The Science of Health, Nutrition and Fitness

Steroids: Facts and Fiction

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In every gymnasium throughout the land, rhetoric and verbalisations surrounding the sphere of anabolic androgenic steroids (AAS). Athlete like Lance Armstrong, Marion Jones and more recently Tyson Gay and Asafa Powell have all brought the drugs and steroid debate in to sharp media focus and into the eyes of the general public. Whenever these drugs are mentioned they are always portrayed as a negative factor. A harbinger of death and destruction! But what is the truth behind these ergogenic aids? What weight should we lend to the claims that these drugs can negatively affect health or even 'kill'? Also, how valid is the claim that these drugs can enhance performance?

In respect of these questions, what follows are three articles. The first article, '*The Science Behind Steroids*' will look at what these drugs are and what their supposed method of action is? The second article, entitled '*Side Effects of Anabolic Steroids'* will look at the negative theories behind steroid utilisation and the third and final article will look at the effectiveness of steroid use, in an article entitled '*Steroids and Performance enhancement'*.

So with our preamble over let us now look at the theories and science behind androgenic-anabolic steroids.

Part 1: The Science Behind Steroids

There are two naturally occurring types of steroid which have widely different functionalities in the maintenance of homeostasis within the human body. These are corticosteroids and androgenic anabolic steroids.

Corticosteroids are produced naturally by the adrenal glands located above the kidneys. Corticosteroids may be found in two formats; those being 'glucocorticoids' and 'mineralocorticoids'. An example of a glucocorticoid is Cortisol. Cortisol has many varied roles including; regulation of blood sugar levels, regulation of metabolism and also anti inflammatory roles. An example of a mineralocorticoid is aldosterone. This corticosteroid controls water and electrolyte levels. In terms of medical applications, corticosteroids can be used to treat medical issues ranging from asthma to rheumatoid arthritis.

However, these steroids are not the ones that the government, sporting bodies and the media denounce and depict as being ' corrupt', 'dangerous' or 'deadly'! That particular brand of steroid is called 'androgenic-anabolic steroids'. Androgenic refers to the premise that the steroid has 'masculinising effects' which could include development of secondary male sexual characteristics such as body hair or development of genitalia. Anabolic refers to the fact that the steroid can create 'anabolism'; a state in which 'macro-molecular' synthesis takes place. For example, in the realm of bodybuilding this could refer to muscle cell hypertrophy.

Now it is not within the remit of this article to identify and discuss the physiological principles by which AAS exert their influence on the various cells of the body. However, an overview of the theory behind the use of AAS might be of benefit at this juncture.

In order to repair damaged muscle tissue a naturally derived hormone termed testosterone is released by Leydig cells in the testes. Testosterone is a 19-carbon steroid hormone which belongs to the androgen class of hormones. Among its many roles testosterone increases the rate of protein synthesis. This results in increased hypertrophy and thus increased strength. In addition to the increased protein synthesis, testosterone also reduces the catabolic effects of Cortisol. The simplistic premise behind the use of AAS is that 'more is better' i.e. injecting AAS increases protein synthesis and reduces catabolism more than by natural means alone. In addition to this premise, by manipulating the 'chemistry' of these synthetically derived hormones, even more exciting physiological outcomes are possible. For example, trenbolone, a synthetic androgenic steroid has the potential not only to increase muscle mass but also to decrease body fat levels.

Having now identified that the principle behind AAS utilisation is to increase the amount of testosterone in the blood and thus availability to muscle; how much testosterone does a male naturally produce per day? The average 20 year old male will produce between 6mg and 8mg of this hormone per day. The predominance of the hormone is produced in the morning and the production tapers off throughout the day. Testosterone production levels peak in males at around the 25 to 30 year old juncture. Another factor that must be considered is that as a male grows more mature in years their natural testosterone production significantly decreases. Decreases occur at around 1.5% per year after peak production has been realised. This means that at the age of 50 years the average male has 30% less testosterone being produced. So how does this relate to AAS utilisation? Well let's reconsider our 20 year old male who is producing approximately 50mg of testosterone per week. If he was to take just a one 1ml injection of Sustanon 250; a highly popular AAS; he would now have, in just that one injection, 165mg of testosterone available to his cells. When you consider that many beginners inject as much as 500 – 750mg per week in their initial cycles you now gain an understanding of the advantage of AAS interventions and the potential for huge increases in anabolism.

There are a few more factors to consider in respect of AAS. Firstly let us deal with the androgenic anabolic ratio. Many steroids have been synthetically produced to reduce the androgenic effects while increasing the anabolic effects. However, it is a general rule of thumb that the steroids with the greatest anabolic effect also have the greatest androgenic effect. For example, Nap 50's or Anadrol (oxymetholone); is one of the strongest anabolic compounds available. However, it also has a highly androgenic effect and would therefore be unsuitable for female athletes who wished to remain 'de-masculinised'.

Now let us discuss one more significant factor, *'aromatization'*. Aromatization refers to the natural process that occurs in the body which results in the conversion of testosterone into oestrogen. This process is carried out to maintain a homeostatic environment. It is termed aromatization due to the fact that an enzyme called

aromatase carries out the conversion. Testosterone converts more readily to oestrogen in later life in males due to increased aromatase activity. In addition to this aromatase is predominantly found in fat cells so individuals with greater fat cell deposition are more likely to suffer testosterone to oestrogen conversions, which in turn can lead to greater deposition of body fat. A vicious circle indeed! With respect to steroid utilisation, the greater the testosterone content in the cells, the greater will be the potential for aromatization to occur. This is an issue because the effects of increased oestrogen production are feminisation of the male. This could include factors such as 'Gynecomastia'; which is the development of 'breast' tissue in the male subject. A somewhat irreversible condition that results in unsightly growths, usually around the nipples.

There are some steroids that do not aromatize such as Anavar (Oxandrolone). But as has been stated previously, the more potent AAS do have a high potential for this mechanism to occur. With this in mind there are methods for minimising such factors. For example, Clomid, a selective oestrogen receptor modulator, while not having the ability to decrease testosterone conversion to oestrogen; can block the cell receptor sites to which oestrogen might attach. It does so by binding to those sites itself. Another more powerful drug called Arimidex (Anastrozole) may also be used in the battle against aromatization. Arimidex is used in the treatment of female breast cancer. Breast cancer is exasperated by the conversion of androgens to oestrogen through the aromatization process. Breast cancer cells flourish within oestrogen environments. Arimidex has the ability to inhibit the aromatase enzyme from carrying out the conversion process and so less oestrogen is produced. In the case of AAS utilisation, Arimidex may be used while on a cycle or post cycle to reduce aromatization of the increased testosterone now present in the body.

Now we have determined a basic understanding of how AAS work and why an individual might gain some advantage from taking them; it is now necessary to examine what side effects an individual might experience if he/she were to implement an AAS cycle into their training regimen. This will be the subject discussed in part 2.

Part 2: The Side Effects of Anabolic Steroids

There has been much debate and conjecture about AAS and their efficacy and safety as an ergogenic aid. Banned in all sports, they are seen as a 'cheat' mechanism that goes against the whole premise behind sport, 'a level playing field'. Over the past few years many individuals have been banned from high level competition due to the use and detection of AAS among other substances. However, this article will not look at the question of whether using AAS is cheating or otherwise. This article will look at what evidence exists to support the various governments, the sporting bodies and most of society's perception that AAS are highly dangerous drugs.

For purposes of that investigative process, the author would first like to state that he does not in any way wish to offend or detract from the loss that individuals have suffered due to the perceived use of AAS; and that any opinions offered herein, are offered as objectively as possible by highlighting all of the details of the cases that are discussed.

With that said, let us now look at why there is such a huge cloud over the use of steroids in terms of health implications. The furore has been fuelled by a string of deaths among sportsmen and bodybuilders over the past two decades or so. What follows is a list of some of the deaths that have arisen in professional athletes in recent years. It is by no means extensive or all inclusive.

Famous Bodybuilders (Deaths)		
Name	**Age**	**Cause**
Mike Mentzer	49	Heart Failure
Andreas Munzer	31	Heart Failure
Charles Durr	44	Enlarged Heart
Robert Benavente	30	Massive Heart Attack
Eduardo Kawak	47	Heart Attack
Luke Wood	35	Kidney Failure
Nasser El Sonbaty	47	Heart Failure

Famous Wrestlers (Deaths)		
Name	**Age**	**Cause**
Randy Savage	58	Massive Heart Attack
Eddie Guerrero	38	Heart Attack
Ultimate Warrior	54	Massive Heart Attack
Rick Rude	40	Heart Failure
British Bulldog	39	Heart Attack
Big Boss Man	41	Heart Attack
Umaga	36	Heart Attack

Most of these athletes competed in America where the average age of death for a male is currently 78.7 years. Some of the athletes named above died in earlier decades but in all cases their deaths can certainly be viewed at best, as being premature. Especially when one considers that many of them were still competing and therefore would have been at the height of their fitness. If one were too statistically analyse the number of deaths per 'sport' population against deaths in general population, I am sure that the findings would be significant in terms of the sporting professionals having a higher than average death rate in relation to age. But this still does not evidence in any way that AAS are the causal influence behind these deaths. The 'Centre for Disease Control Prevention (CDC)' identified that '*congestive heart failure hospitalizations for those under age 65 increased from 23% to 29% over a 10 year period'* (Hall, M. 2012) in America. This indicates that heart disease cases have risen by 6% in under 65 year old individuals in the last 10 years. Is everyone taking steroids?

The media is very quick in jumping on the steroids 'bandwagon' whenever a famous athlete dies. In respect of this comment, although it does not involve an elite athlete, the following story is perhaps a good example of such media ignorance and 'propaganda', heightened by the emotionality of parental love.

On June 26th 2012 the 'Mirror' newspaper ran the headline '*Steroids Killed Our Son'*. The story was reporting on a mother's viewpoint regarding the reasons why her son of 17, very sadly died. The tagline read '*Teenager tried to bulk up his muscles. Within weeks he was dead*'. Anyone with a minuscule knowledge of physiological adaptive processes would understand that for morphological changes to happen to the body, in this case the brain, a far greater time span would be required. Even if steroids were being significantly abused I am not aware of an AAS that has this majorly concerning side effect. Other parts of the story read '*While a post-mortem was inconclusive, Tina believed the muscle-building drugs, which he bought illegally, caused his brain to swell*' (Nilufer, A. 2012). Should the statement of a grieving parent be perceived as evidence over and above that of an experienced medical pathologist? This is subjective sensationalist reporting at its best. The rest of the story explains the symptoms that her young son experienced prior to death after just '3 weeks' on the steroids. Again, why I am not a medical practitioner, I have never experienced any other story that states that AAS could cause such symptoms so fast. Unless the batch was tainted or 'cut' down with another drug. The evidence goes on to state that the two 'anabolic steroids salesman' had been prosecuted for

selling 50 tablets. This would equate to approximately 3 dianabol a day for 3 weeks at the weakest end of the steroid spectrum to 3 nap 50's at the other end of the continuum. This is by no means extreme dosing and should not have caused such tragic events to occur. The summary of this one story epitomises the whole media propagated ignorance with respect to the use of AAS. This bias reporting method nurtures a sociological belief that steroids are harmful drugs. It's now time to look at the science or lack of it, behind this claim.

Deaths as a Result of Using Steroids

There can be no blanket statement that steroids do or do not cause fatal medical issues. Statistics cannot illustrate the likelihood of steroids being a causal factor in recorded deaths. For example, if an individual dies from a heart condition such as heart failure or heart attack; the death will be recorded as exactly that, even if steroids might have been an influential factor in the development of that heart condition. So it is not the intention of this discussion to attempt to interpret such data. Instead, the intention is to provide information which will allow an informed opinion.

The figures below indicate the causes and number of deaths within the United Kingdom in 2012.

UK Causes of Death Statistics (Males) (Source: Office for National Statistics -2012)

Cause of Death (Medical Condition)	Statistic
Heart Disease	37,423
Lung Cancer	16,698
Stroke	14,116
Prostate Cancer	9, 698
Bowel Cancer	7,841
Throat Cancer	4603

Cause of Death (Causal Factors)	Statistic
Smoking	47,300
Alcohol	5,438
Drug Poisoning	1,706
Suicide	4,590
Obesity	151
Steroids	0

During the research for this article, the author could not find one definitive statistic that could categorically determine steroid's as being the influential factor behind any single death. Any yet we are informed that smoking is the influential factor in 47,300 death's, through various methods such as cancer, atherosclerosis and heart disease. Why is this linkage not possible with regard to AAS? The answer is simple. There is not enough research data available, as most governments will not allow such research to be carried out. Some research has been carried out in to deaths from AAS use.

Darke, S. and Torok, M. (2014) researched the deaths of 24 males who all died at a mean age of 31.7 years. The characteristics, pathology and toxicology of the individuals were investigated. Characteristically the causes of death were accidental drug toxicity (62.5%), suicide (16.7%) and homicide (12.5%). In terms of drugs profiles, the following AAS profiles were found. Abnormal testosterone epitestosterone ratios were reported in 62.5%, followed by metabolites of nandrolone (58.3%), stanozolol (33.3%), and methandienone (20.8%). A key fact herein is that 23 of the 24 individuals tested positive for 'other' chemical substances including psychostimulants. In about 50% of the individuals there was also testicular

shrinkage. Several of the individuals had increased left ventricle hypertrophy and narrowed coronary arteries.

The issue with this research is not the method or subsequent findings, it is the way in which other agencies or individuals such as the media, bloggers and anti-steroid proponents have represented this work. Headlines such as '*Sudden or un-natural deaths involving anabolic androgenic steroids*' deviate the findings in a massive way and totally discount all of the evidence that has been represented. For example, nearly 30% of the individuals died as a result of homicide or suicide. In what way is the blood steroid profile relevant to this 'fact'? The other individuals died as a result of drug toxicity which can include anything from overdose, allergic reaction or 'bad' drugs. The fact that many of these subjects used stimulants has been totally discounted from the headline. This is a spurious representation of research findings when that research stated that '*psycho-stimulant toxicity was the direct cause of death in eight of the 24 deaths and opioid overdose was the direct cause in seven'*. The researchers have also not mentioned that stimulants can place a stress on the heart that can also increase left ventricle hypertrophy. Science is often misinterpreted to suit the agendas of individuals and not to divulge the truth.

Another study on adult male mice did provide some quite damning evidence to the proponents for steroid use. Bronson F.H., Matherne C.M. (1997) carried out a study on mice. The test group were exposed to four AAS for a period of 6 months. They were given the AAS at 5 or 20 times the normal levels that would be expected for mice. One year later the mice were around 20 months old. The following markers were found as a result of this experiment. 52% of the mice who were given the higher AAS dose had died. This was compared to 35% who had been given the lower dose. The control group had a far lower 12% deaths. When autopsied the mice who were given' the AAS were typically found to have tumours in their livers and kidneys and some heart damage. Some mice had all of these conditions.

This study clearly identified that AAS do have some negative health implications and might certainly, in this instance, have resulted in premature death rates of the mice. However, it might be assumed that any significant overdose of any drug might have similar negative effects. Also, would the dosage being administered orally or intra-muscularly make a difference to these results? The death rate was dose dependant i.e. the greater the dose the more damaging the effect. This infringes on the premise of 'use' or 'abuse'. Would a bodybuilder using smaller doses in a medically correct way, experience less physiological damage while reaping the muscular benefits? Finally, would the use of aromatase inhibitors such as Arimidex have reduced some of the negative health implications? Sometimes 'answers' require so many more questions. But this is the nature of science!

There are many more studies that are relevant to this specific area; but for now we will move away from the morose study of drugs and death rates and move towards medical implications short of the individual demise.

Physiological Implications of AAS Utilisation

There are many physiological implications that are purportedly related to AAS utilisation. Some of the areas that might be of significance are:

Cardiovascular	Endocrine	Hepatic
Increase in LDL	Gynecomastia	Liver vascular injury
Increased BP	Testicular Atrophy	Tumours
Decreased Myocardial Function	Impotence	Enzyme elevations
	Decreased Testosterone	

There are also some genitourinary issues such as reduced sperm count, menstrual irregularities and masculinization. But for this section we will discuss cardiovascular based issues.

In terms of cardiovascular issues there are many implications that are theorised to occur as a result of AAS use. Some of these include left ventricular hypertrophy, reduced left ventricular function, arterial thrombosis, pulmonary embolism, increased blood pressure and myocardial infarction.

Left ventricle hypertrophy is one of the theorised implications of AAS utilisation. Most studies that use echocardiographic methods of measuring heart size exemplify the fact that bodybuilders or powerlifters have significantly increased heart wall (myocardium) wall thickness. In post mortems this physiological adaptation is also noted and is often attributed to the use of AAS which have been found in greater than normal amounts in the blood. Rather a clear cut argument in the first instance but a study by Dickerman R.D., Schaller F., McConathy W.J. (1998) provided a significant counter argument.

The researchers examined 4 elite resistance-trained athletes using two-dimensional echocardiography. They also examined data from the individual left ventricular dimensions of 13 bodybuilders from previous echocardiographic studies. All 4 elite resistance-trained athletes had left ventricular wall thicknesses beyond 13 mm. From the previous studies, 43% of the drug-free bodybuilders and 100% of the steroid users had left ventricular wall thickness beyond the normal range of 11 mm. In all of the subjects there was no indication of diastolic dysfunction. The research demonstrated that left ventricular wall thicknesses was found routinely in elite resistance-trained athletes who have not used anabolic steroids.

However, in a study by Baggish *et al* (2010), 19 weight lifters of whom 12 were long term AAS users and 7 whom were non AAS users, were examined using echocardiograph to assess left ventricle ejection fraction and left ventricle systolic strain. The research identified that left ventricle structural parameters were the same between the individuals. However, the AAS users had significantly lower left ventricle ejection fraction (50.6% - below normal). The AAS users also demonstrated decreased diastolic function. The researchers arrived at the conclusion that cardiac dysfunction was sufficiently higher in AAS users and might be sufficient enough to increase the risk of heart failure.

Now let us look at lipoproteins. Lipoproteins are special particles made up of droplets of fats surrounded by a single layer of phospholipid molecules. There are several types of lipoproteins, each with specific functions and profiles; for the purposes of this discussion we will focus on Low Density Lipoproteins (LDL) and High Density Lipoproteins (HDL). LDL is considered to be the ‘bad’ lipoprotein as it is a carrier of cholesterol. The rationale behind this premise is that LDL Cholesterol is responsible for the build-up of plaque on arterial walls leading to conditions such as atherosclerosis. HDL’s are considered the good cholesterol and higher levels are desirable. HDL is believed to ‘scavenge’ and take LDL cholesterol away from the arteries. The question now arises is what is the relationship between steroid use and HDL/ LDL profiles.

Hartgens *et al* (2004) established that over a 14-week period AAS administration led to decreases in serum concentrations of HDL. This would be potentially detrimental. Hartgens and Kuipers (2004) also proposed in a further study that illustrated steroid use had profound effects on the cardiovascular system, mediated by the occurrence

of AAS-induced atherosclerosis (due to unfavourable influence on serum lipids and lipoproteins). Many other studies concurred with similar findings regarding the LDL levels. However, it must also be pointed out that some studies are showing AAS interventions might have beneficial effects on LDL and HDL profiles.

As with all areas regarding science and the multi-dimensional human body, studies often show controversial or opposing findings. There is no doubt that anabolic steroids do have significant effects on the body. It is not in the remit of this article to cover each and every one. That would take an entire encyclopaedia of information. But as a conclusion to part two of this article, it must be realised that any drug has side effects and these may be beneficial or damaging. The actions they exhibit are based on a plethora of individual factors. We will discuss the physiological implications of steroid use in further articles.

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