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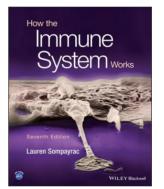
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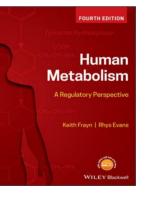


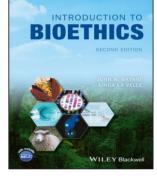
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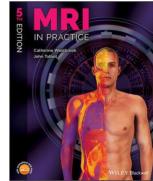
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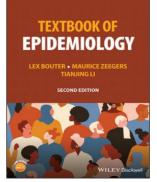
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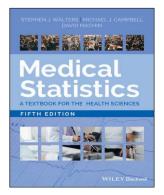












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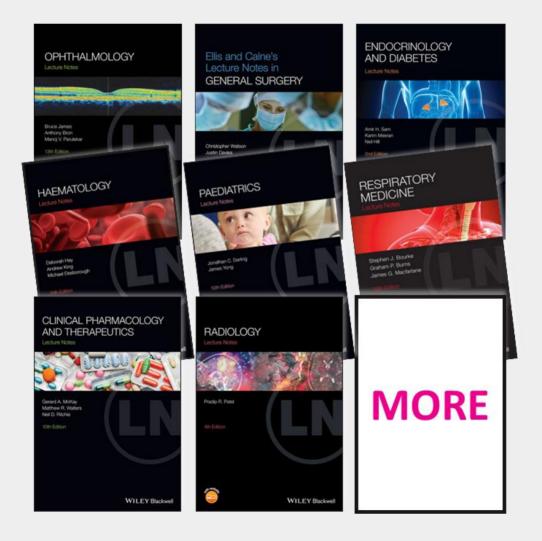
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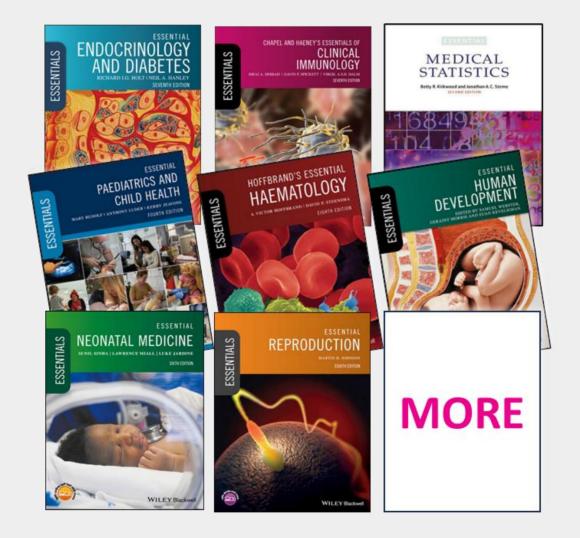
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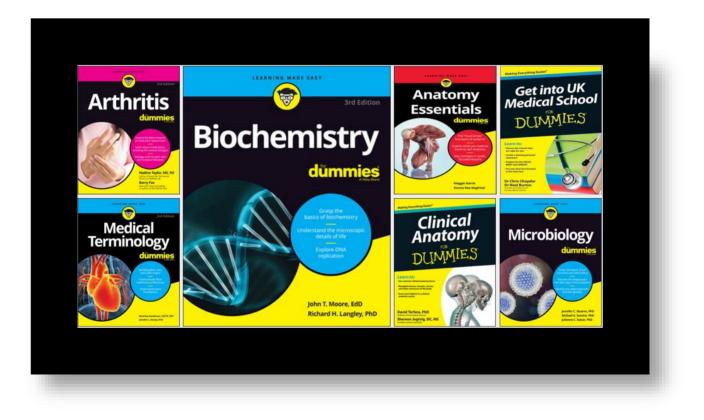
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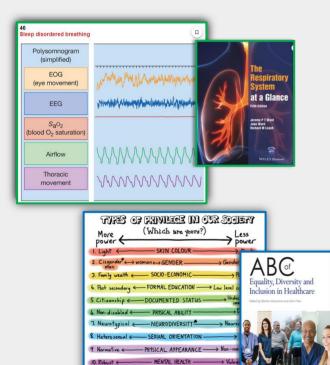
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Examples



Examples relevant to today's students



- RELIGION -

* Gender corresponds with birth sex A Developmental, intellectual & cognitive abilities

-> Non -(

11. Christian

Thrombosis and the COVID-19 pandemic

During the COVID-19 pandemic starting in late 2019, it became apparent that patients with severe COVID-19 infection had a higher rate of venous thrombosis than would be expected for people with a similar level of illness. Some of these thromboses were due to classic risk factors such as immobility and inflammation. However, there was also an increase in small segmental and subsegmental pulmonary emboli without evidence of deep vein thrombosis. These have been termed *immunothromboses* and are thought to arise from inflammation of the pulmonary vasculature rather than from deep vein thromboses breaking away to form pulmonary emboli. The pathological basis of these immunothromboses and whether they need different treatment from classic pulmonary emboli are still topics of debate.

There has also been a risk of an exceptionally rare thrombotic syndrome following COVID-19 vaccination using adenoviral vaccines such as the ChAdOx1 nCoV-19 vaccine. This has been termed *vaccine-induced thrombosis and thrombocytopenia* (VITT). VITT is due to antibodies forming against platelet factor 4 leading to formation of thromboses. This has a similar mechanism to heparin-induced thrombocytopenia. VITT has often manifested as cerebral venous sinus thromboses associated with thrombocytopenia and has been challenging to treat. The mainstay of treatment has been anticoagulation and intravenous immunoglobulin. The risk of death of thrombosis from COVID-19 infection remains very much results from the vaccines.





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