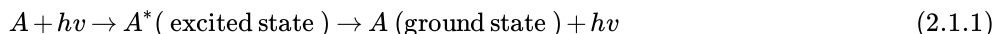
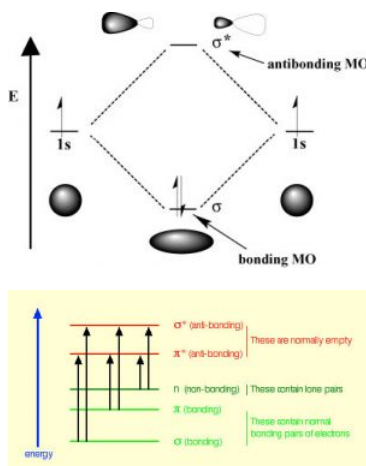


2.1: Interactions of Electromagnetic Radiation and Electrons in Molecules

Now we will extend this idea from atoms to molecules. Just as electrons occupy atomic orbitals in atoms, the electrons in molecules occupy molecular orbitals.

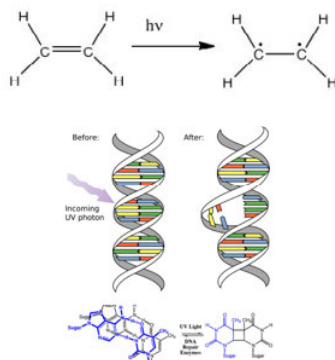


As with atomic orbitals, electrons in molecular orbitals can absorb or release photons of a specific energy as they move from one molecular orbital to another. However, there is a significant difference between the absorption/emission process in isolated atoms (or ions) versus that of molecules. When an electron is promoted to a higher energy level in an atom, the product is an atom in an excited state—generally the excited atom (or ion) will decay back to the ground state by emitting a photon (\downarrow). However, when an electron within a molecule is excited it moves (or is “promoted”) from its original molecular orbital to another. Now there are a number of different consequences that can occur. For example, if the electron absorbs a photon and is promoted from a bonding molecular orbital to an antibonding orbital, the result will be that the bond will break, since there is now no overall stabilizing interaction. Consider $\text{H} - \text{H}$, which is the simplest possible molecule. The set of molecular orbitals for hydrogen includes a σ (sigma) bonding and a σ^* antibonding orbital. In the ground (or lowest-energy) state, molecular hydrogen has a σ bonding orbital containing both of the molecule’s electrons. If one of the bonding electrons absorbs a photon that has just the right amount of energy (the energy difference between the bonding and antibonding orbital) it will be promoted and move into the destabilized antibonding orbital—causing the bond between the atoms to break because there is now no overall bonding interaction. As you might imagine, if chemical bonds were susceptible to breaking merely by being exposed to low-energy electromagnetic radiation (such as that of visible light) the world would be a different (and rather boring) place. For example, life would not be possible since it depends upon the stability of molecules. In fact, the energy of the photons required to bring about bond-breaking is quite large. For example, the energy required to break an $\text{H} - \text{H}$ bond (the bond energy) is 436 kJ/mol . If you calculate the wavelength of a photon that could deliver this amount of energy, the amount of energy required to break one $\text{H} - \text{H}$ bond would be in the far UV section of the electromagnetic spectrum ($\sim 280 \text{ nm}$). The typically strong covalent σ (or single) bond requires quite high-energy photons to break them.

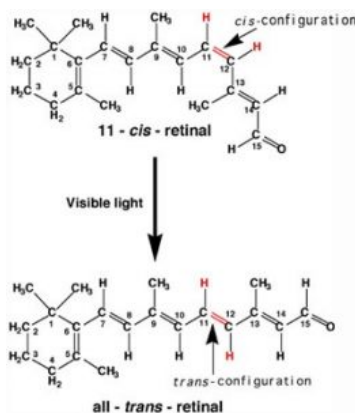


In fact, the Earth’s atmosphere blocks out most ($> 98\%$) high-energy (ultraviolet) photons and most biologically important molecules cannot absorb visible light, so that leaves us with the question of why is there a need for sunscreen, which filters out the UV A (400 - 315 nm) and UV B (315 - 280 nm) photons. The answer is that a number of biological molecules contain more than simple sigma bonds. For example, most complex biological molecules also contain π (pi) bonds and non-bonding electrons in addition to σ bonds; transitions between these orbitals may be observed. The energy gaps between these different orbitals are quite smaller than the $\sigma - \sigma^*$ energy gap. Photons with enough energy to cause these electron transitions are present in sunlight. For example, a double bond typically involves both a σ and a π bond. Absorption of a photon that would promote an electron from a π bonding orbital to a π^* anti-bonding orbital would have the effect of breaking the original pi bond. One way to represent this is shown here (\rightarrow). In this case, one of electrons that was in the pi bond is now in the high-energy π^* antibonding orbital and is far more reactive. Another way to think about it is that the electrons are now unpaired, and are much more likely to react to form a more stable entity.^[1] An obvious way to regain stability is for the electron in the π antibonding orbital to drop back down to the bonding energy level and emit a photon of the same energy, and in most cases this is what happens—ultimately causing no damage. (As we will see later, since double-bonds are rotationally constrained, another possible occurrence is that there can be rotation

around the single $[\sigma]$ bond) and then reformation of the π bond, leading to an isomer of the original alkene). However, in some instances, if there is another potentially reactive species in proximity, reactions between molecules (or in the case of biological macromolecules, between distinct regions of these molecules) can occur and cause problems. For example: most of us are aware that exposure to the sun causes skin damage that can lead to skin cancer. A major mechanism occurs in DNA where two thymine bases are adjacent to one another. A UV photon can be absorbed by a pi bond in one thymine base. This broken pi bond (and resulting unpaired electron) is very reactive. It can react with a pi bond in an adjacent thymine leading to a new bond, a reaction that produces a four-membered carbon ring, known as a thymine dimer. The DNA replication machinery cannot accurately replicate a sequence containing a thymine dimer, resulting in a change in DNA sequence—a mutation. Mutations of this type are a common early step in the generation of cancerous skin cells (carcinomas) and pigment cells (melanomas).^[2]



A more benign example of photon absorption in biological systems underlies the mechanism by which we (and other organisms) detect light—that is how we can see things! While it was originally thought (at least by some) that vision involved rays emitted from eyes,^[3] we now understand that to see we need to detect photons that are reflected or emitted by the objects around us. The process begins when the photons of light fall on cells known as photoreceptors. In our eyes, these cells are located within the retina: a sheet of cells that line the interior surface of the eye. Within a subset of retinal cells are a number of different types of molecules that contain pi bonds. These molecules are proteins known generically as opsins. An opsin is composed of a polypeptide (or apoprotein) that is covalently bound to another molecule, 11-cis-retinal.^[4]



This molecule is derived from vitamin A (all-trans-retinal). The complex of apoprotein and retinal is the functional opsin protein. There are a number of different opsin components that influence the wavelength of the photons absorbed by the functional opsin protein. When a photon is absorbed, it promotes an electron from one of the retinal's pi bonds to an antibonding orbital. Instead of reacting with another molecule, like thymine, there is a rotation around the remaining single (sigma) bond, and then the formation of a new pi bond, this leads to the isomerization of the original 11-cis form into the trans-isomer. This change in retinal shape influences the shape of the opsin protein which initiates a cascade of electrochemical events that carry signals to the rest of the brain (the retina is considered an extension of the brain) that are eventually recognized as visual input.