Søknad om midler til avansert vitenskapelig utstyr/databaser 2011 (over 1 mill kroner)

Prosjektittel:

Søker	**Utstyrsansvarlig/ prosjektansvar**	**Administrativt ansvar**
Navn	**Christian A. Drevon**	Eva H. Mjelde
Stilling & Akademisk grad	Professor, dr med	Kontorsjef
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Forskningsgruppe som skal drifte utstyret	**Christian A. Drevon**	

Prosjektsammendrag (maks. 200 ord):

For several different projects related to whole body energy metabolism, we need advanced calorimetry for rodents (mice and rats). Some of us will use the outfit for studies on diet and physical activity related to energy metabolism. In particular we have discovered that a sulphur-modified fatty acid enhance food intake in parallell with reduced fat depots. This type of finding requires whole body measurements of O2 uptake and CO2 release, combined with food intake and registration of exercise. This can be performed with the new instruments representing a completely new research infrastructure, which will be of marked interest for many national research groups because nobody else to our knowledge has this equipment in Norway. All types of dietary, pharmacological and exercise studies of whole animals (mice and rats) can be performed in these cages. It is required significant expertise to operate this equipment, and it will be available for all national scientists to use the cages on non-profit basis.

Type utstyr (kort beskrivelse av utstyret og hva det skal brukes til):

Advanced metabolic units with ability for monitoring a number of body functions are central to phenotyping of transgenic animals as well as in studies on the role of diet and other environmental factors on basic biomedical processes. To advance our research on energy metabolism we need whole body calorimetry. This instrument is in use in several research laboratories in Europe as well as in USA but we don't get access to this equipment because it is very inconvenient to transport rodents fed different diets to distant places. We have quite active research groups in the forefront of this area at IMB (Drevon CA, Blomhoff R & Nebb HI) but we lack the possibility of performing *in vivo* up-to-date studies of food intake, physical activity and CO2-production and oxygen uptake. We have access to good experimental set-ups for in vitro (molecular and cellular) studies, but we have no access to whole body calorimetry. This is available in Tromsø and Trondheim at present.

The dominating instrument in this field is based on circuit calorimetry (Oxymax, Columbus

Instruments). Heat is derived by assessment of the exchange of oxygen for carbon dioxide that occurs during the metabolic process. The relationship between the volume of gas consumed (oxygen) and of that produced (carbon dioxide), reveals the energy content of the foodstuff utilized by the animal. As an indirect system, the instrument relies on accurate measurements of gas concentrations and flow. Flow is measured by a mass technique that yields data formatted in terms normalized to scientific STP (760 mmHg and zero Centigrade). It is the measurement of mass, not volume, that allows the outfit to be employed under various atmospheric conditions without the need to account for environmental pressure or temperature.

The instrument allows f. ex. housing of 10 mice and 10 rats in separate cages for individual measurement of oxygen uptake and CO2 production. This allows us to monitor very accurately the energy expenditure. Combined with information about food intake, we will have very good view of the metabolic situation of each animal.

The equipment is a fully automated to a connected personal computer and supports numerous set-up options that accommodate many applications like physical activity, feeding mass, drinking (licks and/or volume) body temperature, heart rate, urine collection/ monitoring and special treadmills for monitoring metabolic performance during exercise. Thus, we can measure the specific effects on heart rate, kidney function, food intake, and liquid consumption.

These new instruments will allow whole body metabolic studies on a level that takes systems biology another step further. The new instruments represent a new research infrastructure and will be of marked interest for many research groups at UiO because nobody else to our knowledge has this equipment in Oslo. All types of dietary, pharmacological and exercise studies of whole animals (mice and rats) can be performed in these cages. It is required significant expertise to operate this equipment, and it will be available for collaborating scientists to use the cages on non-profit basis.

Budsjett

Kostnadsplan (i 1000 kr)	**2011**	**2012**	**2013**	**Sum**
Anskaffelsessum	3000			
mva	750			
Brutto anskaffelsesum	**3750**			

Finansieringsplan (i 1000 kr)	**2011**	**2012**	**2013**	**Sum**
Egne midler	375			
NFR-midler				
Andre offentlige midler				
Andre private midler				
Internasjonale midler				
Søkes UiO (max 90%)	3375			
Totalsum	**3750**			
Merk: Totalsum for rubrikkene i kostnadsplan og finansieringsplan skal være like år for år.				

Vedleggsoversikt. Kryss av for vedlegg.			
X	**Prosjektbeskrivelse (maks 5 sider)**	**X**	**Veiledende/uforpliktende pristilbud fra aktuelle leverandører i NOK inkl. mva. (Ikke brosjyrer)**
X	**Beskrivelse av forskningsmiljøet (ca. 10-20 linjer)**	**X**	**Prosjektansvarliges CV (1 side, gjelder nye søkere)**
X	**Samarbeidende miljøer, interne og eksterne, ev. rolle som kjernefasilitet**	**X**	**Publikasjonsliste (gjelder nye søkere)**
	Nødvendig utskifting av gammelt utstyr (beskrivelse)	**X**	**Vurdering av innpassing i fakultetets/enhetens og UiOs strategiske planer.**
X	**Teknisk avdeling IT/Bygg/Innkjøp** Egenmelding om avklaring og synliggjøring av ulike følgekostnader		

Fakultetets/museets/senterets prioritering nr. _____

Underskrifter:		
Dato:		
	Søker/Utstyrsansvarlig	**Fakultetsleder/Muesumsleder/Senterleder**

Fylles ut av fakultet/museum/senter	**Kommentarer**
Fakultetets/museumets/senterets prioriteringer	
UiO's vedtatte strategi	
Faglig kvalitet	
Sambruk med andre, internt og eksternt	
Nødvendig utskifting av gammelt utstyr	
Annet	

Interactions between nutrient intake, exercise and energy metabolism

Prof Christian A. Drevon and Dr. Knut Tomas Dalen

Obesity - imbalance in nutrient intake and energy metabolism

The ability to adjust the organism's energy intake against energy usage is challenged by our society's constant excess of food. Inability to control food intake is nowadays manifested in the worldwide epidemic in obesity, and the long list of associated clinical complications including: insulin resistance, cardiovascular diseases, stroke, heart attack, stroke and cancer. Despite intense research for decades, we still do not understand what contributes most to development of obesity; *nutrient composition*, *excess energy intake* or *insufficient energy usage/exercise*. What is undisputable is a net positive intake. At Department of Nutrition, several interdisciplinary projects are focusing on dissecting the importance of the above:

1) Nutrient composition:

The work of prof. H. Refsum consistently demonstrates a clear positive association between plasma total cysteine levels and body weight within distinct ethnic populations and throughout the entire lifespan (1;2). In a search for the molecular link, we are involved in this project by studying the effect of cysteine in cultured adipocytes and the effect of altered cystein levels in the diet in experimental mice.

2) Energy intake / Exercise:

It is well known that exercise has a beneficial effect, and prevents development of a large number of diseases. Although exercise is likely to prevent obesity by increasing energy consumption, other factors are likely involved. Amongst those are communication between skeletal muscle and the rest of the organism. We have identified a number of cytokines that are secreted by cultured human myotubes, including IL-6, IL-7 and IL-8 are (3). We are searching for additional factors specifically secreted from muscle cells after exercise; either by exercise training of mice or by electric stimulation of cultured muscle cells.

3) The intramyocellular lipid paradox:

Obese individuals, especially those that are insulin resistant, tend to accumulate cytosolic lipid droplets (LDs) outside adipose tissue, in tissues such as liver, pancreas and skeletal muscle (4;5). This peripheral accumulation of LDs is believed to be an important cause of insulin resistance. Paradoxically, athletes/highly exercised individuals also have high levels of intramyocellular LDs, yet these are highly insulin sensitive (6;7). To clarify the role of intramuscular lipids and tissue insulin sensitivity, we have generated mice with disruptions in the perilipin genes (Plin1-5). Lack of perilipins on LDs severely affects the stability of the LD surface, increases hydrolysis of its content, and lower the cells ability to retain LDs.

Muscle - an endocrine organ

Adipose tissue is an important endocrine organ with changes in its secretion prior to or after development of diseases related to obesity/insulin resistance (8;9). Proteins secreted from adipose tissue (leptin, resistin, adiponectin, tumor necrosis factor = TNFa, angiotensinogen, adipsin, acylation-stimulating protein, retinol-binding protein, interleukin 6, plasminogen activator inhibitor-1, fasting-induced adipose factor, a fibrinogen-angiopoietin-related

protein, metallothionein, tissue factor, complement C3, fibronectin, haptoglobin, entactin/nidogen, collagen VI3, pigment epithelium-derived factor = PEDF, hippocampal cholinergic neurostimulating peptide = HCNP, neutrophil gelatinase-associated lipocalin = NGAL, adiponutrin (10)), appear to have many distinct physiological effects on various organs. These secretory proteins are named adipokines.
Skeletal muscle accounts for on average 40 % of body weight, approximately twice the mass of total adipose tissue in a normal weight individual. It is of essential importance for health. Physical activity reduces risk of obtaining several diseases like cardiovascular disease, type 2 diabetes mellitus (T2D) and certain types of cancers. One possibility is that exercise consumes energy, and thus prevents development of obesity by reducing accumulation of fatty acids in adipose tissue. Other possibility is that skeletal muscles secrete peptide hormones (myokines) that influence body tissues beneficially. It has recently been shown that human skeletal muscle release significant higher amounts of interleukins (IL) like IL-6 and IL-8 into the circulation during and after exercise (11;12).
We want to focus on how nutrients influence expression of adipokines and myokines. Using both cell culture systems and animals, we will investigate how nutrients, exercise and changes in secretion of adipokines and myokines can affect cellular and whole organism lipid accumulation and development of insulin resistance.

Beneficial nutrients that prevent development of obesity

During evolution famine has been a big threat. The solution was to store large amounts of energy in the form of fat in specialized cells, adipocytes, which could be mobilized during periods of under-nutrition. Nowadays humans face over-nutrition combined with a sedentary life-style. The negative health consequences liked to excessive intake of energy-dense foods (high fat and/or sugar) and development of obesity is alarming (13).
Only a marginal change in our current diet has the potential of drastically improve public health. But to give the correct advices, we need to understand better how food composition, total energy intake/appetite and exercise influence health. Studies suggests that the amount as well as the type of dietary fatty acids may affect lipid homeostasis and fat deposition (14). As examples, polyunsaturated fatty acids (PUFA) and the FA analogue tetradecylthioacetic acid (TTA) seems to prevent development of obesity and T2D (15). Out collaborator at the Department has more recently discovered a clear positive association between plasma total cysteine levels and body weight.
We want to feed mice diets low or high in cysteine or with compounds that effect cystein metabolism. This will enable us to distinguish the effect of cystein on total body weight (food intake, food absorption, exercise, uncoupling of mitochondria).

Skeletal muscle and insulin resistance

Development of insulin resistance and T2D has been closely linked to defects in skeletal muscle oxidative capacity as well as increased lipid accumulation. Approximately 80 % of the insulin-responsive post-prandial glucose removal from blood is due to skeletal muscle. Skeletal muscles can be made insulin resistant by iv infusion of free fatty acids (FFA) and this can be related to inhibition of insulin signaling (16). Increased accumulation of intramyocellular triacylglycerol (imTAG) is a major clinical feature of insulin resistance. Why TAG accumulates is unclear, but increased lipid availability together with reduced muscular oxidative capacity of fatty acids may contribute. Early detectable signs among relatives of type 2 diabetics include mitochondrial dysfunction and reduced expression of genes involved in oxidative phosphorylation (17). It is not known how fatty acids are able to inhibit insulin signaling and lead to insulin resistance. One common explanation is that lipid derivatives like DAG or acyl-CoAs can activate PKC, which then can phosphorylate IRS1/2

leading to reduced insulin signaling. Another possibility is related to fatty acids activating the serine kinase IKkB that activates NFkB, and thus promotes inflammation (18).

Fatty acids & diabetes
High dietary fat intake probably enhances risk for development of obesity (11). Moreover, it has been shown in animal feeding experiments that high intake of marine fatty acids reduces the amount of perirenal and epididymal adipose tissue (19;20). Dietary intake of less saturated fatty acids may also improve insulin sensitivity without any effect on insulin secretion. There was no beneficial effect of fat quality on insulin sensitivity in humans with a high fat intake (> 37 E%), whereas feeding experiments in rats show that omega-3 fatty acids reduce development of insulin resistance (21;22).

Perilipins - lipid droplet surface proteins

The perilipin proteins cover the surface of cytosolic LDs, analogous to lipoproteins cover the surface of lipoproteins in plasma. These are tissue-specifically expressed, with unique characteristics, but all seems to control the rate of LD hydroplysis (23;24). Cultured cells with lack of perilipins have abnormal LDs with reduced sensitivity to insulin (25).
Dr Dalen has generated mice with disruption of all perilipin genes in mice (plin1-5, unpublished work). The perilipin null mice are excellent to determine the role of LDs in relation to obesity and insulin sensitivity. Mice lacking myocellular perilipins will have defects in their ability to control myocellular LDs.
Using perilipin null mice, mice we will be able to address the importance of LDs for cellular insulin sensitivity and the effect of LD formation during exercise. The perilipin null mice will also be used to determine if the level of stored LDs affect secretion of myokines.

Our available experimental model systems

Cultured human adipose tissue & cells
We have established a method for culturing human adipose tissue (26). Human mature adipocytes are difficult to study because they are very fragile, and little is known about their metabolism. We have also a human preadipocyte cell strain (SGBS) with high capacity for differentiation (27).

Cultured skeletal muscle cells
Cultures of human or mice skeletal muscle cells are obtained by isolating satellite cells (SC) from needle biopsies. The SC can be proliferated in culture, and then cryogenically frozen for long time storage. By careful alteration of the growth medium, SC can differentiate and fuse into multinucleated myotubes sharing many of the classical characteristic features of mature striated skeletal muscle cells, including the ability to contract and increase glucose uptake and glycogen synthesis in response to insulin (28). We investigate effects of different fatty acids and carbohydrates on transcription of genes and secretion of myokines.
We have available primary myotybe cells isolated from perilipin null mice, as well as two different mouse myotube cell lines, Sol8 and c2c12. The cell lines have been manipulated into efficient host cells for stable integration of expression vectors (based on Flp-In technology from Invitrogen).

High through-put analyses of uptake & oxidation of energy-providing substrates
We have developed multi-well assays to monitor accumulation as well as oxidation of 14C-labeled substrates in cultured cells (29). This make it possible to monitor cellular uptake and oxidation of substrates in high through-put 96-well plates for cultured cells. It would be ideal to compare our in vitro data with data obtained in vivo using metabolic cages.

Instrumental need

We are in urgent need of equipment to monitor whole organism biochemistry

In vivo measurement of oxygen uptake, CO2 release (oxygraphy), food intake and physical activity

As demonstrated in the selected ongoing projects described above, we perform many metabolic studies in mice and rats where there will be an interaction between food intake, treatment with drugs, physical activity and progression of a disease. To understand the biological effects of alterations in diet, drugs, genetic constitution and muscle contraction, we need reliable data on energy expenditure, food intake and physical activity.

As an example, monitoring of energy intake and expenditure will enable us to conclude if and observed reduction in body weight by a treatment is due to uncoupling of mitochondrial beta-oxidation to generate heat instead of ATP or lowered appetite (reduced energy intake). *Such interpretations can only be made using metabolic cages*.

Some of our previous results

We have shown that long-term changes in lifestyle including decreased intake of dietary fat and increased physical activity reduce plasma leptin concentrations in humans beyond the reduction expected as a result of changes in fat mass (30), and that long term intake of n-3 PUFAs suppress leptin expression in humans and rats (31). We have demonstrated leptin expression in primary human skeletal muscle cells (32).

Resistin

Adipocytes secrete a signal molecule named resistin (33). Plasma resistin levels are reduced by the anti-diabetic drug rosiglitazone and enhanced in diet-induced and genetic forms of obesity. Moreover, we have observed that insulin strongly inhibits resistin expression in these cells (34), and that arachidonic acid is a strong inhibitor of resistin secretion (35).

Adiponectin

Another secretory protein from the adipose tissue that may be involved in development of T2D is adiponectin (36). Reduced expression of adiponectin correlates negatively with insulin resistance. Adiponectin increases insulin sensitivity by decreasing triglyceride content in muscle and liver in obese mice (37). We have shown that women with gestational diabetes have lower plasma concentration and adipose tissue levels of mRNA adiponectin (38). Moreover, we have analyzed a randomized, intervention trial, including 188 males with metabolic disease, divided into four groups; diet, exercise, a combination of diet and exercise, and a control group. Analysis of changes in plasma adiponectin levels showed that only diet ($p=0.03$) had effect depending on body fat mass (39).

Planned and ongoing experiments

An important area of investigation is our focus on secretion of myokines. We use primary cultures of human skeletal myotubes, based on differentiation of satellite cells from muscle biopsies. It is known that exercise offers protection against chronic disorders such as cardiovascular diseases, T2D, dementia, and depression. It is unclear how contracting skeletal muscles mediate beneficial metabolic effects. It is possible that released myokines may explain how muscle activity can influence mood, performance, and cognitive function (40;41). We have recently shown that IL-6, IL-7 and IL-8 are secreted by cultured human myotubes (3). ***However, we want to advance by studying secretion in relation to whole body energy metabolism, energy status and different degrees of physical activity.***

In order to obtain relevant data on food intake, physical activity and CO2-production and oxygen uptake, we have to get access to calorimetry combined with registration of food intake, body temperature, urine production, body weight, movement, heart rate, CO2-

production, O2 uptake and exercise studies. These measurements will allow us to perform studies on substances that are associated with altered body weight. Our previous data support the hypothesis that feeding with the fatty acid analogue tetradecylthioacetic acid (TTA) may increase hepatic fatty acid β-oxidation, thereby diminishing storage of fat in adipose tissues (42). The increased expression of hepatic UCP3 and of Ucp1 in abdominal adipose tissues may together promote enhanced energy dissipation and reduced weight gain in rats (42). To advance this field further we need whole body calorimetry.
Metabolic cages are in use in several European laboratories as well as in USA but we don't get access to this equipment because it is very inconvenient to transport rodents fed different diets to distant places. Our application is related to completely new scientific metabolic cages to house rodents under feeding and exercise studies. All sorts of dietary, pharmacological and exercise studies of whole animals (mice and rats) can be performed in these cages and we intend to develop a national core function in relation to calorimetry.

Economy and people

We have limited resources to buy expensive equipment, and metabolic research requires the outlined infrastructure. Individuals in this project are research fellows Frode Norheim (UiO), Knut Tomas Dalen (UiO), Torgrim Langleite (Helse Sør-Øst), bioengeneer Anne Randi Enget, professors Rune Blomhoff, Arild C. Rustan, Inst of Pharmacy and Christian A. Drevon University of Oslo (project coordinator). Professors Jørgen Jensen and Truls Raastad at NIH are also partners of our team. In addition we collaborate in three EU-projects (FP7 Food4Me & NutriTech), Nutrigenomics (www.nugo.org) for studies of molecular nutrition and genes. **We apply for money to buy metabolic cages where 10 mice/rats can be individually housed simultaneously for registration of oxygen uptake, carbon dioxide release, food intake and physical activity for an estimated price of 3.0 mill NOK + vat.**

Expectations

We will study whole body energy metabolism in relation to energy status, diet and physical activity. We publish our results in highly rated international journals, in addition to inform the public about our research in a balanced manner via several media. I have supervised 24 PhD students to their degree in my research career, of which 11 have defended their thesis since 2000. I now supervise 9 PhD students, and CAD has published/submitted 77 international papers and 64 articles/chapters in Norwegian since 2006. We communicate our findings to the community by ~ 100 talks/interviews via radio, newspapers & TV yearly.

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Linton Instrumentation

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Email: mail@lintoninst.co.uk
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Attention Professor Drevon
University of Oslo
Email: c.a.drevon@medisin.uio.no

29rd April 2011

PG/PG

Dear Professor Drevon

OUR QUOTATION NUMBER: 9556/11

CLAM-20M--C[HS]-A(xz)-FM(cf)-TR(hr)
And
CLAM-20R--C[HS]-A(xz)-FM(cf)-TR(hr)

Comprehensive Lab Animal Monitor, configured for 20 rats and 20 mice, with high-speed calorimetry (O2, CO2, RER, Heat), X (General) and Z (Rearing Behaviour) Activity Monitoring, Feeding Mass Monitoring with low spill centre feeders, Oxymax Animal Chamber, Telemetry Receiver Antenna Assembly, Battery less Telemetry Implants for monitoring Heart Rate, Includes CLAMS racking and truck assemblies.

Comprising:	Mouse System 20 Chambers	Rat System 20 Chambers
CLAMS Controller/Interface/Software	$ 35,957.00	$ 35,957.00
Oxymax Calorimeter [C]	$102,389.00	$102,389.00
High Speed Gas Analysis Upgrade [HS]	$ 31,800.00	$ 31,800.00
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Activity XY (or XZ)	$106,099.00	$106,099.00
Feeding Mass [FM]	$ 77,870.00	$ 77,870.00
Standard Cage	$ 22,574.00	$ 22,574.00
Centre Feeder Option	$ 24,521.00	$ 24,521.00
Telemetry Receiver	$128,974.00	$128,974.00
Temp & Heart Rate Transmitter [TH]	$ 31,927.00	$ 31,927.00
System sheving	$ 16,385.00	$ 16,385.00
Total	$585,591.00	$585,591.00
Large system discount	*-$117,118.00*	*-$117,118.00*
Total (after discount)	$468,473.00	$468,473.00
Total cost of goods		**$936,946.00**
Delivery charged at cost – estimated at -		$4,500.00
Installation and training		$5,000.00

Prices exclude VAT / Local Taxes & Duties.
This quotation is valid for acceptance for 90 days from issue.

Kindest regards

Paul Gunning
LINTON INSTRUMENTATION

Linton Instrumentation

Unit 11, Forge Business Centre, Upper Rose Lane, Palgrave, Diss, Norfolk, IP22 1AP
Email: mail@lintoninst.co.uk
Telephone +44 1379 651344
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Attention Knut Tomas Dalen

University Oslo

Email: k.t.dalen@medisin.uio.no

Pdg/mar 25 May 2011

Dear Knut

OUR QUOTATION NUMBER: 9633/11

1 x 1055DRM Exer-3/6 Open Treadmill with Shock and Shock Detection for Rats (3) and Mice (6). Includes Software.	€12,253.00
1 x 1012M-4-E54 Modular Enclosed Metabolic Treadmill for Mice, 4 Lanes with Sock.	€23,492.00
1 x 0890M-E54 Rotamex-5 4 Lane Rota-Rod for Mice with Full Detection. Includes Software.	€7,698.00

Prices exclude delivery costs and exclude local sales taxes.

This quotation is valid for 30 days from date of issue.

We hope that this is of interest to you. If we may be of any further assistance, please do not hesitate to contact us.

Kindest regards

Paul Gunning
LINTON INSTRUMENTATION

Egenerklæring
Vedlegg til søknad om vitenskapelig utstyr over 1 mill kr

Gjelder innkjøp av
Kalorimeter koblet til metabolske bur for registrering av fórinntak og registrering av fysisk aktivitet (Navn på utstyret)

Oxymax Open Circuit Calorimeter, Linton Instrumentation (Angi utstyrsenhet)

Dyreavdelingen ved Institutt for Medisinske Basalfag (Enhet utstyret skal plasseres ved)

Behov for IT-støtte
¤ Utstyrenhetenes IT-behov passer inn i universitetets IT-infrastruktur og krever kun styring ved hjelp av en vanlig PC.

¤ Utstyrsenhetens IT-behov er avklart med USIT
Kostnader i forbindelse med tilpasning av infrastruktur vil være ca. NOK 70 000

Behov for laboratorieinvesteringer
¤ Utstyrsenheten vil ikke innbære vesentlige endringer med hensyn til arealdisponering, men det vil kreve et benkeareale på 2 x 160 cm x 55 cm med en høyde på 190 cm.
¤ Utstyrsenheten vil ikke innebære omkostninger til ombygging av lokaler, bygningstekniske-, vvs- eller elektroniske arbeider

Driftskostnader
¤ Det vil ikke påløpe årlig driftskostnader ved utstyret tilsvarende 100.000 NOK

Prosedyrer og retningslinjer for anbud og kjøp
¤ Instituttet er kjent med universitetets retningslinjer for anbud og innkjøp
¤ Instituttet har hatt kontakt med innkjøpsseksjonen for avklaring av retningslinjer for anbud og innkjøp

Underskrift

Christian A. Drevon

…….. ……
Prosjektleder Administrativt ansvarlig

Bekreftelse av sambruk

Utstyrsenhet: **Kalorimeter koblet til metabolske bur for registrering av fórinntak og registrering av fysisk aktivitet**.
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Lokalisering : **Dyreavdelingen ved Institutt for Medisinske Basalfag**

Søker: **Christian A. Drevon**

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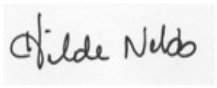
Sambrukserklæring fra Professor Hilde Nebb, avdeling for ernæringsvitenskap.
Dr. Philos Knut Tomas Dalen, avdeling for ernæringsvitenskap.

I samarbeide med National Institutes of Health, USA, har Dr. Dalen laget fire mus hvor hvert av genene i perilipinfamilien er kondisjonelt knocket ut. Perilipin proteinene binder seg til overflaten av intracellulære lipiddråper (LDer) og stabiliserer disse i cytosol. Fjerning av disse genene enkeltvis eller i kombinasjon, påvirker de affiserte organenes evne til å styre metaboliseringen av LDer. Dette vil ha svært sammensatte effekter på organismens fysiologi, herunder pre-diponerbarhet for å utvikle insulin resistens, kardiovaskulære sykdommer og hjerteinfarkt, evne til å lagre energi (i fettvev), utholdenhet, sårheling, og termogenese.

Vi arbeider pr i dag med den basale karakteriseringen av disse fire musemodellene i ulike prosjekter ved Universitetet i Oslo. Forsøk utført i metabolske bur er nærmest et krav ved førstegangs publisering av arbeider hvor gener som er involvert i energiomsetningen er knock-out ut. Bruk av metabolske bur er derfor essensielt for vår forskning. Slikt utstyr vil muliggjøre monotorering av energiomsetning i passiv tilstand, under eller etter trening hos mus med defekter i de ulike perilipin genene. Bruk av dette utstyret vil heve kvaliteten på forskingen betraktelig og demonstrere viktigheten av å kunne lagre LDer i ulike vev i en organisme.

For vår forskningsgruppe står anskaffelse av metabolske bur øverst på prioriteringlisten over utstyr som pr i dag ikke er tilgjengelig i Osloregionen, men som er essensielt for å kunne heve kvaliteten på våre forskningsprosjekter til internasjonalt toppnivå.

Date: 10.06.2011.



Professor Hilde Nebb, avdeling for ernæringsvitenskap
e-postadresse: h.i.nebb@medisin.uio.no

Knut Tomas Dalen, avdeling for ernæringsvitenskap
e-postadresse: k.t.dalen@medisin.uio.no

Bekreftelse av sambruk

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Sambrukserklæring fra Guro Valen, IMB, avdeling for fysiologi

Jeg vil med dette støtte Christian Drevons søknad om metabole bur til studier av mus helkroppsmetabolisme.

De metabole bur det søkes om gir mulighet til monitorering av helkropps energimetabolisme i våkne mus, med måling av oksygen opptak, karbon dioksyd frisetting, inntak av mat og fysisk aktivitetet. Utstyret kan om ønskelig i framtiden kombineres med telemetri for å få enda mer detaljert informasjon. Denne type utstyr er en grunnenhet for fenotyping av genetiske forandrete mus, så vel som utstyr som gir verdifull informasjon ved ulike stimuleringer. I Europa er utstyret et vanlig innslag i sentre som lager genetisk forandrete mus. Det er på høy tid at forskere ved IMB får muligheten til å bruke metabolske bur i sin virksomhet! I vår gruppe er utstyret særlig interessant for å studere helkroppsmetabolisme i mus med indusert hjerteinfarkt, hvor betydning av ulike gener for overlevelse og tilheling studeres ved hjelp av genetisk forandrete mus.

Guro Valen, professor og avdelingsleder

IMB, avdeling for fysiologi

guro.valen@medisin.uio.no

Sambrukserklæring fra ……. (forskningsgruppe, event. avdeling)

Tekst (10-20 linjer, eks. hva dette utstyret betyr for min forskningsgruppe, avdeling osv)

Navn (med underskrift fra ansvarlig forskningsgruppeleder, eller avdelingssjef /klinikksjef)

Fagmiljø (institutt, avdeling

e-mailadresse

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Sambrukserklæring fra Forkningsgruppen Glykobiologi ved Svein O. Kolset, Avdeling for ernæringsvitenskap, IMB, UiO

Serglycin er et proteoglykan som finnes i alle hematopoetiske celler, endotelceller og glatte muskelceller. Vi arbeider med mus hvor genet for serglycin er slått ut (Serglycin knock out mus =SGKO). Vi har tidligere vist at serglycin er det dominerende proteoglykan i makrofager fra disse museneog involvert i kontroll av sekresjon av tumor nekrose faktor. Vi har også vist at serglycin er et dominerende proteoglykan i dyrkede endotelceller og viktig for kontroll av kjemokin sekresjon. Vi vil studere hvilken betydning serglycin har for inflammasjonssvar og metabolisme i SGKO. Studier på aktivering av immunforsvaret hos SGKO med vekt på endotelaktivering er videre under planlegging i samarbeid med endotelforsker e ved Oslo Universitetssykehus-Rikshospitalet. Vi avslutter på sensommeren foringsforsøk med SGKO og resultatene av disse analysene vil danne grunnlag for nye forsøk på slike mus, med spesielt fokus på inflammasjon og aterosklerose.

Våre dyreforsøk vil profitere på tilgang til det utstyret det her søkes om

Navn - Svein Olav Kolset Svein O. Kolset

Fagmiljø - Institutt for medisinske basalfag, Avdeling for ernæringsvitenskap

e-mailadresse s.o.kolsetedisin.uio.no

Bekreftelse av sambruk

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Sambrukserklæring fra Niels Chr. Danbolt (Neurotransporter Group, Avd Anatomi)

Metabolske prosesser inne i cellene er helt avhengige av transport av stoffer gjennom membraner. Det er nødvendig å kunne transportere mellom cytosol og utsiden enten denne inneholder ekstracellulærvæske, pre-urin eller tarminnhold. Det er noe slikt som 400 ulike transportørproteiner som deltar i disse prosessene. Vi har mus hvor transportørgener er endret, og vi antar at tre av de transportørene vi har endret vil ha betydning for funksjonen til nyre og lever, mens andre har betydning for andre organer (hjerne og hjerte). Mulighet for å registrere forinntak, fysisk aktivitet, nyrefunksjon med mer vil være viktig for min gruppes virksomhet. Jeg støtter med dette søknaden fra Drevon.

Mvh,

Niels Chr. Danbolt

Prof. Dr. Med., Gruppeleder

Fagmiljø (IMB, Anatomi)

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Professor dr.med.
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Instituttgruppe for medisinske basalfag
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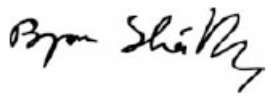
Oslo June 2011.

To whom it may concern.

It is with great enthusiasm I give my support to the application for metabolic cages to measure energy metabolism in live rodents on the project “"Interactions between nutrient intake exercise and energy metabolism".

This instrumentation will be essential for *in vivo* studies of energy metabolism in our transgenic mice at the Department of Nutrition.

Sincerely Yours,



_____
Bjørn Steen Skålhegg
Professor, PhD

Bekreftelse av sambruk

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Søker: **Christian A. Drevon**

E-mailadresse: c.a.drevon@medisin.uio.no

Sambrukserklæring fra Rune Blomhoff.

Vi har en rekke forskningsprosjekter som involverer sykdomsmodeller i forsøksdyr. I disse prosjektene undersøker vi hvordan matvarer og bioaktive stoffer i kostholdet påvirker genekspresjon og signallering knyttet til inflammasjon og oksidativt stress . Muligheter for å benytte et kalorimeter koblet til metabolske bur for registrering av fórinntak og registrering av fysisk aktivitet vil være meget nyttig for et flertall av disse prosjektene. Et slikt utstyr vil også være viktig for å utføre mange andre fenotypiske analyser av forsøksdyr, spesielt transgene mus. Jeg støtter søknaden derfor på det varmeste.

Rune Blomhoff,

Leder, Avdeling for ernæringsvitenskap,

Institutt for medisinske basalfag, UiO

rune.blomhoff@medisin.uio.no

CURRICULUM VITAE CHRISTIAN A. DREVON
Born January 20, 1945, Norwegian citizen

EDUCATION

1964. Examen artium at Teisen Gymnasium, Oslo.
1970. MD at the University of Oslo (UO) with the average grade of 10.26 (laudabilis).
1970-72. Internship at Ålesund County Hospital (1 year) and in a GP office (6 months).
1972. Military service as a lieutenant at the Institute of Work Physiology.
1973-76. Research fellow at the Institute for Nutrition Research at UO.
1976-77. Deputyship as Assistant Professor at the Institute for Nutrition Research.
1977. Dr med thesis on "Cholesteryl ester metabolism."
1977-79. Postdoctoral fellow in Dept of Medicine, University of California, San Diego. Fellowships from San Diego County Heart Ass, John Fogarty's International Fellowship & Royal Norwegian Council for Scientific & Industrial Research.
1979. Ass. Professor at Dept of Pharmacology, Institute of Pharmacy, UO.
1984. Associated head of National Institute of Forensic Toxicology.
1988- Professor (& chairman of Section for Dietary Research), Institute for Nutrition Research, UO.
1996/97. Sabbatical at Department of Biology, San Diego State University, California.

RESEARCH

I have written 272 publications including published, in press & submitted with international reviews, and 244 papers including 4 TV educational programs in Norwegian, and approximately 265 abstracts. I collaborate with researchers from the USA, Spain, The Netherlands, France, Italy, UK, Ireland, Germany, Poland, Bulgaria, Greece, Denmark, Finland & Sweden, in addition to several Norwegian research groups in clinical as well as basic science. We are presently involved in two large EU projects in the 7th Framework Program; a Food4Me and NutriTech, both with 25 partners focused on personalized nutrition and application of molecular nutrion reasearch. I am a cofounder and president of the NuGO Association, which is a follow-up of the NuGO, Network of Excellence in FP6.
My research is related to diet and health, ranging from population-based studies to molecular biology. At present my group is involved in studies of fatty acids and different vitamins related to energy metabolism, obesity, cachexia, diabetes, cardiovascular diseases, cognitive function and cancer. In addition we are deeply involved in studies on how skeletal muscle can communcate with other tissues via signal proteins (myokines), and perhaps explain the beneficial effects physical activity on health. Mechanistically we focus on transport, metabolism and biological effects of nutrients, with special emphasis on interactions between nutrients, physical activity and genes.

RESEARCH SUPERVISION & TEACHING

I have supervised 65 students to their Master degrees in pharmacy, nutrition or biochemistry, and 24 research fellows to PhD (7 medical doctors, 5 nutritionists, 6 pharmacists, 3 biochemists, 1 genetisist and 2 zoophysiologists). Since year 2000 ten of my students have defended their PhD thesis.
At present I have 9 PhD students, one postdoctoral fellow and 1 bioengeneer associated with my group. I have teaching experience in physiology, nutrition & a number of courses & seminars. I am an editor and a main author of “Mat & Medisin, Nordic textbook in basic and clinical nutrition” published in 5 editions, the last in 2007 with a total number of copies of about 12000.

PUBLICATION LIST FOR CHRISTIAN A DREVON, SECTION 1
Original papers, international reviews & contributions in books

196. Haugen F, Ranheim T, Harsem NK, Lips E, Staff AC & Drevon CA: Increased plasma levels of adipokines in preeclampsia: Relationship to placenta and adipose tissue gene expression. Am J Physiol. Endocrinology & Metabolism, 2006, 290, E326-33
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Vedlegg for søknaden: "Interactions between nutrient intake, exercise and energy metabolism"

Beskrivelse av forskningsmiljøet (10-20 linjer)

Individuals in this project are research fellows Frode Norheim (UiO), Knut Tomas Dalen (UiO), Torgrim Langleite (Helse Sør-Øst), bioengeneer Anne Randi Enget, professors Rune Blomhoff, Helga Refsum, Hilde I. Nebb, Arild C. Rustan, Inst of Pharmacy and Christian A. Drevon University of Oslo (project coordinator). Professors Jørgen Jensen and Truls Raastad at NIH are also partners of our team. In addition we collaborate in three EU-projects (FP7 Food4Me & NutriTech), Nutrigenomics (www.nugo.org) for studies of molecular nutrition and genes. We have extensive international collaboration on topics within energy metabolism as can be seen from our publication lists, including most European nutrition institutions working on molecular nutrition. In addition, we also collaborate with professor AJ Lusis at Department of Human Genetics, the Department of Microbiology, Immunology and Molecular Genetics, and the Department of Medicine, University of California, Los Angeles, Los Angeles, California, USA, where PhD student Frode Norheim now is spending a year to work on genetic mouse models on energy metabolism.

Vurdering av innpassing i fakultetets/enhetens og UiOs strategiske planer

Our metabolic studies are right in the core of our prioritized topics at Department of Nutrition, IMB. We have just submitted an application to Norwegian Research Council for a grant as Center of Excellence on this topic. Our Center will be called "Cnetre of energy balance (CEB).