

Table 1 Risk factors for VTE¹¹.

Strong risk factors (odds ratio >10)

Fracture (hip or leg)
Hip or knee replacement
Major general surgery
Major trauma
Spinal cord injury

Moderate risk factors (odds ratio 2–9)

Arthroscopic knee surgery
Central venous lines
Chemotherapy
Congestive heart or respiratory failure
Hormone replacement therapy
Malignancy
Oral contraception
Paralytic stroke
Pregnancy, postpartum
Previous VTE
Thrombophilia

Weak risk factors (odds ratio <2)

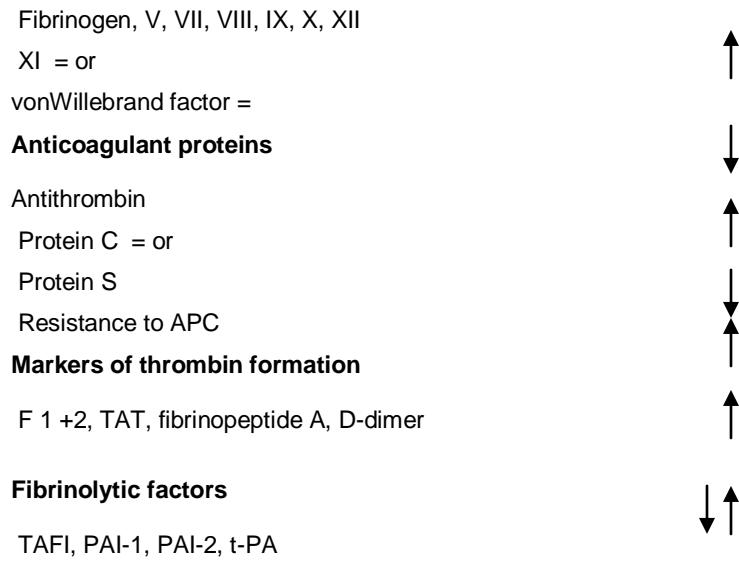
Bed rest >3 days
Immobility due to sitting (e.g., prolonged car or air travel)
Increasing age
Laparoscopic surgery (e.g., cholecystectomy)
Obesity
Pregnancy, antepartum
Varicose veins

Table 2 Haemostatic changes during COC¹³

Factor Change during COC use

Procoagulant factors





↑ increase; ↓ decrease; = no change (vs. non-use of COCs).
 TAT, thrombin–antithrombin complex; TAFI, thrombin-activatable
 Fibrinolysis inhibitor; PAI, plasminogen activator inhibitor ; t-PA, tissue plasminogen activator.

Table 3 Effects of E₂-based COCs (E₂V/DNG and E₂/NOMAC) and DRSP-containing COCs on haemostasis, lipid and carbohydrate metabolism. EE/LNG was used as the comparator.

| Marker | <i>EE/DRSP</i> ⁵¹ | <i>E₂V/DNG</i> ^{45,46} | <i>E₂/NOMAC</i> ^{28,47} |
|---|------------------------------|--|---|
| <i>Haemostatic markers</i> | | | |
| <i>F 1 + 2</i> | - | NS | *↓ |
| <i>d-dimer</i> | - | ↓*† | *‡ |
| <i>APC ratio</i> | - | NS | *↓ |
| <i>Protein S (activity)</i> | - | NS | NS |
| <i>Markers of lipid metabolism</i> | | | |
| <i>HDL-C (or total cholesterol)</i> | ↑* | NS | NS |
| <i>LDL-C</i> | ↑NS | NS | *↓ |
| <i>TG</i> | * | NS | *↓ |
| <i>Markers of carbohydrate metabolism</i> | | | |
| <i>Insulin response (AUC)</i> | NS | NS | |
| <i>Glucose levels (AUC)</i> | - | NS | *↓ |

*Significant vs. EE 30 µg/LNG 150 µg. Another study showed a non-significant difference vs. the comparator⁴⁶.

†Another study showed a non-significant difference vs. EE 30 µg/LNG 150 µg²⁸.

↑*, significant relative increase vs. EE/LNG's effects on the marker evaluated; ↓*, significant relative decrease vs. EE/LNG's effects on the marker evaluated; -, no existing data; NS, not significant

Table 4 Head-to-head studies evaluating effects of EE/LNG, EE/DRSP, E₂V/DNG and E₂/NOMAC on haemostasis, lipid and carbohydrate metabolism. This table also includes two studies that assessed effects of EE/DSG combinations on relevant markers.

| Study and treatment group | Relevant marker studied | | | | | | | | | |
|--|-------------------------|----|---------------------------|------------------|-------|-------|-------------------------|------------------|---------|---|
| | Haemostasis | | | Lipid metabolism | | | Carbohydrate metabolism | | | |
| | Acquired APC | | F 1+ 2 D-dimer resistance | Protein S | HDL-C | LDL-C | TG | Insulin response | Glucose | |
| Oelkers et al. 1995 ⁵¹ EE 15 µg, 20 µg, 30 µg/DRSP 3 mg (n= 20 women in each group) | NA | NA | NA | NA | + | + | + | - | + | |
| EE 30 µg/LNG 150 µg (n= 20) | | | | | | | | | | |
| Middeldorp et al. 2000 ³⁷ EE 30 µg/LNG 150 µg EE 30 µg/DSG 150 µg | + | - | - | - | NA | NA | NA | NA | NA | |
| Klipping& Marr 2005 ⁴⁴ EE 20 µg/DRSP 3 mg (n= 29) | + | + | + | + | + | + | + | NA | NA | |
| EE 20 µg/DSG 150 µg (n= 30) | | | | | | | | | | |
| Klipping et al. 2011 ⁴⁵ E ₂ V 1 mg, 2 mg, 3 mg/DNG 2 mg, 3 mg, placebo (n= 16) | + | + | + | + | NA | NA | NA | NA | NA | |
| EE 30 µg/LNG 150 µg (n= 16) | | | | | | | | | | |
| Junge et al. 2011 ⁴⁶ E ₂ V 1 mg, 2 mg, 3 mg/DNG 2 mg, 3 mg, placebo (n= 30) | + | + | + | + | + | + | + | + | + | + |
| EE 30 µg ,40µg/LNG 50 µg, 75 µg, 125 µg (n=28) | | | | | | | | | | |
| Gaussem et al. 2011 ⁴⁷ E ₂ 1.5 mg/NOMAC 2.5 mg + + + + (n 45) | | | | | NA NA | NA | NA | NA | NA | |
| EE 20 µg/LNG 100 µg (n=45) | | | | | | | | | | |
| Ågren et al. 2011 ²⁸ E ₂ 1.5 mg/NOMAC 2.5 mg + + + + (n 60) | | | | | ++ | + | + | + | + | |
| EE 30 µg/LNG 150 µg (n=58) | | | | | | | | | | |

*33 women were recruited in this study.+, assessed: -, not assessed: NA, not appropriate

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