

Providing good feedback on written work

Aims of providing feedback

1. Facilitates the development of self assessment (reflection) in learning.
2. Encourages teacher and peer dialogue around learning.
3. Helps clarify what good performance is (goals, criteria, expected standards).
4. Provides opportunities to close the gap between current and desired performance.
5. Delivers high quality information to students about their learning.

Examples of **good** feedback

During the early stages of diabetic nephropathy, mediated by the haemodynamic pathway, functional changes begin to occur; for example both glomerular hyperfiltration and hyperfusion, which can remain clinically undetected. The presence of these functional changes facilitates albumin leakage, and the glomerular basement membrane begins to thicken. The kidney shows signs of glomerular hypertrophy, mesangial cell-matrix expansion and podocyte injury predicting the progression to overt nephropathy. These haemodynamic factors are the result of defective autoregulation of the filtration system within the glomerulus, in the form of decreased resistance in the efferent and afferent arterioles, with the latter seeing a greater decrease (Dronavalli, Duka & Bakris, 2008). This decreased resistance is due to the release of prostanoids, nitric oxide, VEGF, transforming growth factor β 1 (TGF- β 1) and by angiotensin-II, as part of the renin-angiotensin-aldosterone system (RAS).

Insert reference here

It would be helpful to describe how GFR is regulated normally and thus what would happen if afferent & efferent arteriole resistance changes.

transplantation therefore is still not a cure for diabetic nephropathy. The toxic effects of immunosuppressant drugs as well as their potential to increase the risk of infection put patients at a high risk of developing cardiovascular disease, which is still one of the most common causes of mortality in patients with ESRD (Fioretto & Mauer, 2012).

nice summary of the treatment options open to DN patients

nephrin, in patients with diabetes compared to without, is a sign of podocyte injury. Decreased volumes of nephrin within the podocytes, due to this secretion, can lead to loss of integrity of the glomerular filtration barrier and therefore loss of function (Dronavalli, Duka & Bakris, 2008).

This is a very dense page: some paragraph breaks &/or a diagram would help to break it up and make it easier for the reader to follow.

Why this is a good example....

- Comment provides specific detail on how to improve the highlighted sentence
- Comment gives some positive feedback to the student
- Examples of how to improve paragraph layout are provided

Introduction

they evolved BY natural selection because they favoured survival

Natural toxins evolved as a defence mechanism developed by bacteria, plants and animals to survive **natural selection**. After the discovery of their therapeutic potential, natural toxins represented the beginning of drug development (Rishton, 2008). Although, natural toxins were used by humans for their poisoning proprieties, they still remain the oldest therapeutic compounds used in medicine. The paradox between their beneficial role in small doses and their poisonous nature in larger amounts **makes them such complex molecules**. The aim of this review is to give an overview of a few important natural toxins used in medicine nowadays and their importance in understanding underlying processes of disease such as cancer, heart failure or pain management.

a complex molecule implies a complex structure or mechanism - this is not necessarily the case.

One of the most potent toxins known is Botulinum neurotoxin. It has been reported that only 1 gram of neurotoxin is capable of killing over 14,000 humans, while one molecule can induce paralysis of a single nerve cell (Katona, 2012). Botulinum toxin is produced by the anaerobic bacteria *Clostridium botulinum* and it was first described in 1895 as a neurotoxin that acts at the presynaptic neuromuscular junctions by inhibiting acetylcholine release (Mahajan, et al., 2007). As a consequence to induced inhibition of neurotransmitter release, toxin exposure **involves** a disease called botulism. The most common symptoms specific to botulism are limb paralysis, facial muscle weakness, dysarthria and shortness of breath. The most frequent case of acquiring botulism is after ingestion of contaminated food which causes bacterial colonization of the gut.

?causes?

how is it absorbed? diffusion? active transport? what sort of molecule is it?

The **absorption site** of botulinum toxin is the gastrointestinal tract and after its release in the circulation, it **inhibits** acetylcholine vesicles discharge by binding to the cholinergic nerve endings (Simpson, 2000). Once it binds to receptors on the apical side of the gut the toxin is released unchanged in the circulation. Due to its large size, the neurotoxin cannot pass through the blood-brain barrier, therefore its primary target tissue is the peripheral motor nerve ending. Botulinum mechanism of action can be presented in three distinct phases. First, the toxin binds to motor nerve endings receptors followed by its **internalization** in the cell. As a result, the process of endocytosis

Why this is a good example...

- comment states specifically what is wrong with the student's opening sentence
- comment provides an accurate alternative interpretation of words used by the student
- comment poses questions which the student has not considered

References

The Higher Education Academy

Enhancing student learning through effective formative feedback

http://www-new2.heacademy.ac.uk/assets/documents/resources/resourcedatabase/id353_senlef_guide.pdf