Two Isoforms of Sister-of-Mammalian Grainyhead Have Opposing Functions in Endothelial Cells and In Vivo

Judith Haendeler,* Arne Mlynek,* Nicole Büchner,* Margarete Lukosz, Martin Graf, Christopher Guettler, Sascha Jakob, Sabrina Farrokh, Kerstin Kunze, Christine Goy, Francisca Guardiola-Serrano, Heiner Schaal, Miriam Cortese-Krott, René Deenen, Karl Köhrer, Christoph Winkler, Joachim Altschmied

Objective—Sister-of-Mammalian Grainyhead (SOM) is a member of the Grainyhead family of transcription factors. In humans, 3 isoforms are derived from differential first exon usage and alternative splicing and differ only in their N terminal domain. SOM2, the only variant also present in mouse, induces endothelial cell migration and protects against apoptosis. The functions of the human specific isoforms SOM1 and SOM3 have not yet been investigated. Therefore we wanted to elucidate their functions in endothelial cells.

Approach and Results—Overexpression of SOM1 in primary human endothelial cells induced migration, phosphorylation of Akt1 and endothelial nitric oxide synthase, and protected against apoptosis, whereas SOM3 had opposite effects; isoform-specific knockdowns confirmed the disparate effects on apoptosis. After reporter assays demonstrated that both are active transcription factors, microarray analyses revealed that they induce different target genes, which could explain the different cellular effects. Overexpression of SOM3 in zebrafish embryos resulted in increased lethality and severe deformations, whereas SOM1 had no deleterious effect.

Conclusions—Our data demonstrate that the splice variant–derived isoforms SOM1 and SOM3 induce opposing effects in primary human endothelial cells and in a whole animal model, most likely through the induction of different target genes. (Arterioscler Thromb Vasc Biol. 2013;33:1639-1646.)

Key Words: apoptosis ▪ cell movement ▪ human umbilical vein endothelial cells ▪ nitric oxide ▪ sister-of-mammalian grainyhead protein, human ▪ transcription factors ▪ zebrafish

Migratory capacity and protection against apoptosis are crucial for proper function of endothelial cells (EC). Both processes depend on an adequate production of NO. After having identified the mammalian transcription factor Grainyhead-like 3 (GRHL3)/sister-of-mammalian grainyhead (SOM) in a screen for tumor necrosis factor z-induced survival genes in a mammary carcinoma cell line,¹ we have shown that it reduces apoptosis, activates the endothelial NO synthase (eNOS), and induces migration of EC,¹² the latter corroborating findings in epidermal cells. There, knockdown experiments in keratinocytes and analyses of keratinocytes isolated from SOM-deficient mice had shown an essential role for this protein in epidermal cell migration.¹³ The grainyhead transcription factor family is highly conserved during evolution, with Drosophila Grainyhead (Grh) being the first described member.² In vertebrates, three Grh-homologs are known, Mammalian Grainyhead (or Grainyhead-like 1), brother-of-mammalian grainyhead (BOM or Grainyhead-like 2), and SOM.⁵,⁷ In contrast to mice, in humans, 3 isoforms of SOM exist. Whereas SOM2 is present in both species, SOM1 and SOM3 are only found in humans. They are translation products from an alternatively spliced primary transcript, which is not found in mice, because their genome lacks the first exon of this pre-mRNA. Alternative splicing is a general phenomenon⁸ and occurs also in many transcription factor families.¹⁰ The resulting protein isoforms can have different effects on cellular functions. SOM1 and SOM3 are coexpressed in several human tissues⁶; however, functions on the cellular level and in vivo have not yet been published. Having shown that SOM2, the human homolog of the single
mouse SOM protein, induces migration and has a protective effect in EC.\textsuperscript{1,2} We have now addressed the question whether SOM1 and SOM3 also regulate essential functions of EC. Here, we demonstrate that both SOM1 and SOM3 are expressed in primary human EC and are regulated reciprocally by physiological levels of NO. Overexpression of SOM1 increases the migratory capacity, inhibits apoptosis, and enhances eNOS activity in EC. In contrast, SOM3 inhibits EC migration, has no antiapoptotic effect, and does not activate eNOS. In contrast to a previous study,\textsuperscript{6} we demonstrate that both isoforms are transcriptional activators. Microarray analyses revealed different target gene spectra between the 2 SOM-isoforms, which could explain the opposite effects of these proteins in EC. To further support our cell culture findings, we also investigated the role of SOM1 and SOM3 in vivo in zebrafish embryos. Here, SOM3 induced severe malformations and diminished the number of normally developing animals, whereas SOM1 had no effect. Thus, we conclude that SOM1 has protective functions in primary ECs, whereas SOM3 has deleterious effects in EC and in vivo.

Materials and Methods

Materials and Methods are provided in the online-only Supplement.

Results

SOM1 and SOM3 Are Both Expressed in ECs

We have identified SOM in a screen for antiapoptotic genes in a mammary carcinoma cell line. In ECs, this transcription factor is required for migration. In addition, it induces eNOS phosphorylation in EC, protects these cells against apoptosis in an eNOS-dependent manner, and is required for basal as well as NO-induced migration.\textsuperscript{1,2} In human, 2 additional protein isoforms with different N termini, SOM1 and SOM3, exist, which are derived from alternative splicing of a primary transcript not present in mice, because the mouse genome lacks the corresponding first exon.\textsuperscript{6} Their role in EC functions has not been addressed so far. Before assessing functional properties of these proteins, we confirmed expression of both alternatively spliced transcripts in EC by reverse transcription-polymerase chain reaction (Figure 1 in the online-only Data Supplement).

Because it is known for several transcription factors that different splice variants have distinct and sometimes opposing functions, we investigated the role of SOM1 and SOM3 in EC.

SOM1 and SOM3 Have Opposing Effects on Endothelial Cell Migration, Apoptosis, and NO-Production

Important properties of EC in the vessel wall are migratory capacity, apoptosis protection, and supply of bioactive NO. To assess the impact of the 2 transcription factor isoforms on EC migration, we expressed SOM1 or SOM3 in primary human EC (Figure 1A) and determined their migratory capacity by setting an artificial wound in the EC monolayer and counting the migrated cells.\textsuperscript{11} As shown in Figure 1B and 1C, SOM1 dramatically induced migration, whereas SOM3 significantly reduced the migratory capacity of EC after 24 hours. These effects were not attributable to enhanced or reduced proliferation of the cells (Figure 2 in the online-only Data Supplement).

In the same experimental setting, overexpression of SOM1 also significantly inhibited apoptosis in EC, whereas SOM3 had no protective effect as measured by annexinV exposure on the outer cell surface (Figure 2A). These findings were corroborated by the respective changes in the levels of the antiapoptotic protein BCL-X\textsubscript{L} (Figure 2B), which were elevated after overexpression of SOM1, but not changed by SOM3. The antiapoptotic effect of SOM1 was not only observed under baseline conditions, but also, when apoptosis was induced with 7-β-hydroxycholesterol (Figure 3 in the online-only Data Supplement). One of the most important antiapoptotic and promigratory stimuli in EC is endogenously derived NO produced by eNOS. To assess, whether the protective function of SOM1 depends on eNOS activity, we overexpressed the SOM-isoforms in the presence of the eNOS-inhibitor L-N\textsuperscript{ω}-nitroarginine methyl ester (L-NAME), which completely blocked the antiapoptotic effect of SOM1 (Figure 2A). A similar effect was observed with respect to the promigratory effect of SOM1 (data not shown). Because overexpression can cause a somewhat artificial situation within a cell, we also knocked down both SOM-isoforms

Figure 1. Sister-of-mammalian grainyhead (SOM1) and SOM3 have opposing effects on endothelial cell migration. EC were transfected with expression vectors for SOM1, SOM3, or an empty vector (EV). A, Representative immunoblots of lysates from transfected cells probed with an anti-V5 antibody to detect the SOM fusion proteins (V5 (SOM)), proteins of the appropriate molecular weight (SOM1, 71 kDa; SOM3, 60 kDa) are indicated by asterisks, Akt1 was used as loading control. B and C, Migration of transfected cells was determined by scratch wound assay. B, Representative fluorescence microscopic pictures. The white line indicates the origin of migration, the migration direction is indicated by an arrow. C, Migrated cells were counted and are shown as number of migrated cells per high power field (HPF). Data are mean±SEM (n=4; *P<0.05 versus EV). The inset shows the data for SOM3 on a different scale.
individually to deplete the cells of the respective endogenous proteins. After transfecting EC with isofrom-specific, fluorescently labeled siRNAs, we determined that the transfection efficiency was sufficient (>95% transfected cells; Figure IV in the online-only Data Supplement), confirmed the isofrom-specific knockdowns by reverse transcription-polymerase chain reaction (Figure 2C), and measured apoptosis rates. As shown in Figure 2D, knockdown of SOM1 resulted in increased apoptosis, whereas the SOM3-knockdown slightly reduced apoptosis rates. After establishing that overexpression and knockdown have a profound influence on EC functions, we asked whether stimuli affecting these functions have an influence on expression of these 2 SOM-isoforms. Therefore, we first established a multiplex real-time polymerase chain reaction approach allowing us to simultaneously detect both transcript variants. To assess the impact of the well-described promigratory and antiapoptotic stimulus NO on expression of SOM1 and SOM3, we treated EC with the NO-donor propylamine propylamine NONOate. We had previously shown that NO increases the migratory capacity of EC which is constitutively activated by protein kinase B/Akt1. Therefore, we analyzed whether the SOM-isoforms have an effect on the activity of both enzymes. Therefore, 24 hours after overexpression of SOM1 and SOM3, we determined

**Figure 2.** Sister-of-mammalian grainyhead (SOM1) and SOM3 have opposing effects on endothelial cell apoptosis. **A** and **B**, EC were transfected with expression vectors for SOM1, SOM3, or an empty vector (EV). **A**, Transfected cells were treated 6 hours after transfection with 100 µmol/L-NG-nitroarginine methyl ester (L-NAME) for another 18 hours or left untreated, and apoptosis was measured flow cytometrically. Data are means±SEM (n=4; *P<0.05 versus SOM1 transfected, untreated cells). **B**, Top, Representative immunoblots of lysates from transfected EC probed with antibodies directed against Bcl-XL and Akt1 (loading control). **Bottom**, Semiquantitative analysis of scanned autoradiographs. Shown are the levels of Bcl-XL normalized to Akt1 relative to EV transfected cells. Data are mean±SEM (n=4; *P<0.05 versus con). **C** and **D**, EC were transfected with siRNAs targeting SOM1 (siRNA SOM1) or SOM3 (siRNA SOM3), respectively. **C**, Expression of SOM1, SOM2, SOM3, and the housekeeping gene RPL32 was assessed by reverse transcription-polymerase chain reaction (RT-PCR). The expected fragment sizes for each transcript are indicated. **D**, Apoptosis was measured flow cytometrically. Data are means±SEM (n=3; *P<0.05 versus neg. control). **E**, Regulation of SOM isoform expression by NO. EC were treated with propylamine propylamine NONOate (PAPA) for 18 hours or left untreated (con). Expression of SOM1 and SOM3 was assessed by multiplex RT-PCR, RPL32 served as control. Top, Representative image of amplification products, the expected fragment sizes for all transcripts are indicated. Bottom, Semiquantitative analysis shown are the levels of the transcripts for the 2 SOM-isoforms normalized to RPL32. Data are means±SEM (n=3; *P<0.05 versus con).
Figure 3. Sister-of-mammalian grainyhead (SOM1) and SOM3 have opposing effects on Akt1 and endothelial NO synthase (eNOS) activation. Endothelial cells (EC) were transfected with expression vectors for SOM1, SOM3, or an empty vector (EV). A, Top, Representative immunoblots of lysates from transfected cells probed with antibodies directed against active Akt1 phosphorylated on serine 473 (P-Akt1 (S473)) and total Akt1 (Akt1). B, Top, Representative immunoblots of lysates from transfected cells probed with antibodies directed against active eNOS phosphorylated on serine 1179 (P-eNOS (S1179)), and total eNOS (eNOS). Shown are the ratios of phosphorylated Akt1 to total Akt1 relative to EV transfected cells. Data are mean±SEM (n=4; *P<0.05 versus EV). B, Bottom, Semiquantitative analysis of scanned autoradiographs. Shown are the ratios of phosphorylated eNOS to total eNOS relative to EV transfected cells. Data are mean±SEM (n=4; *P<0.05 versus EV).

Figure 4. Both sister-of-mammalian grainyhead (SOM)-isoforms are transcriptional activators. A, Schematic representation of the SOM-specific luciferase reporter constructs. The core sequences of the SOM-binding sites with the corresponding mutations are shown. TATA denotes the minimal promoter and LUC the luciferase gene. B and C, Transactivation by SOM1 and SOM3. Shown are the luciferase activities after cotransfection of HEK293 cells with SOM expression vectors (SOM1, SOM3) and reporter plasmids carrying 2 wild-type (wt) or mutated (mut) consensus-binding sites. The values obtained with the luciferase construct with the binding site mutations were set to 1. Data are mean±SEM (n=6; *P<0.05 versus mut). C shows the data for SOM1 on a different scale.

SOM1 and SOM3 Activate Different Target Genes

The most obvious explanation for the opposing effects induced by SOM1 and SOM3 could be the activation of different target genes. Based on 2 hybrid experiments in human 293T cells, it had been proposed that SOM3 is transcriptionally inactive or even a repressor. However, in these experiments, only isolated domains of both proteins were fused to the GAL4 DNA-binding domain. Therefore, it cannot be excluded that other regions of the protein have a function in transcriptional activation, and that fusion to a heterologous DNA-binding domain could mask properties of the full-length protein. To assess whether SOM1 and SOM3 are transcriptional activators, HEK293 cells were cotransfected with expression vectors encoding full-length SOM1 or SOM3 and a SOM-dependent luciferase reporter plasmid. Here, luciferase expression is controlled by a tandem consensus-binding site for SOM fused to a minimal promoter to avoid interactions with other transcription factors. As a specificity control, we included a reporter, in which critical residues in the SOM-binding sites were mutated (Figure 4A). These experiments clearly demonstrated transcriptional activation by both proteins, with SOM3 being the even more potent activator (Figure 4B and 4C). This, together with the disparate effects on EC migration, apoptosis, and NO-production after 24 hours, suggests that the 2 SOM-isoforms activate different targets genes. Therefore, we analyzed gene expression profiles of EC transfected with expression vectors for SOM1, SOM3, or a corresponding empty vector after 18 hours. Pairwise comparisons revealed that SOM1 regulated 367 genes, and SOM3 regulated 261 genes, when compared with empty vector transfected cells.
with only a small overlap of 28 common targets (Figure 5A; Tables I and II in the online-only Data Supplement). To validate the microarray data, we chose 2 different targets, which could affect EC functions, the basic helix-loop-helix protein Max interactor 1 (MXI1) and protein kinase B \( \beta \)/Akt2. MXI1 can inhibit Myc-dependent transcription, and Myc has been shown to induce apoptosis in different cardiovascular cells, including EC,\(^{17}\) smooth muscle cells,\(^{18}\) and fibroblasts.\(^{19}\) Therefore, one could assume that MXI1 acts antiapoptotic. Akt2 is a master regulator of all Akt isoforms and, thus, important for apoptosis inhibition and migration.\(^{20}\) We analyzed the levels of MXI1 and Akt2 in the same setting used for the gene expression analyses. Both proteins were upregulated after overexpression of SOM1 and downregulated by SOM3 (Figure 5B). This clearly indicates that SOM1 activates expression of genes coding for antiapoptotic and promigratory proteins, whereas SOM3 suppresses their accumulation.

**SOM3 Has Deleterious Effects In Vivo**

Finally, we addressed the in vivo relevance of the opposing effects of human SOM1 and SOM3 using zebrafish as a model system. Because the embryos are translucent, they can easily be analyzed through all developmental stages. We injected in vitro transcribed mRNA for human SOM1 and SOM3 in identical molar concentrations into 1-cell stage zebrafish embryos and monitored the resulting phenotypes. The animals were categorized into normal, medium, and severe phenotypes (Figure 6A). The medium phenotype is characterized by slightly smaller size, a bent tail, smaller eyes, and obvious apoptosis in the head region. Massively deformed embryos (classified as severe phenotype) have no heads and show an open spinal cord, bifurcated dorsal tails, and massive cell death. SOM1 did not change the embryonic development significantly, when compared with control animals. In contrast, SOM3 injection with the same molar RNA concentration resulted in a dramatic increase in animals with the medium phenotype and severe malformations. Correspondingly, the number of normally developed embryos was significantly reduced (Figure 6B). These results demonstrate that not only in primary human cells, but also in a whole animal SOM1 and SOM3 exert different functions.

**Discussion**

The present study investigated cellular functions of isoforms 1 and 3 of the human transcription factor SOM, which are translated from an alternatively spliced primary transcript. Here, we show that SOM1 and SOM3 are both transcriptional activators, but exert opposing effects on apoptosis and migration in primary human EC, most likely mediated through the activation of different target genes. In addition, SOM3 overexpression, in contrast to SOM1, has detrimental effects in vivo during zebrafish embryogenesis.

Alternative splicing is more a rule than an exception because it affects at least two thirds of all human protein-coding genes, creating an extreme proteome diversity with a limited number of genes.\(^{19}\) Inclusion or skipping of alternatively spliced exons can be tissue-specific, regulated during development, or in response to physiological or pathophysiological stimuli. One of the most prominent examples for alternative splicing in the cardiovascular system is the pre-mRNA of the vascular endothelial growth factor, leading to several protein isoforms with different properties.\(^{21,22}\)

Alternative splicing is also a common feature of transcripts encoding transcription factors, with \( \approx \)30% of these RNAs in humans being alternatively spliced generating on average 3 protein isoforms.\(^{10}\) In the vasculature, the Inhibitor-of-Differentiation 3 (Id3) pre-mRNA can be alternatively spliced, creating isoforms of this helix-loop-helix transcription factor, which exert disparate functions. Id3 can promote cell cycle entry and thereby induce smooth muscle cell proliferation by inhibiting p21\(^{\text{WAF1}}\) and p16\(^{\text{INK4A}}\) transcription.\(^{23}\) An alternatively spliced transcript translates into an isoform with a different
experiments would not have identified isoform-specific target gene spectra. 

**Figure 6.** sister-of-mammalian grainyhead (SOM3), but not SOM1, induces severe malformations in zebrafish embryos. One-cell stage zebrafish embryos were injected with in vitro transcribed SOM1 or SOM3 mRNA at equimolar concentrations (68 and 80 ng/µL, respectively), scored for phenotypic changes 24 hours post fertilization (hpf) and compared with uninjected embryos. A, Pictures of an uninjected control embryo and 3 embryos injected with SOM3 RNA, representing the different phenotypes at 34 hpf. B, Shown are the percentages of the 4 different phenotypes after injection of SOM1 or SOM3 mRNA (mean±SEM; n=4; 63–97 injected embryos per experiment; *P<0.05). C terminus (Id3a), which is not detected in normal rat carotid arteries, but abundantly expressed in the neointimal layer after balloon injury, like its human homolog Id3L in carotid atherosclerotic plaques. This isoform, in contrast to Id3, seems to attenuate growth of smooth muscle cell during vascular lesion formation by inducing apoptosis. Unlike Id3 and Id3a, SOM1 and SOM3 are coexpressed in normal tissues and in primary human EC (Ting et al and this study), but similar to Id3, they have opposing effects. Whereas Id proteins sequester basic helix-loop-helix factors, thereby preventing them from binding to DNA and activating transcription, both SOM-isoforms are active transcription factors on their own. The difference in the transactivation potential observed in our experiments could be ascribed to the luciferase reporter construct used, which only contains tandem binding sites for SOM in front of a minimal promoter. It is long known that the combination of regulatory DNA elements in promoters and enhancers and, thus, the interactions of the respective transcription factors, determine under which conditions a gene is transcribed. Because SOM1 has an extended N terminus compared with SOM3, they might, despite their identical DNA-binding properties, interact with different coactivators or other transcription factors bound to certain promoters, which would also explain the different target gene spectra. SOM target genes have been identified in a microarray screen before by comparing backskin from wild-type and SOM-deficient mice. However, because migration of keratinocytes depends on proliferation, unlike migration of EC, a completely different set of target genes might be induced. In addition, mice do not express homologs of the human SOM1 and SOM3 proteins since the corresponding first coding exon does not exist in the mouse genome and, therefore, these experiments would not have identified isoform-specific targets. We have validated the upregulation of MXI1 and Akt2 by SOM1 in EC on the protein level, which most likely is due to increased transcription, whereas the downregulation by SOM3 might be a secondary effect. Akt2 has been described as a master regulator of all Akt isoforms, explaining the activation of Akt1 and, thus, eNOS observed after overexpression of SOM1. The second validated target, MXI1, belongs to the Mad family, whose members antagonize Myc functions by sequestering Max. In contrast to Myc-Max dimers, which activate transcription by binding to so-called E-boxes, Mad-Max complexes, including MXI1-Max, serve as transcriptional repressors because of their ability to recruit histone deacetylases, thereby inhibiting Myc-dependent transcription. Myc has been shown to induce apoptosis in several cell types of the cardiovascular system, which could, therefore, be blocked by MXI1.

Here, we show that a physiological stimulus promoting migration and inhibiting apoptosis of EC, namely NO, leads to a change in the expression levels of the 2 alternatively spliced SOM transcripts. Whereas the level of SOM1 mRNA is increased, the amount of SOM3 mRNA is downregulated. Together with the observed cellular effects after overexpression or downregulation of these 2 transcripts, this could, at least to a certain degree, explain the promigratory and antiapoptotic effects of NO in human primary EC.

The different SOM-isoforms can dimerize with each other and other members of the grainyhead-like family, which all recognize the same DNA element. It is conceivable that the dimer composition determines which genes are activated. However, the activation of different target genes might also be explained by differential interactions of the 2 SOM proteins with other regulatory factors. In both cases, the consequence of the overexpression of SOM1 or SOM3 would be an altered gene expression profile in EC and zebrafish embryos. The zebrafish genome contains a unique SOM-gene encoding a single protein, which, like the mouse homolog, is related closer to human SOM2 than to SOM1 (Figure V in the online-only Data Supplement). During zebrafish embryogenesis, it is expressed in the gastrula and is a key regulator of periderm differentiation, indicating a role in barrier formation, similar to what has been shown in mammals. Overexpression of
SOM might, similar to the situation human in ECs, change the transcriptional output in the embryos, leading to an insufficient development of epidermal barriers, which could cause osmotic perturbations explaining the massive deformations observed. Because these events take place before vessel development, it remains unclear whether SOM3 overexpression would also affect the vasculature. Nevertheless, similar to the situation in human EC, the 2 isoforms have different effects in vivo.

Interesting aspects for future studies will be to understand under which other physiological and pathophysiological conditions one or the other SOM isoform is dominantly expressed, and how alternative splicing of the primary transcript is regulated. Because it is also conceivable that not only alternative splicing, but also changes in SOM interaction partners determine which genes are activated under specific conditions, another route of investigation will be to identify and characterize isoform-specific binding partners of the 2 SOM proteins. After having validated 2 proteins differentially regulated by SOM1 and SOM3, it will be important to uncover their exact functions in EC and to further characterize the regulatory networks affected by and affecting the different SOM-isoforms, which could provide clues for therapies aimed at improving endothelial function.

Acknowledgments

We thank Anika Rühl for expert technical assistance.

Disclosures

This work was supported by the Deutsche Forschungsgemeinschaft Research Training Groups (RTG) 1089 and 1033, the collaborative research center 612, start-up grants of the University of Düsseldorf to A. Mlynek is a scholarship holder of RTG 1033, and M. Graf is an NUS/DBS graduate scholar-

References


**Significance**

We have previously shown that the transcription factor sister-of-mammalian grainyhead (SOM) has promigratory and antiapoptotic functions in human endothelial cells. In humans, several isoforms of this protein exist. Two of these isoforms, SOM1 and SOM3, are translation products of an alternatively spliced pre-mRNA. They are not found in mice because their genome lacks the first exon of the corresponding transcript. SOM1 and SOM3 are both expressed in primary human endothelial cells and are regulated reciprocally by physiological levels of NO. Whereas SOM1 induces migration of these cells, inhibits apoptosis in an NO-dependent manner, and leads to an activation of eNOS, SOM3 reduces migration significantly and does not affect apoptosis. Both proteins are transcriptional activators and activate different target genes, which could explain their disparate cellular functions. In a whole animal model, SOM3, in contrast to SOM1, perturbs zebrafish development. Thus, SOM3, but not SOM1, has deleterious effects not only in human endothelial cells, but also in vivo.
Two Isoforms of Sister-of-Mammalian Grainyhead Have Opposing Functions in Endothelial Cells and In Vivo

Judith Haendeler, Arne Mlynek, Nicole Büchner, Margarete Lukosz, Martin Graf, Christoph Guettler, Sascha Jakob, Sabrina Farrokh, Kerstin Kunze, Christine Goy, Francisca Guardiola-Serrano, Heiner Schaal, Miriam Cortese-Krott, René Deenen, Karl Köhrer, Christoph Winkler and Joachim Altschmied

Arterioscler Thromb Vasc Biol. 2013;33:1639-1646; originally published online May 16, 2013; doi: 10.1161/ATVBAHA.113.301428

Arteriosclerosis, Thrombosis, and Vascular Biology is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2013 American Heart Association, Inc. All rights reserved.
Print ISSN: 1079-5642. Online ISSN: 1524-4636

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://atvb.ahajournals.org/content/33/7/1639

Data Supplement (unedited) at:
http://atvb.ahajournals.org/content/suppl/2013/05/16/ATVBAHA.113.301428.DC1

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Arteriosclerosis, Thrombosis, and Vascular Biology can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Arteriosclerosis, Thrombosis, and Vascular Biology is online at:
http://atvb.ahajournals.org//subscriptions/
Materials and Methods

Plasmid constructs

The 5'-ends of the SOM1 and SOM3 coding sequences were amplified from MCF-7 cell cDNA with isoform specific primers and inserted into an expression vector containing the remainder of the coding region with a C-terminal V5-tag as described previously. SOM specific reporter plasmids were constructed by inserting two consensus SOM binding sites or mutants thereof upstream of a minimal promoter in the luciferase vector pTATA-LUC+. Complementary oligonucleotides containing two wildtype SOM binding sites (5'-AGCTTGATACGGATCCAACTAGATAACCGGTTTTACTAGTTAGATCCAACTAGATAAAACCGGTTTTTACTAGTTA-3' and 5'-GATCTAACTAGTAAAAACCGGTTTTATCTAGTTGGATCTAATAGTAAAAACCGGT TTATCAGTTGGATCCGTATCA-3') or two binding sites with mutations in the core SOM recognition motif, which prevent binding of the transcription factor (5'-AGCTTGATACGGATCCAACTAGATAACCGGTTTTTACTAGTTAGATCCAACTAGATAAAACCGGT TTATCAGTTGGATCCGTATCA-3' and 5'-GATCTAACTAGTAAAAACCGGTTTTATCTAGTTGGATCTAATAGTAAAAACGTTTTATCAGTTGGATCCGTATCA-3') were mixed in equimolar ratios, annealed and the resulting double stranded molecules with HindIII- and BamHI-compatible overhangs were inserted between the corresponding restriction sites in pTATA-LUC+. The integrity of all constructs was verified by sequencing.

siRNAs

SOM isoform-specific, 5'-Alexa Fluor 488 labelled siRNAs were synthesized commercially (Qiagen, Hilden, Germany). For SOM1, the junction between exon 1B and exon 2 was targeted (target sequence: 5’-CCTATTTTTTTTCTACGCTT-3’) and for SOM3 the junction between exon 1B and exon 3 (target sequence: 5’-TTTTTCTTGGATCCCAAAGGAA-3’); both exon-exon junctions are found exclusively in the respective transcript encoding SOM1 or SOM3 (see supplemental figure 1).

Cell culture

Human primary umbilical vein endothelial cells (EC) and human embryonic kidney cells (HEK293) were cultured as previously described. After detachment with trypsin, cells were grown for at least 18 h as described.
Transfections
EC were transiently transfected with plasmid DNA using Superfect or with siRNA using HiPerFect (both Qiagen, Hilden, Germany) according to manufacturer’s specifications. HEK293 were transfected with Lipofectamine-PLUS (Invitrogen, Darmstadt, Germany) as described previously\textsuperscript{5}.

Migration assays
Migration was quantified with a scratch wound assay as described previously\textsuperscript{8}.

Apoptosis assays
Detection of cell death was performed by flow cytometry using annexinV-APC or annexinV-FITC binding and 7-amino-actinomycin (7-AAD) staining as described previously\textsuperscript{9}.

Measurement of cell proliferation
Proliferation was measured by the incorporation of 5-bromo-2'-deoxyuridine (BrdU) as a parameter for DNA synthesis using the BrdU Flow Kit (BD Biosciences, Heidelberg Germany) according to manufacturer’s recommendations. BrdU was added to the culture medium 24 h before cell detachment. Incorporated BrdU was labeled with a FITC-coupled anti-BrdU antibody according to Manufacturer's protocol and permeabilized cells were counterstained with 7-amino-actinomycin (7-AAD). Cells double positive for BrdU and 7-AAD were analysed using a FACSCalibur flow cytometer (BD Biosciences, San Jose, CA, USA).

Reverse transcriptase PCR (RT-PCR)
RNA was isolated using Trizol (Invitrogen, Darmstadt, Germany) according to manufacturer’s specifications. RNA concentrations were determined photometrically and RNA integrity was assessed by agarose gel electrophoresis.
2 µg total cellular RNA were reverse transcribed using Superscript™ III (Invitrogen, Darmstadt, Germany) according to manufacturers’ specifications with a primer mix of oligo-dT\textsubscript{20}, pdN\textsubscript{6} (random hexamers) and the gene-specific primer SOM Ex7 rev1 (5'-GGGACCTTCTCATTGTCGAA-3'). The resulting cDNA was used as template for isoform specific amplification of SOM1 (primers: SOM Ex1B for2, 5'-TCTGAGCAGAAGAATGTGGATG-3' and hSOM E4 rev1, 5'-CTTGAGCAATCTGGGTACTCTGG-3', expected amplification product: 412 bp), SOM2 (primers: SOM Ex1A for2, 5'-CTCGACACCCCAACCTCAACAT-3' and SOM Ex2 rev1, 5'-CAGCTGTCAACCGGTTTCT-3', expected amplification product: 254
bp), SOM3 (primers: SOM Ex1B3 for1, 5'-CCTATTTTCTGGTCCCAAGG-3' spanning the junction between exon 1B and exon 3 and Q8TE85 5'-CAATCTGGTACTCTGGGG-3', expected amplification product: 185 bp) and as a control the transcript for the 60S ribosomal protein L32 (RPL32, primers: hmRPL32 Ex02 for1, 5'-GTGAAGCCCAAGATCGTCAA-3' and hmRPL32 Ex03 rev1, 5'-TTGTTGCACATCAGCAGC-3', expected amplification product: 257 bp). For multiplex analyses 5 µg of total cellular RNA were reverse transcribed and the primers SOM Ex1B for2 and hSOM E4 rev1 were used for RT-PCR, yielding an additional amplification product of 225 bp indicative of the SOM3 transcript. The SOM primer positions are schematically shown in supplemental figure 1. The amplification products were resolved on standard 2% agarose gels.

**Luciferase assays**

Two days after transfection cells were lysed with Reporter Lysis Buffer (Promega, Mannheim, Germany) according to manufacturers' instructions. Identical amounts of cellular protein were made up to 20 µl with Reporter Lysis Buffer and luciferase activity was determined by automatic injection of 100 µl luciferase assay mix (20 mM Tricine-KOH, pH 7.8, 0.1 mM EDTA, 8 mM MgCl₂, 33.3 mM DTT, 0.27 mM Coenzyme A, 0.53 mM ATP, 0.47 mM D-luciferin) using a MicroLumat Plus Microplate Luminometer LB 96 V (Berthold, Bad Wildbad, Germany).

**Immunoblotting**

Immunoblotting was performed as described previously⁶ with antibodies directed against Akt1 (1:500), phospho-Akt1 (S473) (1:500), eNOS (1:1000) and phospho-eNOS (S1179) (1:500) (all Becton & Dickinson, Karlsruhe, Germany), Akt2 (1:1000) and phospho-eNOS (T495) (1:500) (both Cell Signalling Technology, Frankfurt, Germany), BCL-Xₐ (1:200) and MXI1 (1:100) (both Santa Cruz Biotechnology, Heidelberg, Germany), the V5 epitope (1:1000, Invitrogen, Darmstadt, Germany) and γ-actin (1:10000, Sigma-Aldrich, Deisenhofen, Germany). Blotting membranes were incubated with primary antibodies overnight at 4°C before they were washed and incubated with secondary antibodies according to standard procedures. Detection was performed by enhanced chemiluminescence using the ECL reagent (GE Healthcare, Freiburg, Germany) and standard X-ray films. Semi-quantitative analyses were performed on scanned X-ray films using ImageJ 1.42q¹⁰.
**Microarray analysis**

RNA was isolated using Trizol (Invitrogen/Life Technologies, Darmstadt, Germany) according to manufacturers' specifications and subjected to a second purification step using RNeasy columns (Qiagen, Hilden, Germany). RNA integrity was checked on an Agilent 2100 Bioanalyzer and concentrations determined by photometric Nanodrop measurement. All samples in this study showed common high quality RNA Integrity Numbers (RIN 9.7-10).

Synthesis of cDNA and subsequent fluorescent labelling of cRNA was performed on three replicates of each experimental condition (transfection with empty vector, expression vectors for SOM1 and SOM3, respectively) with the One-Color Microarray-Based Gene Expression Analysis/Low Input Quick Amp Labeling (Agilent Technologies, Böblingen, Germany) according to the manufacturers' protocol. Briefly, 100 ng of total RNA were converted to cDNA, followed by in vitro transcription and incorporation of Cy3-CTP into nascent cRNA. After fragmentation labelled cRNA was hybridized to SurePrint G3 Human GE 8x60k Microarrays (Agilent Technologies, Böblingen, Germany) for 17 h at 65°C and scanned as described in the manufacturers' protocol.

Signal intensities on 20 bit tiff images were calculated by Feature Extraction software (FE, Vers. 10.7.1.1; Agilent Technologies, Böblingen, Germany). One SOM1 sample did not pass post extraction quality control and was therefore excluded from further analyses. Data analyses were conducted with GeneSpring GX software (Vers. 10.5; Agilent Technologies, Böblingen, Germany). Probe signal intensities were quantile normalized across all samples to reduce inter-array variability. Input data pre-processing was concluded by baseline transformation to the median of all samples. After grouping of replicates according to their respective experimental condition a given transcript had to be expressed above background (i.e. called “detected” by FE) in at least two of three replicates in any one of two or both conditions to be further analysed in pairwise comparisons of conditions. Differential gene expression was statistically determined by Welch’s unpaired T-test.

**In vitro transcription and RNA injection into zebrafish embryos**

The vectors for in vitro transcription were constructed by transferring BamHI/Pmel fragments from the SOM expression vectors into pCS2+\textsuperscript{12} opened with BamHI and SnaBI. Plasmid DNA was purified with the Wizard plus SV miniprep kit (Promega, Mannheim, Germany) according to the manufacturers' manual.
For *in vitro* transcription the plasmids were linearized with NotI and 1 µg of linearized, purified plasmid was used as template for mRNA synthesis. Capped mRNAs were synthesized with SP6 RNA polymerase using the mMMessage mMachine SP6 Kit (Ambion/Life Technologies, Darmstadt, Germany) and freshly produced mRNA was purified via Phenol/Chloroform/Isoamylalcohol, RNeasy columns (Quiagen, Hilden) and precipitated with NaAc/Ethanol. The mRNA concentration was measured spectrophotometrically and appropriate dilutions were prepared (50ng/ul – 150ng/ul). To test the quality of the synthesized mRNAs, they were analyzed by gel electrophoresis.

Different concentrations of SOM1 and SOM3 mRNA were injected into one-cell stage wild-type zebrafish embryos (strain DBS). The concentration of injected mRNA was optimized by injecting various mRNA amounts up to concentrations, where a high death rate was observed or most of embryos were heavily affected. After adjusting the mRNA concentrations in these preliminary experiments, a concentration of 80 ng/µl for SOM1 mRNA was chosen for injections and – due to the size difference between the transcripts - 68 ng/µl for SOM3 mRNA to inject equal molar concentrations of both mRNAs. Injected and fertilized zebrafish embryos were collected and incubated at 28°C in zebrafish medium (58 mM NaCl, 0.7 mM KCl, 0.4 mM MgSO₄, 0.6 mM Ca(NO₃)₂, 5 mM HEPES) over night. Death/life ratios were measured at 24 hours post fertilization (hpf) and surviving embryos were classified according to their phenotype. Uninjected embryos served as controls. Photographs were taken at 34 hpf with a Nikon MSZ 1000 stereomicroscope.

**Statistics**

Statistical analyses were performed with student’s T-test.

**References**


Supplemental figure I: All 3 SOM isoforms are expressed in primary human endothelial cells. (A) Schematic representation of the genomic organization of the SOM 5'-end and the isoform-specific transcripts. The first 3 exons (boxes) are drawn to scale and named according to Ting et al. 2003 Biochem J. 2003;370:953-962, translated regions are shown in black. All exons 3' to exon 3 are identical in SOM1, SOM2 and SOM3. The SOM3 transcript has a frame shift downstream of the SOM1 start codon such that the open reading frame in SOM3 begins in exon 4. (B) Expression of SOM isoforms in primary human endothelial cells. EC RNA was reverse transcribed in the presence (+) or absence (-) of reverse transcriptase and the resulting cDNAs were used as template for isoform-specific amplifications. The positions of the primers used for amplification are shown schematically in A. Amplification products were resolved by agarose gel electrophoresis. The expected fragment sizes are 412 bp (SOM1), 254 bp (SOM2) and 185 bp (SOM3). RPL32 denotes the amplification product (257 bp) of a housekeeping gene coding for a ribosomal protein, M is a DNA size marker, the fragment sizes are indicated on the left.
Supplemental figure II: Overexpression of SOM1 and SOM3 does not induce proliferation of primary human endothelial cells. Primary human endothelial cells were transfected with expression vectors for SOM1, SOM3 or an empty vector (EV) and proliferation was measured as BrdU incorporation by flow cytometry. Data are mean ± SEM (n=3).
Supplemental figure III: Overexpression of SOM1 protects primary human endothelial cells against apoptosis induced by 7-β-hydroxycholesterol. Primary human endothelial cells were transfected with an expression vector for SOM1 or an empty vector (EV), treated for 3 h with 10µg/ml 7-β-hydroxy-cholesterol (+) or left untreated (-) and apoptosis was measured by flow cytometry using annexin V-FITC binding and 7-aminoactinomycin staining. Data are mean ± SEM (n=4-10; *p<0.05 vs.EV, #p<0.05 vs. EV + 7-β-hydroxycholesterol).
Supplemental figure IV: Transfection efficiencies of primary human endothelial cells with SOM-specific siRNAs. Primary human endothelial cells were transfected with 5'-AlexaFluor 488-labelled siRNAs targeting SOM1 or SOM3, respectively, and analyzed by flow cytometry. Transfection efficiencies were 96.2% (siRNA SOM1) and 95.9% (siRNA SOM3).
Supplemental figure V: Comparison between human and zebrafisch SOM proteins. The alignment of the human SOM protein isoforms (hSOM1, hSOM2, hSOM3) and zebrafisch SOM (zfSOM) was performed with CLUSTALW. Complete conservation is indicated by an asterisk (*), conservation between amino acids with similar properties by a colon (:). The DNA binding domain is shown in yellow, the dimerization domain in blue. The alignment indicates that zfSOM is most likely the homologue of human SOM2.
**Supplemental tables: Transcripts regulated by SOM1 and SOM3.** The tables list the transcripts significantly regulated (p≤0.05) after overexpression of SOM1 (supplemental table I, 367 transcripts) or SOM3 (supplemental table II, 261 transcripts) in primary human endothelial cells when compared to cells transfected with an empty vector. The data were obtained from three biological replicates, with the exception of SOM1, where only two samples passed post extraction quality controls (see materials and methods). Shown are the probe identifiers from the microarrays, the gene symbols and RefSeq accession numbers where available, the direction of regulation (up or down) and the fold change in transcript levels.
### Supplemental table I: transcripts regulated by SOM1

<table>
<thead>
<tr>
<th>probe</th>
<th>gene symbol</th>
<th>RefSeq accession no.</th>
<th>regulation</th>
<th>fold change</th>
<th>significance (p-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A_33_P3395650</td>
<td>PCDHGA5</td>
<td>NM_032054</td>
<td>up</td>
<td>6.56</td>
<td>0.0428</td>
</tr>
<tr>
<td>A_33_P3368144</td>
<td>LOC652614</td>
<td>XM_942149</td>
<td>down</td>
<td>4.22</td>
<td>0.0035</td>
</tr>
<tr>
<td>A_19_P00812311</td>
<td></td>
<td></td>
<td>down</td>
<td>4.21</td>
<td>0.0376</td>
</tr>
<tr>
<td>A_19_P00330326</td>
<td></td>
<td></td>
<td>down</td>
<td>3.88</td>
<td>0.0051</td>
</tr>
<tr>
<td>A_24_P339560</td>
<td>SIGLEC11</td>
<td>NM_052884</td>
<td>down</td>
<td>3.58</td>
<td>0.0338</td>
</tr>
<tr>
<td>A_33_P3333003</td>
<td></td>
<td></td>
<td>down</td>
<td>3.37</td>
<td>0.0086</td>
</tr>
<tr>
<td>A_19_P00806490</td>
<td></td>
<td></td>
<td>down</td>
<td>3.36</td>
<td>0.0205</td>
</tr>
<tr>
<td>A_19_P00803051</td>
<td></td>
<td></td>
<td>down</td>
<td>2.97</td>
<td>0.0218</td>
</tr>
<tr>
<td>A_33_P3279708</td>
<td>RNU2-2</td>
<td>NR_002761</td>
<td>down</td>
<td>2.47</td>
<td>0.0393</td>
</tr>
<tr>
<td>A_33_P3374180</td>
<td>LOC645769</td>
<td>XR_015325</td>
<td>down</td>
<td>2.44</td>
<td>0.0174</td>
</tr>
<tr>
<td>A_24_P65060</td>
<td>MEX3B</td>
<td>NM_032246</td>
<td>up</td>
<td>2.35</td>
<td>0.0320</td>
</tr>
<tr>
<td>A_33_P3348992</td>
<td>LOC645769</td>
<td>XR_015325</td>
<td>up</td>
<td>2.28</td>
<td>0.0317</td>
</tr>
<tr>
<td>A_33_P3382595</td>
<td>RN7SK</td>
<td>NR_001445</td>
<td>down</td>
<td>2.26</td>
<td>0.0497</td>
</tr>
<tr>
<td>A_33_P3353622</td>
<td>RBAK</td>
<td>NM_021163</td>
<td>up</td>
<td>2.24</td>
<td>0.0138</td>
</tr>
<tr>
<td>A_23_P431179</td>
<td>HIST1H4A</td>
<td>NM_003538</td>
<td>down</td>
<td>2.24</td>
<td>0.0013</td>
</tr>
<tr>
<td>A_23_P167497</td>
<td>EFNA5</td>
<td>NM_001962</td>
<td>down</td>
<td>2.16</td>
<td>0.0163</td>
</tr>
<tr>
<td>A_33_P3229122</td>
<td>HIST1H2BF</td>
<td>NM_003522</td>
<td>down</td>
<td>2.14</td>
<td>0.0358</td>
</tr>
<tr>
<td>A_33_P329335</td>
<td>HIST3H2BB</td>
<td>NM_175055</td>
<td>down</td>
<td>2.14</td>
<td>0.0430</td>
</tr>
<tr>
<td>A_33_P3404989</td>
<td>HIST1H3H</td>
<td>NM_003536</td>
<td>down</td>
<td>2.04</td>
<td>0.0494</td>
</tr>
<tr>
<td>A_33_P3254695</td>
<td>RNU105A</td>
<td>NR_004404</td>
<td>down</td>
<td>2.03</td>
<td>0.0474</td>
</tr>
<tr>
<td>A_33_P3421674</td>
<td>LOC284620</td>
<td>XR_017656</td>
<td>down</td>
<td>1.99</td>
<td>0.0127</td>
</tr>
<tr>
<td>A_33_P3248962</td>
<td>LOC100131742</td>
<td>JR_078213</td>
<td>down</td>
<td>1.96</td>
<td>0.0355</td>
</tr>
<tr>
<td>A_33_P377380</td>
<td>LOC100130494</td>
<td>XM_001725351</td>
<td>down</td>
<td>1.94</td>
<td>0.0148</td>
</tr>
<tr>
<td>A_24_P555496</td>
<td>OSR2</td>
<td>NM_053001</td>
<td>down</td>
<td>1.94</td>
<td>0.0496</td>
</tr>
<tr>
<td>A_19_P00329986</td>
<td></td>
<td></td>
<td>down</td>
<td>1.90</td>
<td>0.0418</td>
</tr>
<tr>
<td>A_24_P940411</td>
<td>CXorf36</td>
<td>NM_176819</td>
<td>up</td>
<td>1.86</td>
<td>0.0459</td>
</tr>
<tr>
<td>A_33_P3265394</td>
<td>WDR74</td>
<td>up</td>
<td>1.85</td>
<td>0.0475</td>
<td></td>
</tr>
<tr>
<td>A_24_P156748</td>
<td>SLC30A2</td>
<td>NM_001004434</td>
<td>down</td>
<td>1.84</td>
<td>0.0292</td>
</tr>
<tr>
<td>A_23_P359540</td>
<td>HIST1H4F</td>
<td>NM_003540</td>
<td>down</td>
<td>1.82</td>
<td>0.0436</td>
</tr>
<tr>
<td>A_33_P3816266</td>
<td>ZNF253</td>
<td>up</td>
<td>1.80</td>
<td>0.0250</td>
<td></td>
</tr>
<tr>
<td>A_24_P225616</td>
<td>RRM2</td>
<td>NM_001034</td>
<td>up</td>
<td>1.79</td>
<td>0.0351</td>
</tr>
<tr>
<td>A_19_P00319741</td>
<td></td>
<td></td>
<td>down</td>
<td>1.78</td>
<td>0.0375</td>
</tr>
<tr>
<td>A_19_P00324351</td>
<td></td>
<td></td>
<td>down</td>
<td>1.74</td>
<td>0.0090</td>
</tr>
<tr>
<td>A_24_P20873</td>
<td>HIST1H4I</td>
<td>NM_003495</td>
<td>down</td>
<td>1.74</td>
<td>0.0385</td>
</tr>
<tr>
<td>A_33_P3407564</td>
<td>LOC647188</td>
<td>XM_001717128</td>
<td>down</td>
<td>1.73</td>
<td>0.0330</td>
</tr>
<tr>
<td>A_33_P3786807</td>
<td>SNAR-E</td>
<td>up</td>
<td>1.70</td>
<td>0.0242</td>
<td></td>
</tr>
<tr>
<td>A_33_P3393135</td>
<td></td>
<td>down</td>
<td>1.70</td>
<td>0.0199</td>
<td></td>
</tr>
<tr>
<td>A_33_P3421867</td>
<td>MDGA1</td>
<td>up</td>
<td>1.70</td>
<td>0.0499</td>
<td></td>
</tr>
<tr>
<td>A_24_P252794</td>
<td>CUL2</td>
<td>NM_003591</td>
<td>up</td>
<td>1.69</td>
<td>0.0409</td>
</tr>
<tr>
<td>A_23_P430558</td>
<td>CHRN</td>
<td>NM_000751</td>
<td>down</td>
<td>1.69</td>
<td>0.0237</td>
</tr>
<tr>
<td>A_24_P82135</td>
<td>SECISBP2L</td>
<td>NM_014701</td>
<td>up</td>
<td>1.68</td>
<td>0.0322</td>
</tr>
<tr>
<td>A_33_P3380372</td>
<td></td>
<td>down</td>
<td>1.68</td>
<td>0.0169</td>
<td></td>
</tr>
<tr>
<td>A_33_P3289541</td>
<td>MLLT1</td>
<td>NM_005934</td>
<td>up</td>
<td>1.67</td>
<td>0.0075</td>
</tr>
<tr>
<td>A_19_P00812901</td>
<td></td>
<td></td>
<td>down</td>
<td>1.67</td>
<td>0.0322</td>
</tr>
<tr>
<td>A_24_P918843</td>
<td>RALGAPA1</td>
<td>NM_194301</td>
<td>up</td>
<td>1.67</td>
<td>0.0313</td>
</tr>
<tr>
<td>A_33_P3418130</td>
<td>UPRT</td>
<td>NM_145052</td>
<td>up</td>
<td>1.66</td>
<td>0.0470</td>
</tr>
<tr>
<td>A_33_P3345350</td>
<td>DRGX</td>
<td>NM_00180520</td>
<td>down</td>
<td>1.66</td>
<td>0.0270</td>
</tr>
<tr>
<td>A_23_P416894</td>
<td>PION</td>
<td>NM_017439</td>
<td>up</td>
<td>1.66</td>
<td>0.0338</td>
</tr>
<tr>
<td>A_24_P322908</td>
<td>USP27X</td>
<td>NM_001145073</td>
<td>down</td>
<td>1.64</td>
<td>0.0270</td>
</tr>
<tr>
<td>A_19_P00322724</td>
<td></td>
<td></td>
<td>down</td>
<td>1.64</td>
<td>0.0409</td>
</tr>
</tbody>
</table>
### Supplemental table I: transcripts regulated by SOM1

<table>
<thead>
<tr>
<th>probe</th>
<th>gene symbol</th>
<th>RefSeq accession no.</th>
<th>regulation</th>
<th>fold change</th>
<th>significance (p-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A_24_P55148</td>
<td>HIST1H2BJ</td>
<td>NM_021058</td>
<td>down</td>
<td>1.64</td>
<td>0.0331</td>
</tr>
<tr>
<td>A_33_P3273854</td>
<td>NAAALDL2</td>
<td>NM_207015</td>
<td>down</td>
<td>1.62</td>
<td>0.0292</td>
</tr>
<tr>
<td>A_33_P3258382</td>
<td>HBA2</td>
<td>NM_000517</td>
<td>down</td>
<td>1.62</td>
<td>0.0449</td>
</tr>
<tr>
<td>A_33_P3294598</td>
<td>PCDH10</td>
<td>NM_020815</td>
<td>up</td>
<td>1.61</td>
<td>0.0464</td>
</tr>
<tr>
<td>A_33_P3238433</td>
<td>ALDH3A1</td>
<td>NM_01135168</td>
<td>down</td>
<td>1.60</td>
<td>0.0061</td>
</tr>
<tr>
<td>A_33_P3422429</td>
<td></td>
<td></td>
<td>down</td>
<td>1.59</td>
<td>0.0332</td>
</tr>
<tr>
<td>A_33_P3250055</td>
<td></td>
<td></td>
<td>down</td>
<td>1.59</td>
<td>0.0216</td>
</tr>
<tr>
<td>A_19_P00315716</td>
<td></td>
<td></td>
<td>down</td>
<td>1.59</td>
<td>0.0109</td>
</tr>
<tr>
<td>A_23_P148737</td>
<td>MYBPH</td>
<td>NM_004997</td>
<td>down</td>
<td>1.57</td>
<td>0.0211</td>
</tr>
<tr>
<td>A_19_P00329786</td>
<td></td>
<td></td>
<td>down</td>
<td>1.56</td>
<td>0.0068</td>
</tr>
<tr>
<td>A_33_P3241771</td>
<td>LOC440313</td>
<td>XR_041904</td>
<td>down</td>
<td>1.56</td>
<td>0.0405</td>
</tr>
<tr>
<td>A_24_P940149</td>
<td>C2CD2</td>
<td>NM_015500</td>
<td>up</td>
<td>1.56</td>
<td>0.0408</td>
</tr>
<tr>
<td>A_19_P00315943</td>
<td></td>
<td></td>
<td>down</td>
<td>1.53</td>
<td>0.0242</td>
</tr>
<tr>
<td>A_23_P140434</td>
<td>MYO5C</td>
<td>NM_018728</td>
<td>up</td>
<td>1.53</td>
<td>0.0424</td>
</tr>
<tr>
<td>A_33_P3420762</td>
<td></td>
<td></td>
<td>down</td>
<td>1.53</td>
<td>0.0381</td>
</tr>
<tr>
<td>A_33_P3283201</td>
<td></td>
<td></td>
<td>down</td>
<td>1.52</td>
<td>0.0221</td>
</tr>
<tr>
<td>A_33_P3241081</td>
<td>AKNAD1</td>
<td>NM_152763</td>
<td>down</td>
<td>1.52</td>
<td>0.0278</td>
</tr>
<tr>
<td>A_33_P3317731</td>
<td></td>
<td></td>
<td>down</td>
<td>1.51</td>
<td>0.0484</td>
</tr>
<tr>
<td>A_33_P3380751</td>
<td>ST8SIA1</td>
<td>NM_003034</td>
<td>down</td>
<td>1.51</td>
<td>0.0323</td>
</tr>
<tr>
<td>A_23_P146922</td>
<td>GAS6</td>
<td>NM_000820</td>
<td>up</td>
<td>1.51</td>
<td>0.0120</td>
</tr>
<tr>
<td>A_33_P3363091</td>
<td></td>
<td></td>
<td>up</td>
<td>1.50</td>
<td>0.0192</td>
</tr>
<tr>
<td>A_19_P00801387</td>
<td></td>
<td></td>
<td>up</td>
<td>1.49</td>
<td>0.0169</td>
</tr>
<tr>
<td>A_33_P3230526</td>
<td>MPRIP</td>
<td>NM_015134</td>
<td>up</td>
<td>1.49</td>
<td>0.0423</td>
</tr>
<tr>
<td>A_23_P251303</td>
<td>SEC16A</td>
<td>NM_014866</td>
<td>up</td>
<td>1.49</td>
<td>0.0063</td>
</tr>
<tr>
<td>A_23_P141738</td>
<td>SS18</td>
<td>NM_001007559</td>
<td>up</td>
<td>1.49</td>
<td>0.0448</td>
</tr>
<tr>
<td>A_23_P118095</td>
<td>RPL3L</td>
<td>NM_005061</td>
<td>down</td>
<td>1.49</td>
<td>0.0475</td>
</tr>
<tr>
<td>A_33_P3336262</td>
<td></td>
<td></td>
<td>down</td>
<td>1.48</td>
<td>0.0242</td>
</tr>
<tr>
<td>A_24_P374973</td>
<td></td>
<td></td>
<td>up</td>
<td>1.48</td>
<td>0.0028</td>
</tr>
<tr>
<td>A_19_P0080731</td>
<td></td>
<td></td>
<td>down</td>
<td>1.47</td>
<td>0.0007</td>
</tr>
<tr>
<td>A_19_P00315499</td>
<td></td>
<td></td>
<td>down</td>
<td>1.47</td>
<td>0.0440</td>
</tr>
<tr>
<td>A_33_P3416588</td>
<td>RIT2</td>
<td>NM_002930</td>
<td>down</td>
<td>1.47</td>
<td>0.0372</td>
</tr>
<tr>
<td>A_23_P559069</td>
<td>HIST1H2BO</td>
<td>NM_003527</td>
<td>down</td>
<td>1.47</td>
<td>0.0392</td>
</tr>
<tr>
<td>A_23_P161399</td>
<td>MX11</td>
<td>NM_130439</td>
<td>up</td>
<td>1.47</td>
<td>0.0307</td>
</tr>
<tr>
<td>A_33_P3400171</td>
<td>AKT2</td>
<td>NM_001626</td>
<td>up</td>
<td>1.46</td>
<td>0.0279</td>
</tr>
<tr>
<td>A_23_P6398</td>
<td>AP1B1</td>
<td>NM_001127</td>
<td>up</td>
<td>1.46</td>
<td>0.0442</td>
</tr>
<tr>
<td>A_33_P3226755</td>
<td>LOC100131857</td>
<td></td>
<td>down</td>
<td>1.46</td>
<td>0.0057</td>
</tr>
<tr>
<td>A_19_P00320777</td>
<td></td>
<td></td>
<td>up</td>
<td>1.45</td>
<td>0.0320</td>
</tr>
<tr>
<td>A_19_P00807791</td>
<td></td>
<td></td>
<td>down</td>
<td>1.45</td>
<td>0.0105</td>
</tr>
<tr>
<td>A_23_P3956</td>
<td>C1QTNF1</td>
<td>NM_198594</td>
<td>up</td>
<td>1.45</td>
<td>0.0448</td>
</tr>
<tr>
<td>A_23_P388681</td>
<td>ELAVL1</td>
<td>NM_001419</td>
<td>up</td>
<td>1.44</td>
<td>0.0181</td>
</tr>
<tr>
<td>A_23_P111054</td>
<td>HIST1H2BB</td>
<td>NM_021062</td>
<td>down</td>
<td>1.44</td>
<td>0.0461</td>
</tr>
<tr>
<td>A_23_P74391</td>
<td>OPN3</td>
<td>NM_014322</td>
<td>up</td>
<td>1.44</td>
<td>0.0242</td>
</tr>
<tr>
<td>A_33_P3228445</td>
<td>FXYD2</td>
<td>NM_001680</td>
<td>down</td>
<td>1.44</td>
<td>0.0471</td>
</tr>
<tr>
<td>A_23_P33407</td>
<td>HERC2</td>
<td>NM_004667</td>
<td>up</td>
<td>1.44</td>
<td>0.0360</td>
</tr>
<tr>
<td>A_32_P178800</td>
<td>ITGA2</td>
<td>NM_002203</td>
<td>up</td>
<td>1.44</td>
<td>0.0229</td>
</tr>
<tr>
<td>A_33_P3253501</td>
<td>HIST2H2BF</td>
<td>NM_001161334</td>
<td>down</td>
<td>1.43</td>
<td>0.0422</td>
</tr>
<tr>
<td>A_33_P3292915</td>
<td>TCOF1</td>
<td>NM_001008657</td>
<td>up</td>
<td>1.43</td>
<td>0.0291</td>
</tr>
<tr>
<td>A_23_P366216</td>
<td>HIST1H2BH</td>
<td>NM_003524</td>
<td>down</td>
<td>1.43</td>
<td>0.0312</td>
</tr>
<tr>
<td>A_19_P00807159</td>
<td></td>
<td></td>
<td>down</td>
<td>1.43</td>
<td>0.0437</td>
</tr>
<tr>
<td>A_24_P373768</td>
<td>GIPR</td>
<td>NM_000164</td>
<td>down</td>
<td>1.42</td>
<td>0.0254</td>
</tr>
</tbody>
</table>
## Supplemental table I: transcripts regulated by SOM1

<table>
<thead>
<tr>
<th>probe</th>
<th>gene symbol</th>
<th>RefSeq accession no.</th>
<th>regulation</th>
<th>fold change</th>
<th>significance (p-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A_32_P528311</td>
<td>RTN4RL2</td>
<td>NM_178570</td>
<td>down</td>
<td>1.42</td>
<td>0.0289</td>
</tr>
<tr>
<td>A_23_P92349</td>
<td>FGFRL1</td>
<td>NM_001004356</td>
<td>up</td>
<td>1.42</td>
<td>0.0451</td>
</tr>
<tr>
<td>A_33_P3229083</td>
<td>HIST1H2BK</td>
<td>NM_080593</td>
<td>down</td>
<td>1.42</td>
<td>0.0344</td>
</tr>
<tr>
<td>A_24_P944144</td>
<td>DIDO1</td>
<td>NM_080797</td>
<td>up</td>
<td>1.42</td>
<td>0.0154</td>
</tr>
<tr>
<td>A_23_P8013</td>
<td>HIST1H2BL</td>
<td>NM_003519</td>
<td>down</td>
<td>1.42</td>
<td>0.0362</td>
</tr>
<tr>
<td>A_23_P111041</td>
<td>HIST1H2BI</td>
<td>NM_003525</td>
<td>down</td>
<td>1.42</td>
<td>0.0320</td>
</tr>
<tr>
<td>A_24_P399009</td>
<td>EDC4</td>
<td>NM_014329</td>
<td>up</td>
<td>1.41</td>
<td>0.0064</td>
</tr>
<tr>
<td>A_19_P00324163</td>
<td></td>
<td></td>
<td>down</td>
<td>1.41</td>
<td>0.0409</td>
</tr>
<tr>
<td>A_33_P3256033</td>
<td>CBL</td>
<td>NM_005188</td>
<td>up</td>
<td>1.41</td>
<td>0.0169</td>
</tr>
<tr>
<td>A_33_P3306442</td>
<td>QRIC1H2</td>
<td>NM_032134</td>
<td>up</td>
<td>1.41</td>
<td>0.0337</td>
</tr>
<tr>
<td>A_23_P93180</td>
<td>HIST1H2BC</td>
<td>NM_003526</td>
<td>down</td>
<td>1.41</td>
<td>0.0475</td>
</tr>
<tr>
<td>A_23_P340922</td>
<td>ZNF414</td>
<td>NM_032370</td>
<td>up</td>
<td>1.40</td>
<td>0.0411</td>
</tr>
<tr>
<td>A_24_P84880</td>
<td>LOC148709</td>
<td>NR_002929</td>
<td>up</td>
<td>1.40</td>
<td>0.0388</td>
</tr>
<tr>
<td>A_23_P113283</td>
<td>ZMAT3</td>
<td>NM_022470</td>
<td>up</td>
<td>1.39</td>
<td>0.0366</td>
</tr>
<tr>
<td>A_23_P104073</td>
<td>S100A3</td>
<td>NM_002960</td>
<td>down</td>
<td>1.39</td>
<td>0.0393</td>
</tr>
<tr>
<td>A_19_P00811747</td>
<td></td>
<td></td>
<td>down</td>
<td>1.39</td>
<td>0.0214</td>
</tr>
<tr>
<td>A_23_P72068</td>
<td>GMDS</td>
<td>NM_001500</td>
<td>up</td>
<td>1.39</td>
<td>0.0244</td>
</tr>
<tr>
<td>A_19_P00806178</td>
<td></td>
<td></td>
<td>down</td>
<td>1.39</td>
<td>0.0464</td>
</tr>
<tr>
<td>A_23_P4909</td>
<td>SNRNP70</td>
<td>NM_003089</td>
<td>up</td>
<td>1.38</td>
<td>0.0207</td>
</tr>
<tr>
<td>A_19_P00331764</td>
<td></td>
<td></td>
<td>down</td>
<td>1.38</td>
<td>0.0382</td>
</tr>
<tr>
<td>A_33_P3313597</td>
<td>XR_078912</td>
<td>NM_014628</td>
<td>up</td>
<td>1.38</td>
<td>0.0245</td>
</tr>
<tr>
<td>A_24_P392958</td>
<td>TBC1D20</td>
<td>NM_022757</td>
<td>up</td>
<td>1.37</td>
<td>0.0377</td>
</tr>
<tr>
<td>A_33_P3236563</td>
<td>ALDH3B1</td>
<td>NM_001161473</td>
<td>down</td>
<td>1.37</td>
<td>0.0106</td>
</tr>
<tr>
<td>A_19_P00811026</td>
<td></td>
<td></td>
<td>down</td>
<td>1.37</td>
<td>0.0363</td>
</tr>
<tr>
<td>A_33_P3359250</td>
<td></td>
<td></td>
<td>down</td>
<td>1.37</td>
<td>0.0372</td>
</tr>
<tr>
<td>A_23_P12079</td>
<td>KCNC4</td>
<td>NM_153763</td>
<td>up</td>
<td>1.37</td>
<td>0.0394</td>
</tr>
<tr>
<td>A_23_P116587</td>
<td>OMP</td>
<td>NM_006189</td>
<td>down</td>
<td>1.37</td>
<td>0.0436</td>
</tr>
<tr>
<td>A_23_P204052</td>
<td>PCBP2</td>
<td>NM_031989</td>
<td>up</td>
<td>1.37</td>
<td>0.0076</td>
</tr>
<tr>
<td>A_24_P225961</td>
<td>DAG1</td>
<td>NM_004393</td>
<td>up</td>
<td>1.37</td>
<td>0.0056</td>
</tr>
<tr>
<td>A_19_P00329758</td>
<td></td>
<td></td>
<td>down</td>
<td>1.36</td>
<td>0.0389</td>
</tr>
<tr>
<td>A_33_P3280157</td>
<td>SNORD116-19</td>
<td>NR_001290</td>
<td>down</td>
<td>1.36</td>
<td>0.0411</td>
</tr>
<tr>
<td>A_24_P192197</td>
<td>WRNIP1</td>
<td>NM_130395</td>
<td>up</td>
<td>1.35</td>
<td>0.0433</td>
</tr>
<tr>
<td>A_24_P19370445</td>
<td></td>
<td></td>
<td>down</td>
<td>1.35</td>
<td>0.0367</td>
</tr>
<tr>
<td>A_23_P39931</td>
<td>DYSF</td>
<td>NM_003494</td>
<td>up</td>
<td>1.35</td>
<td>0.0306</td>
</tr>
<tr>
<td>A_33_P3269879</td>
<td>FAM160B2</td>
<td>NM_022749</td>
<td>up</td>
<td>1.35</td>
<td>0.0499</td>
</tr>
<tr>
<td>A_33_P3338968</td>
<td>LOC10013373</td>
<td>XM_001722336</td>
<td>down</td>
<td>1.35</td>
<td>0.0206</td>
</tr>
<tr>
<td>A_19_P00316734</td>
<td></td>
<td></td>
<td>down</td>
<td>1.35</td>
<td>0.0288</td>
</tr>
<tr>
<td>A_33_P3269453</td>
<td></td>
<td></td>
<td>up</td>
<td>1.34</td>
<td>0.0478</td>
</tr>
<tr>
<td>A_19_P00327579</td>
<td></td>
<td></td>
<td>down</td>
<td>1.34</td>
<td>0.0092</td>
</tr>
<tr>
<td>A_24_P322709</td>
<td>SNTA1</td>
<td>NM_003098</td>
<td>up</td>
<td>1.34</td>
<td>0.0205</td>
</tr>
<tr>
<td>A_33_P3272663</td>
<td></td>
<td></td>
<td>down</td>
<td>1.34</td>
<td>0.0495</td>
</tr>
<tr>
<td>A_33_P3348469</td>
<td>ANKRD13C</td>
<td>NM_030816</td>
<td>up</td>
<td>1.34</td>
<td>0.0469</td>
</tr>
<tr>
<td>A_33_P3317725</td>
<td></td>
<td></td>
<td>up</td>
<td>1.34</td>
<td>0.0376</td>
</tr>
<tr>
<td>A_19_P00813244</td>
<td></td>
<td></td>
<td>down</td>
<td>1.33</td>
<td>0.0395</td>
</tr>
<tr>
<td>A_23_P121806</td>
<td>ENOPH1</td>
<td>NM_021204</td>
<td>up</td>
<td>1.33</td>
<td>0.0379</td>
</tr>
<tr>
<td>A_19_P00322276</td>
<td></td>
<td></td>
<td>down</td>
<td>1.33</td>
<td>0.0284</td>
</tr>
<tr>
<td>A_23_P32029</td>
<td>SLC35D2</td>
<td>NM_007001</td>
<td>up</td>
<td>1.33</td>
<td>0.0140</td>
</tr>
<tr>
<td>A_33_P3412438</td>
<td></td>
<td></td>
<td>up</td>
<td>1.33</td>
<td>0.0480</td>
</tr>
<tr>
<td>A_24_P176079</td>
<td>WASF3</td>
<td>NM_006646</td>
<td>up</td>
<td>1.33</td>
<td>0.0216</td>
</tr>
</tbody>
</table>
**Supplemental table I: transcripts regulated by SOM1**

<table>
<thead>
<tr>
<th>probe</th>
<th>gene symbol</th>
<th>RefSeq accession no.</th>
<th>regulation</th>
<th>fold change</th>
<th>significance (p-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A_19_P00803850</td>
<td>OR2AG2</td>
<td>NM_001004490</td>
<td>down</td>
<td>1.33</td>
<td>0.0495</td>
</tr>
<tr>
<td>A_33_P3262069</td>
<td>LOC100130987</td>
<td>NR_024469</td>
<td>up</td>
<td>1.32</td>
<td>0.0414</td>
</tr>
<tr>
<td>A_33_P3308764</td>
<td>RXRG</td>
<td>NM_001009598</td>
<td>down</td>
<td>1.32</td>
<td>0.0314</td>
</tr>
<tr>
<td>A_23_P150064</td>
<td>MMRN2</td>
<td>NM_024756</td>
<td>up</td>
<td>1.32</td>
<td>0.0017</td>
</tr>
<tr>
<td>A_23_P368558</td>
<td>TMEM131</td>
<td>NM_015348</td>
<td>up</td>
<td>1.32</td>
<td>0.0273</td>
</tr>
<tr>
<td>A_23_P50946</td>
<td>RAMP1</td>
<td>NM_005855</td>
<td>down</td>
<td>1.32</td>
<td>0.0216</td>
</tr>
<tr>
<td>A_33_P3286616</td>
<td>IRAK1</td>
<td>NM_001569</td>
<td>up</td>
<td>1.32</td>
<td>0.0463</td>
</tr>
<tr>
<td>A_32_P206401</td>
<td>QSOX2</td>
<td>NM_181701</td>
<td>up</td>
<td>1.32</td>
<td>0.0332</td>
</tr>
<tr>
<td>A_24_P148750</td>
<td>SH3BP5</td>
<td>NM_004844</td>
<td>up</td>
<td>1.32</td>
<td>0.0088</td>
</tr>
<tr>
<td>A_19_P00318927</td>
<td></td>
<td></td>
<td>down</td>
<td>1.32</td>
<td>0.0091</td>
</tr>
<tr>
<td>A_19_P00328038</td>
<td></td>
<td></td>
<td>down</td>
<td>1.31</td>
<td>0.0411</td>
</tr>
<tr>
<td>A_19_P00331835</td>
<td></td>
<td></td>
<td>down</td>
<td>1.32</td>
<td>0.0452</td>
</tr>
<tr>
<td>A_19_P00316576</td>
<td></td>
<td></td>
<td>down</td>
<td>1.31</td>
<td>0.0102</td>
</tr>
<tr>
<td>A_19_P00321089</td>
<td></td>
<td></td>
<td>up</td>
<td>1.31</td>
<td>0.0079</td>
</tr>
<tr>
<td>A_33_P3399107</td>
<td>PI4KA</td>
<td>NM_058004</td>
<td>up</td>
<td>1.31</td>
<td>0.0486</td>
</tr>
<tr>
<td>A_33_P3315223</td>
<td>HNRNPA0</td>
<td>NM_006805</td>
<td>up</td>
<td>1.31</td>
<td>0.0478</td>
</tr>
<tr>
<td>A_19_P00809346</td>
<td></td>
<td></td>
<td>down</td>
<td>1.31</td>
<td>0.0267</td>
</tr>
<tr>
<td>A_19_P00801627</td>
<td></td>
<td></td>
<td>down</td>
<td>1.31</td>
<td>0.0399</td>
</tr>
<tr>
<td>A_24_P396375</td>
<td>ECE1</td>
<td>NM_001113347</td>
<td>up</td>
<td>1.31</td>
<td>0.0302</td>
</tr>
<tr>
<td>A_23_P94053</td>
<td>TRRAP</td>
<td>NM_003496</td>
<td>up</td>
<td>1.30</td>
<td>0.0427</td>
</tr>
<tr>
<td>A_24_P235049</td>
<td>MTHFD1L</td>
<td>NM_015440</td>
<td>up</td>
<td>1.30</td>
<td>0.0176</td>
</tr>
<tr>
<td>A_23_P160862</td>
<td>HNRNPU</td>
<td>NM_031844</td>
<td>up</td>
<td>1.30</td>
<td>0.0017</td>
</tr>
<tr>
<td>A_19_P00806063</td>
<td></td>
<td></td>
<td>down</td>
<td>1.30</td>
<td>0.0124</td>
</tr>
<tr>
<td>A_23_P123086</td>
<td>KIAA1908</td>
<td>NR_027329</td>
<td>up</td>
<td>1.30</td>
<td>0.0498</td>
</tr>
<tr>
<td>A_32_P100430</td>
<td>LOC100128737</td>
<td>XM_001715932</td>
<td>down</td>
<td>1.30</td>
<td>0.0044</td>
</tr>
<tr>
<td>A_33_P3414851</td>
<td>RNF220</td>
<td>NM_018150</td>
<td>up</td>
<td>1.30</td>
<td>0.0396</td>
</tr>
<tr>
<td>A_19_P00319843</td>
<td></td>
<td></td>
<td>down</td>
<td>1.30</td>
<td>0.0109</td>
</tr>
<tr>
<td>A_23_P358221</td>
<td>UBXN7</td>
<td>NM_015562</td>
<td>up</td>
<td>1.29</td>
<td>0.0261</td>
</tr>
<tr>
<td>A_19_P00813242</td>
<td></td>
<td></td>
<td>down</td>
<td>1.29</td>
<td>0.0444</td>
</tr>
<tr>
<td>A_33:P3288180</td>
<td>CDC2L1</td>
<td>NM_033489</td>
<td>up</td>
<td>1.29</td>
<td>0.0315</td>
</tr>
<tr>
<td>A_33:P3257784</td>
<td>TSPAN17</td>
<td>NM_012171</td>
<td>up</td>
<td>1.29</td>
<td>0.0381</td>
</tr>
<tr>
<td>A_33:P3373745</td>
<td>BRD4</td>
<td>NM_014299</td>
<td>up</td>
<td>1.29</td>
<td>0.0450</td>
</tr>
<tr>
<td>A_19_P00322525</td>
<td></td>
<td></td>
<td>down</td>
<td>1.29</td>
<td>0.0255</td>
</tr>
<tr>
<td>A_33:P3363100</td>
<td>LOC100294232</td>
<td>XM_002343989</td>
<td>down</td>
<td>1.29</td>
<td>0.0397</td>
</tr>
<tr>
<td>A_33:P3382271</td>
<td></td>
<td></td>
<td>down</td>
<td>1.29</td>
<td>0.0277</td>
</tr>
<tr>
<td>A_23:P501339</td>
<td>CDIPT</td>
<td>NM_006319</td>
<td>up</td>
<td>1.29</td>
<td>0.0323</td>
</tr>
<tr>
<td>A_33:P3317473</td>
<td></td>
<td></td>
<td>down</td>
<td>1.28</td>
<td>0.0215</td>
</tr>
<tr>
<td>A_23:P259012</td>
<td>BAP1</td>
<td>NM_004656</td>
<td>up</td>
<td>1.28</td>
<td>0.0000</td>
</tr>
<tr>
<td>A_23:P168443</td>
<td>EPHB4</td>
<td>NM_004444</td>
<td>up</td>
<td>1.28</td>
<td>0.0334</td>
</tr>
<tr>
<td>A_23:P414252</td>
<td>SNX8</td>
<td>NM_013321</td>
<td>down</td>
<td>1.28</td>
<td>0.0327</td>
</tr>
<tr>
<td>A_33:P3307910</td>
<td></td>
<td></td>
<td>down</td>
<td>1.28</td>
<td>0.0180</td>
</tr>
<tr>
<td>A_23:P74799</td>
<td>SLC25A24</td>
<td>NM_0213651</td>
<td>up</td>
<td>1.28</td>
<td>0.0469</td>
</tr>
<tr>
<td>A_24:P15754</td>
<td>TOMM40</td>
<td>NM_006114</td>
<td>up</td>
<td>1.28</td>
<td>0.0488</td>
</tr>
<tr>
<td>A_33:P3420655</td>
<td>KDM4A</td>
<td>NM_014663</td>
<td>down</td>
<td>1.28</td>
<td>0.0311</td>
</tr>
<tr>
<td>A_23:P331598</td>
<td>IPO7</td>
<td>NM_006391</td>
<td>up</td>
<td>1.28</td>
<td>0.0408</td>
</tr>
<tr>
<td>A_33:P3322945</td>
<td>SPDYC</td>
<td>NM_001008778</td>
<td>down</td>
<td>1.27</td>
<td>0.0453</td>
</tr>
<tr>
<td>A_33:P3260605</td>
<td>CTNNAL1</td>
<td>NM_003798</td>
<td>down</td>
<td>1.27</td>
<td>0.0426</td>
</tr>
<tr>
<td>A_19_P00800805</td>
<td></td>
<td></td>
<td>down</td>
<td>1.27</td>
<td>0.0485</td>
</tr>
</tbody>
</table>
### Supplemental table I: transcripts regulated by SOM1

<table>
<thead>
<tr>
<th>probe</th>
<th>gene symbol</th>
<th>RefSeq accession no.</th>
<th>regulation</th>
<th>fold change</th>
<th>significance (p-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A_33_P325661</td>
<td>LOC100292270</td>
<td>XM_002346039</td>
<td>down</td>
<td>1.27</td>
<td>0.0078</td>
</tr>
<tr>
<td>A_23_P133923</td>
<td>BAT4</td>
<td>NM_033177</td>
<td>down</td>
<td>1.27</td>
<td>0.0446</td>
</tr>
<tr>
<td>A_23_P49674</td>
<td>ARHGEF15</td>
<td>NM_173728</td>
<td>up</td>
<td>1.27</td>
<td>0.0035</td>
</tr>
<tr>
<td>A_23_P64617</td>
<td>FZD4</td>
<td>NM_012193</td>
<td>up</td>
<td>1.27</td>
<td>0.0322</td>
</tr>
<tr>
<td>A_24_P33444</td>
<td>WWAHE</td>
<td>NM_006761</td>
<td>down</td>
<td>1.27</td>
<td>0.0385</td>
</tr>
<tr>
<td>A_23_P69513</td>
<td>RGS12</td>
<td>NM_198229</td>
<td>up</td>
<td>1.27</td>
<td>0.0362</td>
</tr>
<tr>
<td>A_23_P119344</td>
<td>TEAD2</td>
<td>NM_003598</td>
<td>up</td>
<td>1.26</td>
<td>0.0055</td>
</tr>
<tr>
<td>A_19_P00809859</td>
<td></td>
<td></td>
<td>down</td>
<td>1.26</td>
<td>0.0062</td>
</tr>
<tr>
<td>A_24_P82106</td>
<td>MMP14</td>
<td>NM_004995</td>
<td>up</td>
<td>1.26</td>
<td>0.0316</td>
</tr>
<tr>
<td>A_33_P3293529</td>
<td>LOC100129527</td>
<td>XM_001715665</td>
<td>down</td>
<td>1.26</td>
<td>0.0217</td>
</tr>
<tr>
<td>A_33_P335590</td>
<td>CCDC64B</td>
<td>NM_001103175</td>
<td>down</td>
<td>1.26</td>
<td>0.0388</td>
</tr>
<tr>
<td>A_24_P286465</td>
<td>PURB</td>
<td>NM_033224</td>
<td>up</td>
<td>1.26</td>
<td>0.0273</td>
</tr>
<tr>
<td>A_23_P334282</td>
<td>BMP2K</td>
<td>NM_017593</td>
<td>up</td>
<td>1.26</td>
<td>0.0432</td>
</tr>
<tr>
<td>A_33_P3216869</td>
<td>CRAP1</td>
<td>NM_004378</td>
<td>down</td>
<td>1.26</td>
<td>0.0051</td>
</tr>
<tr>
<td>A_33_P3269359</td>
<td>UNQ1887</td>
<td>NM_139015</td>
<td>up</td>
<td>1.26</td>
<td>0.0221</td>
</tr>
<tr>
<td>A_23_P38219</td>
<td>PRPF8</td>
<td>NM_006445</td>
<td>up</td>
<td>1.26</td>
<td>0.0217</td>
</tr>
<tr>
<td>A_19_P00811288</td>
<td></td>
<td></td>
<td>down</td>
<td>1.26</td>
<td>0.0303</td>
</tr>
<tr>
<td>A_23_P157607</td>
<td>INTS10</td>
<td>NM_018142</td>
<td>up</td>
<td>1.26</td>
<td>0.0173</td>
</tr>
<tr>
<td>A_23_P105562</td>
<td>VWF</td>
<td>NM_000552</td>
<td>up</td>
<td>1.26</td>
<td>0.0086</td>
</tr>
<tr>
<td>A_33_P3235568</td>
<td>CAPZB</td>
<td>NM_004930</td>
<td>up</td>
<td>1.25</td>
<td>0.0322</td>
</tr>
<tr>
<td>A_33_P3214597</td>
<td>RP9P</td>
<td>NR_003500</td>
<td>down</td>
<td>1.25</td>
<td>0.0212</td>
</tr>
<tr>
<td>A_23_P55998</td>
<td>SLC1A5</td>
<td>NM_005628</td>
<td>up</td>
<td>1.25</td>
<td>0.0277</td>
</tr>
<tr>
<td>A_23_P342825</td>
<td>FBXW4</td>
<td>NM_022039</td>
<td>up</td>
<td>1.25</td>
<td>0.0163</td>
</tr>
<tr>
<td>A_24_P9321</td>
<td>HIST1H3I</td>
<td>NM_003533</td>
<td>down</td>
<td>1.25</td>
<td>0.0493</td>
</tr>
<tr>
<td>A_32_P114215</td>
<td>COMMD6</td>
<td>NM_203497</td>
<td>down</td>
<td>1.25</td>
<td>0.0285</td>
</tr>
<tr>
<td>A_23_P106016</td>
<td>PRKD1</td>
<td>NM_002742</td>
<td>up</td>
<td>1.25</td>
<td>0.0094</td>
</tr>
<tr>
<td>A_19_P00319537</td>
<td></td>
<td></td>
<td>down</td>
<td>1.25</td>
<td>0.0407</td>
</tr>
<tr>
<td>A_23_P128919</td>
<td>LGALS3</td>
<td>NM_002306</td>
<td>down</td>
<td>1.25</td>
<td>0.0397</td>
</tr>
<tr>
<td>A_23_P37778</td>
<td>FHOD1</td>
<td>NM_013241</td>
<td>up</td>
<td>1.25</td>
<td>0.0479</td>
</tr>
<tr>
<td>A_23_P12849</td>
<td>FBXO18</td>
<td>NM_178150</td>
<td>up</td>
<td>1.24</td>
<td>0.0334</td>
</tr>
<tr>
<td>A_33_P3378031</td>
<td>TMEM18</td>
<td>NM_152834</td>
<td>down</td>
<td>1.24</td>
<td>0.0159</td>
</tr>
<tr>
<td>A_24_P876408</td>
<td>C11orf95</td>
<td>NM_001144936</td>
<td>up</td>
<td>1.24</td>
<td>0.0121</td>
</tr>
<tr>
<td>A_32_P217655</td>
<td>LOC645166</td>
<td>NR_027355</td>
<td>down</td>
<td>1.24</td>
<td>0.0221</td>
</tr>
<tr>
<td>A_19_P00808120</td>
<td></td>
<td></td>
<td>down</td>
<td>1.24</td>
<td>0.0336</td>
</tr>
<tr>
<td>A_19_P00808774</td>
<td></td>
<td></td>
<td>up</td>
<td>1.24</td>
<td>0.0370</td>
</tr>
<tr>
<td>A_23_P58482</td>
<td>MGAT1</td>
<td>NM_002406</td>
<td>up</td>
<td>1.24</td>
<td>0.0236</td>
</tr>
<tr>
<td>A_23_P206369</td>
<td>TMEM208</td>
<td>NM_014187</td>
<td>down</td>
<td>1.24</td>
<td>0.0354</td>
</tr>
<tr>
<td>A_23_P69521</td>
<td>CCNI</td>
<td>NM_006835</td>
<td>up</td>
<td>1.24</td>
<td>0.0363</td>
</tr>
<tr>
<td>A_24_P348989</td>
<td>LILRA1</td>
<td>NM_006863</td>
<td>down</td>
<td>1.23</td>
<td>0.0351</td>
</tr>
<tr>
<td>A_24_P398130</td>
<td>USP6NL</td>
<td>NM_014688</td>
<td>up</td>
<td>1.23</td>
<td>0.0184</td>
</tr>
<tr>
<td>A_23_P136232</td>
<td>IMPAD1</td>
<td>NM_017813</td>
<td>up</td>
<td>1.23</td>
<td>0.0498</td>
</tr>
<tr>
<td>A_33_P3378644</td>
<td>PHC1</td>
<td>NM_004426</td>
<td>up</td>
<td>1.23</td>
<td>0.0328</td>
</tr>
<tr>
<td>A_33_P3411741</td>
<td>SIDT2</td>
<td>NM_001040455</td>
<td>up</td>
<td>1.23</td>
<td>0.0098</td>
</tr>
<tr>
<td>A_24_P497186</td>
<td>IRF2BP2</td>
<td>NM_182972</td>
<td>up</td>
<td>1.23</td>
<td>0.0188</td>
</tr>
<tr>
<td>A_24_P161973</td>
<td>ATP11A</td>
<td>NM_015205</td>
<td>up</td>
<td>1.23</td>
<td>0.0398</td>
</tr>
<tr>
<td>A_33_P3320403</td>
<td>LOC100128528</td>
<td>XM_001724199</td>
<td>down</td>
<td>1.23</td>
<td>0.0218</td>
</tr>
<tr>
<td>A_23_P73457</td>
<td>RUFY1</td>
<td>NM_025158</td>
<td>up</td>
<td>1.23</td>
<td>0.0282</td>
</tr>
<tr>
<td>A_23_P425502</td>
<td>DONSON</td>
<td>NM_017613</td>
<td>up</td>
<td>1.23</td>
<td>0.0354</td>
</tr>
<tr>
<td>A_33_P3299279</td>
<td>C5orf39</td>
<td>NM_001014279</td>
<td>down</td>
<td>1.23</td>
<td>0.0441</td>
</tr>
<tr>
<td>A_23_P74950</td>
<td>RCC2</td>
<td>NM_018715</td>
<td>up</td>
<td>1.22</td>
<td>0.0020</td>
</tr>
<tr>
<td>probe</td>
<td>gene symbol</td>
<td>RefSeq accession no.</td>
<td>regulation</td>
<td>fold change</td>
<td>significance (p-value)</td>
</tr>
<tr>
<td>----------</td>
<td>-------------</td>
<td>----------------------</td>
<td>------------</td>
<td>-------------</td>
<td>------------------------</td>
</tr>
<tr>
<td>A_24_P14367</td>
<td>PTBP1</td>
<td>NM_002819</td>
<td>up</td>
<td>1.22</td>
<td>0.0244</td>
</tr>
<tr>
<td>A_23_P80336</td>
<td>TOMM22</td>
<td>NM_020243</td>
<td>down</td>
<td>1.22</td>
<td>0.0376</td>
</tr>
<tr>
<td>A_19_P00800454</td>
<td></td>
<td></td>
<td>down</td>
<td>1.22</td>
<td>0.0381</td>
</tr>
<tr>
<td>A_23_P142174</td>
<td>FOXA3</td>
<td>NM_004497</td>
<td>down</td>
<td>1.22</td>
<td>0.0372</td>
</tr>
<tr>
<td>A_23_P135857</td>
<td>EIF2AK3</td>
<td>NM_004836</td>
<td>up</td>
<td>1.22</td>
<td>0.0498</td>
</tr>
<tr>
<td>A_33_P3405754</td>
<td></td>
<td></td>
<td>down</td>
<td>1.22</td>
<td>0.0205</td>
</tr>
<tr>
<td>A_24_P271527</td>
<td>JOSD1</td>
<td>NM_014876</td>
<td>up</td>
<td>1.22</td>
<td>0.0383</td>
</tr>
<tr>
<td>A_33_P3281616</td>
<td>C22orf40</td>
<td>NM_207327</td>
<td>down</td>
<td>1.22</td>
<td>0.0321</td>
</tr>
<tr>
<td>A_23_P135857</td>
<td>EIF2AK3</td>
<td>NM_004836</td>
<td>up</td>
<td>1.22</td>
<td>0.0498</td>
</tr>
<tr>
<td>A_23_P130531</td>
<td>CDC37</td>
<td>NM_007065</td>
<td>up</td>
<td>1.21</td>
<td>0.0333</td>
</tr>
<tr>
<td>A_23_P476</td>
<td>MPZL1</td>
<td>NM_003953</td>
<td>up</td>
<td>1.21</td>
<td>0.0292</td>
</tr>
<tr>
<td>A_33_P3232523</td>
<td></td>
<td></td>
<td>down</td>
<td>1.21</td>
<td>0.0381</td>
</tr>
<tr>
<td>A_23_P99811</td>
<td>MDP1</td>
<td>NM_138476</td>
<td>down</td>
<td>1.21</td>
<td>0.0308</td>
</tr>
<tr>
<td>A_23_P15182</td>
<td>ARL2BP</td>
<td>NM_012106</td>
<td>down</td>
<td>1.21</td>
<td>0.0352</td>
</tr>
<tr>
<td>A_23_P155147</td>
<td>ZBED4</td>
<td>NM_014838</td>
<td>up</td>
<td>1.21</td>
<td>0.0249</td>
</tr>
<tr>
<td>A_23_P47073</td>
<td>WDR37</td>
<td>NM_014023</td>
<td>up</td>
<td>1.21</td>
<td>0.0272</td>
</tr>
<tr>
<td>A_19_P00325103</td>
<td></td>
<td></td>
<td>down</td>
<td>1.21</td>
<td>0.0488</td>
</tr>
<tr>
<td>A_33_P3361817</td>
<td></td>
<td></td>
<td>up</td>
<td>1.21</td>
<td>0.0454</td>
</tr>
<tr>
<td>A_32_P183609</td>
<td>ASB1</td>
<td>NM_001040445</td>
<td>up</td>
<td>1.21</td>
<td>0.0334</td>
</tr>
<tr>
<td>A_33_P3377619</td>
<td>C10orf46</td>
<td>NM_153810</td>
<td>up</td>
<td>1.21</td>
<td>0.0364</td>
</tr>
<tr>
<td>A_23_P23924</td>
<td>CAPN2</td>
<td>NM_001748</td>
<td>up</td>
<td>1.20</td>
<td>0.0002</td>
</tr>
<tr>
<td>A_33_P3306327</td>
<td></td>
<td></td>
<td>up</td>
<td>1.20</td>
<td>0.0388</td>
</tr>
<tr>
<td>A_33_P3415216</td>
<td>MPV17</td>
<td>NM_002437</td>
<td>down</td>
<td>1.20</td>
<td>0.0235</td>
</tr>
<tr>
<td>A_19_P00806320</td>
<td></td>
<td></td>
<td>down</td>
<td>1.20</td>
<td>0.0439</td>
</tr>
<tr>
<td>A_33_P3502640</td>
<td>DTX2</td>
<td>NM_020892</td>
<td>up</td>
<td>1.20</td>
<td>0.0051</td>
</tr>
<tr>
<td>A_33_P3239195</td>
<td></td>
<td></td>
<td>down</td>
<td>1.20</td>
<td>0.0450</td>
</tr>
<tr>
<td>A_24_P353289</td>
<td>C22orf13</td>
<td>NM_031444</td>
<td>up</td>
<td>1.20</td>
<td>0.0155</td>
</tr>
<tr>
<td>A_32_P67623</td>
<td>FAM120C</td>
<td>NM_017848</td>
<td>up</td>
<td>1.19</td>
<td>0.0159</td>
</tr>
<tr>
<td>A_33_P3280213</td>
<td>CTS4A</td>
<td>NM_001127695</td>
<td>up</td>
<td>1.19</td>
<td>0.0359</td>
</tr>
<tr>
<td>A_33_P3272563</td>
<td>NMT2</td>
<td>NM_004808</td>
<td>up</td>
<td>1.19</td>
<td>0.0237</td>
</tr>
<tr>
<td>A_23_P4425</td>
<td>FLI1</td>
<td>NM_0002018</td>
<td>up</td>
<td>1.19</td>
<td>0.0265</td>
</tr>
<tr>
<td>A_23_P329112</td>
<td>JAK3</td>
<td>NM_000215</td>
<td>down</td>
<td>1.19</td>
<td>0.0177</td>
</tr>
<tr>
<td>A_33_P3591972</td>
<td>GLYCTK</td>
<td>NM_001144951</td>
<td>up</td>
<td>1.19</td>
<td>0.0483</td>
</tr>
<tr>
<td>A_23_P306933</td>
<td>PPTC7</td>
<td>NM_139283</td>
<td>up</td>
<td>1.19</td>
<td>0.0385</td>
</tr>
<tr>
<td>A_23_P48717</td>
<td>NPC2</td>
<td>NM_006432</td>
<td>down</td>
<td>1.19</td>
<td>0.0178</td>
</tr>
<tr>
<td>A_23_P108244</td>
<td>COX6B1</td>
<td>NM_001863</td>
<td>down</td>
<td>1.19</td>
<td>0.0456</td>
</tr>
<tr>
<td>A_23_P30972</td>
<td>ASCC3</td>
<td>NM_022091</td>
<td>down</td>
<td>1.18</td>
<td>0.0479</td>
</tr>
<tr>
<td>A_23_P115137</td>
<td>HBXIP</td>
<td>NM_006402</td>
<td>down</td>
<td>1.18</td>
<td>0.0098</td>
</tr>
<tr>
<td>A_23_P254944</td>
<td>GSTT1</td>
<td>NM_000853</td>
<td>down</td>
<td>1.18</td>
<td>0.0490</td>
</tr>
<tr>
<td>A_33_P3418010</td>
<td>NUP62</td>
<td>NM_153719</td>
<td>up</td>
<td>1.18</td>
<td>0.0369</td>
</tr>
<tr>
<td>A_33_P3240229</td>
<td>CREBBP</td>
<td>NM_004380</td>
<td>up</td>
<td>1.18</td>
<td>0.0488</td>
</tr>
<tr>
<td>A_33_P3258091</td>
<td>RNPEP</td>
<td>NM_002016</td>
<td>up</td>
<td>1.18</td>
<td>0.0417</td>
</tr>
<tr>
<td>A_23_P129118</td>
<td>PDCD7</td>
<td>NM_005707</td>
<td>up</td>
<td>1.17</td>
<td>0.0220</td>
</tr>
<tr>
<td>A_24_P97197</td>
<td>CTDSP1</td>
<td>NM_182642</td>
<td>up</td>
<td>1.17</td>
<td>0.0452</td>
</tr>
<tr>
<td>A_23_P256682</td>
<td>APEX2</td>
<td>NM_014481</td>
<td>down</td>
<td>1.17</td>
<td>0.0256</td>
</tr>
<tr>
<td>A_33_P3318671</td>
<td>LYPLA2</td>
<td>NM_007260</td>
<td>down</td>
<td>1.17</td>
<td>0.0352</td>
</tr>
<tr>
<td>A_32_P59010</td>
<td>TNFAIP8L3</td>
<td>NM_207381</td>
<td>up</td>
<td>1.17</td>
<td>0.0475</td>
</tr>
<tr>
<td>A_33_P3237775</td>
<td>NR1H3</td>
<td>NM_005693</td>
<td>down</td>
<td>1.17</td>
<td>0.0484</td>
</tr>
<tr>
<td>A_23_P131737</td>
<td>VPS54</td>
<td>NM_016516</td>
<td>up</td>
<td>1.17</td>
<td>0.0320</td>
</tr>
</tbody>
</table>
### Supplemental table I: transcripts regulated by SOM1

<table>
<thead>
<tr>
<th>probe</th>
<th>gene symbol</th>
<th>RefSeq accession no.</th>
<th>regulation</th>
<th>fold change</th>
<th>significance (p-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A_19_P00325284</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A_23_P142750</td>
<td>EIF2AK2</td>
<td>NM_002759</td>
<td>up</td>
<td>1.17</td>
<td>0.0411</td>
</tr>
<tr>
<td>A_33_P3336915</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A_23_P302094</td>
<td>IMP4</td>
<td>NM_033416</td>
<td>down</td>
<td>1.16</td>
<td>0.0489</td>
</tr>
<tr>
<td>A_33_P3216337</td>
<td>FTSJ1</td>
<td>NM_177439</td>
<td>up</td>
<td>1.16</td>
<td>0.0102</td>
</tr>
<tr>
<td>A_23_P208937</td>
<td>TLE6</td>
<td>NM_024760</td>
<td>up</td>
<td>1.16</td>
<td>0.0490</td>
</tr>
<tr>
<td>A_24_P397107</td>
<td>CDC25A</td>
<td>NM_001789</td>
<td>up</td>
<td>1.16</td>
<td>0.0336</td>
</tr>
<tr>
<td>A_33_P3371311</td>
<td>LOC648691</td>
<td>NR_027426</td>
<td>up</td>
<td>1.16</td>
<td>0.0171</td>
</tr>
<tr>
<td>A_23_P110811</td>
<td>COX7C</td>
<td>NM_001867</td>
<td>down</td>
<td>1.16</td>
<td>0.0076</td>
</tr>
<tr>
<td>A_24_P409985</td>
<td>TMEM44</td>
<td>NM_001011655</td>
<td>up</td>
<td>1.16</td>
<td>0.0461</td>
</tr>
<tr>
<td>A_23_P143440</td>
<td>DYNLRB1</td>
<td>NM_014183</td>
<td>down</td>
<td>1.16</td>
<td>0.0200</td>
</tr>
<tr>
<td>A_23_P103476</td>
<td>UBIAD1</td>
<td>NM_013319</td>
<td>down</td>
<td>1.16</td>
<td>0.0005</td>
</tr>
<tr>
<td>A_24_P329600</td>
<td>UBQLN1</td>
<td>NM_013438</td>
<td>up</td>
<td>1.16</td>
<td>0.0474</td>
</tr>
<tr>
<td>A_24_P389959</td>
<td>COP21</td>
<td>NM_016057</td>
<td>down</td>
<td>1.16</td>
<td>0.0468</td>
</tr>
<tr>
<td>A_33_P3272160</td>
<td>REXO4</td>
<td>NM_020385</td>
<td>down</td>
<td>1.16</td>
<td>0.0406</td>
</tr>
<tr>
<td>A_33_P3351914</td>
<td>NDUF10</td>
<td>NM_004544</td>
<td>down</td>
<td>1.16</td>
<td>0.0197</td>
</tr>
<tr>
<td>A_33_P3287922</td>
<td>KIAA0406</td>
<td>NM_014657</td>
<td>down</td>
<td>1.16</td>
<td>0.0399</td>
</tr>
<tr>
<td>A_19_P00316404</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A_33_P3377529</td>
<td>HOXA4</td>
<td>NM_002141</td>
<td>down</td>
<td>1.16</td>
<td>0.0367</td>
</tr>
<tr>
<td>A_23_P81690</td>
<td>COX7A2</td>
<td>NM_001865</td>
<td>down</td>
<td>1.16</td>
<td>0.0366</td>
</tr>
<tr>
<td>A_19_P00316758</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A_23_P34983</td>
<td>JTB</td>
<td>NM_006694</td>
<td>up</td>
<td>1.15</td>
<td>0.0152</td>
</tr>
<tr>
<td>A_23_P17287</td>
<td>IAH1</td>
<td>NM_001039613</td>
<td>down</td>
<td>1.15</td>
<td>0.0109</td>
</tr>
<tr>
<td>A_23_P32135</td>
<td>C9orf9</td>
<td>NM_018956</td>
<td>down</td>
<td>1.15</td>
<td>0.0403</td>
</tr>
<tr>
<td>A_23_P1111487</td>
<td>SRRT</td>
<td>NM_001128853</td>
<td>up</td>
<td>1.14</td>
<td>0.0293</td>
</tr>
<tr>
<td>A_19_P00316326</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A_33_P3309911</td>
<td>PRAMEF5</td>
<td>NM_001013407</td>
<td>down</td>
<td>1.14</td>
<td>0.0358</td>
</tr>
<tr>
<td>A_23_P115223</td>
<td>HAX1</td>
<td>NM_0066118</td>
<td>down</td>
<td>1.14</td>
<td>0.0181</td>
</tr>
<tr>
<td>A_33_P3422812</td>
<td>PL5283</td>
<td>NM_001130929</td>
<td>up</td>
<td>1.14</td>
<td>0.0224</td>
</tr>
<tr>
<td>A_23_P252653</td>
<td>STK25</td>
<td>NM_006374</td>
<td>up</td>
<td>1.13</td>
<td>0.0253</td>
</tr>
<tr>
<td>A_23_P42884</td>
<td>MRPS24</td>
<td>NM_032014</td>
<td>down</td>
<td>1.13</td>
<td>0.0413</td>
</tr>
<tr>
<td>A_33_P3415211</td>
<td>MPV17</td>
<td>NM_002437</td>
<td>down</td>
<td>1.13</td>
<td>0.0211</td>
</tr>
<tr>
<td>A_33_P3415191</td>
<td>ATP8B1</td>
<td>NM_005603</td>
<td>up</td>
<td>1.13</td>
<td>0.0233</td>
</tr>
<tr>
<td>A_33_P3365037</td>
<td>ERCC1</td>
<td>NM_020201</td>
<td>down</td>
<td>1.13</td>
<td>0.0303</td>
</tr>
<tr>
<td>A_23_P113317</td>
<td>P4HTM</td>
<td>NM_177938</td>
<td>up</td>
<td>1.13</td>
<td>0.0390</td>
</tr>
<tr>
<td>A_23_P169838</td>
<td>UNCP8A</td>
<td>NM_025154</td>
<td>up</td>
<td>1.13</td>
<td>0.0198</td>
</tr>
<tr>
<td>A_19_P00320731</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A_33_P3791123</td>
<td>ATP5L2</td>
<td>NM_001168577</td>
<td>down</td>
<td>1.12</td>
<td>0.0373</td>
</tr>
<tr>
<td>A_32_P109794</td>
<td>STYX</td>
<td>NM_145251</td>
<td>up</td>
<td>1.12</td>
<td>0.0231</td>
</tr>
<tr>
<td>A_33_P3241596</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A_23_P102412</td>
<td>PLEKHA3</td>
<td>NM_019091</td>
<td>up</td>
<td>1.12</td>
<td>0.0239</td>
</tr>
<tr>
<td>A_23_P63371</td>
<td>TAL1</td>
<td>NM_003189</td>
<td>up</td>
<td>1.12</td>
<td>0.0290</td>
</tr>
<tr>
<td>A_33_P3651994</td>
<td>GNLS3P</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A_33_P3361182</td>
<td>PDCD11</td>
<td>NM_014976</td>
<td>down</td>
<td>1.12</td>
<td>0.0326</td>
</tr>
<tr>
<td>A_23_P204850</td>
<td>RB1</td>
<td>NM_000321</td>
<td>up</td>
<td>1.12</td>
<td>0.0463</td>
</tr>
<tr>
<td>A_23_P46170</td>
<td>MED8</td>
<td>NM_052877</td>
<td>down</td>
<td>1.11</td>
<td>0.0376</td>
</tr>
<tr>
<td>A_23_P28279</td>
<td>ACTR1B</td>
<td>NM_005735</td>
<td>up</td>
<td>1.11</td>
<td>0.0167</td>
</tr>
<tr>
<td>A_23_P24375</td>
<td>OTUB1</td>
<td>NM_017670</td>
<td>down</td>
<td>1.11</td>
<td>0.0270</td>
</tr>
<tr>
<td>A_23_P421221</td>
<td>R3HCC1</td>
<td>NM_001136108</td>
<td>down</td>
<td>1.11</td>
<td>0.0494</td>
</tr>
<tr>
<td>A_33_P3422298</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
## Supplemental table I: transcripts regulated by SOM1

<table>
<thead>
<tr>
<th>probe</th>
<th>gene symbol</th>
<th>RefSeq accession no.</th>
<th>regulation</th>
<th>fold change</th>
<th>significance (p-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A_33_P3392325</td>
<td>CDC16</td>
<td>NM_001078645</td>
<td>up</td>
<td>1,10</td>
<td>0.0180</td>
</tr>
<tr>
<td>A_33_P3357470</td>
<td>REPS2</td>
<td>NM_004726</td>
<td>down</td>
<td>1,10</td>
<td>0.0460</td>
</tr>
<tr>
<td>A_33_P3280721</td>
<td>GPR177</td>
<td>NM_001002292</td>
<td>down</td>
<td>1,10</td>
<td>0.0391</td>
</tr>
<tr>
<td>A_23_P114826</td>
<td>MRPS15</td>
<td>NM_031280</td>
<td>down</td>
<td>1,10</td>
<td>0.0496</td>
</tr>
<tr>
<td>A_19_P00803943</td>
<td></td>
<td></td>
<td>down</td>
<td>1,09</td>
<td>0.0023</td>
</tr>
<tr>
<td>A_33_P3378126</td>
<td>FBXO32</td>
<td>NM_058229</td>
<td>down</td>
<td>1,08</td>
<td>0.0412</td>
</tr>
<tr>
<td>A_23_P24755</td>
<td>STX5</td>
<td>NM_003164</td>
<td>down</td>
<td>1,08</td>
<td>0.0217</td>
</tr>
<tr>
<td>A_23_P211126</td>
<td>Dyrk1A</td>
<td>NM_130436</td>
<td>up</td>
<td>1,08</td>
<td>0.0499</td>
</tr>
<tr>
<td>A_24_P118376</td>
<td>CEACAM20</td>
<td>NM_001102598</td>
<td>down</td>
<td>1,08</td>
<td>0.0110</td>
</tr>
<tr>
<td>A_32_P155416</td>
<td>ERI3</td>
<td>NM_024066</td>
<td>down</td>
<td>1,08</td>
<td>0.0263</td>
</tr>
<tr>
<td>A_33_P3268466</td>
<td>MPDU1</td>
<td>NM_004870</td>
<td>down</td>
<td>1,07</td>
<td>0.0399</td>
</tr>
<tr>
<td>A_23_P12992</td>
<td>TRMT112</td>
<td>NM_016404</td>
<td>down</td>
<td>1,06</td>
<td>0.0225</td>
</tr>
<tr>
<td>A_33_P3384442</td>
<td></td>
<td></td>
<td>down</td>
<td>1,05</td>
<td>0.0377</td>
</tr>
<tr>
<td>A_23_P152804</td>
<td>NME1</td>
<td>NM_198175</td>
<td>down</td>
<td>1,05</td>
<td>0.0260</td>
</tr>
<tr>
<td>A_23_P79161</td>
<td>PRELID1</td>
<td>NM_013237</td>
<td>up</td>
<td>1,05</td>
<td>0.0214</td>
</tr>
<tr>
<td>A_23_P252322</td>
<td>ATP5E</td>
<td>NM_006886</td>
<td>down</td>
<td>1,04</td>
<td>0.0476</td>
</tr>
<tr>
<td>A_23_P126716</td>
<td>ATP1F1</td>
<td>NM_178191</td>
<td>down</td>
<td>1,04</td>
<td>0.0455</td>
</tr>
<tr>
<td>probe</td>
<td>gene symbol</td>
<td>RefSeq accession no.</td>
<td>regulation</td>
<td>fold change</td>
<td>significance (p-value)</td>
</tr>
<tr>
<td>------------</td>
<td>-------------</td>
<td>----------------------</td>
<td>------------</td>
<td>-------------</td>
<td>-----------------------</td>
</tr>
<tr>
<td>A_23_P110837</td>
<td>IRX4</td>
<td>NM_016358</td>
<td>up</td>
<td>13.44</td>
<td>0.0488</td>
</tr>
<tr>
<td>A_24_P183664</td>
<td>TRIL</td>
<td>NM_0114817</td>
<td>up</td>
<td>11.39</td>
<td>0.0016</td>
</tr>
<tr>
<td>A_23_P121533</td>
<td>SPON2</td>
<td>NM_012445</td>
<td>up</td>
<td>9.05</td>
<td>0.0305</td>
</tr>
<tr>
<td>A_23_P164451</td>
<td>TBX2</td>
<td>NM_005994</td>
<td>up</td>
<td>8.36</td>
<td>0.0244</td>
</tr>
<tr>
<td>A_33_P3383296</td>
<td>OR6C6</td>
<td>NM_001005493</td>
<td>up</td>
<td>6.68</td>
<td>0.0100</td>
</tr>
<tr>
<td>A_33_P3385471</td>
<td>OR4A47</td>
<td>NM_001005512</td>
<td>up</td>
<td>6.56</td>
<td>0.0485</td>
</tr>
<tr>
<td>A_23_P95060</td>
<td>EPHB3</td>
<td>NM_004443</td>
<td>up</td>
<td>5.90</td>
<td>0.0449</td>
</tr>
<tr>
<td>A_33_P3285545</td>
<td>CLDN4</td>
<td>NM_00130305</td>
<td>up</td>
<td>5.86</td>
<td>0.0421</td>
</tr>
<tr>
<td>A_23_P129695</td>
<td>VASN</td>
<td>NM_138440</td>
<td>up</td>
<td>5.18</td>
<td>0.0333</td>
</tr>
<tr>
<td>A_32_P62963</td>
<td>LOC400578</td>
<td>NR_029392</td>
<td>up</td>
<td>4.42</td>
<td>0.0081</td>
</tr>
<tr>
<td>A_23_P32165</td>
<td>LHX2</td>
<td>NM_004789</td>
<td>up</td>
<td>4.24</td>
<td>0.0437</td>
</tr>
<tr>
<td>A_23_P110957</td>
<td>FOXF2</td>
<td>NM_002371</td>
<td>up</td>
<td>4.06</td>
<td>0.0103</td>
</tr>
<tr>
<td>A_33_P3226850</td>
<td>hCG_2025798</td>
<td></td>
<td>up</td>
<td>3.98</td>
<td>0.0258</td>
</tr>
<tr>
<td>A_24_P365515</td>
<td>FOXA2</td>
<td>NM_021784</td>
<td>up</td>
<td>3.97</td>
<td>0.0189</td>
</tr>
<tr>
<td>A_32_P98072</td>
<td>TCHH</td>
<td>NM_007113</td>
<td>up</td>
<td>3.53</td>
<td>0.0186</td>
</tr>
<tr>
<td>A_19_P00316801</td>
<td></td>
<td></td>
<td>up</td>
<td>3.15</td>
<td>0.0388</td>
</tr>
<tr>
<td>A_33_P3253628</td>
<td></td>
<td></td>
<td>down</td>
<td>3.07</td>
<td>0.0457</td>
</tr>
<tr>
<td>A_23_P53370</td>
<td>RND1</td>
<td>NM_014470</td>
<td>up</td>
<td>3.01</td>
<td>0.0269</td>
</tr>
<tr>
<td>A_23_P38537</td>
<td>KRT16</td>
<td>NM_005557</td>
<td>up</td>
<td>2.85</td>
<td>0.0259</td>
</tr>
<tr>
<td>A_33_P3538279</td>
<td>PRO2852</td>
<td></td>
<td>up</td>
<td>2.84</td>
<td>0.0427</td>
</tr>
<tr>
<td>A_33_P3245665</td>
<td></td>
<td></td>
<td>down</td>
<td>2.77</td>
<td>0.0369</td>
</tr>
<tr>
<td>A_19_P00800896</td>
<td></td>
<td></td>
<td>up</td>
<td>2.68</td>
<td>0.0342</td>
</tr>
<tr>
<td>A_23_P256158</td>
<td>ADRA2C</td>
<td>NM_000683</td>
<td>up</td>
<td>2.66</td>
<td>0.0452</td>
</tr>
<tr>
<td>A_23_P40108</td>
<td>COL9A3</td>
<td>NM_001853</td>
<td>up</td>
<td>2.65</td>
<td>0.0193</td>
</tr>
<tr>
<td>A_23_P370454</td>
<td>KCNAB3</td>
<td>NM_004732</td>
<td>up</td>
<td>2.56</td>
<td>0.0116</td>
</tr>
<tr>
<td>A_33_P3382309</td>
<td>PRDM16</td>
<td>NM_022114</td>
<td>up</td>
<td>2.55</td>
<td>0.0382</td>
</tr>
<tr>
<td>A_19_P00803855</td>
<td></td>
<td></td>
<td>up</td>
<td>2.52</td>
<td>0.0314</td>
</tr>
<tr>
<td>A_33_P3359590</td>
<td>DAZAP1</td>
<td>NM_170711</td>
<td>up</td>
<td>2.51</td>
<td>0.0179</td>
</tr>
<tr>
<td>A_19_P00323018</td>
<td></td>
<td></td>
<td>up</td>
<td>2.50</td>
<td>0.0431</td>
</tr>
<tr>
<td>A_24_P93633</td>
<td></td>
<td></td>
<td>down</td>
<td>2.49</td>
<td>0.0314</td>
</tr>
<tr>
<td>A_19_P0031747</td>
<td></td>
<td></td>
<td>up</td>
<td>2.46</td>
<td>0.0147</td>
</tr>
<tr>
<td>A_33_P3326730</td>
<td>KIAA1984</td>
<td>NM_001039374</td>
<td>up</td>
<td>2.46</td>
<td>0.0377</td>
</tr>
<tr>
<td>A_32_P176594</td>
<td>KIAA1614</td>
<td>NM_0020950</td>
<td>down</td>
<td>2.45</td>
<td>0.0237</td>
</tr>
<tr>
<td>A_24_P11436</td>
<td>TTC22</td>
<td>NM_0017904</td>
<td>up</td>
<td>2.43</td>
<td>0.0306</td>
</tr>
<tr>
<td>A_19_P00322557</td>
<td></td>
<td></td>
<td>down</td>
<td>2.29</td>
<td>0.0420</td>
</tr>
<tr>
<td>A_33_P3351920</td>
<td>LOC649941</td>
<td>XR_079479</td>
<td>down</td>
<td>2.28</td>
<td>0.0262</td>
</tr>
<tr>
<td>A_23_P23502</td>
<td>OR6N1</td>
<td>NM_001005185</td>
<td>down</td>
<td>2.27</td>
<td>0.0366</td>
</tr>
<tr>
<td>A_19_P00324550</td>
<td></td>
<td></td>
<td>up</td>
<td>2.17</td>
<td>0.0445</td>
</tr>
<tr>
<td>A_33_P3311845</td>
<td>FLJ38723</td>
<td>XR_041381</td>
<td>down</td>
<td>2.16</td>
<td>0.0086</td>
</tr>
<tr>
<td>A_19_P00329233</td>
<td></td>
<td></td>
<td>down</td>
<td>2.16</td>
<td>0.0095</td>
</tr>
<tr>
<td>A_23_P315836</td>
<td>BAIAAP2</td>
<td>NM_0017451</td>
<td>up</td>
<td>2.08</td>
<td>0.0249</td>
</tr>
<tr>
<td>A_33_P3418992</td>
<td>LOC645769</td>
<td>XR_015325</td>
<td>up</td>
<td>2.08</td>
<td>0.0350</td>
</tr>
<tr>
<td>A_33_P3404281</td>
<td>LOC51145</td>
<td>XR_041655</td>
<td>up</td>
<td>2.08</td>
<td>0.0400</td>
</tr>
<tr>
<td>A_19_P00812091</td>
<td></td>
<td></td>
<td>up</td>
<td>2.05</td>
<td>0.0337</td>
</tr>
<tr>
<td>A_19_P00315668</td>
<td></td>
<td></td>
<td>up</td>
<td>2.05</td>
<td>0.0415</td>
</tr>
<tr>
<td>A_23_P325690</td>
<td>ANKRD35</td>
<td>NM_144698</td>
<td>up</td>
<td>2.02</td>
<td>0.0226</td>
</tr>
<tr>
<td>A_33_P3321462</td>
<td>OR5B21</td>
<td>NM_001005218</td>
<td>up</td>
<td>1.96</td>
<td>0.0368</td>
</tr>
<tr>
<td>A_33_P3215720</td>
<td>PPP1R2</td>
<td>NM_006241</td>
<td>up</td>
<td>1.95</td>
<td>0.0380</td>
</tr>
</tbody>
</table>
### Supplemental table II: transcripts regulated by SOM3

<table>
<thead>
<tr>
<th>probe</th>
<th>gene symbol</th>
<th>RefSeq accession no.</th>
<th>regulation</th>
<th>fold change</th>
<th>significance (p-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A_33_P3354203</td>
<td></td>
<td></td>
<td>down</td>
<td>1.95</td>
<td>0.0004</td>
</tr>
<tr>
<td>A_33_P3295814</td>
<td>LOC100128031</td>
<td>XM_001720914</td>
<td>down</td>
<td>1.95</td>
<td>0.0103</td>
</tr>
<tr>
<td>A_33_P3365305</td>
<td>CHRNA1</td>
<td>NM_001039523</td>
<td>up</td>
<td>1.94</td>
<td>0.0395</td>
</tr>
<tr>
<td>A_33_P3347040</td>
<td>LOC100290344</td>
<td>XM_002347889</td>
<td>down</td>
<td>1.92</td>
<td>0.0453</td>
</tr>
<tr>
<td>A_19_P00315750</td>
<td></td>
<td></td>
<td>down</td>
<td>1.89</td>
<td>0.0284</td>
</tr>
<tr>
<td>A_19_P00318833</td>
<td></td>
<td></td>
<td>up</td>
<td>1.85</td>
<td>0.0259</td>
</tr>
<tr>
<td>A_33_P3377380</td>
<td>LOC100130494</td>
<td>XM_001725351</td>
<td>down</td>
<td>1.83</td>
<td>0.0288</td>
</tr>
<tr>
<td>A_23_P156809</td>
<td>FAM119A</td>
<td>NM_001127395</td>
<td>up</td>
<td>1.81</td>
<td>0.0310</td>
</tr>
<tr>
<td>A_19_P00806418</td>
<td></td>
<td></td>
<td>up</td>
<td>1.77</td>
<td>0.0124</td>
</tr>
<tr>
<td>A_19_P00802314</td>
<td></td>
<td></td>
<td>down</td>
<td>1.77</td>
<td>0.0178</td>
</tr>
<tr>
<td>A_19_P00811052</td>
<td></td>
<td></td>
<td>down</td>
<td>1.74</td>
<td>0.0376</td>
</tr>
<tr>
<td>A_19_P00323091</td>
<td></td>
<td></td>
<td>down</td>
<td>1.73</td>
<td>0.0194</td>
</tr>
<tr>
<td>A_19_P00331895</td>
<td></td>
<td></td>
<td>down</td>
<td>1.73</td>
<td>0.0497</td>
</tr>
<tr>
<td>A_19_P00811821</td>
<td></td>
<td></td>
<td>down</td>
<td>1.73</td>
<td>0.0373</td>
</tr>
<tr>
<td>A_33_P3223825</td>
<td>SRRM3</td>
<td>NM_001110199</td>
<td>up</td>
<td>1.72</td>
<td>0.0477</td>
</tr>
<tr>
<td>A_33_P3351356</td>
<td></td>
<td></td>
<td>up</td>
<td>1.71</td>
<td>0.0499</td>
</tr>
<tr>
<td>A_33_P3214105</td>
<td>ATF3</td>
<td>NM_001674</td>
<td>up</td>
<td>1.71</td>
<td>0.0388</td>
</tr>
<tr>
<td>A_19_P00810374</td>
<td></td>
<td></td>
<td>down</td>
<td>1.69</td>
<td>0.0385</td>
</tr>
<tr>
<td>A_23_P26511</td>
<td>GDPD3</td>
<td>NM_0024307</td>
<td>up</td>
<td>1.67</td>
<td>0.0355</td>
</tr>
<tr>
<td>A_23_P500353</td>
<td>KCNN2</td>
<td>NM_0021614</td>
<td>up</td>
<td>1.65</td>
<td>0.0363</td>
</tr>
<tr>
<td>A_23_P159741</td>
<td>BCOR</td>
<td>NM_017745</td>
<td>up</td>
<td>1.64</td>
<td>0.0422</td>
</tr>
<tr>
<td>A_23_P214144</td>
<td>COL10A1</td>
<td>NM_000493</td>
<td>down</td>
<td>1.63</td>
<td>0.0123</td>
</tr>
<tr>
<td>A_33_P3393135</td>
<td></td>
<td></td>
<td>down</td>
<td>1.63</td>
<td>0.0241</td>
</tr>
<tr>
<td>A_19_P00809217</td>
<td></td>
<td></td>
<td>up</td>
<td>1.61</td>
<td>0.0325</td>
</tr>
<tr>
<td>A_19_P00807595</td>
<td></td>
<td></td>
<td>down</td>
<td>1.61</td>
<td>0.0437</td>
</tr>
<tr>
<td>A_33_P3389363</td>
<td></td>
<td></td>
<td>down</td>
<td>1.61</td>
<td>0.0470</td>
</tr>
<tr>
<td>A_33_P3228709</td>
<td>KRTAP5-7</td>
<td>NM_001012503</td>
<td>up</td>
<td>1.60</td>
<td>0.0268</td>
</tr>
<tr>
<td>A_19_P00809146</td>
<td></td>
<td></td>
<td>down</td>
<td>1.60</td>
<td>0.0354</td>
</tr>
<tr>
<td>A_24_P940310</td>
<td>URB1</td>
<td>NM_014825</td>
<td>up</td>
<td>1.59</td>
<td>0.0105</td>
</tr>
<tr>
<td>A_33_P3305855</td>
<td>LOC645967</td>
<td>XR_040575</td>
<td>up</td>
<td>1.59</td>
<td>0.0269</td>
</tr>
<tr>
<td>A_33_P3273854</td>
<td>NAALADL2</td>
<td>NM_207015</td>
<td>down</td>
<td>1.58</td>
<td>0.0359</td>
</tr>
<tr>
<td>A_33_P3343828</td>
<td>FLJ42627</td>
<td></td>
<td>up</td>
<td>1.57</td>
<td>0.0470</td>
</tr>
<tr>
<td>A_19_P00813254</td>
<td></td>
<td></td>
<td>down</td>
<td>1.57</td>
<td>0.0147</td>
</tr>
<tr>
<td>A_19_P00806500</td>
<td></td>
<td></td>
<td>down</td>
<td>1.57</td>
<td>0.0050</td>
</tr>
<tr>
<td>A_23_P10542</td>
<td>HTRA3</td>
<td>NM_053044</td>
<td>up</td>
<td>1.56</td>
<td>0.0238</td>
</tr>
<tr>
<td>A_19_P00318098</td>
<td></td>
<td></td>
<td>down</td>
<td>1.56</td>
<td>0.0379</td>
</tr>
<tr>
<td>A_33_P3287258</td>
<td></td>
<td></td>
<td>up</td>
<td>1.54</td>
<td>0.0189</td>
</tr>
<tr>
<td>A_24_P314597</td>
<td></td>
<td></td>
<td>up</td>
<td>1.54</td>
<td>0.0268</td>
</tr>
<tr>
<td>A_19_P00315808</td>
<td></td>
<td></td>
<td>down</td>
<td>1.53</td>
<td>0.0488</td>
</tr>
<tr>
<td>A_23_P336929</td>
<td>MED29</td>
<td>NM_017592</td>
<td>up</td>
<td>1.51</td>
<td>0.0312</td>
</tr>
<tr>
<td>A_33_P3394972</td>
<td>OSBPL5</td>
<td>NM_020896</td>
<td>up</td>
<td>1.50</td>
<td>0.0142</td>
</tr>
<tr>
<td>A_24_P17302</td>
<td>UBE2J2</td>
<td>NM_194458</td>
<td>up</td>
<td>1.50</td>
<td>0.0475</td>
</tr>
<tr>
<td>A_24_P301454</td>
<td>NBR2</td>
<td>NR_003108</td>
<td>down</td>
<td>1.49</td>
<td>0.0410</td>
</tr>
<tr>
<td>A_24_P562369</td>
<td></td>
<td></td>
<td>down</td>
<td>1.49</td>
<td>0.0225</td>
</tr>
<tr>
<td>A_32_P211248</td>
<td>LOC100131138</td>
<td>XM_001717925</td>
<td>down</td>
<td>1.49</td>
<td>0.0236</td>
</tr>
<tr>
<td>A_19_P00322375</td>
<td></td>
<td></td>
<td>down</td>
<td>1.48</td>
<td>0.0368</td>
</tr>
<tr>
<td>A_33_P3328726</td>
<td>CCDC33</td>
<td>NM_182791</td>
<td>down</td>
<td>1.48</td>
<td>0.0452</td>
</tr>
<tr>
<td>A_19_P00328934</td>
<td></td>
<td></td>
<td>down</td>
<td>1.48</td>
<td>0.0098</td>
</tr>
<tr>
<td>A_33_P3356092</td>
<td>BTBD9</td>
<td>NM_052893</td>
<td>down</td>
<td>1.47</td>
<td>0.0400</td>
</tr>
<tr>
<td>A_19_P00810799</td>
<td></td>
<td></td>
<td>down</td>
<td>1.46</td>
<td>0.0075</td>
</tr>
</tbody>
</table>
### Supplemental table II: transcripts regulated by SOM3

<table>
<thead>
<tr>
<th>probe</th>
<th>gene symbol</th>
<th>RefSeq accession no.</th>
<th>regulation</th>
<th>fold change</th>
<th>significance (p-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A_33_P3328284</td>
<td>CBL</td>
<td>NM_005188</td>
<td>up</td>
<td>1.46</td>
<td>0.0215</td>
</tr>
<tr>
<td>A_33_P3256033</td>
<td>NCAPH</td>
<td>NM_015341</td>
<td>down</td>
<td>1.45</td>
<td>0.0229</td>
</tr>
<tr>
<td>A_33_P3230259</td>
<td>CBL</td>
<td>NM_005188</td>
<td>up</td>
<td>1.45</td>
<td>0.023</td>
</tr>
<tr>
<td>A_19_P00811771</td>
<td>CBL</td>
<td>NM_005188</td>
<td>up</td>
<td>1.44</td>
<td>0.0229</td>
</tr>
<tr>
<td>A_33_P3283201</td>
<td>CBL</td>
<td>NM_005188</td>
<td>up</td>
<td>1.44</td>
<td>0.0171</td>
</tr>
<tr>
<td>A_33_P3371769</td>
<td>CBL</td>
<td>NM_005188</td>
<td>up</td>
<td>1.44</td>
<td>0.0471</td>
</tr>
<tr>
<td>A_23_P388681</td>
<td>ELAVL1</td>
<td>NM_001419</td>
<td>up</td>
<td>1.44</td>
<td>0.0136</td>
</tr>
<tr>
<td>A_19_P00802881</td>
<td>CBL</td>
<td>NM_005188</td>
<td>up</td>
<td>1.44</td>
<td>0.0319</td>
</tr>
<tr>
<td>A_33_P3256267</td>
<td>CBL</td>
<td>NM_005188</td>
<td>up</td>
<td>1.43</td>
<td>0.0418</td>
</tr>
<tr>
<td>A_33_P3343260</td>
<td>LOC100132433</td>
<td>XR_038594</td>
<td>down</td>
<td>1.43</td>
<td>0.0338</td>
</tr>
<tr>
<td>A_32_P95462</td>
<td>STXBP5L</td>
<td>NM_014980</td>
<td>down</td>
<td>1.42</td>
<td>0.0028</td>
</tr>
<tr>
<td>A_33_P3461416</td>
<td>GP6</td>
<td>NM_001083899</td>
<td>up</td>
<td>1.42</td>
<td>0.0436</td>
</tr>
<tr>
<td>A_24_P497186</td>
<td>IRF2BP2</td>
<td>NM_182972</td>
<td>up</td>
<td>1.42</td>
<td>0.0012</td>
</tr>
<tr>
<td>A_19_P00810613</td>
<td>CBL</td>
<td>NM_005188</td>
<td>up</td>
<td>1.42</td>
<td>0.0427</td>
</tr>
<tr>
<td>A_19_P00318165</td>
<td>CBL</td>
<td>NM_005188</td>
<td>up</td>
<td>1.41</td>
<td>0.0446</td>
</tr>
<tr>
<td>A_23_P33407</td>
<td>SEC16A</td>
<td>NM_006467</td>
<td>up</td>
<td>1.41</td>
<td>0.0242</td>
</tr>
<tr>
<td>A_32_P1533</td>
<td>LOC202181</td>
<td>NR_026921</td>
<td>down</td>
<td>1.40</td>
<td>0.0463</td>
</tr>
<tr>
<td>A_33_P3325439</td>
<td>USP16</td>
<td>NM_001001992</td>
<td>down</td>
<td>1.39</td>
<td>0.0437</td>
</tr>
<tr>
<td>A_19_P00327579</td>
<td>CBL</td>
<td>NM_005188</td>
<td>up</td>
<td>1.39</td>
<td>0.0199</td>
</tr>
<tr>
<td>A_23_P360209</td>
<td>FZD1</td>
<td>NM_003505</td>
<td>up</td>
<td>1.38</td>
<td>0.0402</td>
</tr>
<tr>
<td>A_24_P38276</td>
<td>CBL</td>
<td>NM_005188</td>
<td>up</td>
<td>1.38</td>
<td>0.0415</td>
</tr>
<tr>
<td>A_19_P00800805</td>
<td>CBL</td>
<td>NM_005188</td>
<td>up</td>
<td>1.37</td>
<td>0.0232</td>
</tr>
<tr>
<td>A_33_P3416231</td>
<td>HOXA9</td>
<td>NM_152739</td>
<td>down</td>
<td>1.37</td>
<td>0.0419</td>
</tr>
<tr>
<td>A_33_P3325871</td>
<td>hCG_1993592</td>
<td>NR_027436</td>
<td>down</td>
<td>1.37</td>
<td>0.0199</td>
</tr>
<tr>
<td>A_23_P251303</td>
<td>SEC16A</td>
<td>NM_014866</td>
<td>up</td>
<td>1.37</td>
<td>0.0241</td>
</tr>
<tr>
<td>A_23_P66241</td>
<td>MT1M</td>
<td>NM_176870</td>
<td>down</td>
<td>1.37</td>
<td>0.0209</td>
</tr>
<tr>
<td>A_33_P3389376</td>
<td>MDM4</td>
<td>NM_002393</td>
<td>up</td>
<td>1.37</td>
<td>0.0318</td>
</tr>
<tr>
<td>A_19_P00800161</td>
<td>CBL</td>
<td>NM_005188</td>
<td>up</td>
<td>1.37</td>
<td>0.0449</td>
</tr>
<tr>
<td>A_33_P3310371</td>
<td>LOC283788</td>
<td>NR_027436</td>
<td>down</td>
<td>1.37</td>
<td>0.0475</td>
</tr>
<tr>
<td>A_23_P316042</td>
<td>ERN1</td>
<td>NM_001433</td>
<td>up</td>
<td>1.36</td>
<td>0.0436</td>
</tr>
<tr>
<td>A_33_P3236563</td>
<td>ALDH3B1</td>
<td>NM_001161473</td>
<td>down</td>
<td>1.35</td>
<td>0.0064</td>
</tr>
<tr>
<td>A_19_P00329691</td>
<td>CBL</td>
<td>NM_005188</td>
<td>up</td>
<td>1.35</td>
<td>0.0386</td>
</tr>
<tr>
<td>A_24_P69538</td>
<td>TLR4</td>
<td>NM_138554</td>
<td>down</td>
<td>1.34</td>
<td>0.0409</td>
</tr>
<tr>
<td>A_19_P00804546</td>
<td>CBL</td>
<td>NM_005188</td>
<td>up</td>
<td>1.33</td>
<td>0.0330</td>
</tr>
<tr>
<td>A_33_P3317473</td>
<td>CBL</td>
<td>NM_005188</td>
<td>up</td>
<td>1.33</td>
<td>0.0106</td>
</tr>
<tr>
<td>A_19_P00321685</td>
<td>CBL</td>
<td>NM_005188</td>
<td>up</td>
<td>1.33</td>
<td>0.0433</td>
</tr>
<tr>
<td>A_33_P3313652</td>
<td>CBL</td>
<td>NM_005188</td>
<td>up</td>
<td>1.33</td>
<td>0.0142</td>
</tr>
<tr>
<td>A_24_P119545</td>
<td>CBL</td>
<td>NM_005188</td>
<td>up</td>
<td>1.32</td>
<td>0.0274</td>
</tr>
<tr>
<td>A_33_P3363100</td>
<td>LOC100294232</td>
<td>XM_002343989</td>
<td>down</td>
<td>1.32</td>
<td>0.0194</td>
</tr>
<tr>
<td>A_19_P00320721</td>
<td>CBL</td>
<td>NM_005188</td>
<td>up</td>
<td>1.32</td>
<td>0.0066</td>
</tr>
<tr>
<td>A_23_P417282</td>
<td>IGF1R</td>
<td>NM_000875</td>
<td>up</td>
<td>1.32</td>
<td>0.0380</td>
</tr>
<tr>
<td>A_33_P3288774</td>
<td>RC3H1</td>
<td>NM_172071</td>
<td>up</td>
<td>1.32</td>
<td>0.0202</td>
</tr>
<tr>
<td>A_33_P3289541</td>
<td>MLLT1</td>
<td>NM_005934</td>
<td>up</td>
<td>1.32</td>
<td>0.0462</td>
</tr>
<tr>
<td>A_33_P3214027</td>
<td>CYTSA</td>
<td>NM_015330</td>
<td>down</td>
<td>1.32</td>
<td>0.0275</td>
</tr>
<tr>
<td>A_19_P00324577</td>
<td>CBL</td>
<td>NM_005188</td>
<td>up</td>
<td>1.31</td>
<td>0.0273</td>
</tr>
<tr>
<td>A_23_P112159</td>
<td>EIF2C2</td>
<td>NM_012154</td>
<td>up</td>
<td>1.31</td>
<td>0.0330</td>
</tr>
<tr>
<td>A_23_P69908</td>
<td>GLRX</td>
<td>NM_002064</td>
<td>down</td>
<td>1.31</td>
<td>0.0301</td>
</tr>
<tr>
<td>A_33_P3418516</td>
<td>E2F3</td>
<td>NM_001949</td>
<td>down</td>
<td>1.31</td>
<td>0.0234</td>
</tr>
</tbody>
</table>
Supplemental table II: transcripts regulated by SOM3

<table>
<thead>
<tr>
<th>probe</th>
<th>gene symbol</th>
<th>RefSeq accession no.</th>
<th>regulation</th>
<th>fold change</th>
<th>significance (p-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A_23_P86182</td>
<td>MRPS21</td>
<td>NM_031901</td>
<td>down</td>
<td>1.30</td>
<td>0.0240</td>
</tr>
<tr>
<td>A_33_P320827</td>
<td>PEA15</td>
<td>NM_003768</td>
<td>up</td>
<td>1.30</td>
<td>0.0312</td>
</tr>
<tr>
<td>A_19_P00320970</td>
<td></td>
<td></td>
<td>down</td>
<td>1.30</td>
<td>0.0226</td>
</tr>
<tr>
<td>A_33_P3802558</td>
<td>LOC441245</td>
<td></td>
<td>up</td>
<td>1.30</td>
<td>0.0437</td>
</tr>
<tr>
<td>A_23_P20894</td>
<td>EHMT1</td>
<td>NM_024757</td>
<td>up</td>
<td>1.30</td>
<td>0.0212</td>
</tr>
<tr>
<td>A_19_P00321664</td>
<td></td>
<td></td>
<td>down</td>
<td>1.30</td>
<td>0.0106</td>
</tr>
<tr>
<td>A_33_P3240787</td>
<td>LOC100131910</td>
<td></td>
<td>down</td>
<td>1.30</td>
<td>0.0383</td>
</tr>
<tr>
<td>A_24_P602507</td>
<td>AGPHD1</td>
<td>NM_001031619</td>
<td>down</td>
<td>1.30</td>
<td>0.0360</td>
</tr>
<tr>
<td>A_33_P3811287</td>
<td>HUWE1</td>
<td>NM_031407</td>
<td>up</td>
<td>1.30</td>
<td>0.0178</td>
</tr>
<tr>
<td>A_33_P3296991</td>
<td>FLJ42393</td>
<td>NR_024413</td>
<td>down</td>
<td>1.29</td>
<td>0.0216</td>
</tr>
<tr>
<td>A_33_P3313640</td>
<td></td>
<td></td>
<td>down</td>
<td>1.29</td>
<td>0.0102</td>
</tr>
<tr>
<td>A_33_P3377550</td>
<td>KLC3</td>
<td>NM_177417</td>
<td>up</td>
<td>1.29</td>
<td>0.0484</td>
</tr>
<tr>
<td>A_33_P3211633</td>
<td>WDR3</td>
<td>NM_006784</td>
<td>down</td>
<td>1.29</td>
<td>0.0246</td>
</tr>
<tr>
<td>A_24_P230938</td>
<td></td>
<td></td>
<td>down</td>
<td>1.28</td>
<td>0.0464</td>
</tr>
<tr>
<td>A_33_P3296497</td>
<td></td>
<td></td>
<td>up</td>
<td>1.28</td>
<td>0.0228</td>
</tr>
<tr>
<td>A_33_P3367073</td>
<td>LOC100128142</td>
<td>XM_001715360</td>
<td>down</td>
<td>1.28</td>
<td>0.0423</td>
</tr>
<tr>
<td>A_23_P351320</td>
<td>CDC40</td>
<td>NM_015891</td>
<td>down</td>
<td>1.28</td>
<td>0.0468</td>
</tr>
<tr>
<td>A_33_P3290714</td>
<td>HS6ST2</td>
<td>NM_001077188</td>
<td>down</td>
<td>1.28</td>
<td>0.0315</td>
</tr>
<tr>
<td>A_19_P00331719</td>
<td></td>
<td></td>
<td>down</td>
<td>1.28</td>
<td>0.0271</td>
</tr>
<tr>
<td>A_23_P143006</td>
<td>PRLH</td>
<td>NM_015893</td>
<td>down</td>
<td>1.27</td>
<td>0.0467</td>
</tr>
<tr>
<td>A_23_P424513</td>
<td>RANBP9</td>
<td>NM_005493</td>
<td>up</td>
<td>1.27</td>
<td>0.0425</td>
</tr>
<tr>
<td>A_23_P161352</td>
<td>PTPLA</td>
<td>NM_014241</td>
<td>down</td>
<td>1.27</td>
<td>0.0277</td>
</tr>
<tr>
<td>A_33_P3247392</td>
<td>TPTE</td>
<td>NM_199261</td>
<td>up</td>
<td>1.27</td>
<td>0.0297</td>
</tr>
<tr>
<td>A_33_P3239195</td>
<td></td>
<td></td>
<td>down</td>
<td>1.27</td>
<td>0.0183</td>
</tr>
<tr>
<td>A_19_P00811540</td>
<td></td>
<td></td>
<td>down</td>
<td>1.27</td>
<td>0.0358</td>
</tr>
<tr>
<td>A_19_P00319426</td>
<td></td>
<td></td>
<td>down</td>
<td>1.27</td>
<td>0.0496</td>
</tr>
<tr>
<td>A_19_P00811495</td>
<td></td>
<td></td>
<td>down</td>
<td>1.27</td>
<td>0.0026</td>
</tr>
<tr>
<td>A_33_P361000</td>
<td>ZBTB10</td>
<td>NM_001105539</td>
<td>down</td>
<td>1.27</td>
<td>0.0220</td>
</tr>
<tr>
<td>A_19_P00801843</td>
<td></td>
<td></td>
<td>down</td>
<td>1.26</td>
<td>0.0443</td>
</tr>
<tr>
<td>A_33_P3410284</td>
<td></td>
<td></td>
<td>down</td>
<td>1.25</td>
<td>0.0017</td>
</tr>
<tr>
<td>A_33_P3281196</td>
<td>MRPS36</td>
<td>NM_033281</td>
<td>down</td>
<td>1.25</td>
<td>0.0379</td>
</tr>
<tr>
<td>A_33_P3259821</td>
<td>DOCK9</td>
<td>NM_001130050</td>
<td>down</td>
<td>1.25</td>
<td>0.0325</td>
</tr>
<tr>
<td>A_33_P3270639</td>
<td></td>
<td></td>
<td>down</td>
<td>1.25</td>
<td>0.0317</td>
</tr>
<tr>
<td>A_33_P3339066</td>
<td>RNPC3</td>
<td>NM_017619</td>
<td>down</td>
<td>1.24</td>
<td>0.0367</td>
</tr>
<tr>
<td>A_19_P00326177</td>
<td></td>
<td></td>
<td>down</td>
<td>1.24</td>
<td>0.0488</td>
</tr>
<tr>
<td>A_33_P3346473</td>
<td>PHF21A</td>
<td>NM_001101802</td>
<td>up</td>
<td>1.24</td>
<td>0.0400</td>
</tr>
<tr>
<td>A_33_P3311907</td>
<td>TBC1D7</td>
<td>NM_016495</td>
<td>down</td>
<td>1.24</td>
<td>0.0172</td>
</tr>
<tr>
<td>A_24_P235049</td>
<td>MTHFD1L</td>
<td>NM_015440</td>
<td>up</td>
<td>1.24</td>
<td>0.0240</td>
</tr>
<tr>
<td>A_33_P3414362</td>
<td>USP32</td>
<td>NM_032582</td>
<td>down</td>
<td>1.24</td>
<td>0.0488</td>
</tr>
<tr>
<td>A_33_P3372368</td>
<td>LOC100129362</td>
<td>XM_001721298</td>
<td>down</td>
<td>1.24</td>
<td>0.0488</td>
</tr>
<tr>
<td>A_23_P150379</td>
<td>MPZL2</td>
<td>NM_144765</td>
<td>down</td>
<td>1.24</td>
<td>0.0278</td>
</tr>
<tr>
<td>A_24_P253251</td>
<td>SLC7A1</td>
<td>NM_003045</td>
<td>up</td>
<td>1.23</td>
<td>0.0084</td>
</tr>
<tr>
<td>A_19_P00805076</td>
<td></td>
<td></td>
<td>down</td>
<td>1.23</td>
<td>0.0481</td>
</tr>
<tr>
<td>A_23_P110811</td>
<td>COX7C</td>
<td>NM_001867</td>
<td>down</td>
<td>1.22</td>
<td>0.0064</td>
</tr>
<tr>
<td>A_19_P00327603</td>
<td></td>
<td></td>
<td>down</td>
<td>1.22</td>
<td>0.0297</td>
</tr>
<tr>
<td>A_33_P3404775</td>
<td>PABPC4</td>
<td>NM_001135653</td>
<td>up</td>
<td>1.22</td>
<td>0.0460</td>
</tr>
<tr>
<td>A_33_P3380387</td>
<td>AMZ1</td>
<td>NM_133463</td>
<td>up</td>
<td>1.22</td>
<td>0.0404</td>
</tr>
<tr>
<td>A_23_P359854</td>
<td>BEND3</td>
<td>NM_001080450</td>
<td>up</td>
<td>1.22</td>
<td>0.0409</td>
</tr>
<tr>
<td>A_24_P944444</td>
<td>DIO1</td>
<td>NM_080797</td>
<td>up</td>
<td>1.21</td>
<td>0.0471</td>
</tr>
<tr>
<td>A_33_P3331426</td>
<td>LOC100133086</td>
<td>XM_001723511</td>
<td>down</td>
<td>1.21</td>
<td>0.0294</td>
</tr>
<tr>
<td>probe</td>
<td>gene symbol</td>
<td>RefSeq accession no.</td>
<td>regulation</td>
<td>fold change</td>
<td>significance (p-value)</td>
</tr>
<tr>
<td>------------</td>
<td>-------------</td>
<td>----------------------</td>
<td>------------</td>
<td>-------------</td>
<td>------------------------</td>
</tr>
<tr>
<td>A_33_P3286616</td>
<td>IRAK1</td>
<td>NM_001569</td>
<td>up</td>
<td>1.21</td>
<td>0.0340</td>
</tr>
<tr>
<td>A_33_P3216337</td>
<td>FTSJ1</td>
<td>NM_177439</td>
<td>up</td>
<td>1.21</td>
<td>0.0461</td>
</tr>
<tr>
<td>A_33_P3381410</td>
<td>ORC2L</td>
<td>NM_006190</td>
<td>down</td>
<td>1.21</td>
<td>0.0252</td>
</tr>
<tr>
<td>A_24_P322908</td>
<td>USP27X</td>
<td>NM_001145073</td>
<td>down</td>
<td>1.21</td>
<td>0.0474</td>
</tr>
<tr>
<td>A_23_P88865</td>
<td>CMTM3</td>
<td>NM_144601</td>
<td>up</td>
<td>1.21</td>
<td>0.0279</td>
</tr>
<tr>
<td>A_33_P3357573</td>
<td>MAML3</td>
<td>NM_018717</td>
<td>down</td>
<td>1.20</td>
<td>0.0452</td>
</tr>
<tr>
<td>A_33_P3246505</td>
<td></td>
<td></td>
<td>down</td>
<td>1.20</td>
<td>0.0339</td>
</tr>
<tr>
<td>A_23_P210869</td>
<td>COX4I2</td>
<td>NM_032609</td>
<td>down</td>
<td>1.20</td>
<td>0.0482</td>
</tr>
<tr>
<td>A_33_P3235987</td>
<td>PIN4</td>
<td>NM_006223</td>
<td>down</td>
<td>1.20</td>
<td>0.0263</td>
</tr>
<tr>
<td>A_19_P00315892</td>
<td></td>
<td></td>
<td>down</td>
<td>1.20</td>
<td>0.0350</td>
</tr>
<tr>
<td>A_23_P304543</td>
<td>NFX1</td>
<td>NM_147133</td>
<td>down</td>
<td>1.20</td>
<td>0.0405</td>
</tr>
<tr>
<td>A_23_P3775</td>
<td>OGFOD1</td>
<td>NM_018233</td>
<td>down</td>
<td>1.20</td>
<td>0.0139</td>
</tr>
<tr>
<td>A_33_P3359543</td>
<td>ZNF561</td>
<td>NM_152289</td>
<td>down</td>
<td>1.20</td>
<td>0.0486</td>
</tr>
<tr>
<td>A_23_P15272</td>
<td>ABCC6</td>
<td>NM_01079528</td>
<td>down</td>
<td>1.20</td>
<td>0.0103</td>
</tr>
<tr>
<td>A_19_P00316758</td>
<td></td>
<td></td>
<td>down</td>
<td>1.20</td>
<td>0.0031</td>
</tr>
<tr>
<td>A_19_P00327731</td>
<td></td>
<td></td>
<td>down</td>
<td>1.20</td>
<td>0.0050</td>
</tr>
<tr>
<td>A_23_P362824</td>
<td>CSTF1</td>
<td>NM_001324</td>
<td>down</td>
<td>1.20</td>
<td>0.0429</td>
</tr>
<tr>
<td>A_23_P81880</td>
<td>CTDSP2</td>
<td>NM_005730</td>
<td>up</td>
<td>1.20</td>
<td>0.0255</td>
</tr>
<tr>
<td>A_33_P3320301</td>
<td>LOC388789</td>
<td>NR_015432</td>
<td>down</td>
<td>1.19</td>
<td>0.0225</td>
</tr>
<tr>
<td>A_19_P00809343</td>
<td></td>
<td></td>
<td>down</td>
<td>1.19</td>
<td>0.0350</td>
</tr>
<tr>
<td>A_33_P3411384</td>
<td></td>
<td></td>
<td>up</td>
<td>1.19</td>
<td>0.0453</td>
</tr>
<tr>
<td>A_19_P00321143</td>
<td></td>
<td></td>
<td>down</td>
<td>1.18</td>
<td>0.0227</td>
</tr>
<tr>
<td>A_33_P3257358</td>
<td>AGPHD1</td>
<td>NM_001083612</td>
<td>down</td>
<td>1.18</td>
<td>0.0324</td>
</tr>
<tr>
<td>A_23_P98532</td>
<td>ZDHHC5</td>
<td>NM_015457</td>
<td>up</td>
<td>1.18</td>
<td>0.0115</td>
</tr>
<tr>
<td>A_24_P305467</td>
<td>GATAD2A</td>
<td>NM_017660</td>
<td>up</td>
<td>1.18</td>
<td>0.0368</td>
</tr>
<tr>
<td>A_33_P3322504</td>
<td></td>
<td></td>
<td>down</td>
<td>1.17</td>
<td>0.0115</td>
</tr>
<tr>
<td>A_33_P3264780</td>
<td>CDK8</td>
<td>NM_001260</td>
<td>up</td>
<td>1.17</td>
<td>0.0134</td>
</tr>
<tr>
<td>A_23_P120744</td>
<td>MCM3AP</td>
<td>NM_003906</td>
<td>up</td>
<td>1.17</td>
<td>0.0266</td>
</tr>
<tr>
<td>A_24_P98086</td>
<td>GNA12</td>
<td>NM_007353</td>
<td>up</td>
<td>1.17</td>
<td>0.0215</td>
</tr>
<tr>
<td>A_23_P127175</td>
<td>SAR1A</td>
<td>NM_0020150</td>
<td>down</td>
<td>1.17</td>
<td>0.0192</td>
</tr>
<tr>
<td>A_23_P64083</td>
<td>SF3B2</td>
<td>NM_006842</td>
<td>up</td>
<td>1.16</td>
<td>0.0334</td>
</tr>
<tr>
<td>A_33_P3244181</td>
<td>HBP1</td>
<td>NM_001537</td>
<td>down</td>
<td>1.16</td>
<td>0.0484</td>
</tr>
<tr>
<td>A_23_P210763</td>
<td>JAG1</td>
<td>NM_000214</td>
<td>up</td>
<td>1.16</td>
<td>0.0416</td>
</tr>
<tr>
<td>A_19_P00806152</td>
<td></td>
<td></td>
<td>up</td>
<td>1.16</td>
<td>0.0413</td>
</tr>
<tr>
<td>A_24_P229536</td>
<td>C21orf34</td>
<td>NR_027791</td>
<td>down</td>
<td>1.15</td>
<td>0.0255</td>
</tr>
<tr>
<td>A_33_P3351934</td>
<td>MSTO2P</td>
<td>NR_024117</td>
<td>up</td>
<td>1.15</td>
<td>0.0251</td>
</tr>
<tr>
<td>A_32_P99902</td>
<td>C15orf40</td>
<td>NM_144597</td>
<td>down</td>
<td>1.15</td>
<td>0.0057</td>
</tr>
<tr>
<td>A_33_P3286318</td>
<td>LOCC440043</td>
<td>X0_015812</td>
<td>up</td>
<td>1.15</td>
<td>0.0474</td>
</tr>
<tr>
<td>A_24_P228667</td>
<td>MRPL40</td>
<td>NM_003776</td>
<td>down</td>
<td>1.15</td>
<td>0.0432</td>
</tr>
<tr>
<td>A_32_P82623</td>
<td>AGBL3</td>
<td>NM_178563</td>
<td>down</td>
<td>1.14</td>
<td>0.0276</td>
</tr>
<tr>
<td>A_23_P159833</td>
<td>NDUFA1</td>
<td>NM_004541</td>
<td>down</td>
<td>1.14</td>
<td>0.0235</td>
</tr>
<tr>
<td>A_33_P3347387</td>
<td></td>
<td></td>
<td>down</td>
<td>1.14</td>
<td>0.0341</td>
</tr>
<tr>
<td>A_33_P3209706</td>
<td>ARGFX</td>
<td>NM_001012659</td>
<td>down</td>
<td>1.13</td>
<td>0.0341</td>
</tr>
<tr>
<td>A_33_P3287922</td>
<td>KIAA0406</td>
<td>NM_014657</td>
<td>down</td>
<td>1.13</td>
<td>0.0500</td>
</tr>
<tr>
<td>A_23_P71148</td>
<td>BLVRA</td>
<td>NM_000712</td>
<td>down</td>
<td>1.12</td>
<td>0.0233</td>
</tr>
<tr>
<td>A_24_P41042</td>
<td>C12orf52</td>
<td>NM_032848</td>
<td>up</td>
<td>1.12</td>
<td>0.0430</td>
</tr>
<tr>
<td>A_23_P68866</td>
<td>UCRC</td>
<td>NM_001003684</td>
<td>down</td>
<td>1.12</td>
<td>0.0151</td>
</tr>
<tr>
<td>A_23_P102925</td>
<td>PWP2</td>
<td>NM_005049</td>
<td>up</td>
<td>1.11</td>
<td>0.0374</td>
</tr>
<tr>
<td>A_24_P101629</td>
<td>FAM127B</td>
<td>NM_001078172</td>
<td>down</td>
<td>1.11</td>
<td>0.0324</td>
</tr>
<tr>
<td>A_23_P62907</td>
<td>ATP6</td>
<td>NM_007348</td>
<td>down</td>
<td>1.11</td>
<td>0.0321</td>
</tr>
</tbody>
</table>
Supplemental table II: transcripts regulated by SOM3

<table>
<thead>
<tr>
<th>probe</th>
<th>gene symbol</th>
<th>RefSeq accession no.</th>
<th>regulation</th>
<th>fold change</th>
<th>significance (p-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A_33_P3239587</td>
<td>MXRA7</td>
<td>NM_001008529</td>
<td>up</td>
<td>1.11</td>
<td>0.0162</td>
</tr>
<tr>
<td>A_32_P224149</td>
<td>FKB15</td>
<td>NM_015258</td>
<td>down</td>
<td>1.11</td>
<td>0.0101</td>
</tr>
<tr>
<td>A_23_P45108</td>
<td>QRICH1</td>
<td>NM_017730</td>
<td>up</td>
<td>1.11</td>
<td>0.0225</td>
</tr>
<tr>
<td>A_33_P3376828</td>
<td>CMTM7</td>
<td>NM_138410</td>
<td>down</td>
<td>1.11</td>
<td>0.0193</td>
</tr>
<tr>
<td>A_23_P34983</td>
<td>JTB</td>
<td>NM_006694</td>
<td>up</td>
<td>1.10</td>
<td>0.0159</td>
</tr>
<tr>
<td>A_33_P3269408</td>
<td>FOXI2</td>
<td>NM_207426</td>
<td>up</td>
<td>1.10</td>
<td>0.0012</td>
</tr>
<tr>
<td>A_23_P80362</td>
<td>NHP2L1</td>
<td>NM_005008</td>
<td>down</td>
<td>1.09</td>
<td>0.0286</td>
</tr>
<tr>
<td>A_23_P17287</td>
<td>IAH1</td>
<td>NM_001039613</td>
<td>down</td>
<td>1.09</td>
<td>0.0345</td>
</tr>
<tr>
<td>A_23_P43566</td>
<td>NDUFA8</td>
<td>NM_014222</td>
<td>down</td>
<td>1.09</td>
<td>0.0448</td>
</tr>
<tr>
<td>A_32_P75299</td>
<td>TOMM5</td>
<td>NM_001134484</td>
<td>down</td>
<td>1.06</td>
<td>0.0302</td>
</tr>
<tr>
<td>A_23_P152107</td>
<td>UBE2I</td>
<td>NM_194259</td>
<td>up</td>
<td>1.05</td>
<td>0.0482</td>
</tr>
</tbody>
</table>