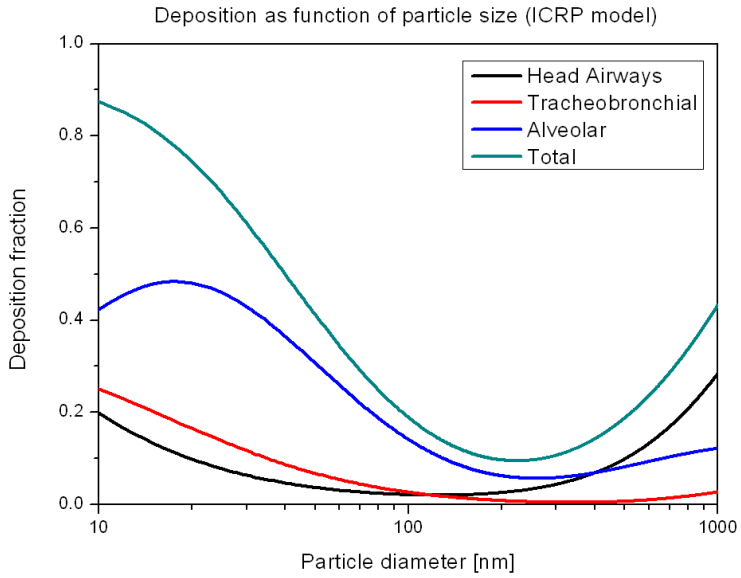
miniDiSC application note #8: lung-deposited and active surface area

Particulate matter can be measured in many different ways. Traditionally, the particle mass per unit air volume is measured, usually with an upper size limit of x microns (PMx - with PM10, PM2.5, PM1 most common). Alternative metrics are e.g. particle number, or particle surface area per volume. These are purely physical metrics. Chemistry can also be taken into account, e.g. by measuring the amount of black carbon, or polyaromatic hydrocarbons (PAH). There is no such thing as the "best" metric to use - it always depends on the application.
One general observation when discussing health effects caused by particles is that the traditional reporting of any quantity per unit volume of air is not too meaningful. What is more interesting is the amount of this quantity that ends up in the human body. The deposition fraction as function of particle size for three different areas of our airways is shown in the figure below.



We can see that the total deposition has a clear minimum at about 200-300nm, where only about 10% of the particles present in the air are deposited in our body, while at 40nm diameter, about half the particles end up in our body. On a mass basis, a single 200nm particle (unit density, spherical) is 125x heavier than a 40nm particle, and contributes 125x more to the measured PMx, although it contributes "only" 20x more to the mass ending up in the human body. We can thus conclude that - at least concerning health effects - we should look at deposited particles only.
Several laboratory studies have demonstrated that on a mass-basis, smaller particles appear to be more toxic than larger particles. This is usually explained by the larger surface area of the smaller particles: the particle surface is the place where our body interacts with the particles. Particles can transport adsorbed toxins on their surface, and their surface can act as catalyst inside a cell, creating reactive oxygen species (ROS). It has been shown that toxic effects scale well with particle surface area in in-vitro tests. Of course, these statements only pertain to biopersistent particles, and not to soluble particles. In my opinion, there is enough evidence to claim that the surface area is a more important metric than particle mass for biopersistent particles. We should therefore measure the **lung-deposited surface area (LDSA),** it appears to be the most relevant physical metric for quantifying exposure to particles.

Measuring the LDSA in principle requires a measurement of the entire particle size distribution, followed by a summation of particle surface in each size bin weighted by its lung-deposition probability, i.e. this would require an SMPS and some calculation. However, by what can only be called a lucky coincidence, LDSA can be measured directly by diffusion charging. Diffusion chargers impart a size-dependent charge q on particles passing through them, which can be well described by

$q \sim= d^{1.1}$

where d is the particle diameter. In the lung deposition curve, one can see that the particle deposition in the lower airways is approximately inversely proportional to particle diameter in the diameter range of 20-300nm. LDSA is thus approximately proportional to the diffusion charger signal:

LDSA = surface area * deposition probability = $d^2 * d^{-1} = d^1 = \sim q$

Obviously, this calculation is only approximately true, and only for spherical particles. Nevertheless, it seems a sensible way of interpreting the DC signal. Another measure of the surface area was invented by my Ph.D. advisor, H.C. Siegmann: the active surface (AS). It is a measure of the surface area of the particle which interacts with the surrounding gas. For particles smaller than the mean free path of the gas molecules (60nm at standard conditions), this is the same as the geometric surface area ($AS \sim d^2$), for particles larger than the mean free path, it is proportional to the particle diameter ($AS \sim d^1$). In between the two regimes, there is a smooth transition. AS is an interesting property, e.g. it is directly proportional to the rate of condensation of volatile species on the particles ("condensation sink"). However, it seems irrelevant in the context of nanoparticle interactions in cell fluids. A common misconception (invented by H.C. and propagated by some instrument manufacturers) is that diffusion chargers directly measure a signal proportional to the AS. This is wrong on three counts:

1) The mean free path of ions responsible for charging the particles is much smaller than the mean free path of neutral gas molecules, thus shifting the transition from free molecular regime to continuum regime to a lower diameter.

2) The coulomb repulsion inhibits particle charges larger than 1 elementary charge, whereas there is no such barrier for neutral species. This inhibition leads to a lower charge for larger particles and a correspondingly lower slope in the charge-vs-diameter curve.

3) The ubiquitous image force between particle and ion is attractive for uncharged particles, and therefore increases charging for the first charge to arrive on a particle. This leads to a $d^{1.5}$ dependence of the average particle charge instead of d^2 as expected in the free molecular regime.

For a further discussion on particle surface area and health effects, as well as LDSA measurement, look at the two following papers:

M.Auffan et al: Towards a definition of inorganic nanoparticles from an environmental, health and safety perspective. Nature Nanotechnology, 4:634-640 (2009).

C. Asbach et al: Conceptual limitations and extensions of lung-deposited Nanoparticle Surface Area Monitor (NSAM). Journal of Nanoparticle Research, 11:101-109 (2009).