

Index

Note: Page numbers of article titles are in **boldface** type.

A

- Acute chest syndrome, as clinical example of sickle ischemia-reperfusion injury, 190–191
- Adenosine, as cytoprotective mediator in sickle cell disease, 270
- Adenosine 2A receptor ($A_{2A}R$), role in sickle cell disease, 289–293
- Adenosine 2A receptor ($A_{2A}R$) agonist, and inflammation in sickle cell disease, 277–278
- Adenosine 2B receptor ($A_{2B}R$), role in sickle cell disease, 293–294
- Adenosine signaling, role in sickle cell therapeutics, **287–299**
- future directions in, 296
 - limitations of adenosine therapeutics, 296
 - pathway, 288–289
 - protective and deleterious roles, 294–295
 - role of adenosine A_{2A} receptor ($A_{2A}R$) in, 289–293
 - role of adenosine A_{2B} receptor ($A_{2B}R$) in, 293–294
- Adhesion. See Cellular adhesion.
- Aes-103 (5-HMF), clinical development of, to treat sickle cell disease, 224–225
- Allosteric modifiers, of hemoglobin, development of to treat sickle cell disease, 220–224
- Allosteric states, of hemoglobin and sickle cell disease, 218–219
- Anti-P-selectin aptamer, and inflammation in sickle cell disease, 276
- Anti-P-selectin monoclonal antibody (SelG1), and inflammation in sickle cell disease, 275
- Anticoagulant level, physiologic, reduction of in sickle cell disease, 358
- Anticoagulant therapy, for sickle cell disease, 364–366
- Arginase, increased activity in sickle cell disease, 305–306
- Arginine metabolome, alterations of, in sickle cell disease, **301–321**
- altered arginine homeostasis, 304–306
 - altered nitric oxide homeostasis, 303–304
 - arginine coadministration with hydroxyurea, 307–308
 - arginine therapy for clinical complications, 308–313
 - leg ulcers, 308
 - priapism, 311
 - pulmonary hypertension risk, 308–311
 - vaso-occlusive pain episodes, 311–313
 - impact of arginine therapy on nitric oxide production, 304–305
 - rationale for, 314
 - safety data for arginine supplementation, 313–314
- Arginine therapy, for sickle cell disease, coadministration with hydroxyurea, 307–308
- for clinical complications of, 308–313
 - leg ulcers, 308
 - priapism, 311
 - pulmonary hypertension risk, 308–311
 - vaso-occlusive pain episodes, 311–313
 - impact on nitric oxide production, 304–305

Arginine (*continued*)

- rationale for, 314

- safety data for, 313–314

Arterial vasculopathy, as clinical example of sickle ischemia-reperfusion injury, 191

Aselizumab, in targeted therapy of sickle cell disease, 351

B

Beta-hemoglobinopathies

- erythropoietin's role in treatment of, **249–263**

- erythropoiesis and, 250

- fetal hemoglobin and, 250–252

- iron overload and, 252–253

- malignancy and, 256–257

- nonhematopoietic cells and, 255–256

- oxidative stress and, 253–255

- targeted fetal hemoglobin induction for treatment of, **233–248**

- dual-action inducers including translation and enhanced erythroid cell survival, 241–242

- experience in trials of prior generation HbF inducers, 234–236

- HbG globin transcription and the fetal globin program, 236–237

- BCL11A, 237

- HBS1L-MYB intergenic interval, 237

- KLF-1 (EKLF), 237

- influence of quantitative trait loci, 242–244

- novel mechanism of HDAC3 displacement and recruitment of EKLF, 240–241

- targeted gamma globin activation through CACCC element, 237–238

- therapeutic approaches directed to increasing gamma globin transcription, 238–240

Beta-thalassemia

- gene therapy for, 207–209

- human gene therapy for, 208–209

- initial development of lentivirus-based vectors, 208

- initial vector development, 207–208

- modulation of hepcidin as therapy for, 394–396

- targeted fetal hemoglobin induction for treatment of, **233–248**

- dual-action inducers including translation and enhanced erythroid cell survival, 241–242

- experience in trials of prior generation HbF inducers, 234–236

- HbG globin transcription and the fetal globin program, 236–237

- BCL11A, 237

- HBS1L-MYB intergenic interval, 237

- KLF-1 (EKLF), 237

- influence of quantitative trait loci, 242–244

- novel mechanism of HDAC3 displacement and recruitment of EKLF, 240–241

- targeted gamma globin activation through CACCC element, 237–238

- therapeutic approaches directed to increasing gamma globin transcription, 238–240

Bimosiamose, in targeted therapy of sickle cell disease, 350

Blood flow, impaired, role of P-selectin in sickle cell disease, 323–327

Download English Version:

<https://daneshyari.com/en/article/3331225>

Download Persian Version:

<https://daneshyari.com/article/3331225>

[Daneshyari.com](https://daneshyari.com)