ERN ITHACA

Webinar 2025

ARID1B Associated Coffin-Siris Syndrome

Diagnosis and Management

TUESDAY, MARCH 18, 2025 FROM 5PM TO 6.30 PM CEST

Chaired by : André Reis

European Commission

EUROPEAN REFERENCE NETWORKS

Helping patients with rare or low-prevalence complex diseases

ITHACA Project Manager Anne Hugon

2025

European Reference Networks ITHACA

Welcome – Technical points

- **We are please to be numerous >** 140 registrations
- **Webinar being recorded**

- **Thank you for**
  - Turn off your microphone and disconnect your camera
  - Raise your hand at the time of the questions and discussions
  - We will answer the questions sent in the registration form
  - A satisfaction survey will be sent to you :

- **Webinars # will be available on ITHACA's Website**
- **https://ern-ithaca.eu/webinars/**

- **Anne Hugon Project Manager ERN ITHACA - anne.hugon@aphp.fr**

Welcome and Introduction

- **ARID1B Associated Coffin-Siris Syndrome – Diagnosis and Management**

- Coffin-Siris syndrome (CSS) is a relatively frequent syndrome with developmental or cognitive delay, classically characterized by small or missing nails and distinctive facial appearance, among several other signs.
- Since the discovery of mutations in *ARID1B*, a component of the BAF chromatin remodelling complex, as the main cause of the syndrome, our knowledge on diagnosis, natural history and management have greatly improved.
- In this webinar we aim to explore these recent developments.

Agenda

Welcome and Introduction

- André Reis, University Hospital Erlangen, Erlangen, Germany

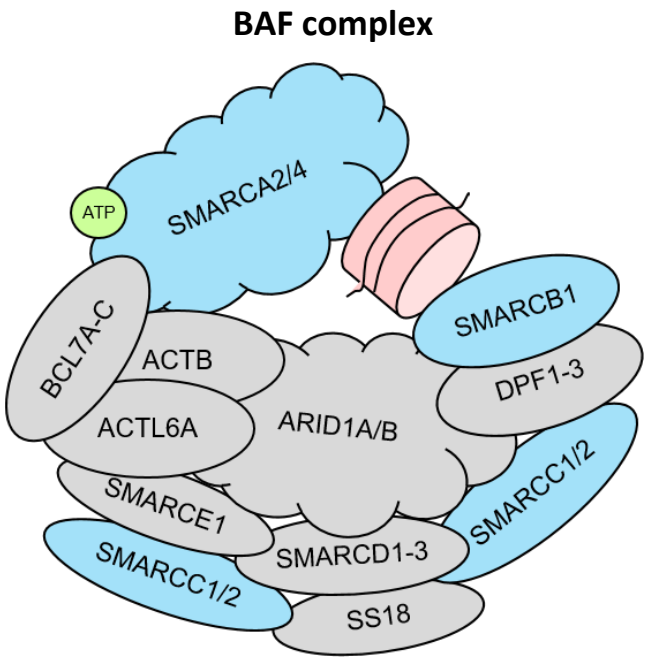
- Clinical and molecular diagnosis in children
  - Georgia Vasileiou , University Hospital Erlangen, Erlangen, Germany
- ARID1B-related disorder in adults
  - Gijs Santen, Leiden University Medical Center, Leiden, The Netherlands
- CARE4ARID1B
  - Dagmar Wieczorek, Medical Faculty and University Hospital, Düsseldorf, Germany
- The patient's perspective
  - Gal Lazarus, Ph.D., Department of Psychology, The Hebrew University of Jerusalem, Israel
- Discussion time - Conclusion with speakers and moderator

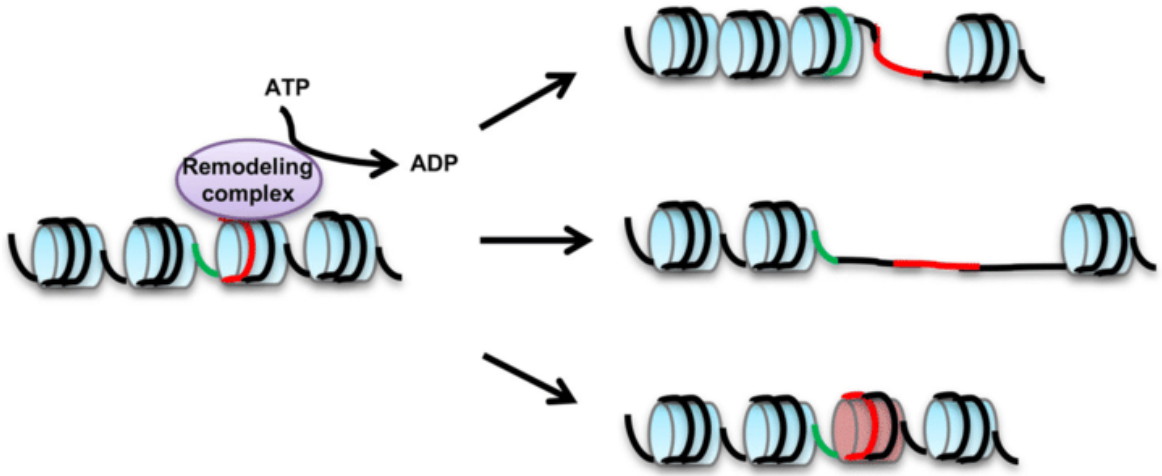
Clinical and molecular diagnosis in children

Georgia Vasileiou , Institute of Human Genetics, University Hospital Erlangen, Germany

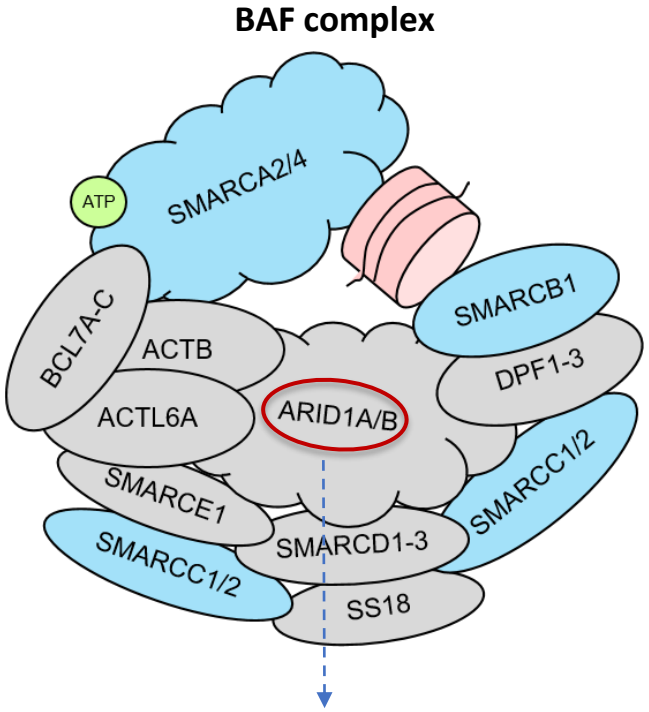
2025

BAF complex
(also known as SWI/SNF complex)





BAFopathies



1% of neurodevelopmental delay cases

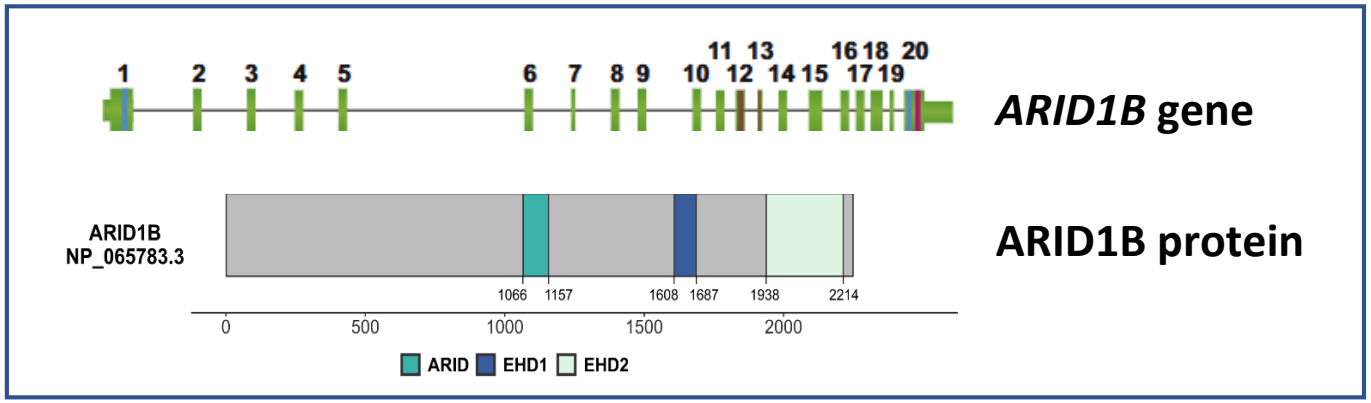
Coffin-Siris syndrome (CSS)

ARID1B: 50-83% (CSS 1)
ARID1A: 6% (CSS 2)
SMARCB1: 7% (CSS 3)
SMARCA4: 7% (CSS 4)
SMARCE1: 2% (CSS 5)
ARID2: 53 patients (CCS 6)
DPF2: 12 patients (CSS 7)
SMARCC2: 65 patients (CSS 8)
SMARCD1: 7 patients (CCS 11)
BICRA: 16 patients (CSS 12)

Hoyer et al. 2012; Santen et al. 2012, 2013; Wieczorek et al. 2013; Tsurusaki et al. 2014;
Kosho et al. 2014a; van der Sluijs et al. 2019; Gillentine et al. 2022; Vasko et al., 2021, Valencia et al. 2023

ARID1B

- ARID1B (AT-rich interactive domain-containing protein 1B) is located in chromosome 6q25 (20 exons)



- 3 main domains
  - ✓ ARID: DNA-binding domain
  - ✓ EHD1/EHD2: interact with each other-formation of ARID1A/B homo-/heterodimers
  - ✓ EHD2: interacts with SMARCA4

- Key stabilizer of the BAF complex
- It is expressed in brain and a wide range of tissues

Hurlstone at al., Biochem J, 2002
Bosch et al., Human Genetics, 2024
Hoyer et al., AJHG, 2012

European Reference Networks ITHACA

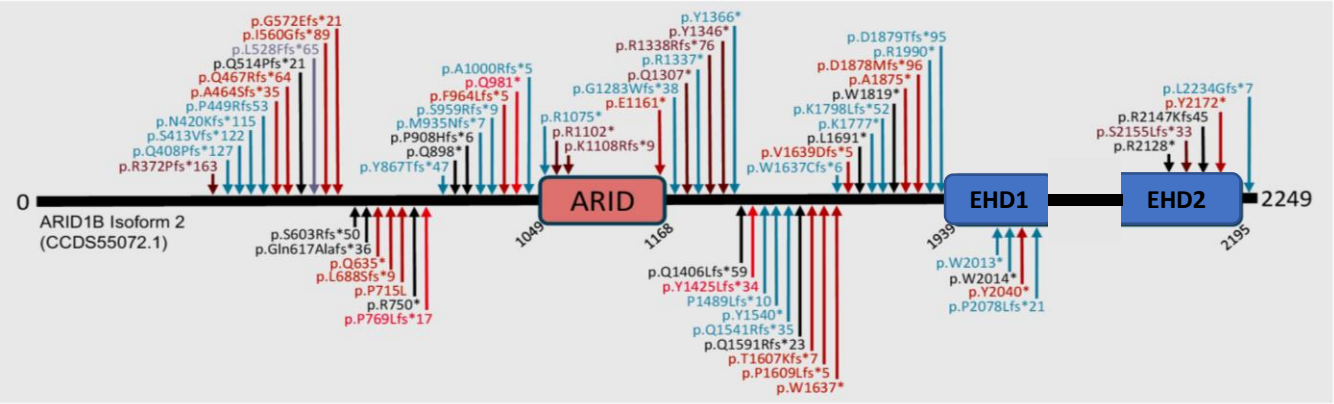
Molecular aspects of CSS 1 (ARID1B-associated CSS)

- autosomal dominant disorder
- complete penetrance
- heterozygous mutations/aberrations
- *de novo* mutations/aberrations
  - ✓ recurrence risk <1%
- germline mosaicism
  - ✓ reccurence risk up to 50%
- inherited mutations from mildly affected parents
  - ✓ recurrence risk 50%

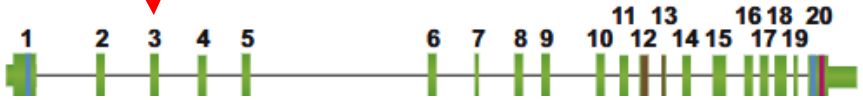
Truncating variants

- ❖haploinsufficiency gene
- ❖loss-of-function (truncating) variants
  - frameshift, nonsense, splice-site → protein degradation
  - intragenic deletions/duplications → protein degradation

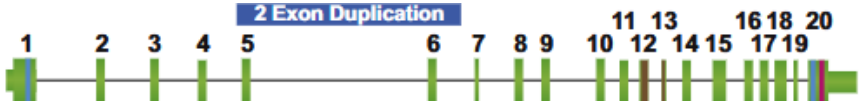
ARID1B protein



Not pathogenic



***ARID1B* gene**



***ARID1B* gene**

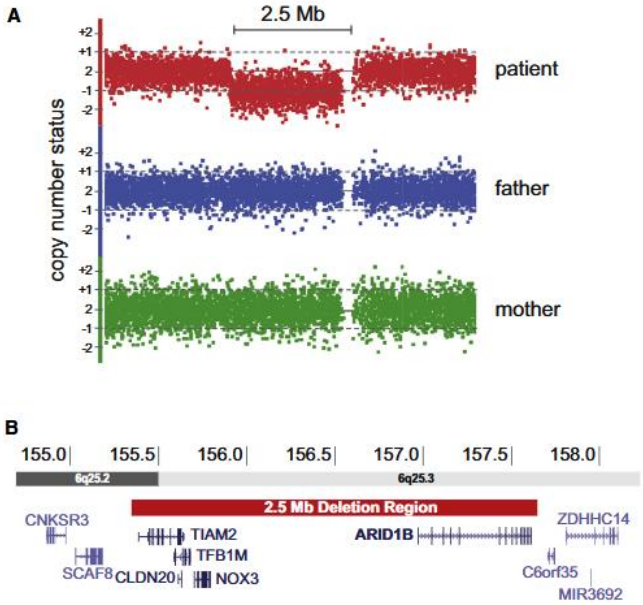
Modified figure from Sim *et. al.*, Intractable & Rare Diseases Research, 2015
Hoyer et al., AJHG, 2012
Van der Sluijs et al., Genetics in Medicine, 2019

2025

European Reference Networks ITHACA

Deletions- structural rearrangements

- whole-gene deletions in 6q25 (0.73- 2.7Mb)
- paracentric inversion-translocations: *ARID1B* disruption



Hoyer et al., AJHG, 2012

Grochowski *et. al.*, Front Genet., 2021

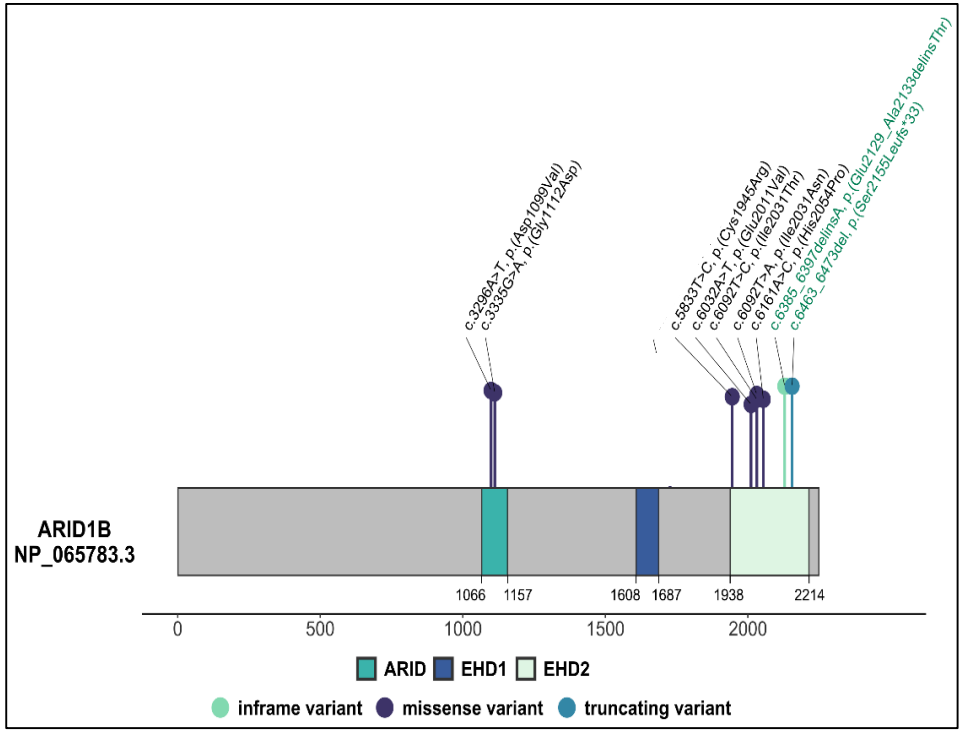
2025

Non-truncating variants

- ❖non-truncating variants (inframe, missense) in ARID and EHD2 domain
- ❖truncating variants not leading to protein degradation (exon 20)

} pathogenic

- pathomechanism is loss-of-function



Bosch et al., Human Genetics, 2024

2025

European Reference Networks ITHACA

ARID1B-associated phenotype and clinical manifestations

Non-syndromic neurodevelopmental delay (NDD)/intellectual disability (ID)

≠ **dysmorphic features**

Classic CSS

Hoyer et al., AJHG, 2012
Mannino et. al., AJMG, 2018
Van der Sluijs et al., Genetics in Medicine, 2019
Vasko et al., genes, 2021

Genetic testing ensures diagnosis

Gene panel testing

Exome analysis

Genome analysis

2025

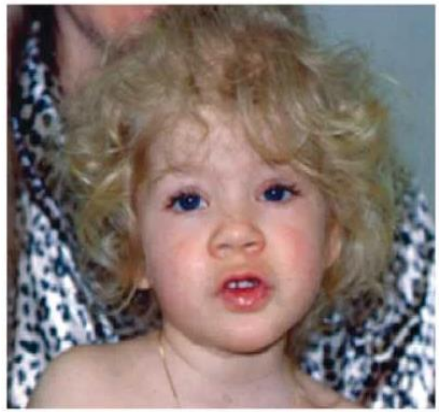
Mannino et al., AJMG, 2018

European Reference Networks ITHACA

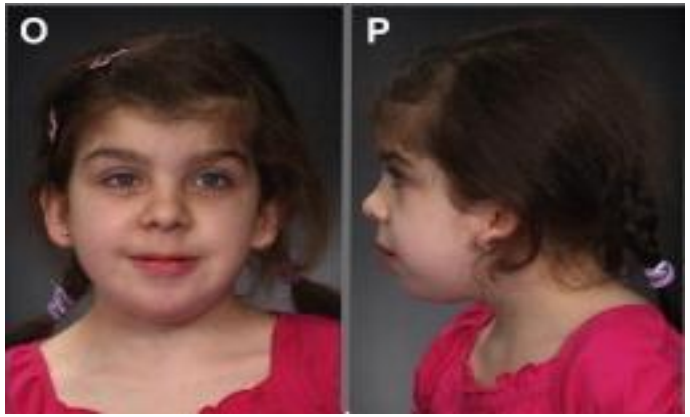
CSS: recognisable phenotype

Coarse facial features (>80%)

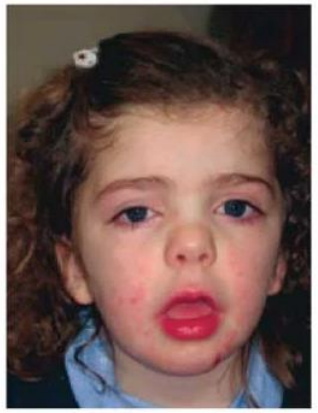
- ✓ Thick eyebrows, long eyelashes, wide mouth with thick lips, everted lower lip, broad nose, downslanted palpebral fissures, low-set ears



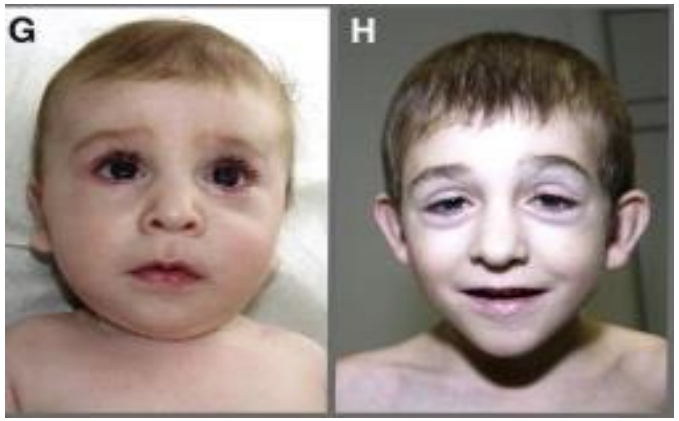
2y 5m



3y 10m

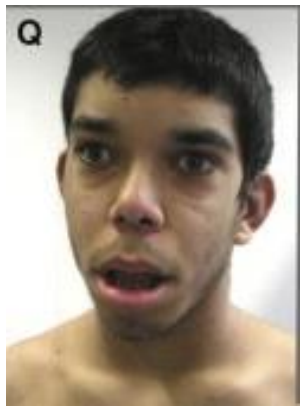


4y 5m



5m 7y 3m

19y



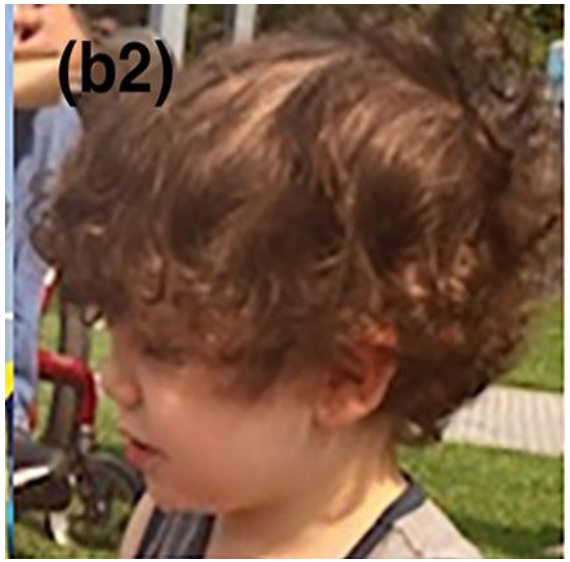
17y

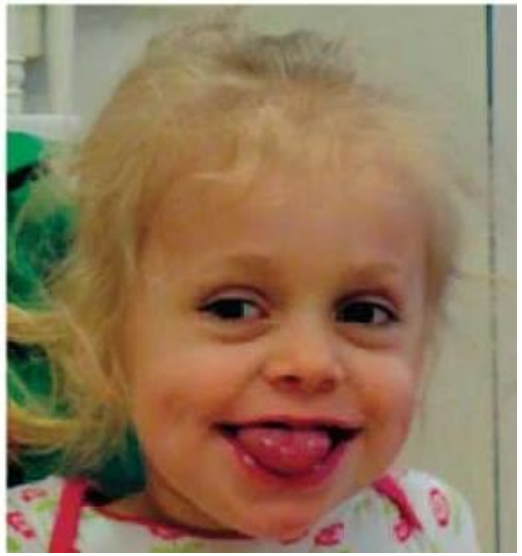
Hoyer et al., AJHG, 2012
Santen et al., Nature Genetics, 2012
Mannino et al., AJMG, 2018

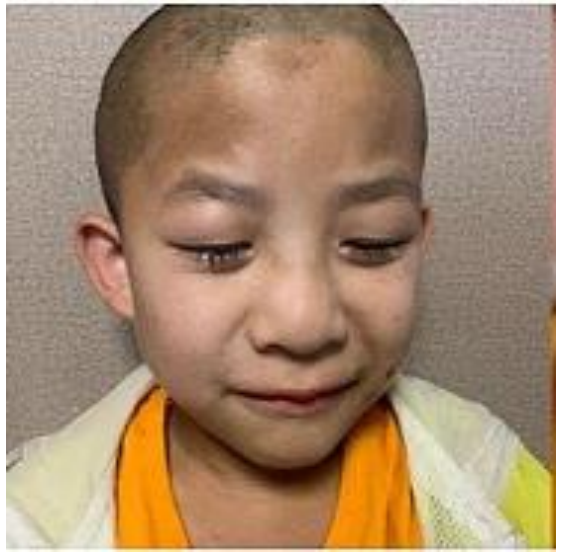
2025

European Reference Networks ITHACA

Sparse/thin scalp hair (50-80%)



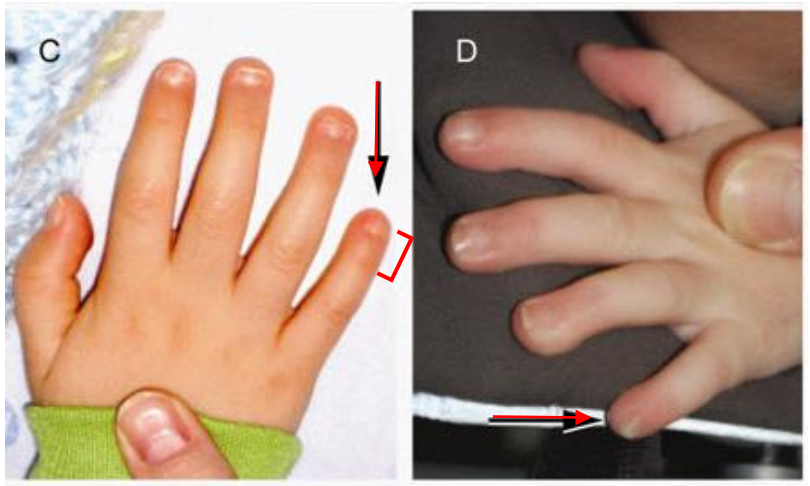


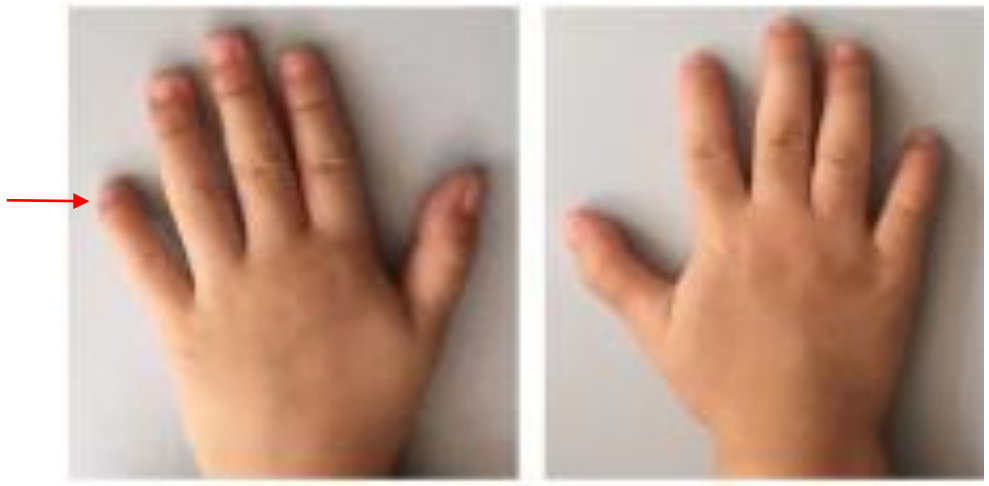


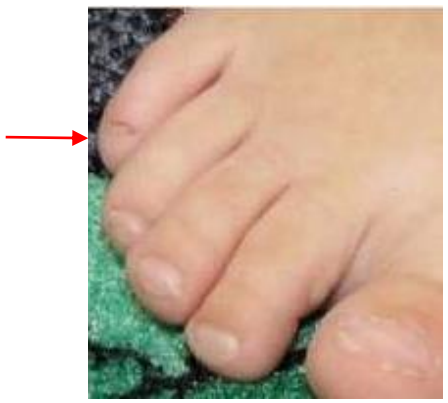
Mannino et al., AJMG, 2018
Vasko et al., Genes, 2021
Zapata-Aldana et al., Clinical Genetics, 2023
Huang et al., Med Genomics, 2024
Santen et al., Nature Genetics, 2012

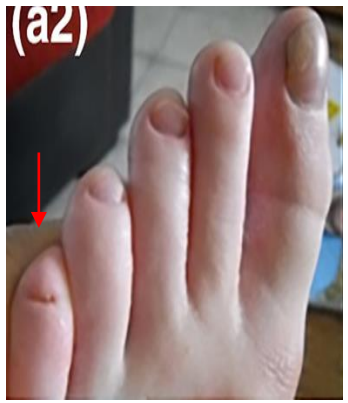
Hypoplasia of the 5th toe or fingernail and/or additional digits (60-80%)

Short/absent 5th distal phalanx (33-40%)



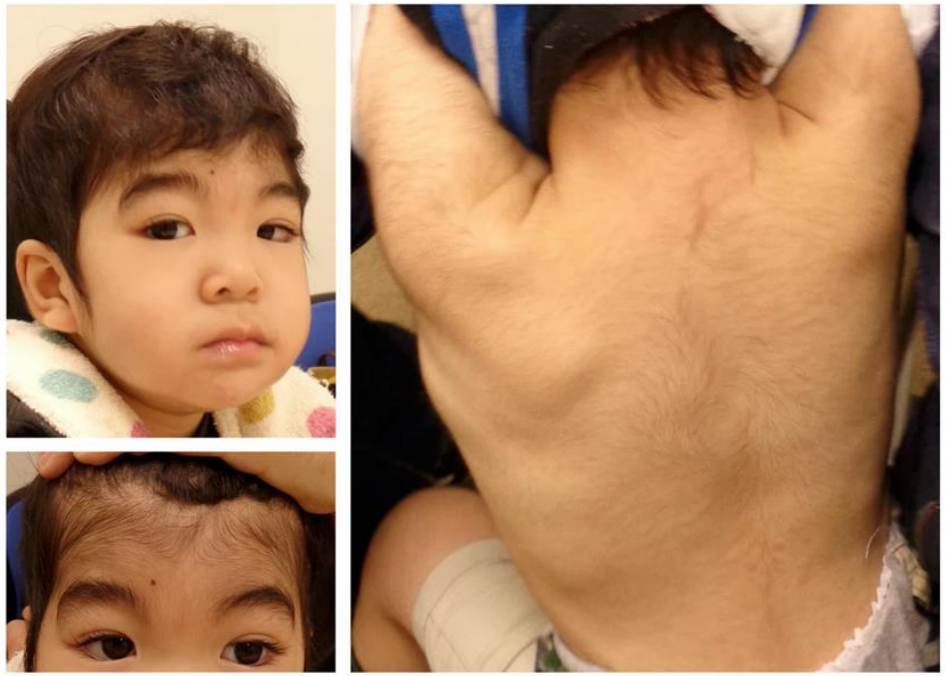






Lu et al., BMC Medical Genomics, 2021
Mannino et al., AJMG, 2018
Bosch et al., Human Genetics, 2024
Vasko et al., Genes, 2021
Gene Reviews, Coffin-Siris syndrome, 2021

Hypertrichosis (60-86%)

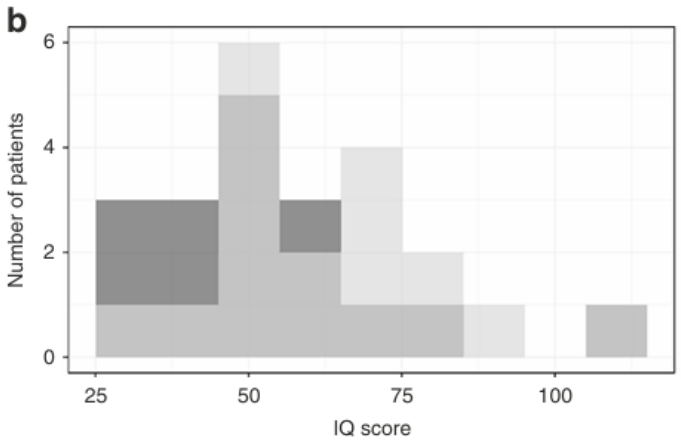


Van der Sluijs et al., Genetics in Medicine, 2019
Vasko et al., Genes, 2021
Sofronova et al., Human Genome Variation, 2022

CSS clinical manifestations

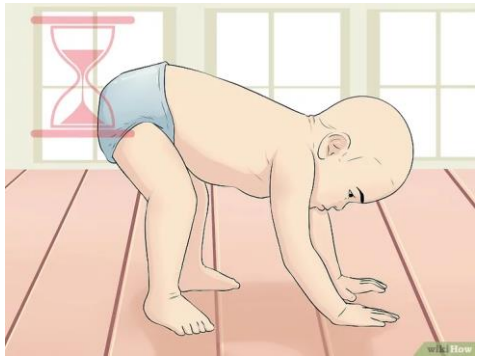
variable NDD/ID- almost all individuals (98%)

- ✓ 3% borderline-normal intelligence
- ✓ 38% mild/8.6% mild-moderate
- ✓ 22% moderate/17% moderate-severe
- ✓ 10% severe/profound



speech delay (98%)

- ✓ the majority of individuals
- ✓ no speech development (25%)



gross and fine motor delay (98%)

behavioral anomalies (80-85%)

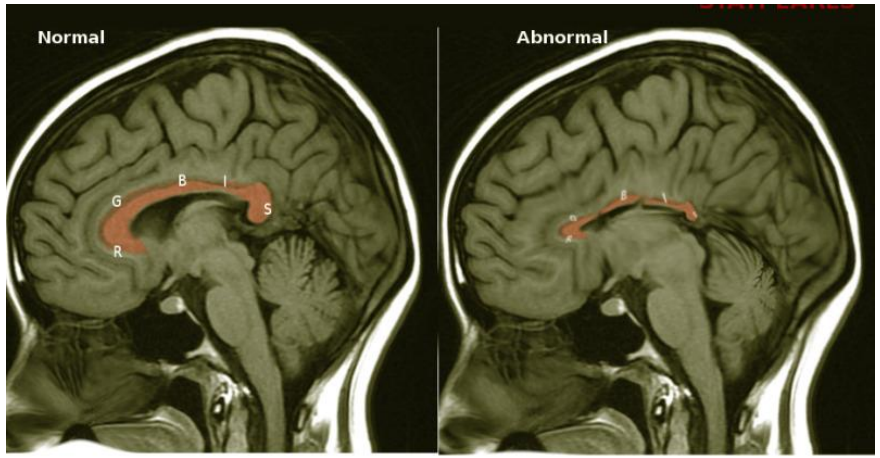
- ✓ autistic traits/autism
- ✓ ADHD
- ✓ hyperactivity

Van der Sluijs et al., Genetics in Medicine, 2019
Gene reviews, ARID1B-related disorder, 2019
https://de.wikihow.com

European Reference Networks ITHACA

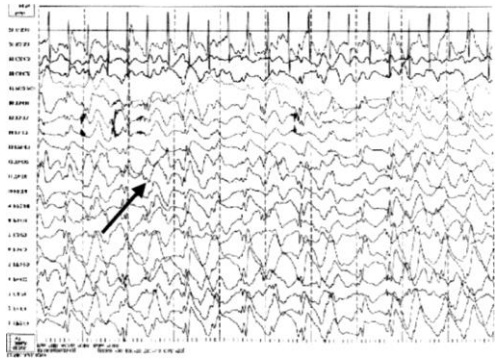
Other neurological anomalies

muscular hypotonia (80%)



brain anomalies

- ✓ Corpus callosum hypoplasia/agenesis (40-47%)



epilepsy (28%)

- ✓ birth-mid teenage years
- ✓ abnormal EEG (6%)

Van der Sluijs et al., Genetics in Medicine, 2019
Gene reviews, ARID1B-related disorder, 2019
Gene reviews, Corpus Calosum Agenesis, 2023
https://de.wikihow.com

European Reference Networks ITHACA

Growth-feeding-infections

growth parameters

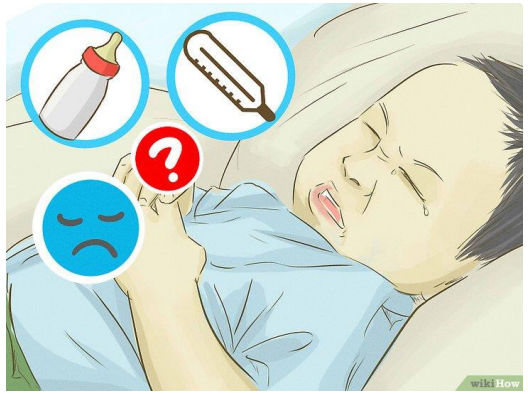
✓ birth: normal for the majority

short stature (30%)

✓ growth hormone deficiency
✓ underweight rare
✓ microcephaly rare

feedings problems (60-70%)

✓ birth (70 %)
✓ short duration (40%)
✓ several years



recurrent infections (57%)

✓ upper respiratory-viral
✓ otitis media

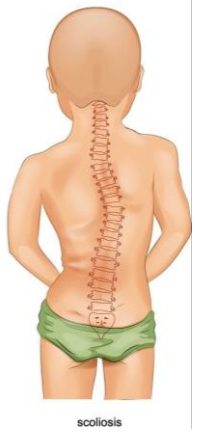
Van der Sluijs et al., Genetics in Medicine, 2019
Gene reviews, ARID1B-related disorder, 2019
https://de.wikihow.com

Organ system abnormalities- multisystem disorder

ophthalmologic abnormalities (48%)

- ✓ myopia
- ✓ strabismus
- ✓ hypermetropia
- ✓ astigmatism

hearing loss (20-30%)



musculoskeletal problems

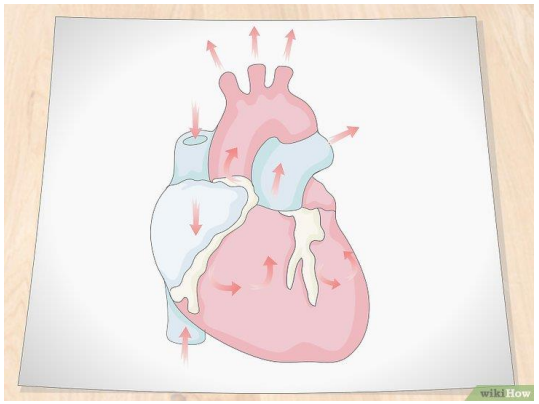
- ✓ delayed bone age (47%)
- ✓ clinodactyly (36%)
- ✓ scoliosis (26%)
- ✓ joint laxity
- ✓ brachydactyly
- ✓ Pes planus

delayed dentition (45%)

- ✓ primary (44%)
- ✓ permanent (48%)
- ✓ widely spaced teeth

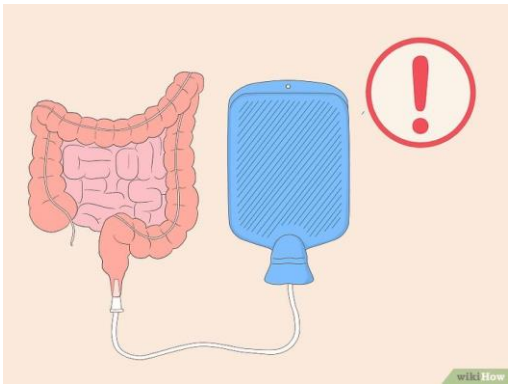
Van der Sluijs et al., Genetics in Medicine, 2019
Gene reviews, ARID1B-related disorder, 2019
Calcus et al., eLife, 2019
https://de.wikihow.com
https://bdiplayhouse.com/scoliosis-intervention/

Organ system abnormalities



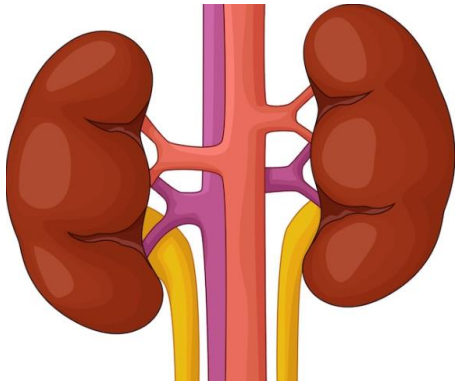
gastrointestinal problems (48%)

- ✓ constipation
- ✓ gastrointestinal reflux



congenital heart defects (20%)

- ✓ atrial septal defect
- ✓ ventricular septal defect

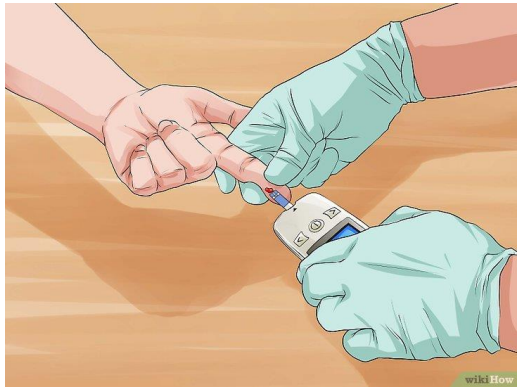


renal defects (12%)

- ✓ nephrolithiasis
- ✓ hydronephrotic kidney

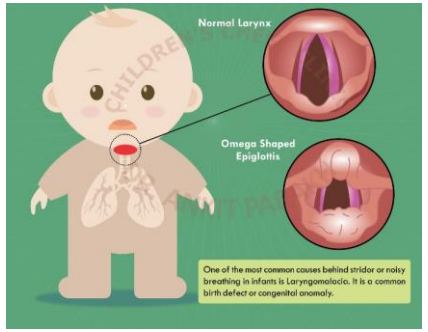
urogenital defects

- ✓ cryptorchidism (44-55%)



endocrinological abnormalities

- ✓ Hypothyreoidism



Laryngomalacia (20%)

Van der Sluijs et al., Genetics in Medicine, 2019
Gene reviews, ARID1B-related disorder, 2019
https://de.wikihow.com
Premium Vector | Human kidney cartoon

European Reference Networks ITHACA

Genotype-phenotype correlation

loss-of-function (truncating) variants in exon 1 of *ARID1B*

↓

milder phenotypes/ID

---

non-truncating variants in EHD2 and ARID domains of *ARID1B*

truncatings variants in exon 20-aberrant transcripts

↓

same phenotypic and clinical features with *ARID1B* truncating variants

Van der Sluijs et al., Genetics in Medicine, 2019
Van der Sluijs et al., Genetics in Medicine, 2024
Bosch et al., Human Genetics, 2024

ARID1B-related disorder in adults

Gijs Santen, Leiden University Medical Center, Leiden, The Netherlands

2025

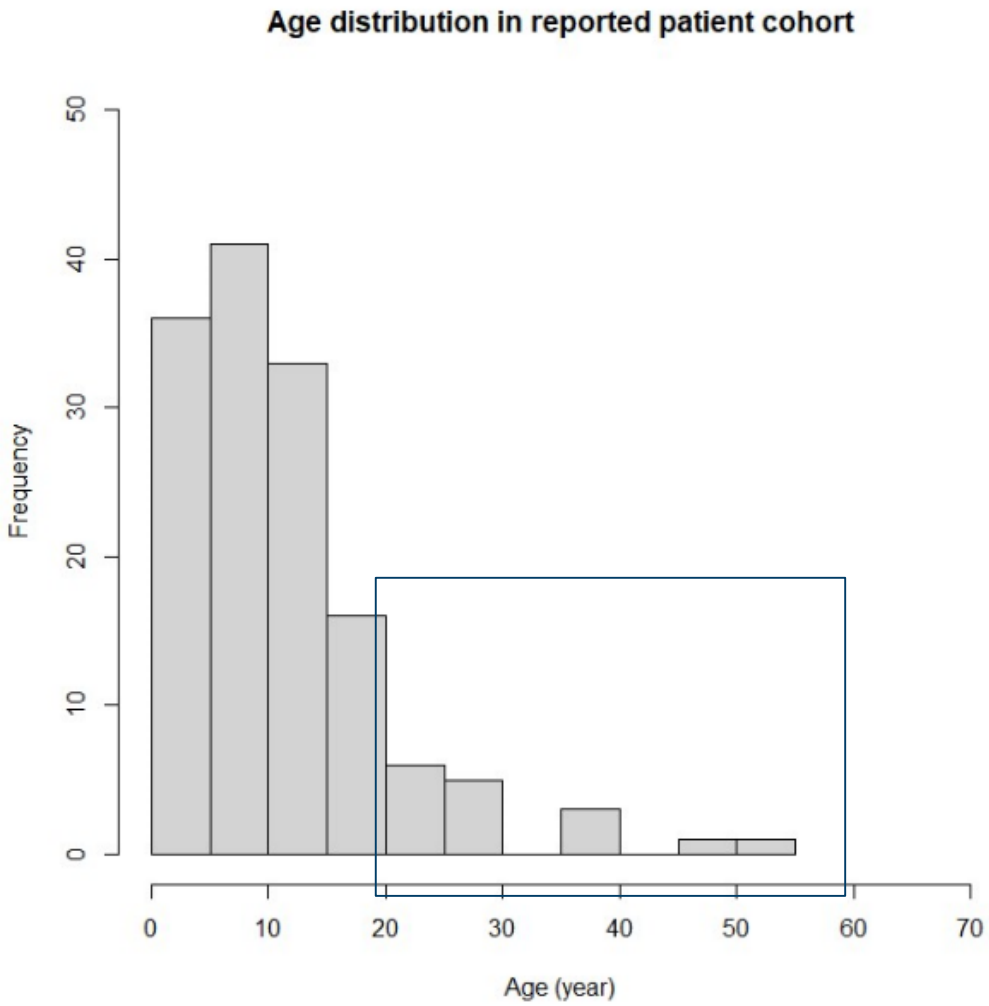
Leiden University
Medical Center

ARID1B-related disorder in adults

https://doi.org/10.1016/j.gimo.2024.101873

Prof. Gijs Santen
Department of Clinical Genetics
LEIDEN UNIVERSITY MEDICAL CENTER
Leiden, the Netherlands

ARID1B in adults: a knowledge gap



- Small number of adult patients included
- Knowledge representative for the adult population?
  - E.g. seizure frequency?

van der Sluijs *et al* 2019

Outline

- Study set-up
- Medical findings
- Self-sustainability
- Exon 1 variants vs other variants

Study set-up

ARID1B in adults: a study

Inclusion criteria

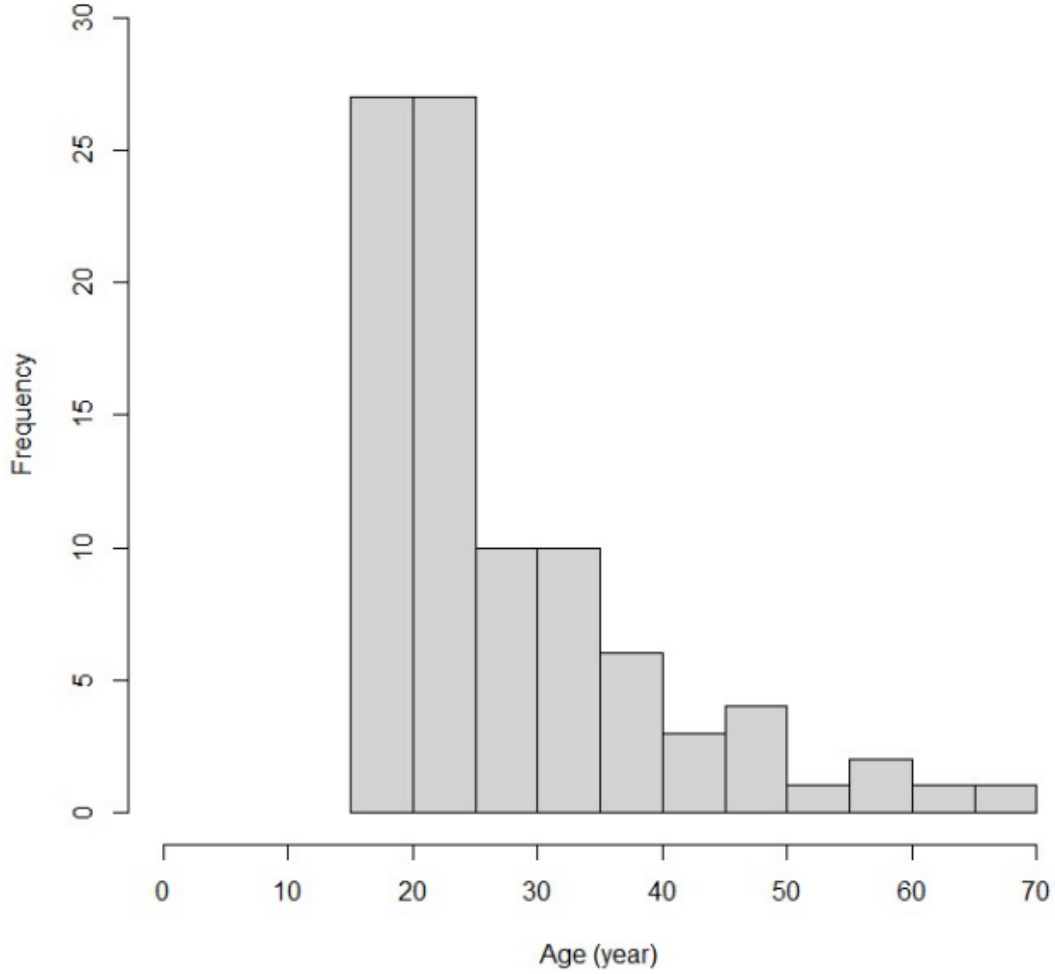
- Pathogenic ARID1B variant
- Aged 18 years or older

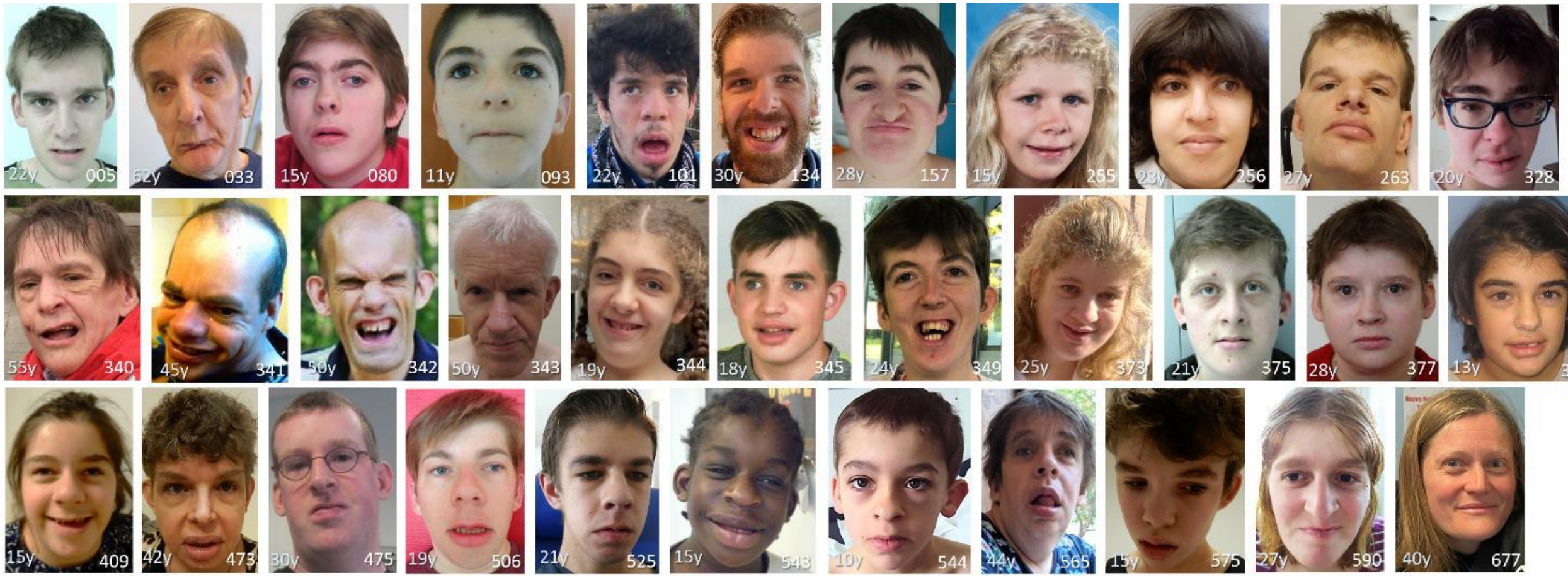
Aims:

- Phenotyping
- Describe functioning

Cohort:

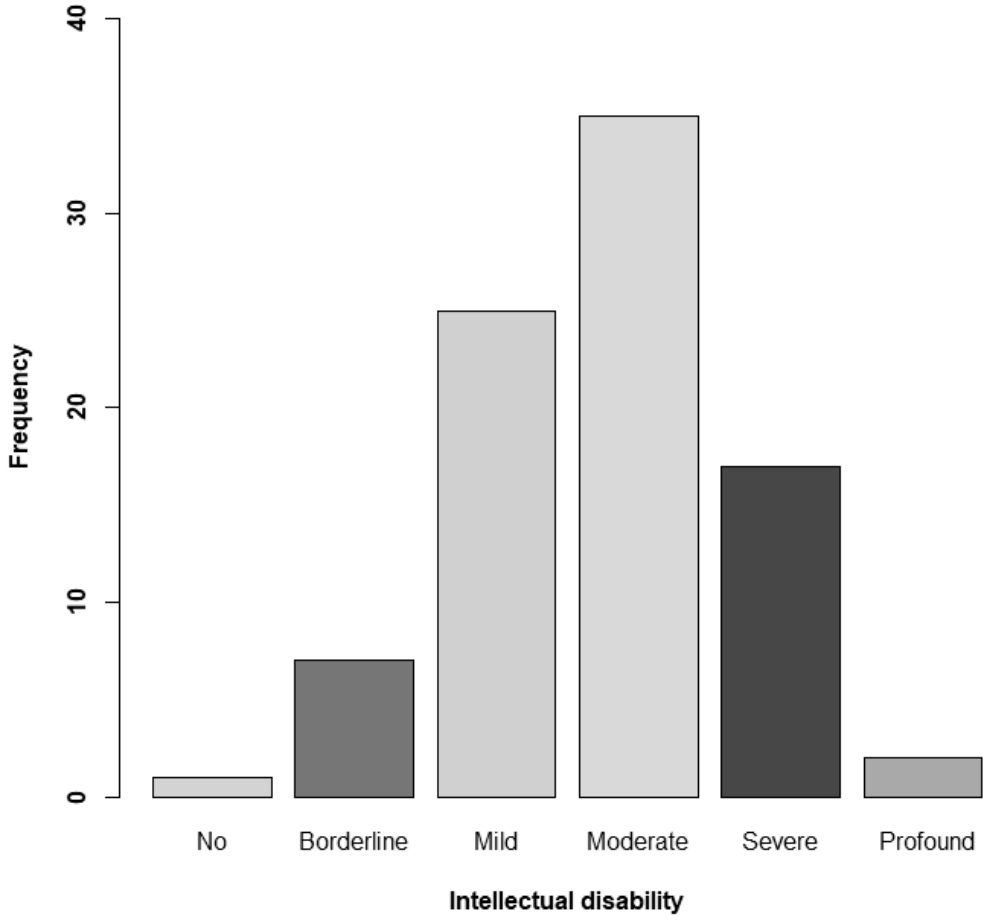
- 87 patients



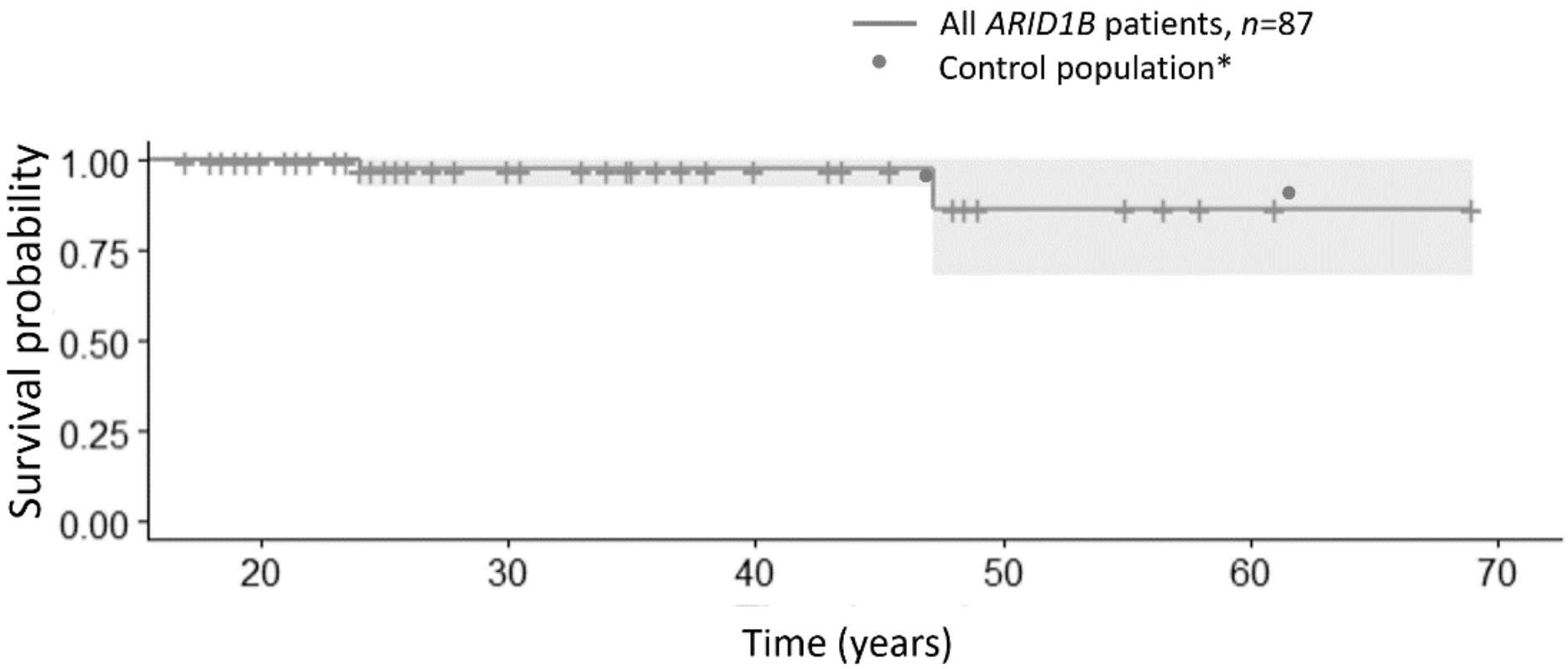

22y 005
62y 033
15y 080
11y 093
22y 101
30y 134
28y 157
15y 255
23y 256
27y 263
20y 328
55y 340
45y 341
50y 342
50y 343
19y 344
18y 345
24y 349
25y 373
21y 375
28y 377
13y 395
15y 409
42y 473
30y 475
19y 506
21y 525
15y 543
10y 544
44y 565
15y 575
27y 590
40y 677

Medical findings

Results: degree of ID

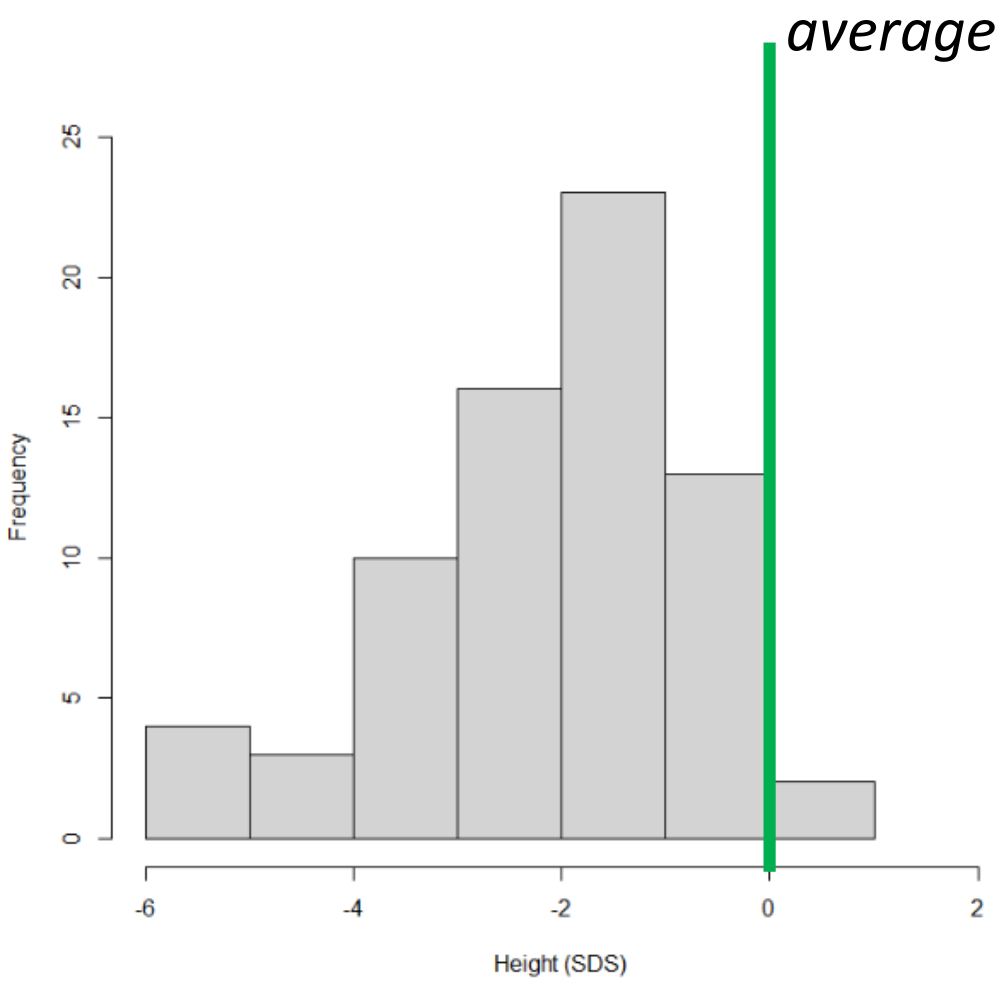


Results: Life expectancy

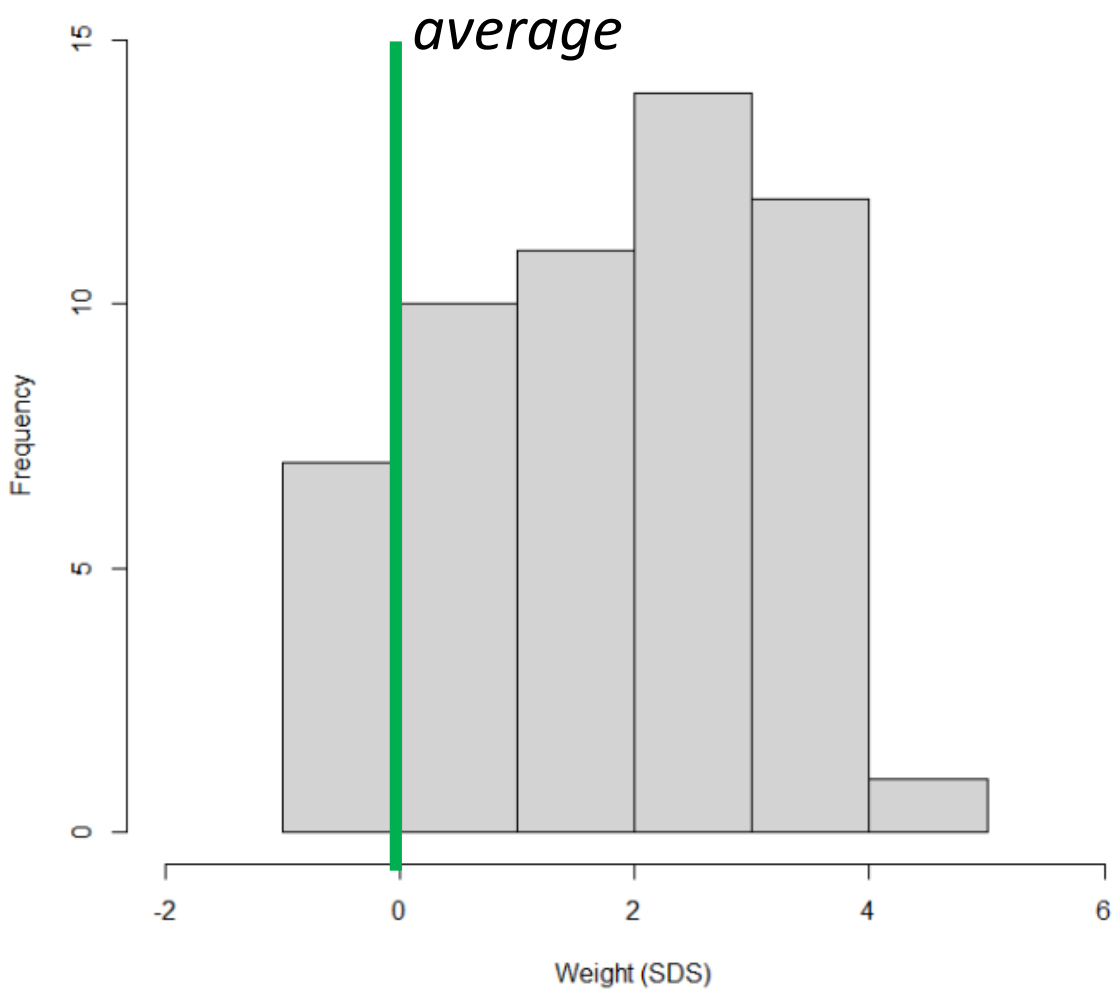


Life expectancy similar to people without an *ARID1B* mutation!

Results: biometry

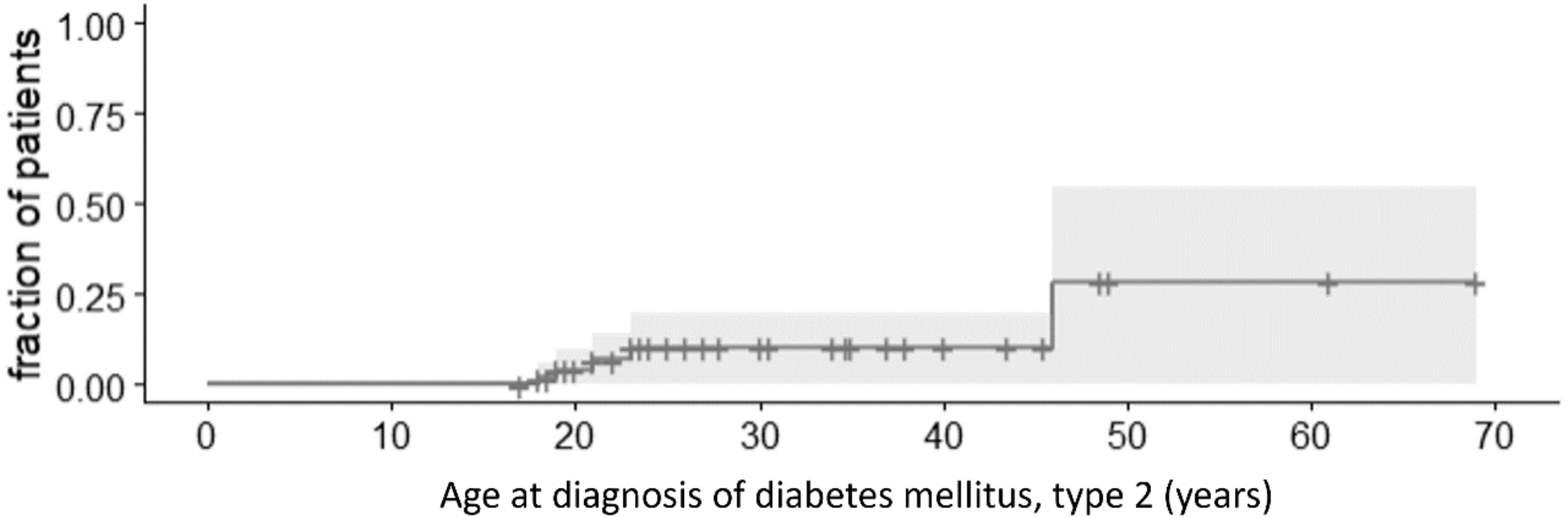


Height: lower than average



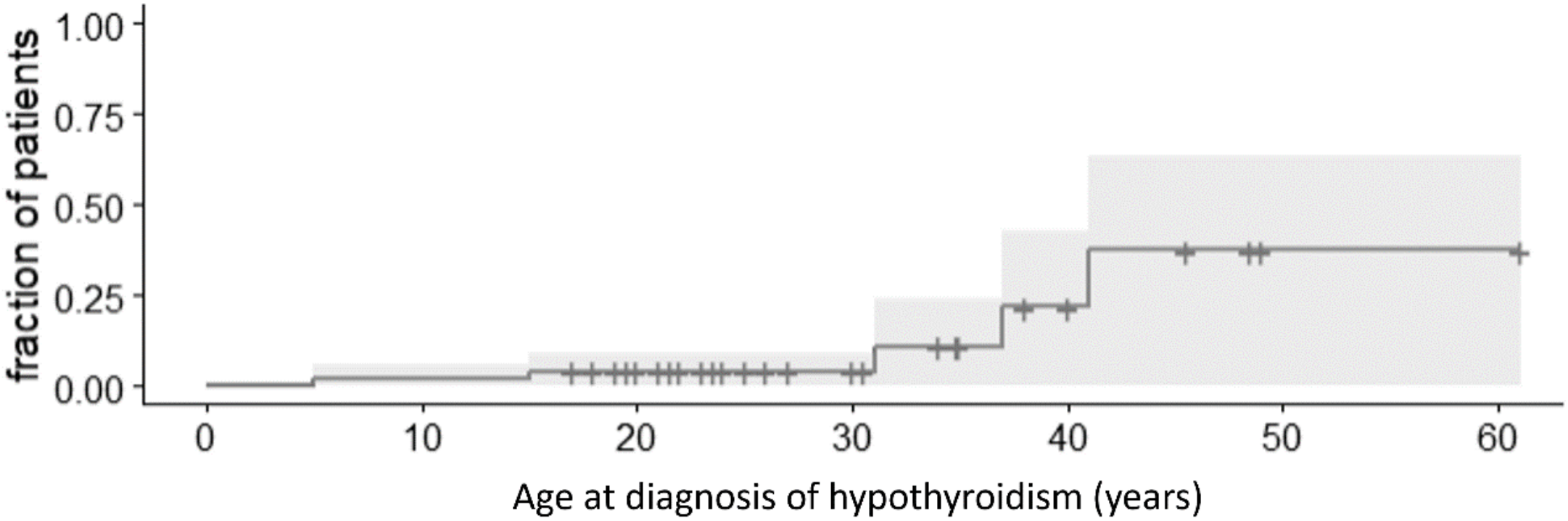
Weight: higher than average

Results: Diabetes Mellitus

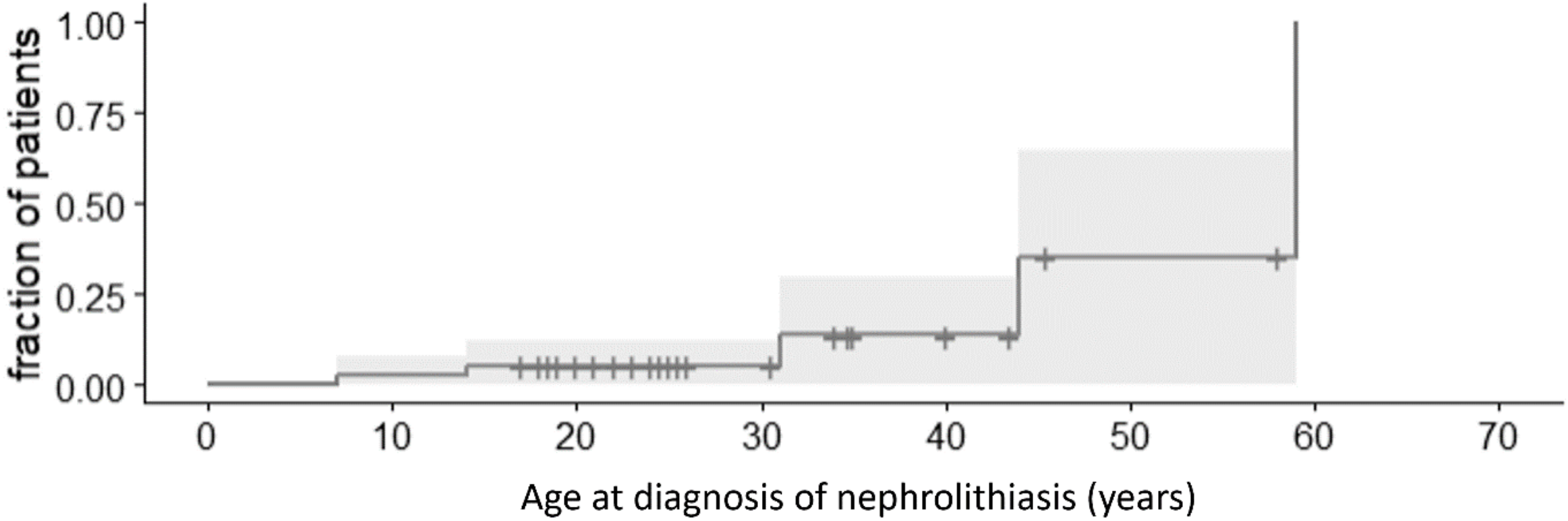


Results: Seizures

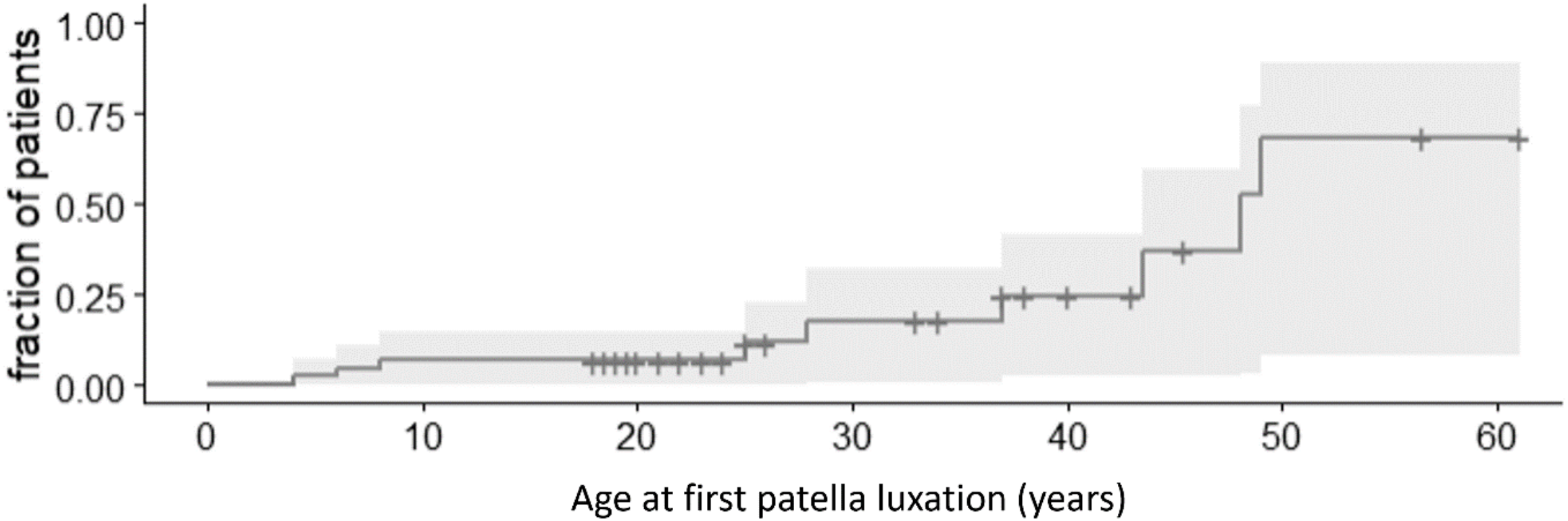
Results: Hypothyroidism



Results: Nephrolithiasis



Results: Patellar luxation



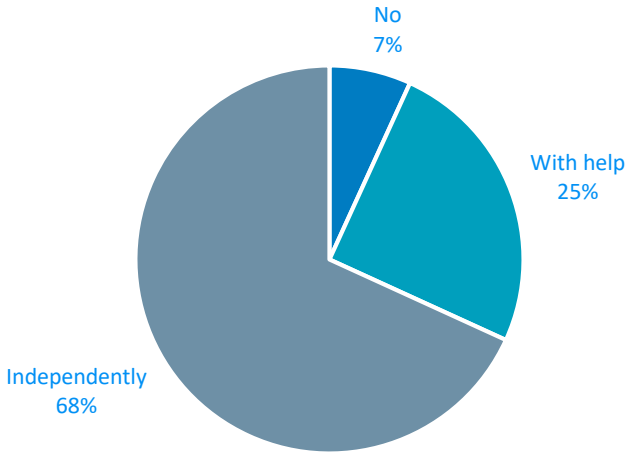
Results: Loss of skills

Loss of skills was noted in 25% (16/64) of patients. No specific triggering event was reported. The age of onset was documented in four cases. This age ranged from 31 to 54 years. Loss of motor skills, particularly in walking ability with increased tripping and decreased balance, was the most common (7/16), followed by loss of speech (5/16). Some patients required the use of a wheelchair (5/62).

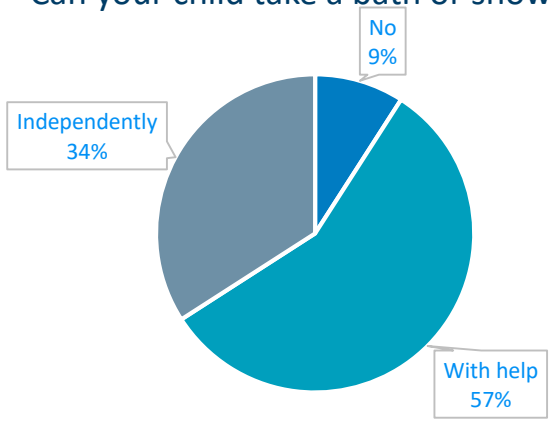
Self-sustainability

Some examples

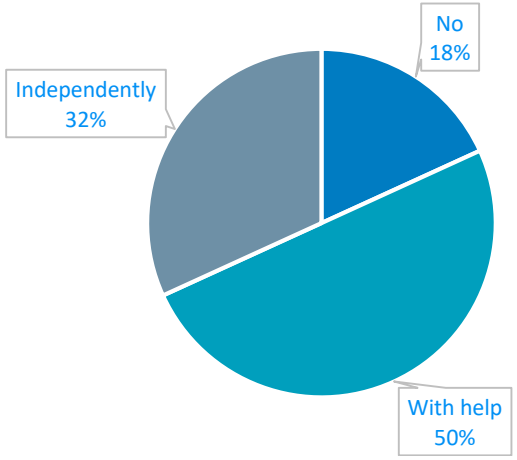
Can your child dress and undress him/herself?



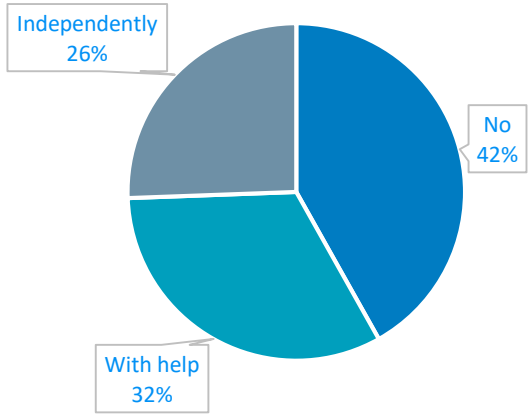
Can your child take a bath or shower?



Can your child brush his/her teeth and comb his/her hair?

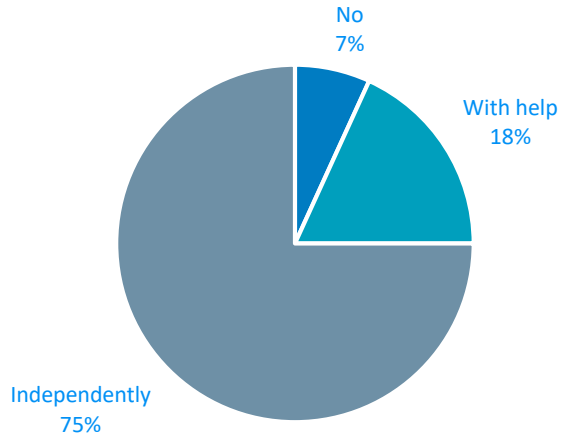


Can your child do the groceries?

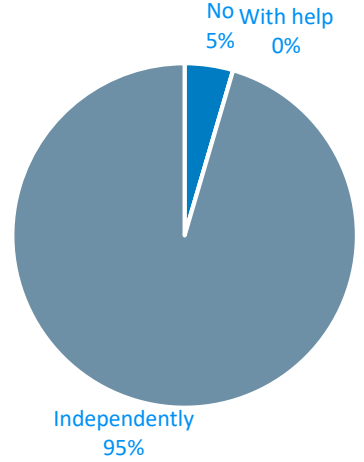


Some more examples

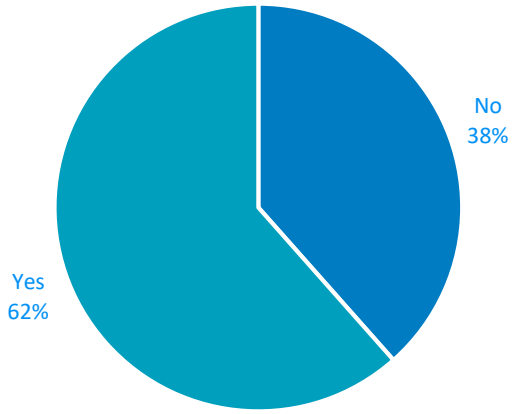
Can your child go to the toilet?



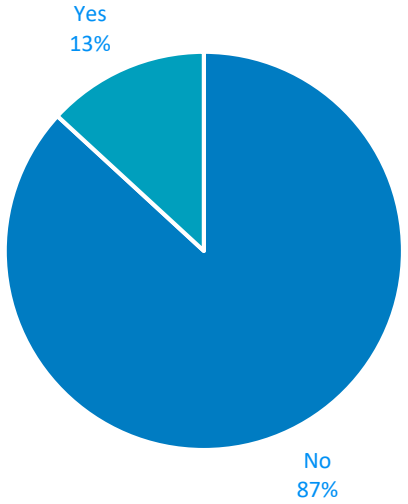
Can your child drink from a cup?



Can your child stay home alone for 30 minutes?

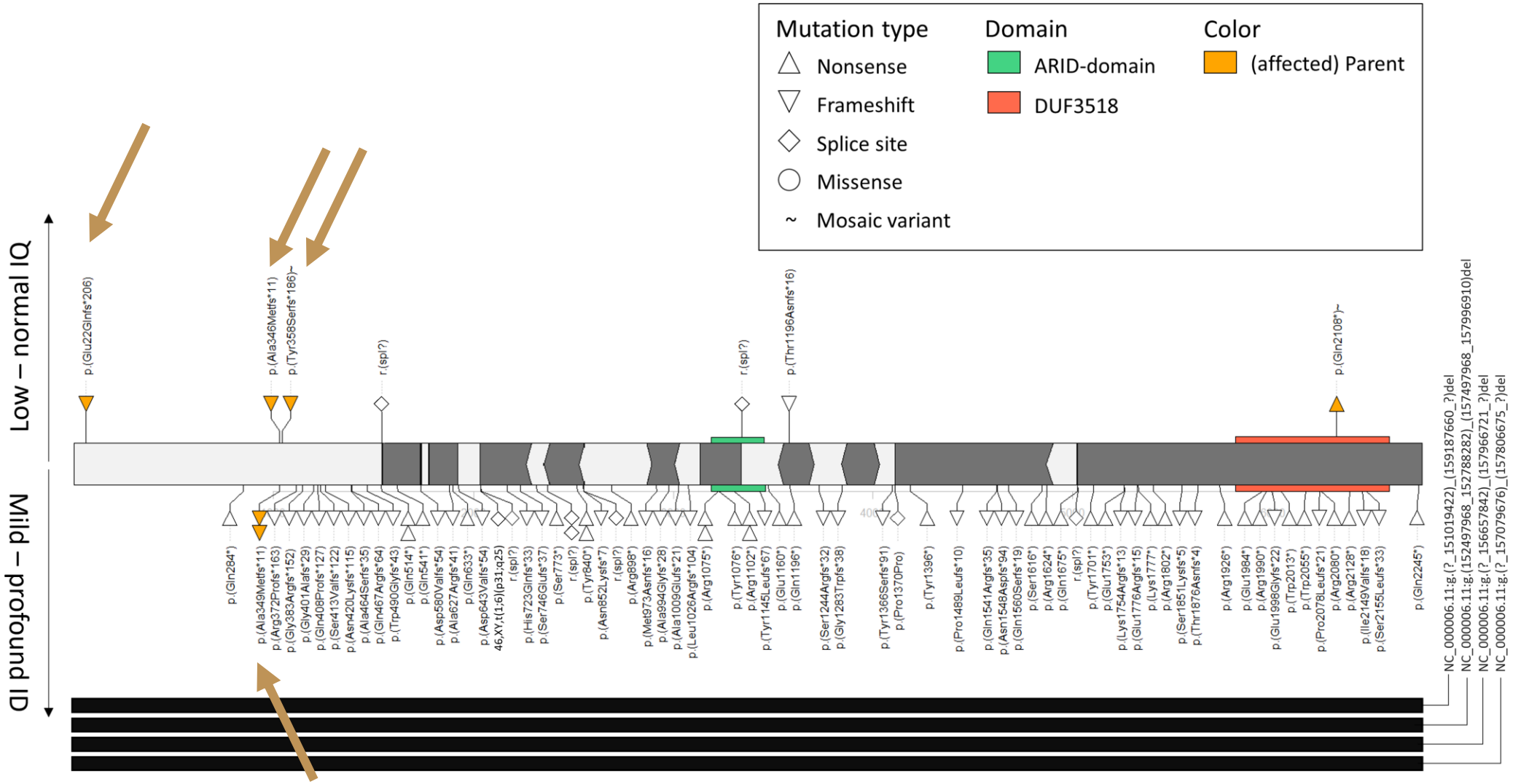


Can your child travel alone by public transport?



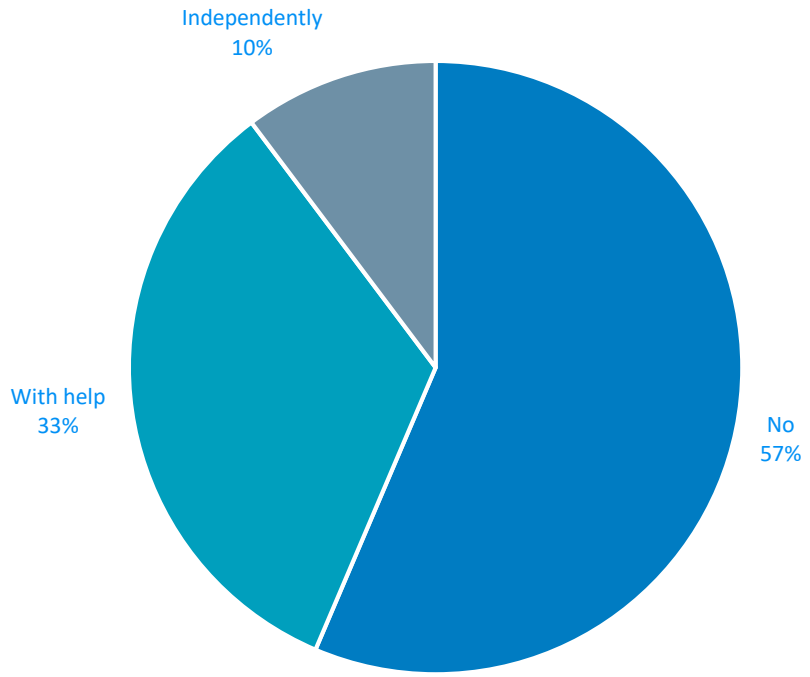
Exon 1 varants in adults

Data from previous work



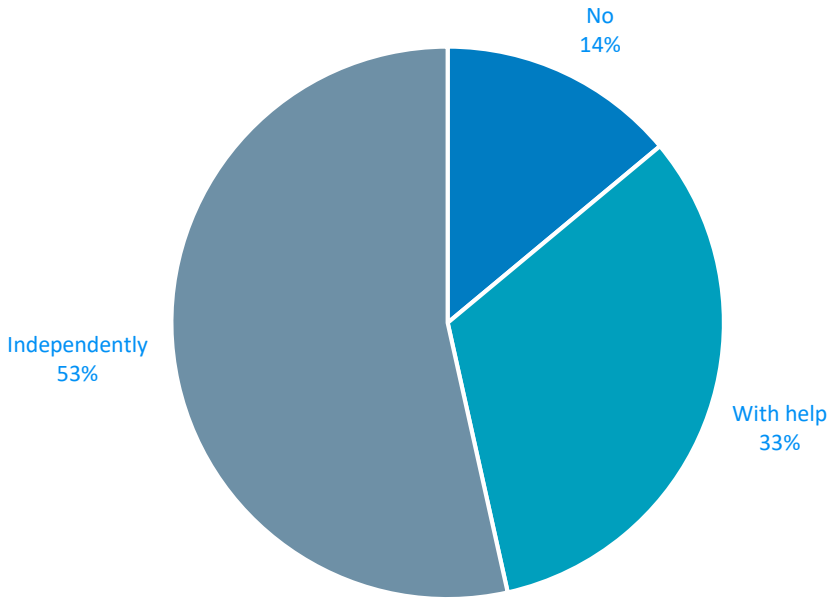
Can your child handle money, pay in the store?

> Exon 1

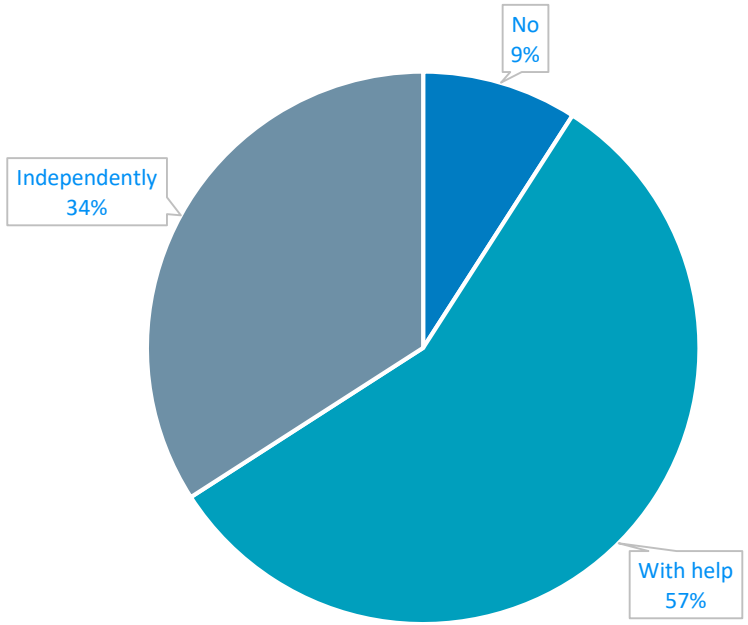


Can your child set and clear the table?

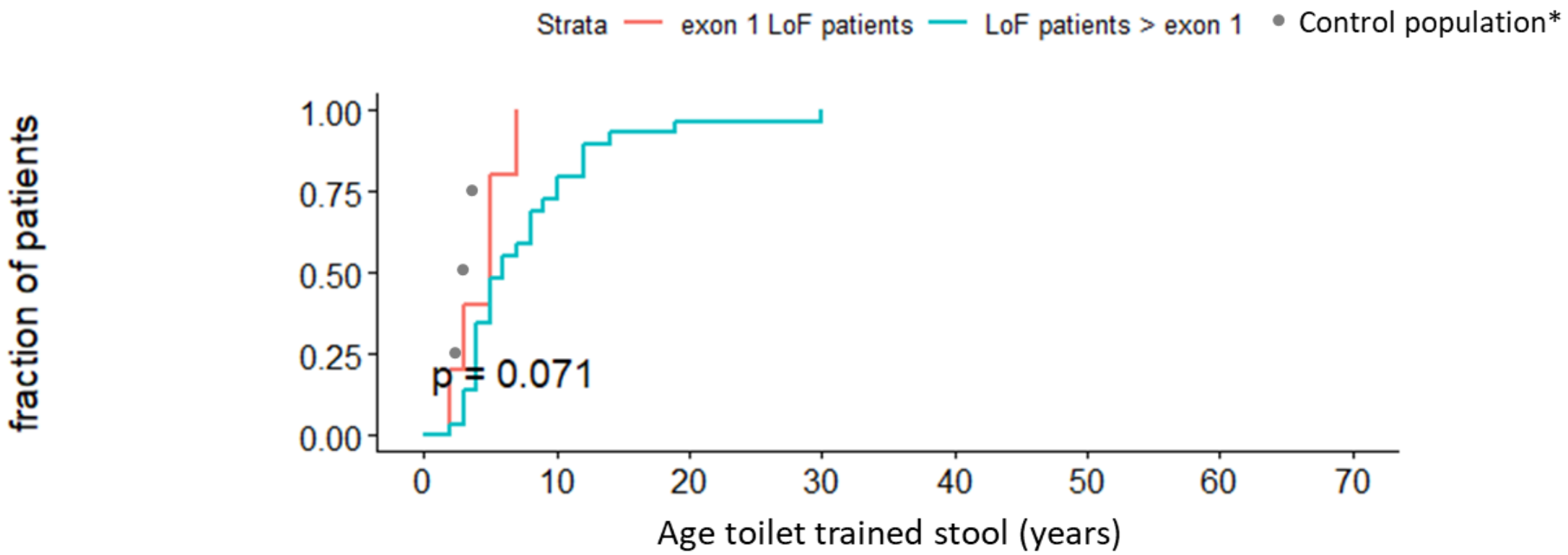
> Exon 1



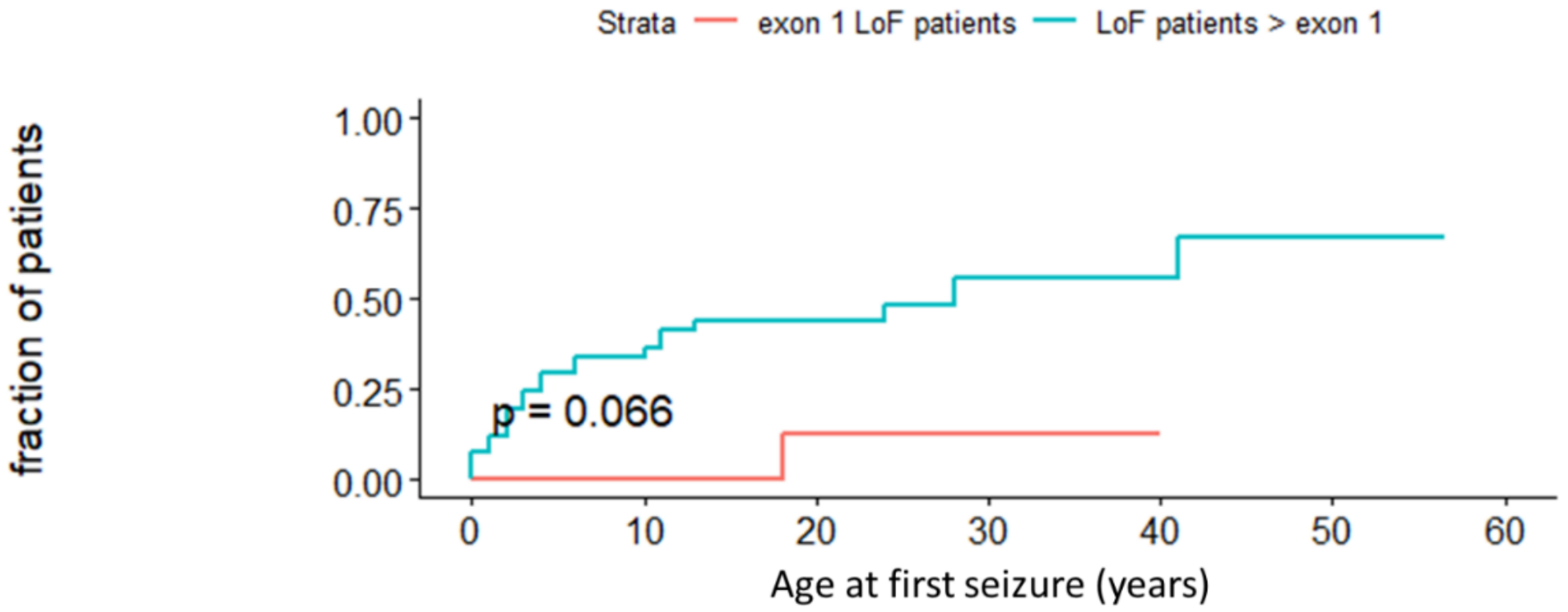
> Exon 1



Toilet training (stool)



Seizures


Strata
exon 1 LoF patients
LoF patients > exon 1
fraction of patients
1.00
0.75
0.50
0.25
0.00
p = 0.066
0
10
20
30
40
50
60
Age at first seizure (years)

Conclusions

- Shift in medical aspects as patients age
- First glimpse of self-sustainability
- Difference in exon 1 vs > exon 1 confirmed
- Recommendations given (see paper)
- More data needed on loss of skills

Leiden University
Medical Center

Acknowledgements

ARID1B-related disorder in 87 adults: Natural history and self-sustainability

P.J. van der Sluijs[1], M. Gösgens[1], A.J.M. Dingemans[2], P. Striano[3,4], A. Riva[4,5], C. Mignot[6], A. Faudet[7], G. Vasileiou[8,9], M. Walther[8], S.A. Schrier Vergano[10,11], M. Alders[12], F.S. Alkuraya[13,14], I. Alorainy[15], H.S. Alsaif[13,16], B. Anderlid[17], I. Bache[18], I. van Beek[12], M. Blanluet[19], B.W. van Bon[20], T. Brunet[21,22], H. Brunner[2], M.L. Carriero[23], P. Charles[6], N. Chatron[24,25], E. Coccia[26], C. Dubourg[27,28], R.K. Earl[29], E.E. Eichler[30,31], L. Faivre[32,33], N. Foulds[34], C. Graziano[35], A.M. Guerrot[36], M.O. Hashem[13], S. Heide[7], D. Heron[7], S.E. Hickey[37,38], S.M.J. Hopman[39], A. Kattentidt-Mouravieva[40], J. Kerkhof[41], J.S. Klein Wassink-Ruiter[42], E.C. Kurtz-Nelson[29,43], K. Kušíková[44], M. Kvarnung[17], F. Lecoquierre[36], G.S. Leszinski[21], L. Loberti[23,45], P.L. Magoulas[46], F. Mari[23], I. Maystadt[47], G. Merla[48,49], J.M. Milunsky[50], S. Moortgat[47], G. Nicolas[36], M.O.' Leary[51], S. Odent[28,52], J.R. Ozmore[53], K. Parbhoo[37,54], R. Pfundt[2], M. Piccione[55,56], A.M. Pinto[23], B. Popp[57], A. Putoux[24], H.L. Rehm[51], A. Reis[8,9], A. Renieri[23,45], J.A. Rosenfeld[46,58], M. Rossi[24], E. Salzano[55], P. Saugier-Veber[36], M. Seri[26], G. Severi[26], F.M. Sonmez[59], G. Strobl-Wildemann[60], K.E. Stuurman[61], E. Uctepe[62], H. Van Esch[63], G. Vitetta[26], B.B.A. de Vries[2], D. Wahl[64], T. Wang[30,65,66,67], P. Zacher[68], K.R. Heitink[69], F.G. Ropers[70], D. Steenbeek[71], T. Rybak[72], G.W.E. Santen[1,*]

CARE4ARID1B

Dagmar Wieczorek, Medical Faculty and University Hospital, Düsseldorf, Germany

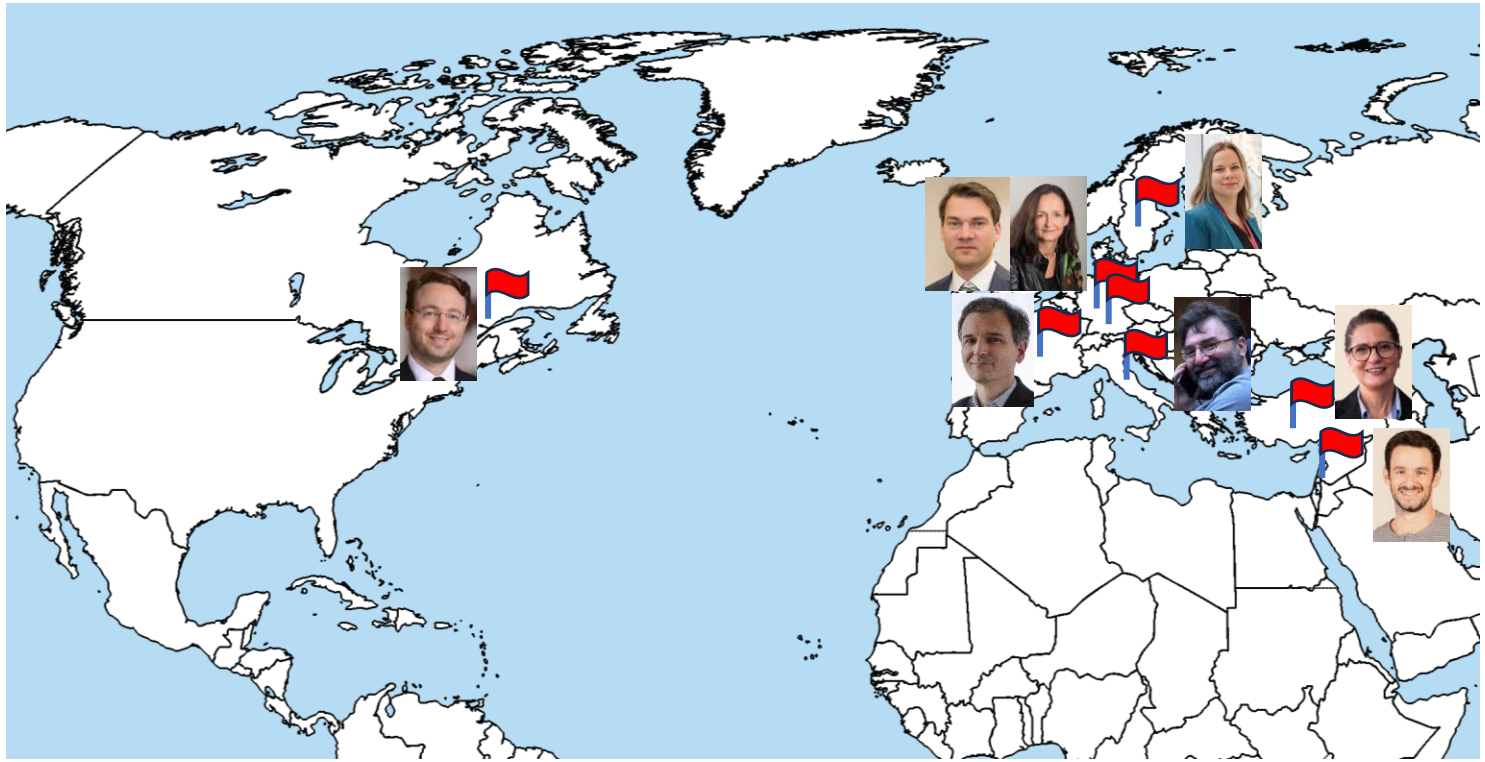
2025

CARE4ARID1B: Developmental Trajectories in *ARID1B*-Related Disorder – a Multi-Method Multi-Site Prospective Natural History Study

Call for patients – Study starts now

Dagmar Wieczorek
Institute of Human Genetics, Medical Faculty and University Hospital,
Heinrich Heine University, Düsseldorf, Germany
17.03.2025

Partners involved in CARE4ARID1B



Research partners (funded):

Gal Lazarus (Coordinator)
Jerusalem, Israel

Philippe Campeau
Montreal, Canada

Gaetano Cantalupo
Verona, Italy

Peter Krawitz
Bonn, Germany

Vincent des Portes
Lyon, France

Kristiina Tammimies
Stockholm, Sweden

Yasemin Alanay
Istanbul, Turkey

Collaborators (not funded)
Amanda Seidl, West Lafayette, Indiana, USA
Audrey Thurm, Bethesda, Maryland, USA
Eva Meisenzahl, Düsseldorf, Germany

SWI/SNF chromatin remodeling complex

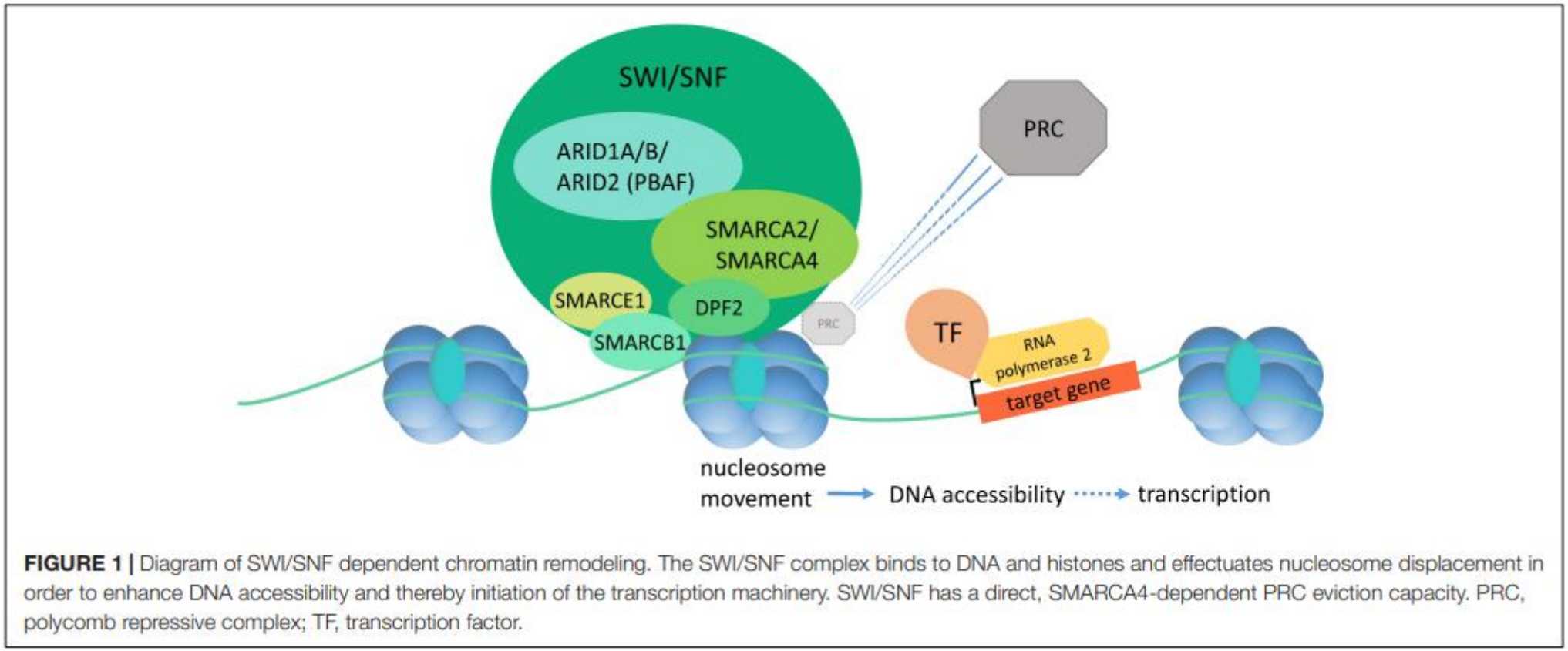


FIGURE 1 | Diagram of SWI/SNF dependent chromatin remodeling. The SWI/SNF complex binds to DNA and histones and effectuates nucleosome displacement in order to enhance DNA accessibility and thereby initiation of the transcription machinery. SWI/SNF has a direct, SMARCA4-dependent PRC eviction capacity. PRC, polycomb repressive complex; TF, transcription factor.

Bögershausen & Wollnik, 2018

Haploinsufficiency of *ARID1B*, a Member of the SWI/SNF-A Chromatin-Remodeling Complex, Is a Frequent Cause of Intellectual Disability

Juliane Hoyer,[1,8] Arif B. Ekici,[1,8] Sabine Endele,[1,8] Bernt Popp,[1] Christiane Zweier,[1] Antje Wiesener,[1] Eva Wohlleber,[2] Andreas Dufke,[3] Eva Rossier,[3] Corinna Petsch,[1] Markus Zweier,[1] Ina Göhring,[1] Alexander M. Zink,[2] Gudrun Rappold,[4] Evelin Schröck,[5] Dagmar Wieczorek,[6] Olaf Riess,[3] Hartmut Engels,[2] Anita Rauch,[1,7] and André Reis[1,*]

The American Journal of Human Genetics *90*, 565–572, March 9, 2012

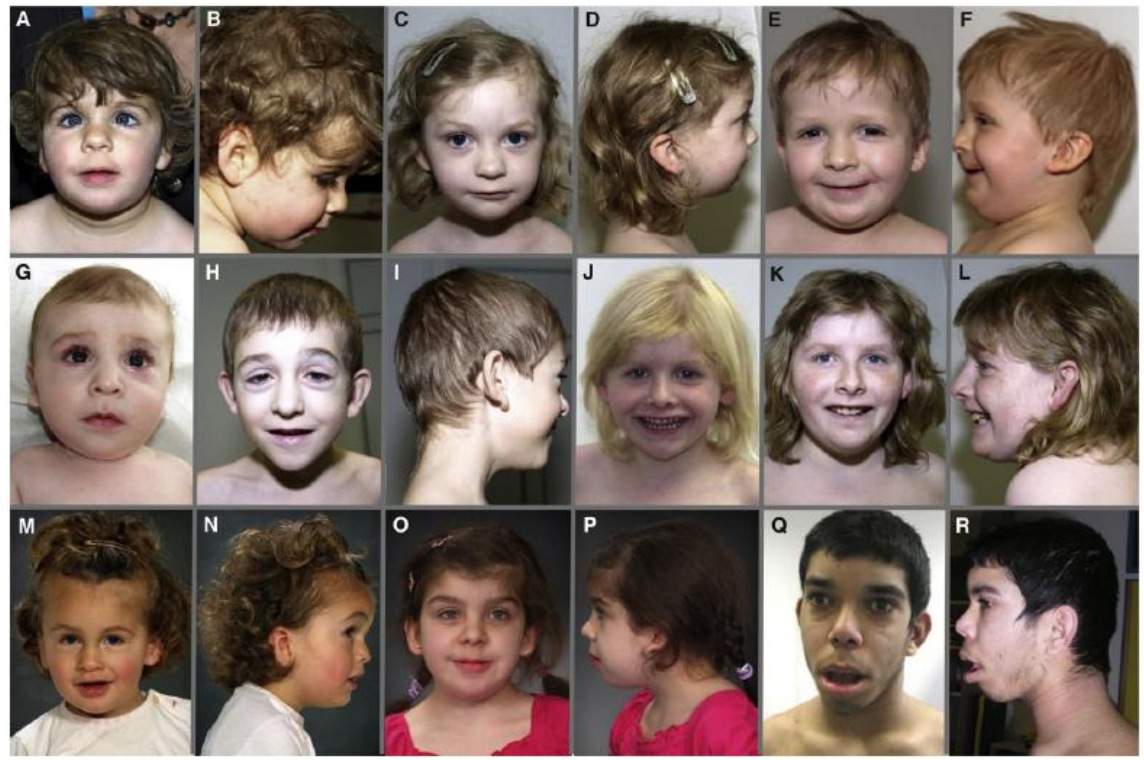


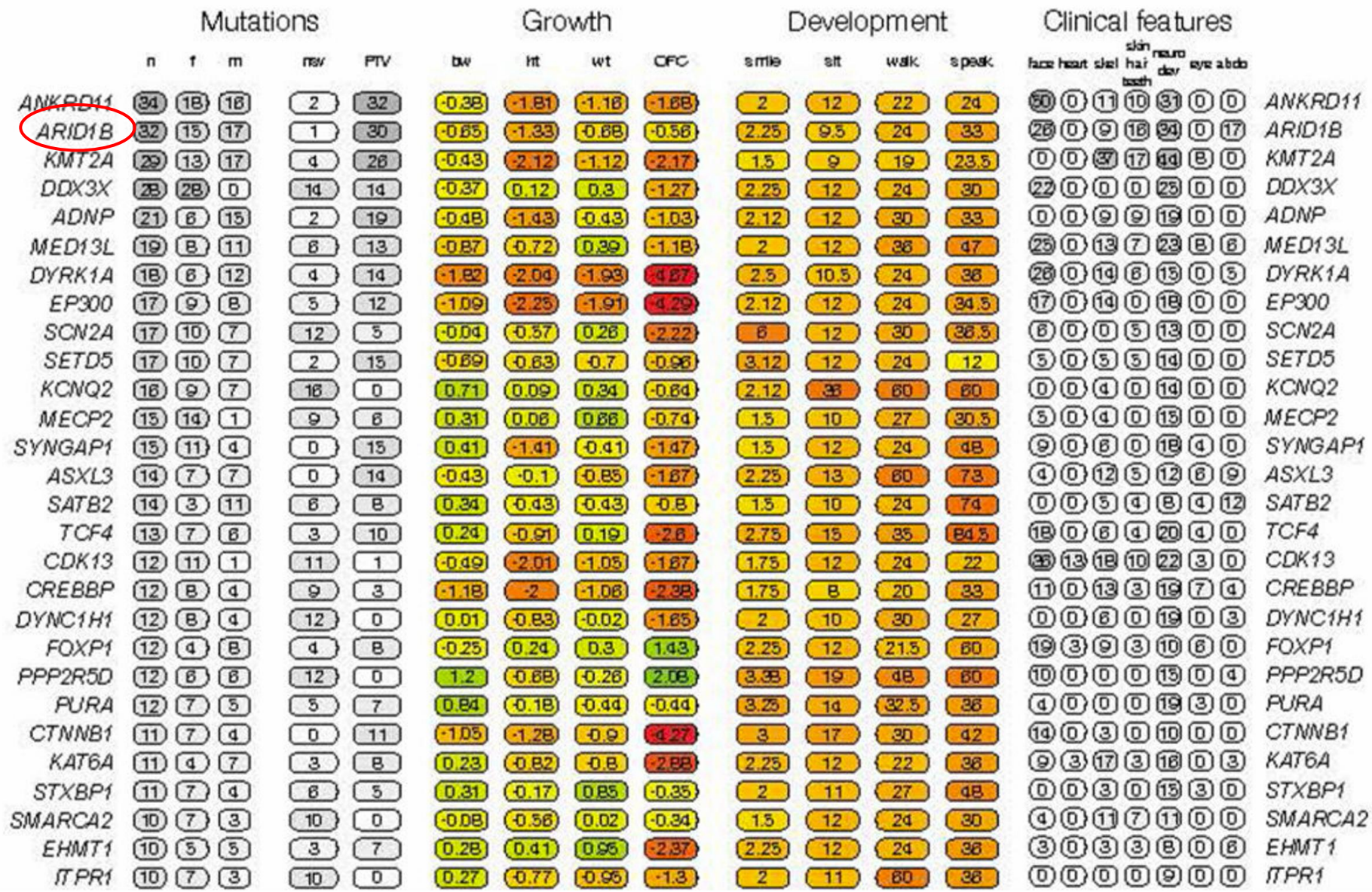
Table 1.

Molecular Genetic Testing Used in Coffin-Siris Syndrome

Gene [1]	Proportion of CSS Attributed to Pathogenic Variants in Gene [2]	Proportion of Pathogenic Variants [3] Detected by Method	
		Sequence analysis [4]	Gene-targeted deletion/duplication analysis [5]
ARID1A	<5%	100% [6]	Unknown [7]
ARID1B	~37%	~95%	~5% [8]
ARID2	Rare [9]	100%	Unknown [7]
DPF2	Rare [10]	100%	Unknown [7]
PHF6 [11]	Rare [12]	100%	Unknown [7]
SMARCA2 [13]	~2%	>90%	1 affected person
SMARCA4	~7%	100%	Unknown [7, 14]
SMARCB1	~7%	100%	Unknown [7, 14]
SMARCC2	Rare [15]	~75%	4 affected persons
SMARCE1	~2%	100%	Unknown [7, 14]
SOX4	Rare [16]	100%	Unknown [7]
SOX11	~2% [17]	~40% [18]	7 persons w/deletions & a CSS phenotype reported to date [19]
Unknown [20]	~40%	NA	

Schrier Vergano *et al.*, 2021

WES in
4293 families
with NDD



Prevalence and architecture of de novo mutations in developmental disorders
The Deciphering Developmental Disorders Study, Nature 2017

State of the art

- No published natural history data derived from repeated assessments of children with ARID1B-RD are available
- Published data were gathered from multiple sources or did not involve clinician-administered standardized developmental assessment (e.g. Vineland Adaptive Behavior Scales)
- Knowledge on behavioural difficulties, communication and daily living skills is limited
- Matching of *ARID1B* epigenetic profile to deep phenotyping is lacking
- Extensive proteomic studies in ARID1B-RD are lacking

Study population

Participants (n=135)

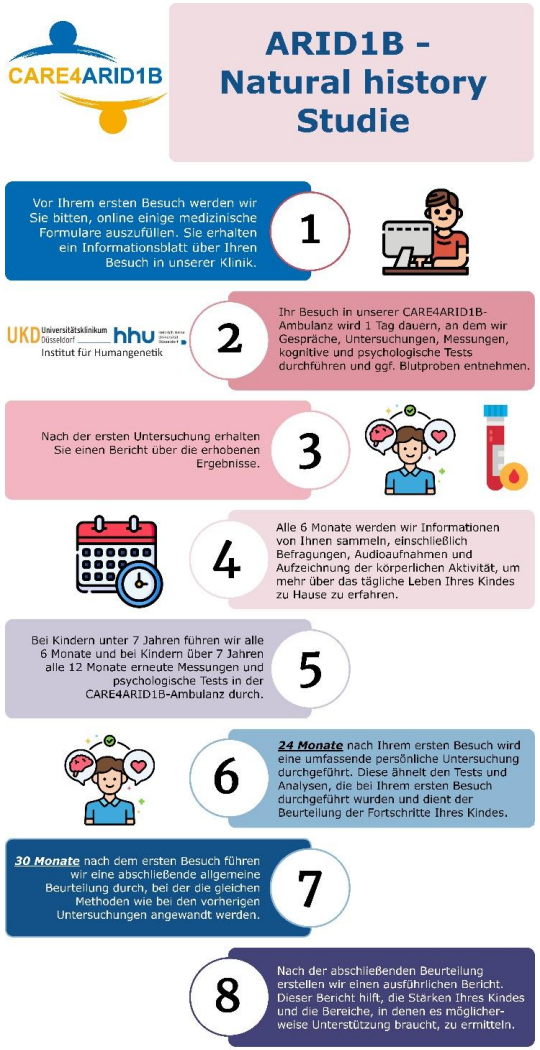
- must be between 2 and 18 years at screening
- must have a documented pathogenic or likely-pathogenic variant in *ARID1B*
- must have a caretaker sufficiently fluent in the site-specific language

Exclusion criteria

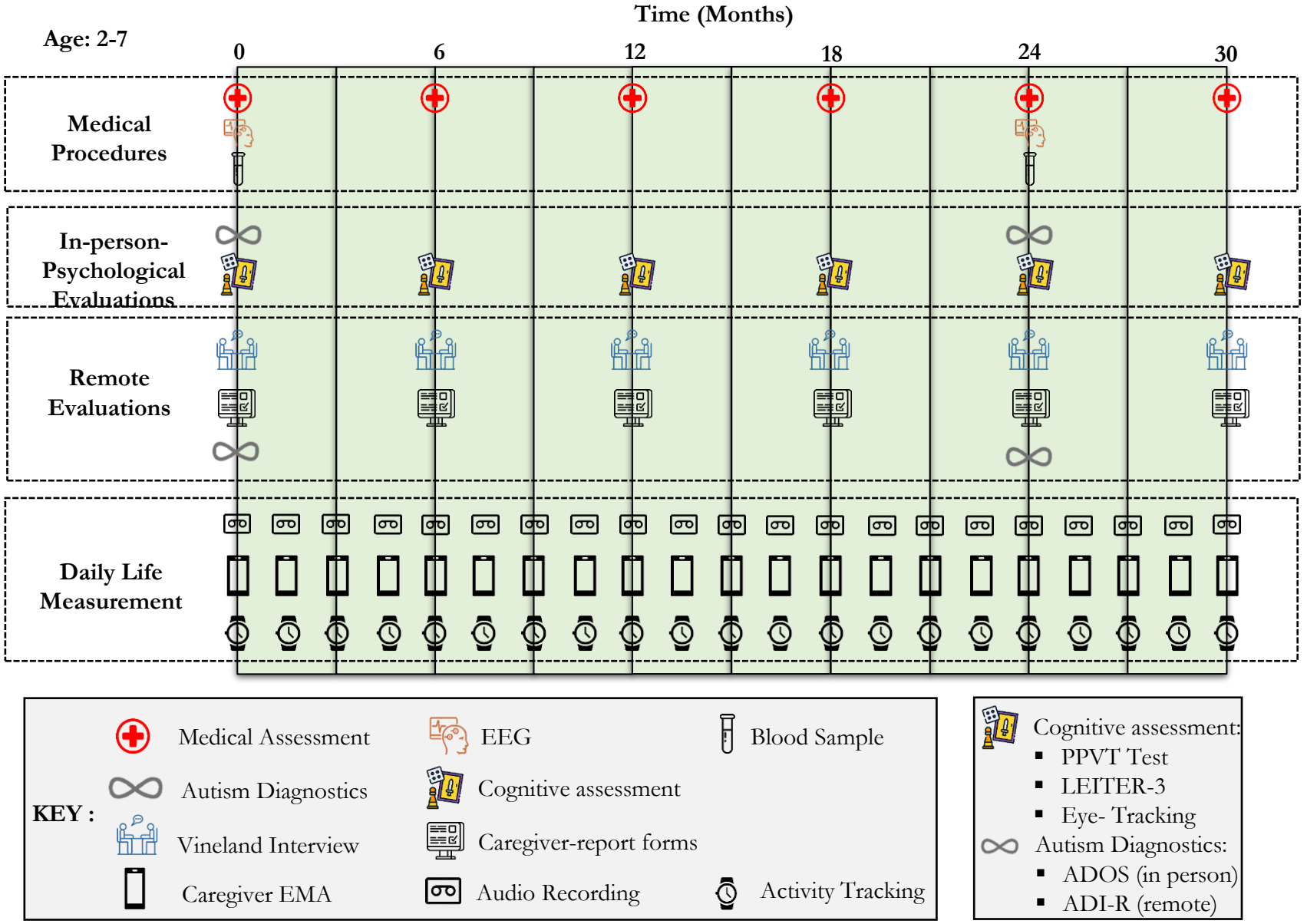
- any other significant disease/disorder
- deletions that involve additional genes beyond *ARID1B*
- current enrollment in any experimental treatment

Study duration

- The study is scheduled to span three years
- Thirty months of active participant monitoring

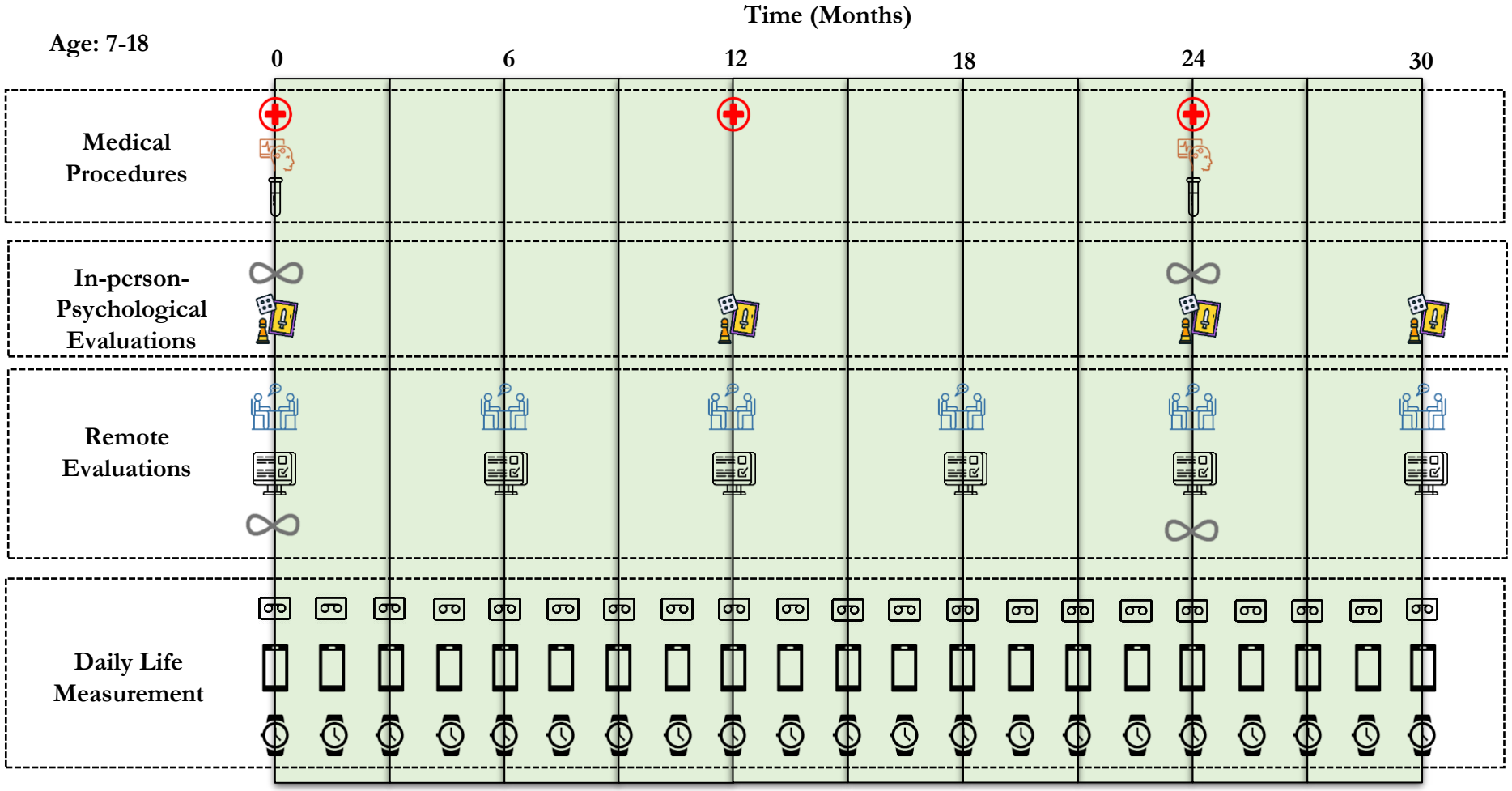


Study protocol (for individuals < 7 years)



We will monitor the development of 135 children and adolescents with ARID1B-RD in seven sites, for 30 months.

Timeline for children 7-18 years



KEY :

- Medical Assessment
- Autism Diagnostics
- Vineland Interview
- Caregiver EMA
- EEG
- Cognitive assessment
- Caregiver-report forms
- Audio Recording
- Blood Sample
- Activity Tracking

Cognitive assessment:
- PPVT Test
- LEITER-3
- Eye- Tracking

Autism Diagnostics:
- ADOS (in person)
- ADI-R (remote)

UKD Universitätsklinikum Düsseldorf
Institut für Humangenetik

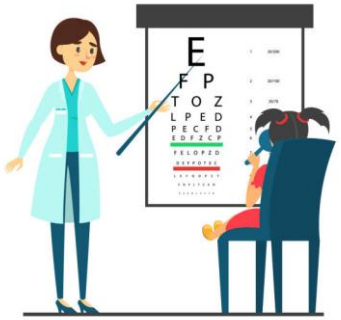
Medical Procedures

EEG

Neuropediatric/
Medical genetic examination

Photographs for
Gestaltmatcher

Hearing test

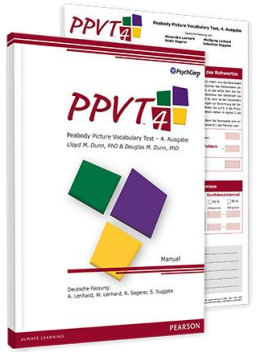


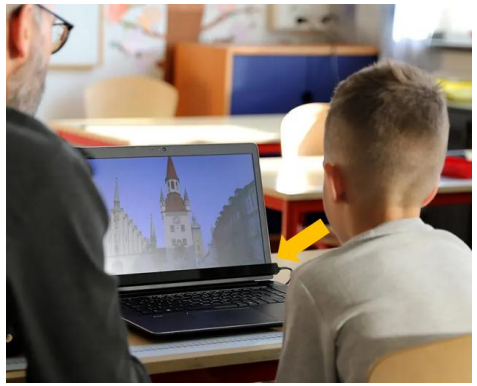
Ophthalmological
examination

Blood sampling

In person psychological evaluations

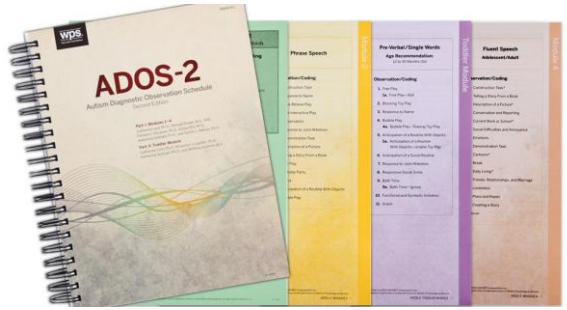
Cognitive assessment



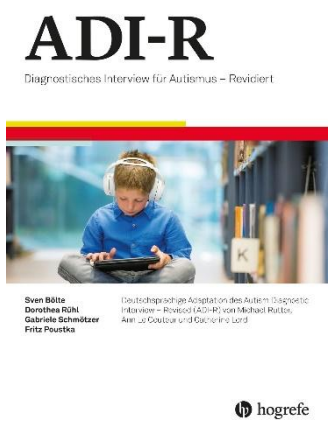


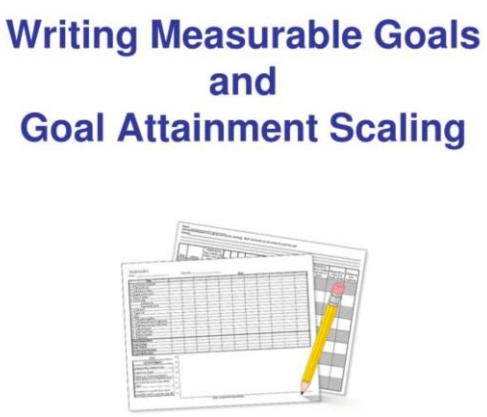
Eyetracker

Autism Diagnostics



Remote evaluations





Daily life measurements

Physical activity and sleep monitors

Vocal development tracking from daylong recordings

Monthly ecological momentary assessment

Homepage

www.care4arid1b.org

Dissemination and communication

Kick-off meeting

Summer school

Regular webinars

Patient community engagement

Career support activities

Primary objectives

- to establish a comprehensive, integrative understanding of the development of ARID1B-RD encompassing all relevant functional domains and physiological systems
- to characterize multiple traditional and novel clinical endpoints for upcoming targeted ARID1B-RD clinical trials.
- to identify biosignatures of ARID1B-RD that may be utilized for stratification and prediction of the disorder's progression.

For further information: www.care4arid1b.org

If you have interested families, please contact:
arid1b@mail.huji.ac.il or
dagmar.wieczorek@med.uni-duesseldorf.de

Team Düsseldorf

Prof Eva Meisenzahl-Lechner

Ioulia Ziavrou

Prof Felix Distelmaier

SHK N.N.

Paul Contzen

Manfred Beier

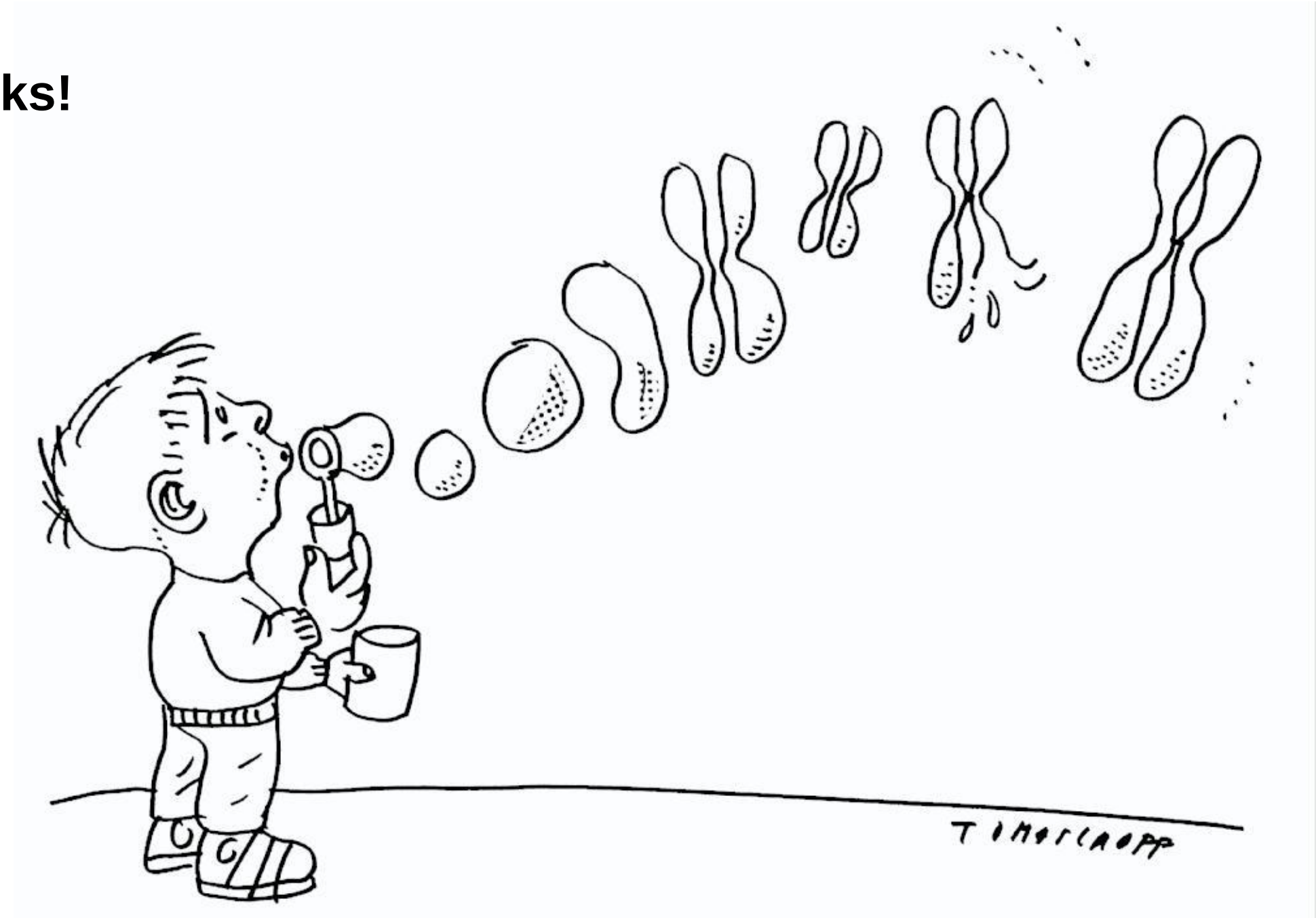
Manuel Michels

Dr. Svenja Daschkey

Dr. Melanie Sapp

CARE4ARID1B

Thanks!



The patient's perspective

Gal Lazarus, Ph.D., Department of Psychology, The Hebrew University of Jerusalem, Israel

Discussion time - Conclusion with speakers and moderator

Discussion & Conclusion

- Time for questions

- Satisfaction Survey :
  - https://forms.office.com/e/iXLm6EadF2
- Website :
  - https://ern-ithaca.eu
  - https://ern-ithaca.eu/webinars/

Thank you for your participation