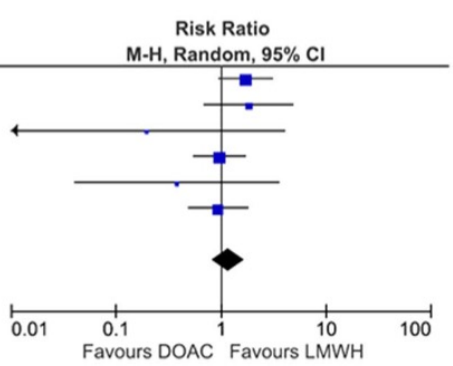
Heterogeneity: Tau² = 0.00; Chi² = 4.36, df = 5 (P = 0.50); I² = 0%
Test for overall effect: Z = 3.22 (P = 0.001)




### B. Major bleeding

| Study or Subgroup | DOAC Events | DOAC Total | LMWH Events | LMWH Total | Weight | Risk Ratio M-H, Random, 95% CI |
|---|---|---|---|---|---|---|
| HOKUSAI-VTE CANCER | 29 | 522 | 17 | 524 | 29.2% | 1.71 [0.95, 3.08] |
| SELECT-D | 11 | 203 | 6 | 203 | 12.2% | 1.83 [0.69, 4.86] |
| ADAM-VTE | 0 | 145 | 2 | 142 | 1.4% | 0.20 [0.01, 4.04] |
| CARAVAGGIO | 22 | 576 | 23 | 579 | 30.3% | 0.96 [0.54, 1.71] |
| CASTA-DIVA | 1 | 74 | 3 | 84 | 2.5% | 0.38 [0.04, 3.56] |
| CANVAS | 17 | 330 | 17 | 308 | 24.5% | 0.93 [0.49, 1.80] |
| **Total (95% CI)** | | **1850** | | **1840** | **100.0%** | **1.17 [0.82, 1.67]** |
| Total events | 80 | | 68 | | | |

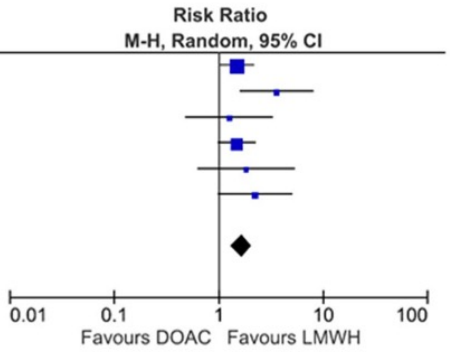
Heterogeneity: Tau² = 0.02; Chi² = 5.66, df = 5 (P = 0.34); I² = 12%
Test for overall effect: Z = 0.85 (P = 0.39)



## C. Clinically relevant non major bleeding

| Study or Subgroup | DOAC Events | DOAC Total | LMWH Events | LMWH Total | Weight | Risk Ratio M-H, Random, 95% CI |
|---|---|---|---|---|---|---|
| HOKUSAI-VTE CANCER | 64 | 522 | 43 | 524 | 40.6% | 1.49 [1.04, 2.16] |
| SELECT-D | 25 | 203 | 7 | 203 | 8.2% | 3.57 [1.58, 8.07] |
| ADAM-VTE | 9 | 145 | 7 | 142 | 5.9% | 1.26 [0.48, 3.29] |
| CARAVAGGIO | 52 | 576 | 35 | 579 | 32.1% | 1.49 [0.99, 2.26] |
| CASTA-DIVA | 8 | 74 | 5 | 84 | 4.8% | 1.82 [0.62, 5.31] |
| CANVAS | 19 | 330 | 8 | 308 | 8.3% | 2.22 [0.98, 4.99] |
| **Total (95% CI)** | | **1850** | | **1840** | **100.0%** | **1.66 [1.31, 2.09]** |
| Total events | 177 | | 105 | | | |

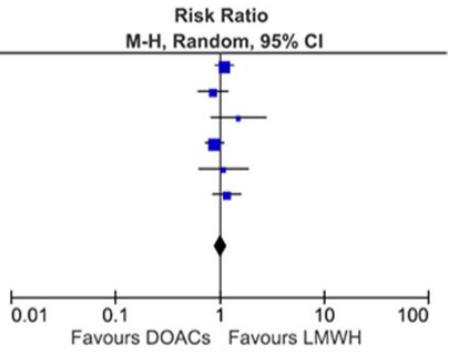
Heterogeneity: Tau² = 0.00; Chi² = 4.82, df = 5 (P = 0.44); I² = 0%
Test for overall effect: Z = 4.23 (P < 0.0001)



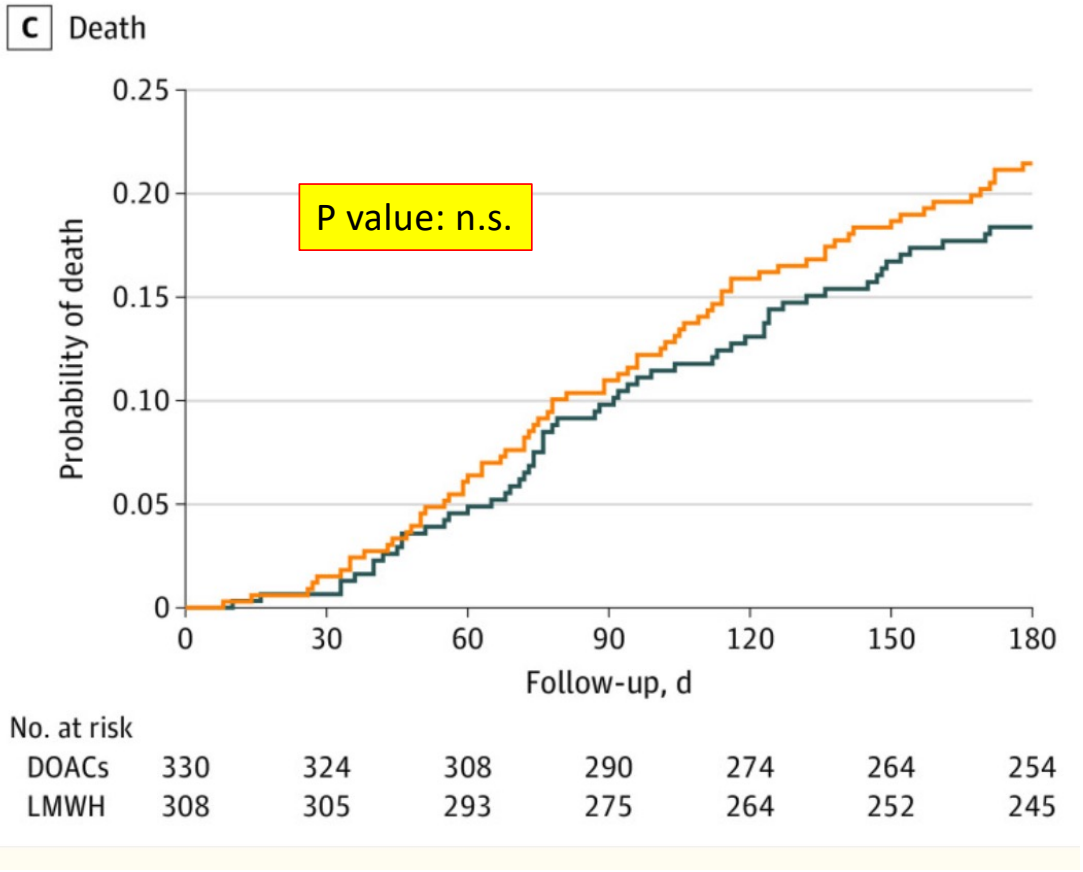

## D. Overall Mortality

| Study or Subgroup | DOAC Events | DOAC Total | LMWH Events | LMWH Total | Weight | Risk Ratio M-H, Random, 95% CI |
|---|---|---|---|---|---|---|
| HOKUSAI-VTE CANCER | 140 | 522 | 127 | 524 | 29.9% | 1.11 [0.90, 1.36] |
| SELECT-D | 48 | 203 | 56 | 203 | 13.6% | 0.86 [0.61, 1.20] |
| ADAM-VTE | 23 | 145 | 15 | 142 | 4.4% | 1.50 [0.82, 2.76] |
| CARAVAGGIO | 135 | 576 | 153 | 579 | 31.4% | 0.89 [0.73, 1.08] |
| CASTA-DIVA | 19 | 74 | 20 | 84 | 5.5% | 1.08 [0.63, 1.86] |
| CANVAS | 71 | 330 | 57 | 308 | 15.3% | 1.16 [0.85, 1.59] |
| **Total (95% CI)** | | **1850** | | **1840** | **100.0%** | **1.02 [0.89, 1.16]** |
| Total events | 436 | | 428 | | | |

Heterogeneity: Tau² = 0.00; Chi² = 5.76, df = 5 (P = 0.33); I² = 13%
Test for overall effect: Z = 0.25 (P = 0.80)



# Overall Survival: DOACs vs LMWH



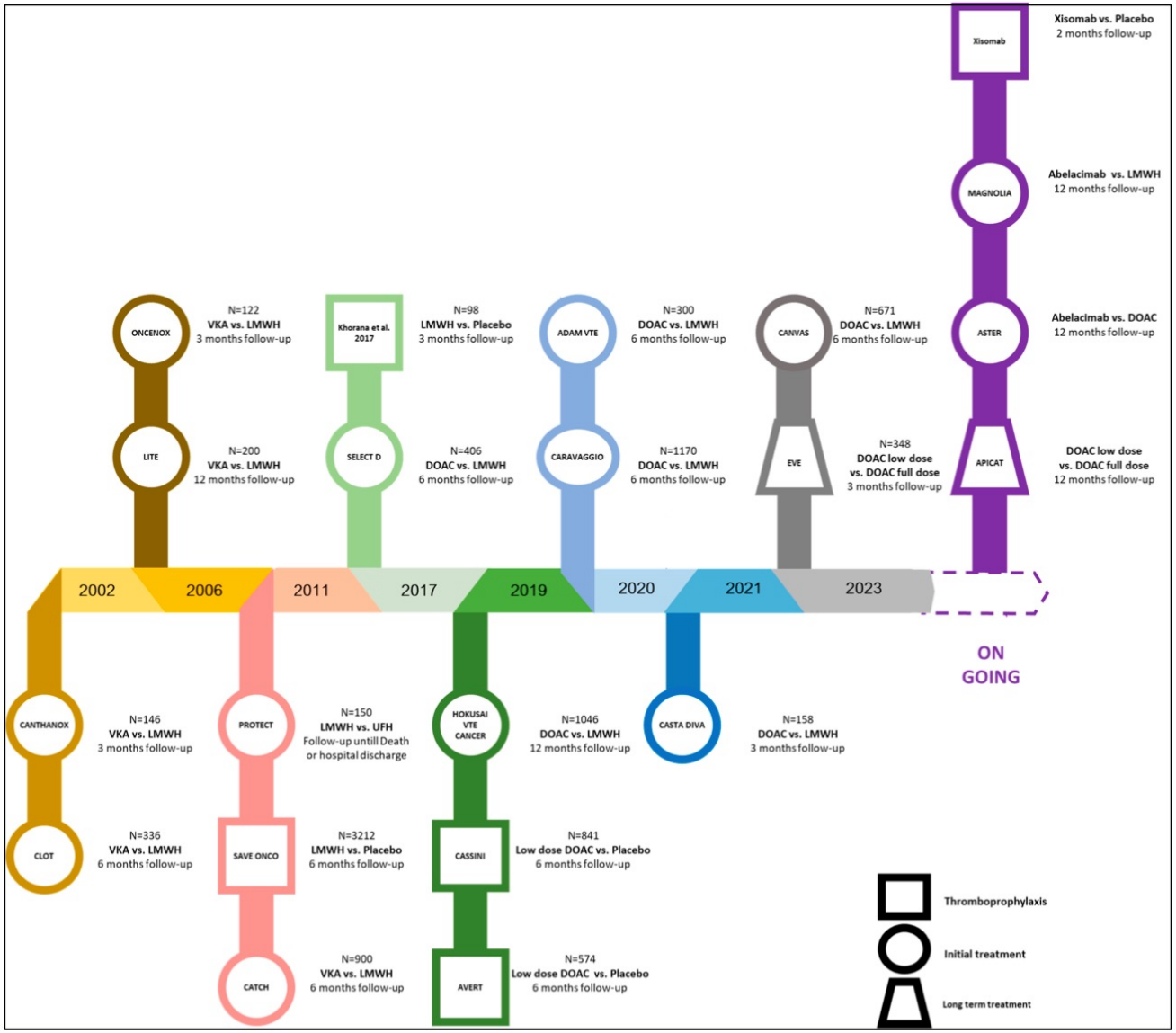

*JAMA 2023 Jun 13;329(22):1924-1933.*

**Q: Better LMWH or DOACs?**

**A: Almost the same!**
**LMWH better in high risk patients**
**(i.e. thrombocytopenic patients)**

# I Nuovi Anticoagulanti

# Il viaggio della terapia anticoagulante nella CAT

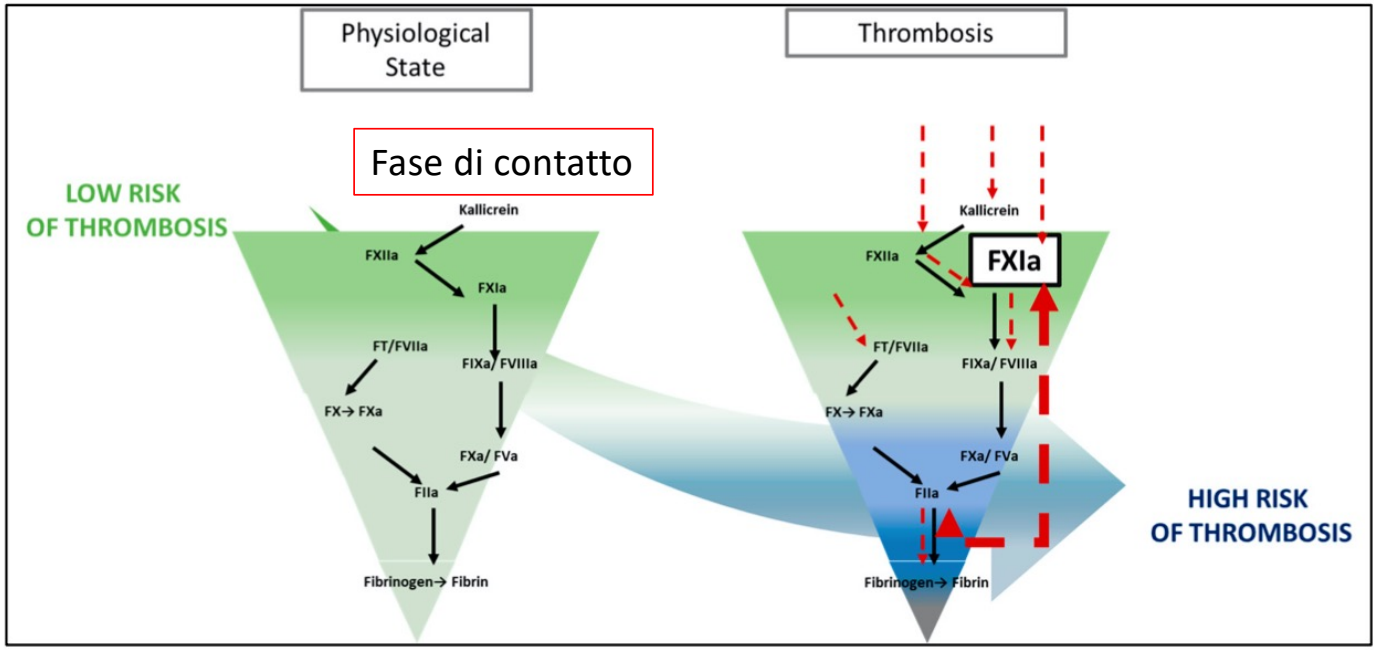


# FXI and FXII inhibitors

1. Oligonucleotidi antisenso (anti-sense oligonucleotides, ASOs): riducono la sintesi epatica di FXI (IONIS-FXIrx e fesomersen).

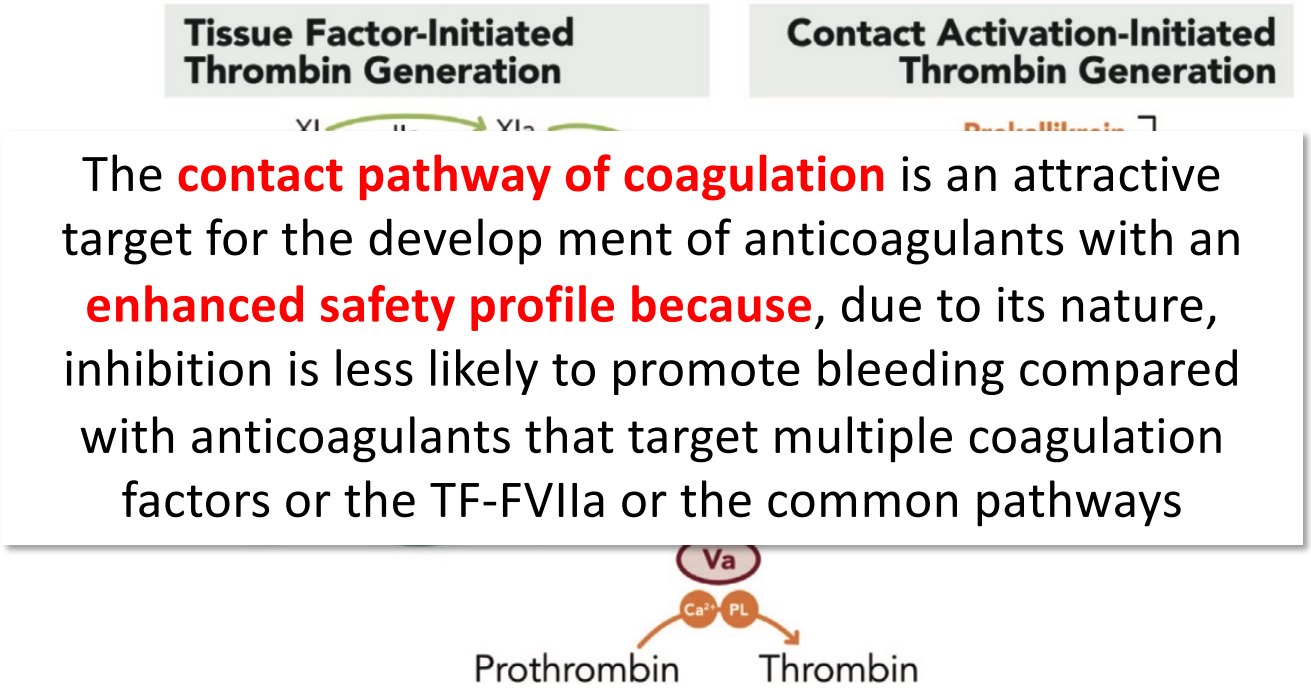
2. Anticorpi monoclonali (monoclonal antibodies, mAbs): si legano al FXI o FXIa bloccandone l'attivazione o l'attività (osocimab, **abelacimab**, xisomab 3G3 e MK-2060)

3. Piccole molecole: legano reversibilmente il FXIa bloccandone l'attività (milvexian , asundexian ed EP-7041)

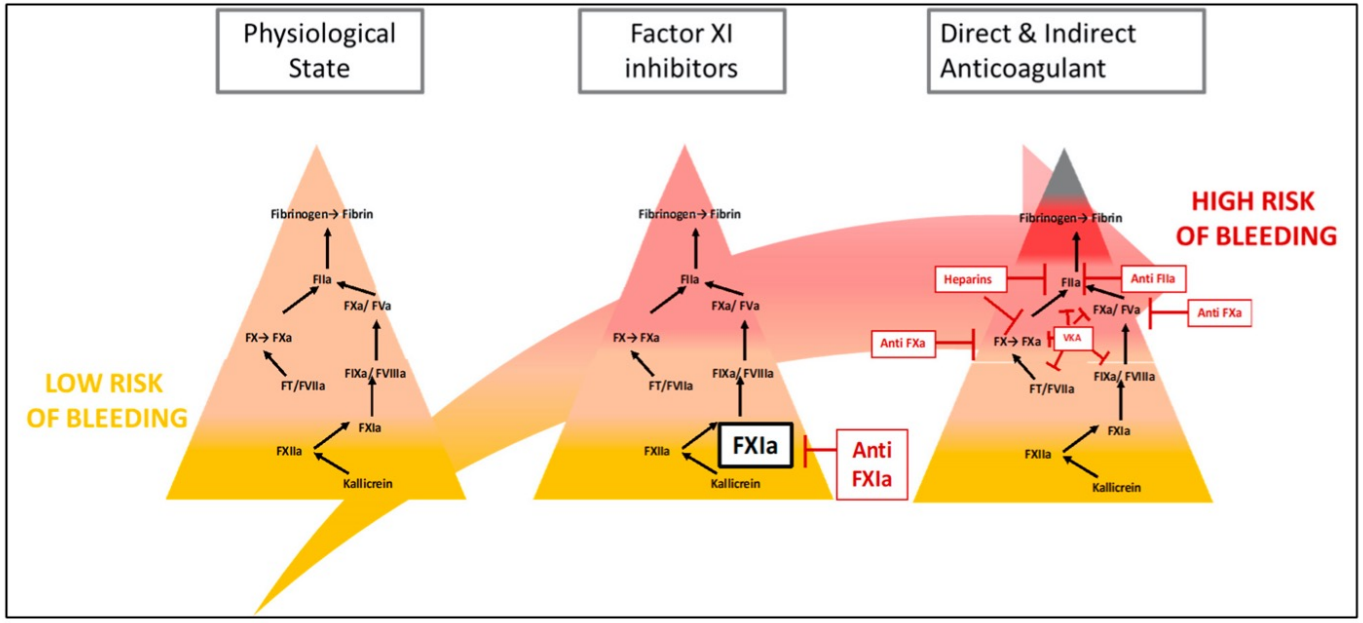



Nei pazienti oncologici, il tumore attiva il FXI (normalmente poco attico nelle trombosi non neoplastiche)

# Ruolo del FXI nella genesi della trombosi e sua inibizione




The **contact pathway of coagulation** is an attractive target for the develop ment of anticoagulants with an **enhanced safety profile because**, due to its nature, inhibition is less likely to promote bleeding compared with anticoagulants that target multiple coagulation factors or the TF-FVIIa or the common pathways



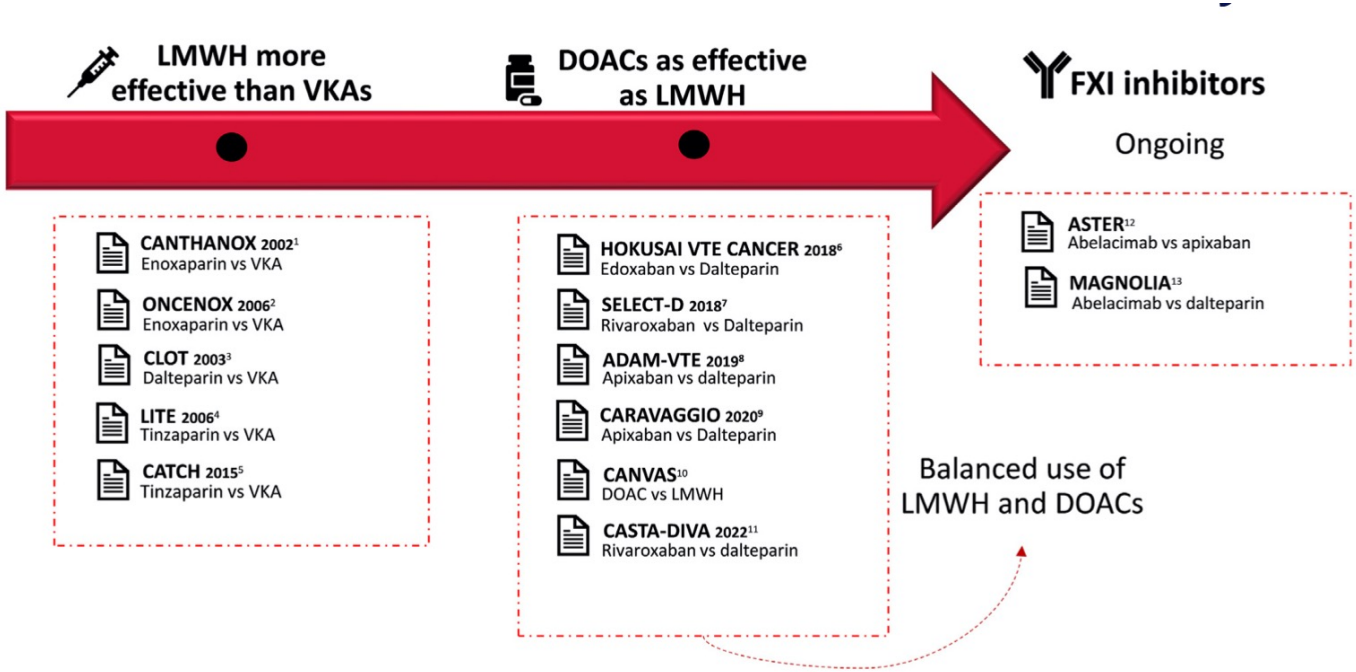

L’inibizione del FXI comporta una possibile riduzione del rischio emorragico rispetto ad altri anticoagulanti

# Characteristics, mechanism of action, and pharmacologic properties of FXI and FXII inhibitors (II)

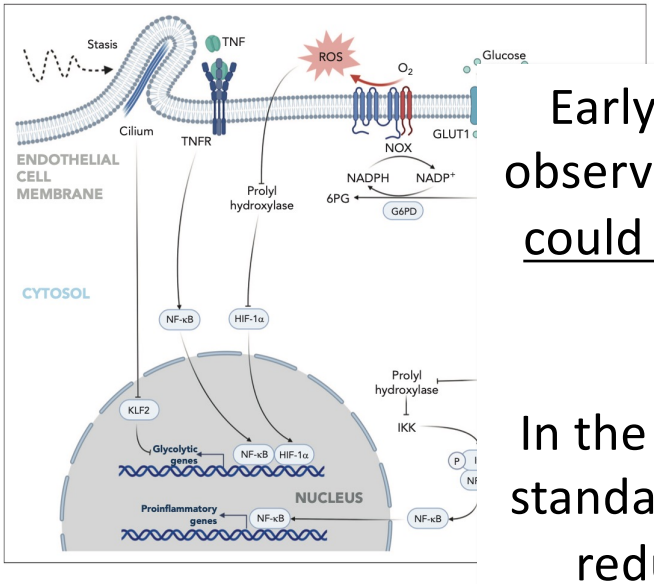
| Antibodies | Mechanism of action | Route of administration | Onset of action | Half-life | Administration frequency | Renal excretion | Metabolism by CYP | Potential for food and drug interactions |
|---|---|---|---|---|---|---|---|---|
| Abelacimab (MAA868) | Binds to the catalytic domain of FXI and locks it in the inactive zymogen confor-mation, preventing its activation by FXII/thrombin | IV or SC | Rapid (hours) | Long (weeks) | Once monthly | No | No | No |
| Osocimab (BAY 1213790) | Binds next to the active site of FXIa and inhibits the activation of factor IX | IV or SC | Rapid (hours) | Long (30–44 days) | Once monthly | No | No | No |
| Xisomab (AB023) | Inhibits FXIIa-mediated activation of FXI but not FXI activation by thrombin | IV | Rapid (hours) | Days to weeks, half-life increases with high doses | Once monthly | No | No | No |
| Garadacimab (CSL312) | Binds to the catalytic domain of FXIIa and inhibits its protease activity | IV or SC | Rapid (hours) | Long (weeks) | Once monthly | No | No | No |

# Ongoing clinical trials with FXI and FXII inhibitors

| Agent | Registry number (name) | Clinical trial phase and indication | No. of patients | Comparator | Additional therapies | Efficacy outcome | Safety outcomes | Study recruitment status* |
|---|---|---|---|---|---|---|---|---|
| IONIS FXI-Rx (ISIS 416858) | NCT03358030 (EMERALD) | Phase 2 Patients with ESRD on hemodialysis | 315 | Placebo | NA | PK, PD | Major bleeding or CRNMB, treatment-emergent AEs | Completed, awaiting results |
| Fesomersen (IONIS-FXI-LRx and BAY2976217) | NCT03582462 | Phase 1 Healthy volunteers | 66 | Placebo | NA | PK, PD | Treatment-emergent AEs | Completed, awaiting results |
| | NCT04534114 (RE-THINc ESRD) | Phase 2 Prevention of cardiovascular events in patients with ESRD | 307 | Placebo | NA | PK, PD | Major bleeding or CRNMB, treatment-emergent AEs | Active, not recruiting |
| Antibodies | | | | | | | | |
| Abelacimab (MAA868) | NCT04213807 | Phase 2 Atrial fibrillation | 28 | Placebo | NA | Dose-range finding study—PK, PD | Safety, tolerability, and immunogenicity | Completed, awaiting results |
| | NCT04755283 (AZALEA-TIMI 71) | Phase 2 Atrial fibrillation | Estimated enrollment 1200 | Rivaroxaban 15 mg and 20 mg OD | NA | NA | Safety and tolerability, major bleeding, and CRNMB | Active, not recruiting |
| | NCT05171049 (ASTER) | Phase 3 Treatment of cancer-associated VTE | Estimated enrollment 1655 | Apixaban 10 mg BID followed by 5 mg BID | NA | Centrally adjudicated VTE recurrence | Major bleeding or CRNMB | Active, recruiting |
| | NCT05171075 (MAGNOLIA) | Phase 3 Treatment of GI and GU cancer-associated VTE | Estimated enrollment 1020 | Dalteparin 200 IU/kg/d followed by 150 IU/kg/d | NA | Centrally adjudicated VTE recurrence | Major bleeding or CRNMB | Active, recruiting |
| Osocimab (BAY 1213790) | NCT03787368 | Phase 1 Safety in patients with ESRD | 55 | Placebo | NA | PK, PD | Major bleeding or CRNMB | Completed, awaiting results |
| | NCT04523220 (CONVERT) | Phase 2 Safety in patients with ESRD | 686 | Placebo | NA | PK, PD | Major bleeding or CRNMB | Active, not recruiting |
| Xisomab (AB023) | NCT04465760 | Phase 2 Prevention of CAT in patients with cancer receiving chemotherapy | Estimated enrollment 50 | None | NA | Incidence of CAT | Major bleeding or CRNMB | Active, recruiting |


LMWH more effective than VKAs
DOACs as effective as LMWH
FXI inhibitors
Ongoing
CANTHANOX 2002[1]
Enoxaparin vs VKA
ONCENOX 2006[2]
Enoxaparin vs VKA
CLOT 2003[3]
Dalteparin vs VKA
LITE 2006[4]
Tinzaparin vs VKA
CATCH 2015[5]
Tinzaparin vs VKA
HOKUSAI VTE CANCER 2018[6]
Edoxaban vs Dalteparin
SELECT-D 2018[7]
Rivaroxaban vs Dalteparin
ADAM-VTE 2019[8]
Apixaban vs dalteparin
CARAVAGGIO 2020[9]
Apixaban vs Dalteparin
CANVAS[10]
DOAC vs LMWH
CASTA-DIVA 2022[11]
Rivaroxaban vs dalteparin
ASTER[12]
Abelacimab vs apixaban
MAGNOLIA[13]
Abelacimab vs dalteparin
Balanced use of LMWH and DOACs

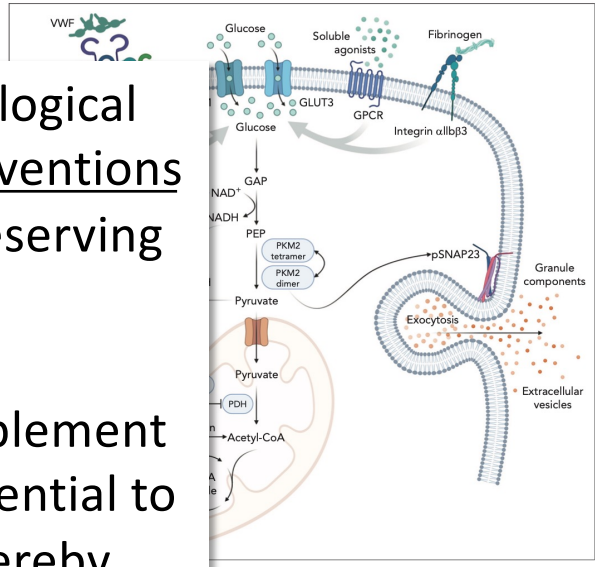
# Metabolic pathways in DVT: a new frontier for therapeutic intervention




**Metabolic reprogramm activated ECs**

Early preclinical evidence and epidemiological observations suggest that <u>metabolic interventions could reduce thrombus burden</u> while preserving hemostasis and lowering DVT risk

In the future, such approaches may complement standard-of-care treatments with the potential to reduce exposure to anticoagulants, thereby decreasing bleeding risk and improving overall treatment efficacy




**ic reprogramming in vated Platelets**

# Conclusions

- It is estimated that 20% of all VTE are related to cancer and anticoagulant treatment in these vulnerable patients is often challenging
- Cancer patients with VTE are at increased risk of developing recurrent VTE compared to non-cancer patients, but also have a higher risk of major bleeding
- LMWH/DOACs are currently recommended over VKA for treatment of cancer-associated VTE
- Need for individual parameters to help in the choice of the optimal duration of anticoagulants
- New anticoagulants targeting contact pathway of anticoagulation became an attractive treatment for the CAT since the low risk for bleedding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