

Intreerede / Inauguration address – Prof. JL du Plessis

4 Augustus 2017 / 4 August 2017

Die VEL in velblootstelling / The SKIN in skin exposure

Verwelkoming en inleidende opmerkings

Ek wil een en elkeen van u ter harte dank vir u teenwoordigheid.

Wat 'n voorreg is dit om vanaand my intreerede te kan lewer. Voordat ek daarmee voortgaan wil ek graag die volgende met u deel:

(1) Eerstens, 'n deel van die intreerede gaan in Afrikaans, my moedertaal gelewer word. Ek kry baie min die geleentheid om Afrikaans as 'n wetenskapstaal te beoefen.

Vir die dele wat ek in Afrikaans aanbied, sal die universiteit se knap tolkdienste my boodskap in Engels oordra. Die skyfies sal deurgaans in Engels wees, net om dit vir myself moeiliker te maak.

(2) Tweedens, die intreerede skep ook die geleentheid om te reflekteer oor waar ek vandaan kom, hoe ek hier uitgekom het, en sleutelfigure hierin te identifiseer, te erken en te bedank.

Die uitleg van my intreerede is as volg:

- My agtergrond en onderbou
- Die vel: struktuur en funksie
- Velsiektes en Beroepsvelsiektes
- Velblootstelling en Beroepsgesondheid en veiligheidswetgewing
- Velblootstelling
- Velgrensfunksie
- Veldeurlaatbaarheid
- Huidige en toekomstige navorsing
- Samevattende opmerkings

My agtergrond en onderbou

Laerskool

As ek terug dink, is my belangstelling in wetenskap en biologie aangewakker deur my Standaard 5 (nou Graad 7) wetenskap en gesondheidsopvoeding onderwyser, mnr. Gerrie ("Grysmuis") Germishuys. In 'n klein laerskool het hy ons uitgedaag en met tye alreeds blootgestel aan Standaard 9 Biologie vakinhoud.

Hoërskool

In Standaard 9 moes ons 'n Biologie taak voltooi oor enige onderwerp en ek het kanker as onderwerp gekies. Gedryf deur my eie ouma as gevalle studie het ek 'n handgeskrewe taak (in hoegesonde Sondagskool handskrif) geskryf, 83 bladsye in lengte, getiteld: Kankerfobie – kanker kan genees word. Lissinda het opgemerk dat dit my eerste oorsig artikel was. Dit het 'n blywende indruk gemaak.

Universiteit

Aan die einde van my derde jaar spandeer ek 'n maand in prof. Willie van Aardt se Dierfisiologie laboratorium. As ek reg onthou moes ek die dissosiasie (ontbinding) van suurstof vanaf strandkrappe se bloedselle vasstel. Wat ek goed onthou, is dat ek R5 per uur betaal is.

Aan die begin van my Honneurs in 1997 het prof. Nico Malan (as Departementshoof) aan personeel meegedeel dat hy 'n dosent benodig vir 'n module. Ewe braaf het ek myself gaan verkoop en begin om Fisiologie aan te bied vir Farmasie studente.

Onder prof. Jurg van der Walt en later Dr. Karin Dyason het ek 'n Honneurs en Meestersgraad verwerf waarin my navorsing aangetoon het dat daar sekere toksiene teenwoordig is in Suid-Afrikaanse skerpioenspesies wat porieë (gaatjies) in selmembrane maak en so inmeng met die selle se funksionering (membraanpotensiaal).

Met die aftrede van prof. Jurg van der Walt en Dr. Karin Dyason wat na Pretoria verhuis het, het Membraan en -elektrofisiologie min kans op oorlewing gehad. Ek het met 'n Doktorsgraad begin, maar elektrofisiologie was duur navorsing, tegnies moeilik en alleen onmoontlik. Met my voltydse aanstelling in 2004 was die skrif aan die muur en ek het gekies om my te bekwaam in Beroepshigiëne. As ek terugdink, was dit die toksikologie deurslaggewend in die "omskakeling", met velblootstelling aan nikkel (n metaal wat ons gereeld in elektrofisiologie gebruik het om ionkanale te blokkeer) as 'n navorsingsprojek in 2007.

Voordat ek aandag gee aan velblootstelling en die rol van die vel in velblootstelling, is dit nodig om konteks te verskaf. Daarom 'n kort oorsig van die struktuur en funksie van die vel en die voorkoms beroepsvelsiektes.

Die vel: struktuur en funksie

Die vel is die grootste orgaan in die menslike liggaam. In 'n gemiddelde volwassene (65 kg), beslaan dit 10-15% (± 7 kg) van die totale liggaamsmassa. Dit bedek 'n oppervlakte van ongeveer 2 m² (Sithampanadarajah, 2008).

Elke 1 cm² bevat:

- 3 miljoen selle
- 10 hare
- 100 sweetkliere, 15 olie kliere
- 14 temperatuurreseptore, 200 pynreseptore, 25 drukreseptore
- 3000 senuwee eindpunte, 1.5 m senuwees
- 1 m bloedvate (Sithampanadarajah, 2008)

Die vel funksioneer as 'n twee-rigting beskermende "grens" tussen die liggaam en die eksterne omgewing. Dit is voortdurend aan die self-herstel/hernuwe, 'n metabolies aktiewe grens ("barrier"), wat dit wat binne is, binne hou, en dit wat buite is aan die buitekant hou (Sithampanadarajah, 2008).

Behalwe vir die beskermende funksies, speel die vel 'n belangrike rol in liggaamstemperatuurregulering en die beperking van water verlies (Sithampanadarajah, 2008). Die ander funksies van die vel is as volg:

- Die vel is verantwoordelik vir die uitskeiding van substansie saam met sweet.
- Dit verskaf ook meganiese ondersteuning.
- Dit vervul verskeie immunologiese funksies, waarvan sommige prominent is in beroepsvelsiektes.
- Dit is ook 'n sensoriese orgaan – en neem druk, hitte, koue en pyn waar.
- Die vel is ook verantwoordelik vir Vitamien D sintese.

Die vel bestaan volgens Sithampanadarajah (2008) uit drie lae, die hipodermis aan die binnekant, die dermis in die middel en die epidermis.

Die binneste hipodermis, wat 'n paar millimeter dik is, dien as brug tussen die onderliggende liggaamsdele en die middelste dermis. Dit anker die middelste dermis aan spiere en been en bestaan uit vetweefsel. Ook opvallend is die teenwoordigheid van bloedvate.

Die dermis (in die middel) is ongeveer 3-5 mm dik en bestaan uit bindweefsel en elastiese weefsel wat meganiese sterkte en buigbaarheid/plooibaarheid verskaf. Ingebed in, of wat deur die dermis strek, is haarfollikels, olie kliere, sweetkliere, senuwee eindpunte, bloedvate en limfvate.

Die buitenste epidermis, van belang omdat dit blootgestel is aan die eksterne omgewing, is meerlagig. Die dikte verskil vanaf 0,05 mm (die dikte van 'n snesie) tot 0,8 mm (op die handpalms en voetsole). Daar is geen bloedvate in die epidermis nie en enige substans sal oor die laag moet diffundeer om die dermis te bereik. Die epidermis bestaan uit vier tot vyf lae:

- Basale laag – stratum germinativum: verskaf selle aan die buitenste lae. Ander selle hier teenwoordig, sluit Langerhansselle (immuunselle) en melanosiete (sekreteer melanien, verantwoordelik vir velkleur) in.
- Stekelsellaag – stratum spinosum: bestaan uit 2-6 lae selle wat keratien sekreteer.
- Granulêre sellaag – stratum granulosum: selle differensieer en word platter en het 'n korrelagtige voorkoms.
- Horinglaag – stratum corneum: Dit neem ongeveer 14 dae vir basale laag selle om korneosiete te word. In nog 14 dae skilfer die selle vanaf die liggaam. Die stratum corneum word beskryf as 'n "brick-and-mortar" (baksteen en sement) struktuur, wat bestaan uit lae "dooie" gekeratiniseerde selle (korneosiete, "bricks") wat ingebed is in 'n vetterige (lipiedryke) matriks ("mortar"). Dit is 2-10 µm dik wanneer dit droog is. Die stratum corneum word beskou as die primêre grens ("barrier"), verantwoordelik vir die regulering van liggaamswaterverlies en die beskerming teen eksterne stressors/gevare.

Skin diseases

The Global Burden of Disease Study of 2013, published earlier this year, estimated that skin conditions contributed 1.79% to the global burden of disease (as measured in DALYs – Disability-Adjusted Life Years¹) (Karimkhani *et al.*, 2017). This ranked skin and subcutaneous diseases as the 18th leading cause of disease (41.0m DALYs). As a reference point, the leading cause has been ischemic heart disease (150.2m DALYs). Dermatitis, which includes atopic and contact dermatitis contributed 0.38%, malignant skin melanoma 0.06% and keratinocyte carcinoma 0.03% to this burden. In Southern sub-Saharan Africa, dermatitis contributed the most to the burden.

Occupational skin diseases

Occupational skin diseases are caused by chemical, physical and biological agents and mechanical trauma (NIOSH, 2012).

- Chemicals – irritants, sensitisers and wet work (water – dihydrogen oxide)
- Physical stressors – extreme temperature (hot and cold) and UV-radiation
- Biological stressors – micro-organisms, parasites, plants and animal material
- Mechanical trauma – friction, pressure, abrasions, lacerations and contusions (scrapes, cuts and bruises).

In the case of chemicals, there are three chemical-skin interactions (Semple, 2004):

- A chemical may become systemic after passing through the stratum corneum (eventually having an effect elsewhere in the body).
- It may act locally, causing irritation or burns.
- It may induce allergic reactions.

Diffusion through the stratum corneum via the transcellular route, extracellular route or via the "shunt" route (hair follicles) (Sithamparanadarajah, 2008).

The in-depth details of the prevalence and incidence of skin disease and that of occupational skin disease globally, and in South Africa is beyond the scope of my inauguration address. However, I want to convey the following:

Europe

Occupational skin diseases constitute up to 40% of all notified OD involving contact dermatitis, contact urticarial and in some countries skin cancer (Coenraads *et al.*, 2011). It is characterised by under-diagnosing and under-reporting (Alfonso *et al.*, 2017). The average incidence of occupational contact dermatitis is around 0.5-1.9 cases/1000 workers/year (Coenraads *et al.*, 2011).

¹ DALY = YLL (Years of Life Lost to a disease) + YLD (Years Lived with Disease). One DALY equivalent to 1 year of healthy life lost.

Occupational skin disease related costs exceed 5 billion Euro/year, due to lack of productivity, causing detrimental socio-economic consequences due to often high rates of sickness absence, job loss and life-long disability (EU25 Report, 2009; Wulfhorst *et al.*, 2011).

United Kingdom (1999-2014)

In 2015, about 1500 new cases of occupational skin diseases were recorded. Working with wet hands, i.e. “wet work” and contact with soaps and cleaners accounting each for 13-14% of all cases, but increased to around 25% in 2013-2015. Other common agents include rubber chemicals and materials, personal protective equipment (including latex gloves), preservatives, fragrances and cosmetics and nickel. Occupations with the highest rates are florists, hairdressers, cooks, beauticians, and certain manufacturing and health care related occupations (HSE, 2016):

South Africa:

The South African Occupational Skin Disease burden has been described in 2008 as a “hidden epidemic” characterised by under-reporting, under-diagnosing and inadequate compensation (Carman and Kruger, 2008) and nothing has really changed since.

Two regional clinics provide vital, but limited prevalence statistics on Occupational Skin Diseases:

- The Occupational Skin Disease Clinic at the National Institute for Occupational Health (NIOH, Gauteng, Johannesburg) diagnosed 141 patients (65%) of 216 referred patients with occupational skin diseases, mainly contact dermatitis, in 2006-2009
- The Occupational Dermatology Clinic at Groote Schuur Hospital in Cape Town diagnosed 31 (52.5%) of 59 referred patients with occupational skin diseases, with 25 cases being contact dermatitis.

Compensation for occupational diseases in South Africa is complex, with the co-existence of the National Compensation Fund and private insurers for specific industries. Data is also not freely available. In 2009, the incidence of occupational skin diseases according to the Compensation Fund was 0.016 cases/1000/year, for the mining industry 0.043 and construction 0.097 (Carman and Fourie, 2010).

The incidence of Occupational Contact Dermatitis is, therefore, about 30 times lower than that reported in Europe (0.016 vs 0.5 cases/1000/year).

Velblootstelling en Beroepsgesondheid en veiligheidswetgewing

Legislation [as contained in Regulations of the Occupational Health and Safety Act (Act 85 of 1993) or the Mine Health and Safety Act (Act 29 of 1996)] addressing the skin as a route of exposure is very limited when compared to respiratory exposure. The reasons thereof are however, beyond the scope of this presentation.

A few guidance notes on skin absorption, precautions to be taken in the event of skin exposure and that control measures should be put in place, are provided. In addition, two types of notations (“warning labels”) are given.

- A Sk-notation is assigned to substances that have the ability to penetrate the intact skin and thus become absorbed in the body. However, the focus is on skin absorption and not on substances having local effects on the skin. As an example, nickel does not have an assigned skin notation, yet it is the leading cause of allergic contact dermatitis.
- A Sen-notation. Certain substances may cause sensitisation of the respiratory tract if inhaled or if skin contact occur. Unfortunately, a Sen notation is assigned only to those sensitisers that may cause sensitisation through inhalation.

We have shown that skin notations as indicated in the South African Regulations matches (agree) those of other leading countries by less than 50% and for sensitization notations it was only 3.6% (du Plessis *et al.* 2010).

Velblootstelling / Skin exposure

Ons hande is instrumente, en dit is baie moeilik om aan 'n werk ('n beroep) te dink, waar ons nie ons hande gebruik nie. Op die oog af, klink velblootstelling ook eenvoudig (so het ek ook aan die begin gedink), maar is alles behalwe eenvoudig.

Back in 1999, Schneider *et al.* (1999) developed a conceptual model for assessment of skin exposure. This model indicates:

- Contamination of the skin may arise in many different ways.
- A contaminant emitted from a source (through splashing, spilling or ejection of particles) may land directly on the skin.
- It may be emitted from a source to the air, from where it may deposit on clothing or the skin or on other (workplace) surfaces.
- They may be transferred to the skin from contact with contaminated (workplace) surfaces or by submersion of a body part into the contaminant.
- The contaminant may also be resuspended from the skin or surfaces to air.
- A contaminant may also be removed from the skin through washing (decontamination) or from air (ventilation of room air)
- A contaminant may also be redistributed, i.e transported from one part of the skin to another (touching face with contaminated finger).
- The presence of personal protective clothing and clothing may alter contact of the contaminant with the skin.

As acknowledged by the authors, this model has a simplistic view of the skin contamination layer. In the following research we "played" around within the domains of this model, but we also ventured into the stratum corneum barrier and skin permeation.

My skin exposure research stemmed from a leading mining company indicating that they wanted to know whether there is skin exposure to nickel at one of their base metal refineries, and if so, they wanted a method to assess skin exposure.

In a base metal refinery an electrolytic process is used to recover metallic nickel from a nickel sulphate (sulphuric acid) solution. Findings from my first study (du Plessis *et al.*, 2010):

- We implemented an easy-to-use, practical protocol for assessing skin exposure to nickel and concurrent workplace surface contamination.
- Even with the supposed use of protective gloves, skin exposure to nickel occurred on the hands (index finger, palm of hand), but also on the neck and forehead, areas not covered by personal protective clothing. Neck and forehead contamination resulted from transfer from contaminated hands and gloves.
- Exposure was highly variable (0.045 to 229.86 $\mu\text{g}/\text{cm}^2$ on palm) between individuals, which is a characteristic of skin exposure in general.
- Skin exposure was 5.4 to 6.3x higher than that of the only other comparative study, conducted in Europe, and published in the same issue of the journal.
- Selection of the correct type and correct use of personal protective gloves were of concern.
- Surfaces in the workplace were also contaminated – door handles, taps and tables.
- Also, contamination of supposed in "clean areas" such as the break room and change house (where overalls are collected).
- Detectible levels of nickel was measured on the skin even before the shift commenced – due to change house contamination and other contaminated workplace surfaces.
- Fifteen recommendations to eliminate/reduce exposure to nickel were made.

In another base metal refinery, with a different chemical process, dermal co-exposure to nickel and cobalt, also a well-known contact allergen, was shown (du Plessis *et al.* 2013):

- This was a novel study indicating skin exposure to cobalt in base metal refineries.
- Exposure to both nickel and cobalt occurred on the hands, wrists and forehead.
- Yet again exposure was highly variable between workers.
- We indicated skin exposure for different occupations, even those not directly involved in the refinery processes.
- Surfaces in the workplace were contaminated.
- 63.6% of nickel samples exceeded a proposed, but highly debatable, threshold for sensitization and likewise 22.7% for cobalt.
- Nine recommendation were made to eliminate/reduce exposure.

Similar studies were conducted in the packaging areas of a base metal refinery, for both cobalt (Eloff *et al.*, 2011) and nickel and in the refining of cobalt. In all instances, dermal exposure to either cobalt or nickel was confirmed.

Die vel in velblootstelling

Skin barrier function

Prof. Fritz Eloff and mr. Petrus Laubscher were of the opinion that occupational hygiene research should have some sort of “physiological” approach/angle. In planning the first dermal/skin exposure study, we stumbled on a device, called an EDS 12 from EnviroDerm Services in the United Kingdom, which had the capability of measuring skin hydration and transepidermal water loss (TEWL). This provided the “physiology” that was needed.

Skin hydration and TEWL have been widely used as indices in evaluating skin barrier function (Darlenski *et al.*, 2009).

Skin hydration, in normal skin around 20%, represents the water content of the stratum corneum (Pirot and Falson, 2004). TEWL represents the outward permeation of condensed water through the stratum corneum by diffusion, but excludes perspiration (Rogiers, 2001). In healthy and intact skin TEWL is low.

In the initial base metal refinery study we have shown that skin on the hands were normally hydrated or slightly dry at the start of a work shift, it significantly deteriorated to dry during a work shift, but recovered to normal levels by the end of the shift (du Plessis *et al.*, 2010).

High TEWL on the hands, even at the start of the shift were indicative of a low barrier function and deteriorated significantly during the shift. At the end of the shift barrier function was very low (Du Plessis *et al.*, 2010).

We have proposed that these refinery workers are exposed to a combination of skin irritants (sulphuric acid, pH = 3.5) and contact allergens (nickel). The irritants are responsible for the deterioration of the skin barrier, and will facilitate with time the permeation of nickel. Thus, there is an increased risk of sensitization. However, the ethnicity (lower incidence of ACD in black South Africans) and a “healthy worker” (with time having a group of workers not-sensitized) effect may be significant contributors toward the low incidence of ACD in the refineries included in this study. However, one can not accurately predict when a worker will become sensitized. So the risk remains as long as there is exposure. The concept of irritant dermatitis preceding and facilitating the development of allergic contact dermatitis, with impaired barrier function contributing to the concurrence of irritant and allergic contact dermatitis is now widely acknowledged (Kasemsarn *et al.*, 2016).

The acidic nature of base metals refineries processes triggered interest in the potential influence thereof on skin surface pH. The stratum corneum is covered by skin surface film liquids, consisting of aqueous sweat, sebum, by-products from skin surface maturation and desquamation processes and metabolites from skin bacteria (Stefaniak, 2014). The skin surface pH is acidic in nature (4.5 to 5.6), and the maintenance thereof play an important roles in stratum corneum integrity and cohesion, regulation of epidermal barrier homeostasis and maintenance of microbial flora balance (Stefaniak *et al.*, 2013). In cobalt refining, skin surface pH decreased by up to 13% during a work shift (du Plessis *et al.*, 2013). This means approximately 2.5 times more hydrogen ions present on the skin surface. At a lower pH the ionisation, solubility and skin permeation of nickel and cobalt increases (Larese Filon *et al.*, 2007; Larese Filon *et al.*, 2008). Therefore, the decrease in skin surface pH of refinery workers in all likelihood further facilitates the skin permeation and absorption of cobalt and nickel.

Two significant contributions to the measurement of skin barrier function in occupational settings resulted from a workshop hosted prior to the 5th Occupational and Environmental Exposure of Skin to Chemicals Conference in 2011 (Toronto, Canada):

- International guidelines for the in vivo assessment of skin properties in non-clinical settings: Part 1: pH (Stefaniak *et al.*, 2013).
- International guidelines for the in vivo assessment of skin properties in non-clinical settings: Part 2: transepidermal water loss and skin hydration (du Plessis *et al.*, 2013).

These papers provided guidelines on the endogenous (internal), exogenous (external), environmental and instrument factors involved and how they may influence measurement, recommendations on how to measure TEWL, skin hydration and pH, and how to report data as to promote consistent data reporting in future studies.

In principle they postulate measuring the consequence of exposure to skin contaminants.

Veldeurlaatbaarheid

Platinumsoute is wel bekend as respiratoriese sensitiseerders (allergene) wat lei tot onder andere asma en rinitis, maar gesensitiseerde persone kan ook presenteer met velkondisies soos ekseem en urtikarie (Calverley *et al.*, 1995; Linnett en Hughes, 1999; Merget *et al.*, 2000; Kiilunen en Aitio, 2007). Baie navorsing is gedoen op die respiratoriese blootstelling aan platinumsoute, maar velblootstelling is nie werklik in ag geneem nie. Dit terwyl Maynard *et al.* (1997) alreeds in 1997 voorgestel het dat velblootstelling as 'n alternatiewe roete van blootstelling kan wees wat bydra tot sensitisering. Hulle argument was, dat respiratoriese blootstelling baie laer as die respiratoriese beroepsblootstellingsdrempel is, maar dat blootstelling nogsteeds sensitisering van raffinadery werkers veroorsaak het.

Soute van rodium, een van die ander platinum groep metale, word ook beskou as 'n respiratoriese sensitiseerder wat respiratoriese simptome veroorsaak, maar ook urikarie en kontak dermatitis (Santucci *et al.*, 2000; Goossens *et al.*, 2011).

Anja Franken het in haar PhD, deur gebruik te maak van *in vitro* eksperimente (Franken *et al.*, 2014; Franken *et al.*, 2015), bevind dat:

- Platinum en rodium deur blanke vel kan beweeg, hoewel die deurlaatbaarheid baie laag is, maar ongeag hiervan nogsteeds sistemies versprei kan word en kan bydra tot sensitisering.
- Dat platinum (en rodium) in die vel akkumuleer/ophoop, wat as 'n "reservoir" kan dien en met tyd sistemies beskikbaar word of selfs verantwoordelik is vir die ontstaan van die velkondisies. Wat ons wel nog moet vasstel is presies in watter sellag/lae dit akkumuleer.
- Siende dat Suid Afrika se werksmag, en die in raffinaderye ook, nie net Kaukasiërs insluit nie, is die veldeurlaatbaarheid van platinum ook bepaal vir swart vel. Die deurlaatbaarheid van platinum en die massa wat in die vel akkumuleer was betekenisvol hoër in swart vel wanneer vergelyk word met blanke vel. Swart raffinadery werkers loop dus 'n hoër risiko indien velblootstelling sou plaasvind.

Sané Jansen van Rensburg het in dieselfde tipe eksperimente aangetoon dat die deurlaatbaarheid van rodium betekenisvol hoër is by 'n pH van 4,5 in vergelyking met 'n pH van 6,5 (Jansen van Rensburg *et al.*, 2017). Dit stem ooreen met die van ander metale soos nikkel, kobalt en chroom, wat by 'n laer pH beter ioniseer, hul oplosbaarheid en gevolglike veldeurlaatbaarheid verhoog (Larese Filon *et al.*, 2007, Larese Filon *et al.*, 2008). 'n Afname van een eenheid in pH kan lei tot 'n 100 voudige toename in veldeurlaatbaarheid (Larese Filon *et al.*, 2009). Die afname in veloppervlak pH, soos bevind in raffinaderye, fasiliteer dus velabsorpsie van metale soos nikkel, kobalt en rodium.

Huidige en toekomstige navorsing

Ons is tans besig om die bydrae van die respiratoriese- en velblootstellingsroetes tot die "totale" blootstelling aan platinum van raffinadery werkers te bepaal. In die projek bepaal ons respiratoriese blootstelling, velblootstelling en doen ook biologiese monitering, waar ons die platinum vlakke in urine bepaal. Hiermee saam ook die bepaling van velgrensfunksie (TEWL, hidrasie en pH). Ek wil nie in die detail hiervan ingaan nie, aangesien die data nog nie gepubliseer, en deur die wetenskaplike gemeenskap beoordeel, is nie:

- 25% van respiratoriese blootstellingsmonsters oorskry die beroepsblootstellingsdrempel van 2 $\mu\text{g}/\text{m}^3$.
- Daar is velblootstelling aan platinum en die ander platinum groep metale. Die hoogste vlak van velblootstelling is 6.79 $\mu\text{g}/\text{cm}^2$ ('n baie bekende beroepsgeneeskundige het 'n paar jaar gelede vir my gesê ons mors ons tyd om velblootstelling te meet, want daar sal nie velblootstelling by die betrokke raffinadery wees nie!).
- Die platinumkonsentrasie in urine wissel tussen <0.1 en 3 $\mu\text{g}/\text{g}$ kreatinien.

Ander beroepe in die visier is gesondheidswerkers, haarkappers en skoonheidskundiges. Ons stel ook belang in die blootstelling van werkers aan die son se UV-straling, siende dat dit velkanker veroorsaak.

Samevatting

Die insidensie en voorkoms van Beroepsvelsiektes in Suid-Afrika is in werklikheid onbekend. Dit terwyl daar in Europa alreeds gefokus word op voorkoming en daar groot voorkomingsprogramme in sleutelindustriële gedryf word.

Daar is duidelik tekortkominge in die Beroepsgesondheid- en Veiligheidswetgewing rakende velblootstellingsriglyne en die vel- en sensitiseringsnotasies.

Daar is unieke geleenthede, nie net vir navorsing nie, maar ook vir onderrig-leer en opleiding.

Dit is belangrik dat navorsingsbevindinge 'n verskil maak in die praktyk, Suid Afrikaanse werksplekke, en uiteindelik in geaffekteerde werkers se gesondheid.

In terme van onderrig-leer het ons nodig om nie net ons studente nie, maar ook mede-beroepshigiëne praktisyns, te onderrig en op te lei sodat hulle risiko's van velblootstelling korrek kan bepaal en uiteindelik blootstelling effektief kan beheer.

Ons moet waar moontlik geleenthede om "voorkomend" op te tree nie verby laat gaan nie.

Ons velblootstellingsnavorsing het deurentyd die "persoon", die werker self wat blootgestel word in ag geneem. Daar is binne die veld van beroepshigiëne kritiek teenoor hoe ons velblootstelling meet en wat dit beteken, maar tog is ek van opinie dat ons benadering tot velblootstelling, deur die "persoon" in ag te neem noodsaaklik is. Dit terwyl ons tydens respiratoriese blootstellingsmonitering, blootstelling meet buite respirators, en nie die respiratoriese gesondheid of fisiese aktiwiteit, die respiratoriese tempo, van die werker in ag neem wanneer ons bevindinge maak nie.

Daar is geleentheid om 'n verskil te maak.

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